HANDBOOK OF CENTRAL AUDITORY PROCESSING DISORDER

Auditory Neuroscience and Diagnosis

VOLUME 1

SECOND EDITION

Frank E. Musiek
Gail D. Chermak

PLURAL PUBLISHING INC.

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Volume I
Auditory Neuroscience and Diagnosis

Second Edition

Editor in Chief for Audiology
Brad A. Stach, PhD

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FOREWORD

The concept of an auditory-specific perceptual disorder in children is more than 60 years old. It was first suggested by Helmer Myklebust, a psychologist originally trained in the evaluation of deaf children. At Northwestern University in the 1950s, Myklebust set up a clinic to examine children suspected of hearing loss and to counsel their parents. As a graduate student in audiology at the time, I was privileged to participate in these pediatric evaluations and to learn, at first hand, the variety and breadth of auditory disorders. As child after child came through the clinic, Myklebust began to realize that some of the children with apparent hearing problems had normal audiograms. The symptoms described by parents and teachers could not be attributed to peripheral hearing loss. They heard faint sounds normally in the controlled acoustic environment of the test booth, but in the real acoustic world, with its many sources of noise and competing speech, these children seemed unable to successfully suppress this background competition in order to focus on a particular source. Myklebust supposed that such problems reflected an auditory-specific perceptual deficit, making it difficult to attend selectively.

In the more than six decades that have elapsed since those seminal observations, the reality of the problem has been verified many times over, but discovering the underlying mechanism of such auditory processing disorders, efficiently diagnosing the malady, and providing effective treatment remain elusive goals. Into this void many ideas have flowed. Some have been helpful; others less so. Concepts founded on careful research and based on a plausible conceptual framework have generally advanced our understanding of the phenomenon. Ideas of what a committee thinks might be the problem, based on anecdotal evidence and exhaustive lists of symptoms, have not been as helpful and have often set us back.

Within the past decade, however, two important advances can be cited. The first is a growing understanding of the influence of extra-auditory cognitive processes, especially attention, on the fundamental nature of the disorder and on our diagnostic tests and instruments. The second is the emergence of innovative treatment options, many of which actually seem to work.

The editors of this handbook, Drs. Frank Musiek and Gail Chermak, have pioneered the kinds of thoughtful studies basic to understanding, diagnosing, and treating auditory processing disorders. In the two volumes of this second edition of their handbook, they have assembled a truly impressive stable of national and international contributors with expertise in virtually every aspect of the problem. They bring us up to date on the latest findings from the standpoint of behavioral assessment, electrophysiological measures, effective treatment options, and the neurobiology of the disorder; a truly impressive achievement.

James Jerger
Dallas, Texas
July 5, 2013

PREFACE

Central auditory processing disorder (CAPD) is a deficit in neural processing of auditory stimuli that is not due to higher-order language, cognitive, or related factors, yet CAPD may lead to or be associated with difficulties in higher-order language, learning, cognitive, and communication functions. The comorbidity of CAPD with a range of language, learning, and communication disorders is the result of brain organization, about which we have learned much in recent years. The perspectives contained in the second edition of our two-volume Handbook reflect continuing advances in auditory neuroscience and cognitive science since a group of Italian otolaryngologists led by Ettore Bocca recognized that pure tone audiometry was insufficient for detecting central auditory lesions. This led them to develop a battery of *sensitized speech tests* to diagnose disorders of the central auditory nervous system. Through their clinical studies over a span of 20 years, they increased our understanding of the relationship between the site and extent of lesion and the nature and severity of auditory dysfunction, including the now well known *contralateral effect* in which temporal lobe lesions show a deficit in the ear contralateral to the lesion. Indeed, many of the tests in use today bear considerable resemblance to those developed by these physicians. Some 40 years later, then President George H. Bush proclaimed the 1990s the "Decade of the Brain." Since that proclamation, we have witnessed great strides in basic and clinical research in neuroscience and cognitive science with considerable impact on the diagnosis and treatment of CAPD.

Perhaps a watershed event, leading clinicians and scientists from around the world convened in Boston, Massachusetts, in March 2012 at the first global conference on CAPD (held in conjunction with the annual convention of the American Academy of Audiology). Presenters and over 250 attendees shared knowledge, perspectives, and practices, which exposed areas of agreement, as well as differences. This conference sealed a commitment from all to seek greater opportunities for collaborative research and exchange. Indeed, the multidisciplinary efforts of thousands of scientists and health care professionals have led to greater insights regarding the nature of CAPD, brain organization and function, and directions for more accurate diagnosis and efficacious and effective interventions. With the recognition that neuroplastic changes in the brain underlie learning and rehabilitation, and that significant neural reorganization can occur in response to injury or learning even in mature nervous systems, there is every reason to embrace an aggressive and optimistic approach to intervention knowing that behavioral interventions that appropriately stimulate plastic neural tissues should lead to positive change.

The complexity and heterogeneity of CAPD, combined with the heterogeneity of learning and related disorders, challenge scientists and clinicians as they attempt to understand and differentially

diagnose individuals with listening deficits, language comprehension problems, attention deficits, learning disabilities, and other related behavioral, emotional, and social difficulties. This Handbook offers the most up-to-date and comprehensive coverage of the auditory neuroscience and the clinical science needed to accurately diagnose, assess, and treat the auditory and related deficits of individuals with CAPD. As in the first edition, the second edition of the Handbook melds science and practice, providing comprehensive coverage of the field of CAPD in children, adults, and older adults, involving the range of developmental (i.e., neurobiological) and acquired origins (i.e., aging and neurological diseases, disorders, and insults, including neurodegenerative diseases). Both volumes include many new chapters, new contributing authors (including international scientists and clinicians), and varied perspectives, and all chapters appearing in the first edition have been revised. Volume 1 provides an expanded section (four new chapters) on auditory neuroscience, new chapters and/or authors discussing diagnostic principles and procedures and multidisciplinary assessment, a chapter of case studies, and two chapters projecting future directions for the field. Volume 2, which concentrates on multidisciplinary rehabilitation and professional issues, contains nine new chapters, including one focused on the efficacy of auditory training, another on school policies, processes, and services, one providing a retrospective of the evolution of research and clinical practices in CAPD, three new chapters on specific treatment approaches, a chapter of case studies, and two chapters reflecting on the current issues requiring additional research and consensus as we consider the future opportunities for even greater treatment effectiveness and efficacy.

The Handbook is intended to serve three primary audiences. The contributing authors have written a comprehensive set of manuals for clinicians, primarily audiologists, speech-language pathologists and psychologists, and other related health care professionals. The Handbook also should serve as a reference source for a range of clinical scientists engaged in research related to audition and speech perception. Finally, we hope our Handbook can serve graduate students in the classroom and in support of their clinical experiences.

The approaches and recommendations offered in this Handbook are not intended to serve as a sole source of guidance for the differential diagnosis and intervention of individuals with CAPD. Rather, the views and methods of the 47 authors contributing to the Handbook are designed to assist the clinician by providing a framework for decision making and implementing diagnostic and treatment strategies. They are not intended to replace clinical judgment or to establish a protocol for all individuals with CAPD. Individual differences and circumstances, including the presence of comorbid conditions, require flexibility and adaptation.

Notwithstanding considerable scientific, technological, and clinical strides forward, we still have much more to learn. As was the case when our first edition was published in 2007, continued research is needed to resolve certain longstanding questions, and address the new questions that arise continually as new knowledge begets new questions. Collaboration between clinicians and scientists—combining the clinician's firsthand knowledge of clinical needs

with the researcher's expertise in the scientific method—provides a powerful approach to asking the right questions and obtaining enduring answers. Indeed, the contributing authors to the Handbook reflect this very collaboration between scientists and clinicians. Only through continued collaboration can we truly generate innovative approaches to questions and problems and accelerate the pace of discovery. In so doing, we will continue to advance our understanding of the central auditory nervous system and its intersections with cognitive and language domains that lead to the complex and heterogeneous clinical profiles of CAPD. It is imperative to seize the momentum that has taken us to our current level of understanding and clinical practice, as described in the Handbook. To ensure that we are able to deliver the best clinical services to our patients and their families, we as individuals and the professional organizations that represent us must remain engaged in developing strategic responses to and preparing for the rapidly changing health care landscape, changes that will focus on value-based service delivery and the convergence of patient-centered outcomes and cost-effectiveness.

In closing, we hope to contribute to enhanced *patient-reported* outcomes and the improved overall quality of the lives of individuals with CAPD, their families, and their communities with the knowledge and practices shared in this second edition of the Handbook.

Frank E. Musiek
Gail D. Chermak

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F. E. M

We also wish to offer our sincere appreciation to each of our chapter authors and the Plural team who have stood by us and worked hard to see this project through.

F. E. M and G. D. C

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Frank E. Musiek is Professor and Director of Auditory Research, Department of Speech, Language and Hearing Sciences and Professor of Otolaryngology, School of Medicine, University of Connecticut. He is the 2007 recipient of the American Academy of Audiology (AAA) James Jerger Career Award for Research in Audiology, the 2010 recipient of "The Honors of the American Speech-Language-Hearing Association for his contributions to Audiology and Auditory Neuroscience," and recipient of the "Book of the Year Award" for the *2007 Handbook of (Central) Auditory Processing Disorder*, Vols. I and II (with Gail Chermak coeditor). He has published over 200 articles and book chapters in the areas of auditory evoked potentials, central auditory disorders, neuroaudiology, and auditory neuroanatomy and has authored or edited 9 books. He has served on numerous national and international committees, editorial boards, and task forces, including chairing the 2010 AAA task force for clinical practice guidelines for central auditory processing disorder.

Gail D. Chermak, PhD

Gail D. Chermak is an internationally recognized authority on central auditory processing disorder (CAPD). She has published extensively and lectured around the world on differential diagnosis and treatment of CAPD. Dr. Chermak is professor of audiology and chair of the Department of Speech and Hearing Sciences at Washington State University. She is the recipient of numerous honors and awards, including the American Academy of Audiology's (AAA) Distinguished Achievement Award and the "Book of the Year Award" for the *Handbook of (Central) Auditory Processing Disorder* Vols. I and II (with Frank Musiek coeditor). She is a Fellow of the American Speech-Language-Hearing Association (ASHA) and is included in several major American and international biographical listings. She has served on a number of editorial boards and national professional committees and task forces, including the 2010 AAA task force which published evidence-based clinical practice guidelines for CAPD. She has authored over 100 articles and book chapters, and authored or edited 6 books.

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I want to extend a profound thanks and admiration to my colleague and dear friend Gail Chermak who I have worked with on so many projects. Her work ethic, foresight and knowledge have been and continue to be truly amazing to me. Gail has helped me (and many of our contributors) through many tough spots on this second edition with her sage advice.

Finally, I want to mention Linda Guenette, audiologist superb, who passed away this past spring. Over the years, Linda and I saw many cases of CAPD together and she strongly supported efforts to advance our knowledge of the central auditory system. She was a dear friend to me and many others.

—Frank E. Musiek

To my brother Steven and my dear friend Ruth, whose unwavering support and love have grounded me throughout my life.

To my sister, Luann, my original source of inspiration.

To Frank Musiek, my esteemed colleague and dear friend, with whom I have enjoyed the most stimulating, productive, and enduring collaboration of my career.

And to my children, Isaac and Alina, who compel me to expand my boundaries.

—Gail D. Chermak

SECTION 1

[redacted]tory Neuroscience

CHAPTER 1

AUDITORY NEUROSCIENCE AND CENTRAL AUDITORY PROCESSING DISORDER

An Overview

FRANK E. MUSIEK and GAIL D. CHERMAK

The initial section of the first volume of this two-volume Handbook is devoted to selected aspects of auditory neuroscience as they relate to the diagnosis of central auditory processing disorder (CAPD). This emphasis on science is consistent with the view of the Handbook editors and contributors that those who are involved in the diagnosis (as well as the intervention) of CAPD must be well grounded in auditory neuroscience.

Knowledge of auditory neuroscience is at times difficult to attain. By its nature, science is complex and not always easily accessible. In addition, it is often not high on the "to do" list of busy clinicians juggling what may seem at times like an endless array of competing priorities and demands. Nonetheless, clinicians must find the time necessary to examine the science that underlies their clinical practices, in particular the science underlying central auditory tests' construction and their interpretation before administering a central auditory processing test battery in the clinical setting.

In this second edition of the Handbook, we have increased the coverage of auditory neuroscience considerably, reflecting the growing importance and impact of fundamental science for clinicians, students, and researchers. By placing the science chapters before the chapters focused on clinical practice, it is our intention that they will be read first. Our focus on science also is intended to communicate the important role of the scientific "attitude" and the scientific method of inquiry for the clinical process. The scientific "attitude" is one that appreciates the scientific method, which is objective, open, and data based. The

scientific attitude begets an approach to knowledge acquisition that is broad and inclusive, recognizing that knowledge pertinent to one's particular area of interest can originate from many different disciplines and methodologies. Scientific thinking begins with curiosity that leads to identifying a problem, generating an hypothesis to solve the problem, testing the hypothesis, and ultimately applying the solution to benefit people. These same attitudinal and thought processes and methodologies are as critical to clinical practice as they are to science. Science cannot be separated from clinical services and vice versa—advances in one lead to commensurate progress in the other domain. Conversely, lagging developments in one delay progress in the other. Clinicians tell scientists what is important to examine. Scientists deploy strong research designs to obtain enduring answers to clinicians' questions. Working together, we benefit from a synergy by which knowledge expands and deepens, and clinical services improve.

Overview of the Science Section

The science section begins with Ray Hurley's and Annette Hurley's chapter on psychoacoustic considerations and implications for diagnosis of CAPD. Their long-standing interest in speech perception and CAPD makes them ideal authors to explore these relationships. Together, they bring considerable research and clinical experience to this chapter. They guide the reader through the number of psychoacoustic principles that are key to developing and using behavioral central auditory processing tests.

The neuromaturation of the central auditory nervous system (CANS), neurologic origins of CAPD, and neuroanatomic and neurophysiologic bases of central auditory tests are examined in three new chapters. Jos Eggermont reviews the development of the CANS. In addition to providing an overview of human auditory maturation, Dr. Eggermont summarizes the anatomic and physiologic correlates underlying the central auditory processes. His review underscores the normal but protracted course of CANS development, providing the reader with an appreciation for the complex neuroanatomy and neurophysiology that underlie auditory perceptual skills, as well as the consequences of delayed maturation of these structures (e.g., the corpus callosum) so critical to auditory processing for CAPD. Next, we examine the neuroanatomic abnormalities, neurologic disorders, and neuromaturational delays that may lead to CAPD in adults and children. In a third new chapter in this section, Dr. Kenneth Hugdahl and Dr. Turid Helland describe the neuroanatomic and neurophysiologic bases of central auditory tests. Known for their body of work on dichotic listening, Drs. Hugdahl and Helland illustrate the role science serves in test development and interpretation of test findings. As Hugdahl and Helland elaborate in Chapter 6 of Volume 1 of the Handbook, central auditory processing can be viewed as the "interaction of bottom-up, sensory and top-down, cognitive factors that shape and modulate how a simple speech sound, like a CV-syllable, is processed in natural surroundings, as when people are talking to each other, which requires decoding of the phonology but also correct attention and focus and selective cognitive filtering of the signal to be processed." They note: " In

central auditory processing deficits, one or several of the components of the interaction is not functioning, causing failure of either low-level perceptual processing or higher level cognitive modulation of the signal, or both." As elaborated throughout the Handbook, this complex interaction carries significant implications for both diagnostic and treatment approaches.

Dennis Phillips has revised his chapter from the first edition, which explains the neurobiology of the CANS, and demonstrates his unmatched ability as an auditory neuroscientist to bridge the gap between science and clinic. Dr. Phillips' interests in the auditory system are broad and far ranging, both from the standpoint of the nature of the processing executed by the system (i.e., spatial, spectral, and temporal) and from the standpoint of the perspective and level of analysis with which it is studied (e.g., psychophysical, neurophysiological). In his chapter, he covers the basic neuroscience of the ascending auditory system, with some emphasis on how the brain establishes a sensory representation of the stimulus. Phillips also covers neurotransmission. In our previous edition, there was little attention paid to CANS neurotransmitters. A rapidly evolving area of study, the neurotransmitters underlying communication in the CANS are being identified and quantified (Altschuler & Shore, 2010). This neuropharmacological information has great potential impact on diagnosis of and intervention for CAPD. For example, might it be possible that a drug that serves as an agonist to certain excitatory neurons in the CANS could be prescribed to improve hearing and auditory processing? Dr. Phillips applies his experience in auditory neurophysiology and psychophysics to provide an informed view of auditory temporal processing and auditory system plasticity and closes his chapter with a critical analysis of the neurophysiologic relationship between central auditory processing and language. Dr. Karen Banai and Dr. Nina Kraus have updated their chapter on the neurobiology of CAPD based on their large body of work with speech, music, and language processing. They examine neurobiologic processes underlying the perception of speech and learning-associated brain plasticity. Germane to this chapter is their line of research demonstrating a fundamental, biological basis for speech-sound perception deficits in some school-age children with learning disabilities and CAPD. This chapter reflects some of their latest research regarding neural encoding of speech in the auditory brainstem with implications for the diagnosis of CAPD and for auditory training remediation strategies.

Next, we include Dr. Teri James Bellis' revised chapter on the nature of central auditory processing, the original of which had appeared in Volume 1 of the first edition of the Handbook. Given the fundamental issues covered in this chapter, the editors determined that it should appear alongside the other core chapters in the auditory neuroscience section. Dr. Bellis defines CAPD and describes the functional characteristics, prevalence, etiology, and comorbidities frequently seen, noting how important understanding these elements are to diagnosis and intervention. Dennis Phillips' conceptualization of central auditory processing and CAPD (Chapter 22) offers additional perspective to the topics covered in Dr. Bellis' chapter. The auditory neuroscience section closes with a fourth new chapter, this one authored by Dr. Jeffrey Weihing and coauthors Dr. Teri James Bellis and

ourselves. Here we critically examine a number of clinical and research issues in CAPD that must be resolved to advance the field and ensure that the best clinical services are provided to patients.

[redacted]itory Neuroscience: [redacted]plications for CAPD

Many technological breakthroughs have impacted our ability to study the brain, and many of the developments in neuroscience carry implications crucial to our understanding of and clinical advances in CAPD. There have been numerous links between advances in auditory neuroscience and CAPD. One of the classic links between auditory neuroscience and CAPD was seen in the development of auditory pattern perception tests. The early animal studies performed by Neff, Butler, and Diamond demonstrated that ablation of the auditory cortex in cats resulted in their inability to correctly recognize patterns of sound (Neff, 1961). Even with training, these animals could not reach the levels of performance documented before surgical ablation. These findings were utilized by Ptacek and Pinheiro (1971) in the development of the frequency (pitch) pattern test for humans. Clinical research studies in humans essentially yielded the same results that the Neff, Butler, and Diamond studies had produced with animals (Musiek, Pinheiro, & Baran, 1987). Hence, the development of an efficient and popular CAPD test that has been used for many years was directly linked to early neuroscience research.

Dichotic testing is another example of the linkage between auditory neuroscience and clinical practice. Dichotic testing, which has long been used to assess the CANS, reveals deficits in the ear contralateral to the compromised hemisphere (Figure 1–1). Doreen Kimura's (1961) original research identified the basis for interpreting dichotic laterality effects. In her original study, Kimura (1961) recruited individuals with well-defined lesions limited to the temporal lobe in one hemisphere. She clearly demonstrated that temporal lobe damage affected dichotic listening performance —primarily in the ear contralateral to the lesioned hemisphere. She explained this finding on the basis of physiologically stronger contralateral neural pathways that contain more fibers than the ipsilateral pathways. When the contra-

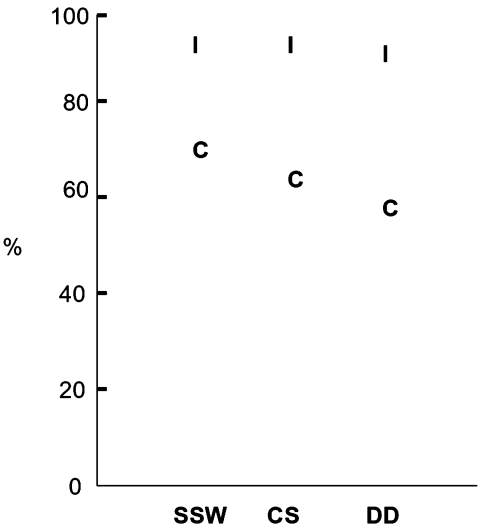

Figure 1–1. The performance (in percent correct) on three dichotic listening tests (CS = competing sentences, SSW = staggered spondaic words, DD = dichotic digits) for patients with confirmed cortical lesions primarily involving auditory regions in one hemisphere. Note that there is significantly better performance for the ear ipsilateral (I) to the lesion than for the ear contralateral (C) to the lesion.

lateral route was compromised due to the left temporal lobe lesion, the remaining intact pathways (i.e., both ipsilateral pathways and the contralateral pathway routed to the opposite side) were dominant, resulting in a right ear deficit on the dichotic listening task in patients with left hemisphere lesions. Kimura (1961) also showed that left hemisphere damage exerted a greater effect on dichotic listening for speech than did right hemisphere lesions. Many years later, functional magnetic resonance imaging (fMRI) data obtained by Hugdahl et al. (1998) supported Kimura's original observations and theoretical explanation regarding the suppression of the ipsilateral neural tracts and dominance of the contralateral neural tracts during dichotic listening. The fMRI data also indicated more activity in the posterior left temporal plane than in the right plane for dichotic speech stimuli. Hugdahl (2003, 2011) has expanded our understanding of dichotic listening mechanisms through systematic studies using both behavioral tests and imaging procedures in normally hearing subjects and those with various pathologies. Indeed, as Hugdahl and Helland suggest in Chapter 6, the forced-attention dichotic listening task is well suited to tap the interactive aspects of central auditory processing that can be used in both research and clinical settings. One must credit the systematic efforts of both Kimura and Hugdahl et al. for providing the scientific grounding for the clinical use of dichotic listening tests.

The auditory brainstem response (ABR) demonstrates yet another linkage between science and clinic. The ABR has long been used as an audiological test for brainstem dysfunction. It is well known that certain patterns of ABR results are reliably associated with central auditory dysfunction of the brainstem. That is, when the later waves (III, IV, V) of the ABR are compromised with the earlier waves unaffected, there is a high probability of CANS involvement at the level of the brainstem (Figure 1–2). The basis for these patterns and their interpretation derive from basic studies on generator sites and lesion effects on animals (Moller, 2000). This basic research has enabled clinicians using ABR to differentiate peripheral from central auditory involvement. More recently, ABR has been elicited by *complex* sound, such as a speech syllable, generating a cABR. These recordings are strikingly similar to the stimulating sound, reproducing transient events (e.g., syllable onsets and offsets) as well as periodic aspects (e.g., voicing cycles of vowels; see Figure 1–2). In contrast to cortical responses

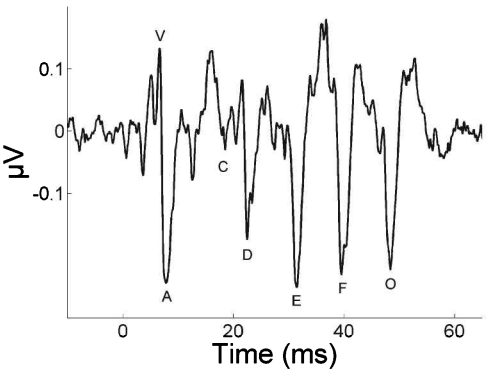

Figure 1–2. Complex ABR of a 21-year-old female with normal hearing and normal neurologic history obtained from the presentation of a 40 ms "da" at 80 dB nHL to the right ear at 10.9 Hz. V = ABR wave V; A = onset of stop consonant; D, E, F = three major peaks within the frequency following response (the period between D, E, F corresponds to the wavelength of the fundamental frequency of the syllable); O = syllable offset.

that provide abstract representations of stimuli, the cABR's fidelity to the stimulus offers opportunities to examine how stimulus attributes are biologically transduced (Skoe & Kraus, 2010), as well as an electrophysiologic measure to assist in diagnosis of CAPD (Wible, Nicol, & Kraus, 2004).

The recent development of the Gaps-in-Noise (GIN) and other gap detection procedures demonstrate the continuing linkage between science and practice (Musiek et al., 2005). Although data on the psychoacoustic underpinnings of many gap detection procedures have been available for many years (Plomp, 1964), the real source of information needed to develop a clinical gap detection procedure has come from animal data showing major effects of auditory cortex ablations (Syka, Rybalko, Mazeloca, & Druga, 2002). Similar results were obtained from humans with cortical lesions and animals with cortical ablations (Bamiou et al., 2006). This is yet another case where the animal data were predictive of clinical findings with humans. So, too, the effects of central auditory involvement due to aging is reflected in gap detection measures in animals and human subjects (Barsz, Ison, Snell, & Walton, 2002; Lister, Besing, & Koehnke, 2002). (See Chapter 18 for discussion of CANS changes due to aging.)

The clarification that auditory neuroscience provides relative to pathophysiology is another example of the link between basic science and clinic. For example, research demonstrating the effects of auditory cortex ablations on auditory detection in primates has informed our understanding of the underlying pathophysiology of CANS dysfunction. Although it has been well documented over the years that central lesions do not influence pure tone detection thresholds, Heffner and Heffner (1986) presented contrary findings that certainly were thought-provoking. These researchers demonstrated poorer pure tone detection thresholds after auditory cortex ablations in the ear contralateral to the lesion, although the animals did recover their hearing sensitivity in time. The clinical implication is an important one in that it poses the possibility that in severe cortical damage (other than central deafness), there may be an effect on pure tone detectability, which may be recovered over time.

Also revealing the role of auditory neuroscience in clarifying the pathophysiology of auditory dysfunction is the work by Masterton and colleagues (1992), who by sectioning ipsilateral versus contralateral lateral lemnisci (LL), demonstrated that the contralateral LL serves a more significant role for detection and frequency discrimination than does the ipsilateral LL. This contralateral dominance is consistent with long-standing anatomic data indicating more contralateral than ipsilateral fibers in the LL and underlies clinical data showing contralateral deficits on behavioral central auditory tests for lesions in the more rostral pons (Musiek et al., 1988).

Yet another example of the role that basic science serves in increasing our understanding of CAPD and related disorders is Galaburda et al.'s (1985) demonstration of the cell abnormalities linked to dyslexia. Polymicrogyria (multiple, underdeveloped gyri in the brain) have been linked to cell migrational problems and have been associated with dyslexia (Galaburda et al., 1985). Of recent import is the finding that these morphologic abnormalities have been observed in children with dyslexia by radiologic

means, and these findings have often been located in the perisylvian (i.e., auditory) region of the brain. In addition, it has now been shown that children with polymicrogyri present deficits on tests of auditory processing (Boscariol et al., 2009). Such findings suggest a rather strong relationship among a genetically based learning problem, an anatomic abnormality, and measurable central auditory dysfunction (see Chapter 4).

The pathophysiology of traumatic brain injury (TBI) is our final illustration of how basic science affects clinical practice. TBI is a leading cause of death and lifelong disability in children and adults. Our understanding of the biomechanical properties of the brain and skull (as well as greater brain plasticity in children) has led to a deeper understanding of head injuries and their potential impact for central auditory as well as peripheral hearing. The pressing need for accurate evaluation and effective treatment of TBI has intensified the basic and clinical scientists' efforts to elucididate the mechanisms and consequences of TBI. (See Chapter 4 of this volume and Pinto et al., 2012 for a general review of the mechanisms of TBI in children.)

Implications of Clinical Findings for Auditory Neuroscience

Although there are many more examples demonstrating the impact of auditory neuroscience on clinical practice, we move on to provide examples of the reverse effect, whereby clinical findings impact auditory neuroscience. One recent example of the impact of clinical findings with human subjects on auditory neuroscience concerns the function of the insula.

For years, our knowledge of the function of the insula has been uncertain. A few studies suggested that the insula might be involved in audition; however, these studies were mostly ignored (Bamiou et al., 2003). A rather dramatic clinical report was published in 1995 that did much to change the thinking about the functional role of the insula: Habib et al. (1995) studied an individual who incurred a stroke involving the insula on the right side and then a few days later a stroke involving the insula on the left side. Interestingly, after the second stroke, the patient demonstrated central deafness, despite the fact that Heschl's gyrus was unaffected and intact on both sides. Clearly, this clinical report has had a resonating impact on auditory neuroscience related to function of the insula.

Human behavioral findings involving the corpus callosum also have influenced science. Although animal research demonstrated electrophysiologically the importance of the corpus callosum (Berlucchi, 1972), findings with human subjects ultimately clarified the underlying science. Although Berlucchi's data demonstrated the influence of myelin on interhemispheric transfer times, it was not until humans with "split brains" were studied that the importance of the corpus callosum was realized. The original studies demonstrated that split brain patients showed essentially no responses to left ear stimuli—only in a dichotic listening mode using speech stimuli. If speech stimuli were presented monaurally, no deficits were noted (Musiek et al., 1984). The dichotic listening findings demonstrated the role of the corpus callosum in the transfer of information from one

hemisphere to the other. In conjunction with other test battery findings, interhemispheric transfer deficits related to damage of the callosum are now rather routinely inferred from left ear deficits on clinical dichotic listening tests.

Another example of clinical findings influencing auditory neuroscience can be seen in the recent reports (also noted above) that have demonstrated the neuroanatomic source of CAPD in some children (Boscariol et al., 2009, 2010, 2011). In particular, these children showed similar central auditory test performance patterns as those seen in adults with documented lesions of the CANS, thereby providing compelling evidence that CAPD originates in some dysfunction of the CANS across many patient populations, including children. Further examination of this neuroanatomic anchor will significantly impact our understanding of CAPD in the future.

New findings on noise exposure and the CANS also are intriguing. While Morest (1982) demonstrated that noise-induced hearing loss in animals was accompanied by damage to the (central) auditory brainstem neurons, highlighting transynaptic degeneration from the periphery to the central auditory system, recent findings demonstrate that exposure to moderate intensity levels of noise that do not cause peripheral hearing loss can nonetheless, over a period of time, result in damage to the CANS, with performance deficits seen on measures of temporal processing and speech recognition in noise (Kumar, Ameenudin, & Sangamanatha, 2012; Pienkowski & Eggermont, 2012; Zhou & Merzenich, 2012). The Zhou and Merzenich study suggests the possibility that the auditory symptoms reported by a patient with history of noise exposure but with a normal audiogram might be related to compromise of the CANS. This possibility should compel audiologists to at least consider central auditory involvement in those with noise-induced hearing loss, and perhaps even those with a history of noise exposure and auditory symptoms.

Clinical Decision Analysis

Clinical decision analysis (CDA) provides the clinician with a quantitative and systematic approach to evaluate the performance of diagnostic tests (see Turner, Robinette, & Bauch, 1999 for review). CDA focuses on a test's sensitivity (its ability to identify correctly those individuals who have the dysfunction), specificity (its ability to identify correctly those individuals who do not have the dysfunction), and efficiency (its combined sensitivity and specificity). Although specificity typically decreases as sensitivity increases, tests can be constructed that offer high sensitivity with an acceptable degree of specificity needed for clinical use. The alteration of pass/fail criteria can influence the sensitivity and specificity of a test. This changing of criteria can help the clinician achieve the desired outcome for the test by manipulating the sensitivity and specificity. If one measures test sensitivity and specificity for a progression of criteria, a receiver operating characteristic (ROC) curve can be constructed to visualize the variation of sensitivity (i.e., hit rate, or true positive rate) versus the false alarm/false positive rate for different pass/fail criteria. (See Chapter 12 for a primer on CDA.)

Also related to a test's clinical utility are the concepts of validity and reliability.

Reliability (i.e., consistency or stability of a measure) is essential to validity; however, reliability does not ensure validity. Validity, and in particular construct validity, which is probably the most important form of validity, refers to the fundamental question: *What* does the test measure? In the case of central auditory testing, the clinician must employ tests and procedures that not only provide a *consistent* measure of function, but also provide a *true, legitimate* measure of CANS integrity. Ascertaining that a test is valid—for instance: (i) through comparison of its content to the domain being measured, (ii) correlation or factor analysis with other supposedly valid tests of that domain, and (iii) amassing of convergent, divergent, and other evidence that the presumed construct is what is being measured—does not mean that the test is sensitive (or specific). CDA is necessary to quantify a test's sensitivity and specificity. The audiologist's central auditory test battery must consist of valid (and therefore reliable) measures of CANS function. Such tests and procedures will not be useful clinically, however, unless they are also highly sensitive to CANS dysfunction and at the same time capable of excluding as normal those individuals who do not present CANS dysfunction. Such measures present the test efficiency needed to accurately diagnose CAPD.

Neuroanatomy of the Human Brain Revisited

Both the scientist and the clinician interested in CAPD are highly dependent on the anatomic correlates of their laboratory or clinical test findings, respectively. In fact, interest in anatomic correlates has fueled advances in functional imaging, which have in turn heightened interest in the anatomy of the human brain. As the anatomy of the human brain has reentered the spotlight, the limitations of anatomic methods have come under scrutiny. It appears that the anatomic knowledge needed to establish important correlations may fall a bit short. Much of this knowledge shortcoming is the result of the fact that much of the information on human neuroanatomy is based on animal models, which in actuality are quite different from those of humans—especially at the cortical level (Musiek & Baran, 2007).

Interesting questions have evolved about the human brain—especially in regard to auditory regions (Leonard, Puranik, Kildau, & Lombardino, 1998). It has been shown that the primary auditory region in humans, Heschl's gyrus, can be two or three gyri (Musiek & Reeves, 1990). Also, at times it is very difficult to know where Heschl's gyrus ends and the planum temporale begins because of the morphology of the tissue in that area of the brain (Rubens, 1977). Perhaps the biggest question mark about human brain anatomy centers on the morphology of the sylvian fissure. The sylvian fissure, which is a guide to most of the auditory regions of the cortex, courses along the superior fringe of the superior temporal gyrus. The difficulty lies in the fact that the sylvian fissure is highly variable in its morphology. At times it courses straight back, but at times the posterior quarter may also turn up (termed an ascending ramus) or down (descending ramus). This can change the precise position of associated structures, including the planum temporale,

supramarginal gyrus, and angular gyrus (for review, see Rubens, 1977). These morphological alterations have implications for establishing anatomic correlates to test findings, evoked potentials, and, yes, even functional imaging. Clearly, the anatomic variability of the human brain must be studied further so that clinicians and scientists have a solid dependable reference to support a better working knowledge for clinical practice.

Although not an entirely new perspective, more recent views regarding the structure of the primary auditory cortex are gaining momentum (Kaas & Hackett, 2000). This newer anatomic perspective considers the auditory cortex to have a core structure surrounded by a "belt" and "parabelt" areas extending farther from the core. This view of auditory cortex anatomy has been demonstrated in primates and is thought by some to present in humans, though the latter view at present still evokes some controversy (see Chapter 5).

An evolving understanding of brain organization and function builds on the fact that the CANS is rather extensive and overlaps with areas of the brain known to support other sensory systems, language, cognition, and motor control (Petacchi, Kaernbach, Ratnam, & Bower, 2011; Poldrack et al., 2001; Poremba et al., 2003; Wong et al., 2009). In fact, the brain is organized in terms of multipurpose representations that different regions support (Lopez-Aranda et al., 2009). Networks are nonmodular, nultisensory, interconnected, and synchronized. They are organized to subserve process-specific (e.g., language) processing rather than domain-specific (e.g., auditory or visual) processing (Buschman & Miller, 2007; Price, Thierry, & Griffiths, 2005).

[redacted]munication Between [redacted]ician and Scientist

The many examples of linkage between auditory neuroscience and clinic underscore the importance of this relationship to advances in the field of CAPD. There is no denying that these links exist and that the linkages are strengthened through the hard work of clinicians and scientists. It would also be fair to say that communication among clinicians, clinical investigators, and basic scientists often is lacking. Given the importance of this communication, we propose several means to improve the communication process.

As discussed in other chapters in this Handbook, attitude plays a key role in communication. Auditory neuroscientists must take an interest in clinical problems, and clinicians must be aware of the issues confronting the basic researcher. Effort must be expended so that colleagues active in each area are aware of each other's progress (see Chapter 23).

Probably one of the biggest hurdles to communication is understanding each other's "language." For example, the acronym CAP is interpreted by physiologists as *compound action potential*; however, to most clinicians, CAP is likely to mean central auditory processing! Familiarizing oneself with terminology is a first step toward improving communication. One way to become familiar with terminology and to achieve the larger goal of communication would be served by clinicians and scientists attending sessions in the other's area at major conferences. Also, clinicians and scientists should at least browse some of the key journals in each other's areas on a more regular basis, focusing on those reports carrying

implications for one's clinical practice or research. For example, *Hearing Research*, the *Journal of the Acoustical Society of America*, the *Journal of Neuroscience*, and the *Journal of the Association for Research in Otolaryngology* are science publications that frequently contain articles pertinent to clinicians. Likewise, auditory neuroscientists should become familiar with the *Journal of the American Academy of Audiology*, the *International Journal of Audiology*, and the *American Journal of Audiology*, which are primarily clinically oriented journals. A journal such as *Ear and Hearing* publishes both basic research and clinical reports and should serve as informative "crossover" reading.

Perhaps one of the most useful approaches to foster communication between the clinician and scientist is for these practitioners to spend time in each other's lab or clinical setting, observing each other's daily routine. This is an invaluable experience that can establish rapport and provide knowledge and insights that benefit the scientist's research and the clinician's patients. This in turn can lead to shared information sessions and even collaborative projects that bridge clinical and research interests.

Summary

In this introductory chapter, we emphasized the important role of the scientific "attitude" and the scientific method of inquiry for the clinical process. Following a brief overview of the auditory neuroscience chapters in this volume, we provided examples of the impact of advances in auditory neuroscience for CAPD diagnostic approaches and the reciprocal role of clinical studies in fueling basic science. The chapter concludes with several suggestions to improve interdisciplinary communication and augment the mutually beneficial relationship between scientists and clinicians.

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CHAPTER 2

PSYCHOACOUSTIC CONSIDERATIONS AND IMPLICATIONS FOR THE DIAGNOSIS OF CENTRAL AUDITORY PROCESSING DISORDER

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The purpose of this chapter is threefold: (1) review selected aspects of traditional and contemporary psychoacoustics; (2) focus on psychoacoustic phenomena that may have a direct bearing on the evaluation of central auditory processing disorder (CAPD); and (3) convince the reader that the study of psychoacoustics within the context of CAPD is needed. Beginning with the third purpose, we would note that the rationale that Zeng, Oba, Garde, Sininger, and Starr (2001) provided for the psychoacoustic study of auditory neuropathy can apply equally well to the study of CAPD. First, the basic auditory function of a listener with CAPD needs to be described if we are ever to discover the reason for the common observation that an individual with CAPD hears but does not understand. Second, we need to develop behavioral tests that can provide a more accurate differential diagnosis and identify underlying physiological processes related to CAPD. Third, the psychoacoustic study of CAPD may provide insight into the development of (re)habilitation programs for the successful treatment of the listener with CAPD. For a more conventional review/update on psychoacoustics, the reader is referred to selected textbooks (Gelfand, 2009; Moore, 2012, 1998; Yost, Popper, & Fay, 1993) and textbook chapters (Humes, 1994; Kidd, 2002; Yost, 2007).

Psychoacoustics is the branch of psychophysics that is concerned with the relationship between a stimulus and the perception of that stimulus by the listener's sensory system. Psychoacoustics deals with how changes in the physical parameters of sound affect the psychological detection of that change

and attempts to infer from a listener's responses what the stimulus has evoked in the auditory system. Naturally, each response from the listener consists of two aspects: (1) a change in the listener's sensory perception (sensitivity), and (2) the listener's response criteria, that is, biases that contribute to the response. Psychoacoustic methodology is driven by attempts to control or at least minimize the effect of the listener's response bias on the actual response.

[redacted]sical Psychoacoustic Methods

The three classical psychoacoustic methods are: (1) method of limits, (2) method of adjustment, and (3) method of constant stimuli. Each method has positive and negative aspects, and each is used for absolute and difference threshold measures. Audiologists are familiar with the Békésy tracking method, which is an adaptation of both the method of limits and the method of adjustment.

Method of Limits

The method of limits is in part the foundation on which the well-known modified Hughson–Westlake procedure (Carhart & Jerger, 1959) is based. All the acoustic stimuli are under the control of the examiner, who presents to the listener (subject) a series of acoustic stimuli that change in a predetermined step. The listener either responds, indicating perception of the stimulus change, or fails to respond, signifying that the stimulus change was not perceived. The procedure to estimate absolute threshold may be used in an ascending series, with the starting signal being at a level predetermined to be below the listener's threshold. Or, conversely, a descending series may be used, beginning at a signal level predetermined to be above the listener's threshold. Conventionally, alternating descending and ascending series are utilized with a predetermined number of trials used in each series (run). The listener's response is recorded for each trial and the response reversal point is determined for each series (Figure 2–1). The mean of the response reversal points defines the 50% threshold.

In the measurement of differential threshold, the method of limits test paradigm utilizes the presentation of two stimuli for each trial, with the level of the first stimulus fixed and the level of the second stimulus varied in a predetermined step size. The subject's task is to determine whether the second stimulus is greater than, less than, or the same as the first stimulus. The results of a differential threshold measure using the method of limits results in a range of values in which the second stimulus is: (1) greater than the first stimulus, (2) less than the first stimulus, or (3) equal to the first stimulus. The difference between the 50% point of the values between greater than and equal to and the 50% point between equal to and less than is referred to as the "interval of uncertainty" and is used in determining the just noticeable difference (jnd).[1] If the pluses and minuses in Figure 2–1 are substituted with the word "greater," "lesser," or "equal," then the figure would illustrate a differential sensitivity measure using the method of limits.

[1]The jnd is considered to be one-half of the interval of uncertainty (Gelfand, 2004).

dB	Run	1	2	3	4	5	6	7
	Direction	Descend	Ascend	Descend	Ascend	Descend	Ascend	Descend
20		Yes						
18								
16		Yes						
14				Yes				
12		Yes	Yes	Yes		Yes		Yes
10			No	Yes	Yes	Yes	Yes	Yes
8		Yes	No	Yes	No	No	No	No
6			No	No	No		No	
4		No	No		No			
2			No					
	Threshold	6 dB	11 dB	7 dB	9 dB	9 dB	9 dB	9 dB

Figure 2–1. Method of limits that results in an average threshold of 9 dB with the first descending trial not included in the average. Each *yes* indicates that the listener responded to the stimulus and each *no* indicates that the listener did not respond to the stimulus.

The method of limits suffers from two types of bias. First, since the stimulus presentation is under the control of the examiner, tester bias can come into play as it can in pure-tone audiometry. Second, the listener's bias for anticipating the stimulus level at which the response reversal will occur in either the ascending or descending stimulus series can affect the results. In an attempt to control for these bias effects, an equal number of ascending and descending trials are often utilized, or alternatively a random ascending-descending presentation schedule is used. In addition to these biases, the accuracy of the method of limits is affected by the step-size used in the trial series. Too large a step-size results in an imprecise estimate of threshold, and too small of a step-size results in an ineffective use of test time. Step size selection by the experimenter is a balance between selecting a step-size that is large enough to quickly arrive at threshold and small enough to offer the precision necessary for an accurate measure of threshold. Last, the method of limits does not allow a distinction in the listener's response between a true sensory change perceived by the listener and a change in the listener's response criterion (bias).

Method of Adjustment

The psychophysical method of adjustment differs from the method of limits

in that the listener, not the examiner, controls the stimulus, although the examiner sets the initial level of the stimulus and the subject adjusts the stimulus according to the test instructions. The subject's adjustment of the stimulus variable is continuous via an unmarked dial and is not stepped as in the method of limits. Typically, the examiner has a control dial that is used to set the initial level of the stimulus, which is unknown to the listener. This initial level is either below or above some preselected starting point that is varied by the examiner. Obviously, if the initial level is above the listener's threshold, the trial will be a descending one, with the listener decreasing the level of the stimulus until it is below threshold, and conversely if the starting level is below the listener's threshold, the trial will be an ascending one, with the listener increasing the level of the stimulus until it is above threshold. Like the method of limits, the method of adjustment is applicable to both absolute and differential threshold measures.

Although, the method of adjustment removes examiner bias, it is not without other problems. As the stimulus is for the most part under the control of the listener, the test paradigm can be affected by an undetected change in the listener's response criterion during a trial. Listeners have a tendency to undershoot or overshoot their threshold level by excessively decreasing or increasing the stimulus level. To control or at least minimize this exaggerated threshold effect, testers have used alternating ascending and descending trials. Furthermore, like the method of limits, the method of adjustment does not allow a distinction in the listener's response between a true sensory change perceived by the listener and a change in the listener's response criterion.

Method of Constant Stimuli

The method of constant stimuli differs from the method of limits and adjustment by using a random stimulus presentation mode from a pretest selection of at least 10 stimulus levels (Humes, 1994), thus not utilizing an ascending and descending test paradigm. Typically, a minimum of 10 presentations per stimulus level are used in the test paradigm (Humes, 1994). In an absolute threshold measure, the examiner will preselect the stimulus levels and step size and randomize the presentation sequence. In addition to measuring absolute threshold, the method of constant stimuli can be used to measure differential threshold using a two stimuli paradigms, requiring the subject to make a judgment about the second stimulus in reference to the first stimulus. Again, the examiner will preselect the stimulus values for the two stimuli, the step-size, and the presentation order. An advantage of the method of constant stimuli is that the examiner can get an estimation of a listener's guessing behavior by inserting blank or "catch" trials in the predetermined test schedule. The listener is to indicate yes or no through either a verbal or a manual response for each stimulus presentation during the response interval, which is usually designated by two visual markers, which are viewed by the subject. From the listener's responses, the percentage of correct responses is computed for each stimulus level and displayed in an S-shaped psychometric function (Figure 2–2). From the psychometric function, the 50% point or a more stringent criterion, for example, the 75% point, can define the threshold. The weakness of the method of constant stimuli is the test time required to obtain sufficient data points to construct the psychometric function.

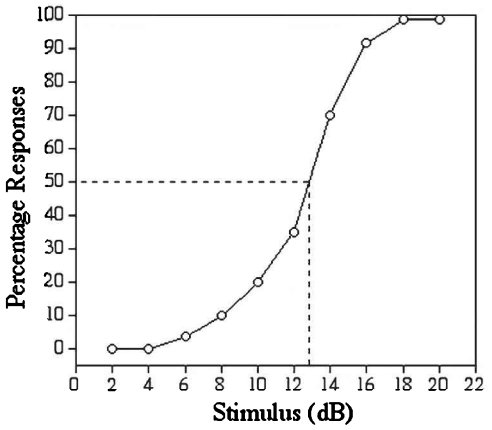

Figure 2–2. Psychometric function with a 50% threshold of 13 dB.

Selective Adaptive Methods

Adaptive methods have increased the efficiency of absolute and differential threshold measures by utilizing test paradigms that converge on the threshold value by quickly eliminating values that are distant from the threshold value. In adaptive methods, the level of any given stimulus is determined by the listener's response to the previous stimulus. Adaptive test strategies often initially use a large step size that is reduced as the stimulus value gets closer to threshold.

Staircase Method

The staircase method (Levitt, 1971, 1978) is similar to the method of limits in that the stimulus value is decreased until the listener gives a negative response and then is increased until the listener gives a positive response. The difference between the method of limits and the staircase method is that the testing sequence does not stop when the listener's response changes in the staircase method. Similar to the method of limits, a specific step-size is utilized in the staircase method. A test sequence (run) is begun using a large step-size in decreasing value until the listener gives a negative response. The test run reverses direction using a smaller step size that is generally one-half of the original one (Figure 2–3). The direction of the runs continues to be reversed at each change in the listener's response from positive to negative and negative to positive until six to eight reversals have occurred; the first reversal is not included. The 50% threshold point is determined by taking the average of the positive and negative reversal values, excluding the first reversal value. The staircase method is very efficient, as it converges on the 50% point quickly, if the step size is neither too small nor too large. Too small a step size would result in unnecessary runs, while too large a step size would result in a less than precise 50% threshold point.

Point Estimation by Sequential Testing

Adaptations of the staircase method are the point estimation by sequential testing (PEST) method (Taylor & Creelman, 1967) and the transformed up-down procedure (Gelfand, 2009; Levitt, 1971, 1978). The PEST procedure utilizes a set of testing rules to either half the stimulus level or double the stimulus level depending upon the listener's response behavior. Although the staircase method can measure only the 50% threshold point, the PEST procedure can obtain any preselected point on the psychometric function. Similarly, a transformed up-down

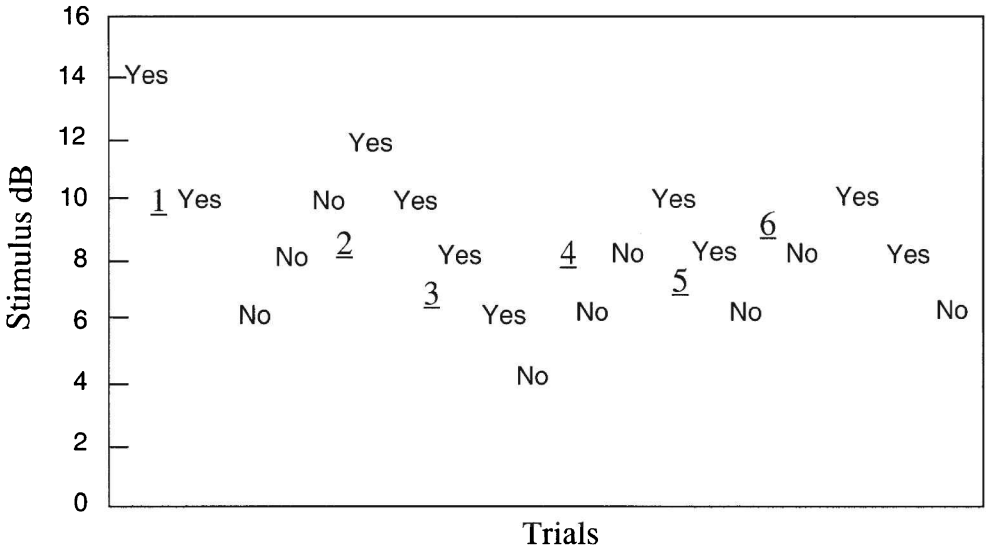

Figure 2–3. The adaptive staircase method with each yes signifying a positive response and each no signifying a negative response. The first descending trial is not included in the average.

procedure can be used to estimate a point on the psychometric function that is above the 50% point; the 71% (70.7%) point is commonly used. The more stringent 71% criterion is obtained by altering the up-down rules such that an increase in stimulus value requires a different response criterion than a decrease in stimulus value. For example, an increase in stimulus value would require a negative response or a positive response followed by a negative response, whereas a decrease in stimulus value would require two positive responses. Like the traditional staircase method, neither the PEST nor the transformed up-down procedure account for the listener's guessing behavior or a change in the listener's response criterion.

Alternative Forced-Choice Methods

A yes/no response mode similar to the method of constant stimuli lends itself to evaluation using an interval forced-choice paradigm. A response interval is established during which the listener must respond yes or no to each presentation; a one-alternative (interval) forced choice. More often than not, however, a multiple alternative (interval) forced-choice paradigm is utilized, such as a two-alternative forced-choice procedure during which the listener must indicate in which of two intervals the stimulus is present. The multiple alternative (interval) forced-choice paradigm can be extended to three or four response intervals and can be paired with an adaptive procedure. In this combined paradigm, the stimulus value is adjusted according to a predetermined rule such as a two-down one-up criterion during which the stimulus value is reduced only after the listener has two successive correct responses. The stimulus value is increased after one error response that would result in the 71% point on the psychometric function. Other adaptive rules

estimate different points on the psychometric function. For example, three-down one-up and four-down one-up rules will estimate the 79% and 84% correct points, respectively, on the psychometric function (Levitt, 1971).

[redacted]eory of Signal Detection

As mentioned in the previous sections, the "classical" and adaptive psychophysical methods do not separate a change in sensitivity from a change in the listener's response criterion (bias). On the other hand, the theory of signal detection (TSD) (Green & Swets, 1974; MacMillan & Creelman, 1991) does provide a procedure that separates a change in the listener's sensitivity from a change in the listener's response criterion (bias). Similar to the method of constant stimuli, the TSD procedure can utilize "catch" trials to estimate a listener's guessing behavior. For example, in a 100 trial run, a single stimulus value is presented during 50 trials and the rest of the trials contain no stimulus, in other words, catch trials. This test paradigm will result in four possible outcomes: (1) the signal is present and the subject says yes, (2) the signal is not present and the subjects says no, (3) the signal is present and the subject says no, and (4) the signal is not present and the subjects says yes. These four possible outcomes can be expressed in a 2 × 2 matrix. Figure 2–4 displays these four outcomes using the TSD nomenclature of hit rate, correct rejection rate, miss rate, and false alarm rate. Similar tables would subsequently be constructed for each stimulus value. From these matrices, a receiver operator characteristic (ROC) curve is constructed (Figure 2–5), which plots the hit rate on the vertical axis (ordinate) against the false alarm rate on the horizontal axis (abscissa). The diagonal line in Figure 2–5 represents chance performance. Curves 1 and 2 depicted in Figure 2–5 represent the listener's sensitivity to two different stimulus values, whereas points A, B, C and D, E, F represent the listener's response criterion to the two different stimulus values. In short,

Stimulus	Response: Yes	Response: No
Present	Hit	Miss
Absent	False Alarm	Correct Rejection

Figure 2–4. 2 × 2 matrix that would summarize the four possible stimulus-response outcomes for a series of test trials at the same stimulus value.

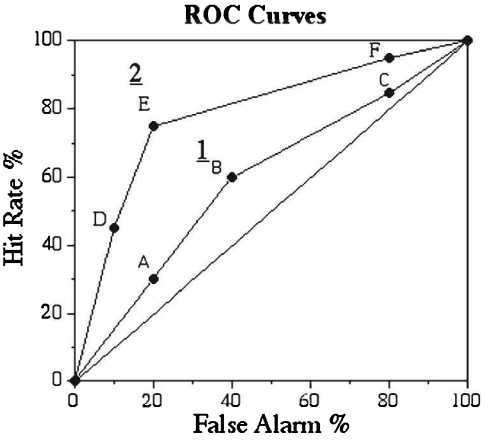

Figure 2–5. ROC curves with the diagonal line representing a d′ value of zero. Curves 1 and 2 differ in sensitivity, whereas points A, B, C, and D, E, F reflect different response criteria.

curves 1 and 2 depict different sensitivity to the stimulus value, whereas points A, B, C and D, E, F depict changes in the listener's response bias. If points A, B, C are at the same location as points D, E, F on their respective ROC curves, the listener's response bias would be considered the same for the two ROC curves. A true change in the listener's sensitivity is reflected in an increase/decrease in the hit rate and no change in the false alarm rate. Conversely, a change in the listener's response bias results in proportional changes in both the hit and false alarm rates.

The listener's response bias can, however, be manipulated by the examiner through instructions given to the listener. For example, the listener can be encouraged to guess, which would result in an increase in the hit rate; however, since the listener's response bias is being manipulated, there would be a proportional increase in the false alarm rate. Or the listener could be instructed that guessing is frowned upon, which would decrease the false alarm rate but would increase the miss rate by changing the listener's response bias. The listener's response bias can also be manipulated by pairing a reward or conversely a penalty to each response, such as a financial reward for each hit and a financial fine for each false alarm.

The index of sensitivity in TSD is the d′ value, which is the amount of separation between the distributions for the stimulus and nonstimulus (catch trials) conditions, that is, separation between the hit rate and correct rejection rate; d′ is a measure of the overlap of the hit rate and false alarm rate probability distribution curves, which in theory are normally distributed and have equal variance. As the overlap of the hit rate and false alarm rate probability distribution curves decreases, the d′ value becomes larger, signifying an increase in sensitivity. The most expedient way to determine d′, assuming that the hit and false alarm rates are known, is to use available tables (Swets, 1964). Pictorially, the ROC curve that has the greatest displacement from the diagonal (d′ = 0.0) toward the upper left corner, where the hit rate is high and the false alarm rate is low, shows greater sensitivity to a stimulus value and produces the largest d′ value (see Figure 2–5).

Clinical Decision Analysis

An outgrowth of TSD is clinical decision analysis (CDA), which has been used in the past to determine the efficacy of CAPD tests (Hurley & Musiek, 1997; Singer, Hurley, & Preece 1998). In the CDA model, the hit rate is the percentage of subjects correctly identified by a test score; the false alarm rate is the percentage of subjects incorrectly identified as positive by a test score; the miss rate is the percentage of subjects incorrectly identified as negative by a test score; the correct rejection rate is the percentage of subjects correctly identified as negative by a test score; and d′ or A′ is a measure of the overall performance of various test scores (Hyde, Davidson, & Alberti, 1991; Turner, Robinette, & Bauch, 1999). Although d′ is dependent on the form of the hit rate and false alarm rate probability distribution cures, A′ is not, as it is a nonparametric measure with values ranging from 0.50 for a test of no diagnostic value to 1.00 for the perfect diagnostic test. The formula to compute A′ may be found in Turner et al. (1999). As audiology tests are known to have hit rates and false alarm rates that are not normally distrib-

uted or of equal variance (Turner et al., 1999; Turner & Nielsen, 1984), the nonparametric A′ value is used in CDA as an indication of overall performance. For a comprehensive discussion of d′ and A′, see Turner and Nielsen (1984), and for an expanded treatment of CDA, the reader is referred to Hyde et al. (1991) and Turner et al. (1999). (See Chapter 12 for application of CDA in the central auditory test battery.)

[redacted]erential Sensitivity: [redacted]uency and Intensity

Many studies have sought to quantify the number of hertz (Hz) or decibels (dB) that are needed for the listener to detect the respective change in frequency or intensity. In short, the investigations have asked the question: How large of a Hz or dB difference must there be between two tones before the listener can detect a change? These measures of differential sensitivity are referred to as the jnd or the difference limen (DL) and can be characterized as an absolute quantity or a relative quantity, that is, a ratio. The ratio, which is often referred to as the Weber fraction, is computed by dividing the absolute DL (Δ) value by the base value, that is, $\Delta f/f$ and $\Delta I/I$ for frequency (f) and intensity (I), respectively. For example, if the base value was 1000 Hz and the DL (Δ) was 10 Hz, the $\Delta f/f$ ratio would be 0.01.

One might ask, why should differential sensitivity be studied within the context of CAPD? Kidd (2002) identified three strong reasons for investigating DLs (jnds) for frequency and intensity, which can be readily applied to the investigation of CAPD. First, DLs will provide insight into how effective a listener's auditory system codes the acoustic parameters of frequency and intensity. (See this chapter's section on differential sensitivity for frequency.) Second, DLs can be used either to develop models of hearing or to test normal/abnormal models of auditory function through such techniques as computer simulation. And third, a listener's DLs can be compared with normative standard values, providing further insight into an impaired listener's auditory dysfunction(s). The above three reasons are certainly sufficient to justify studying DLs in individuals suspected of having CAPD (see Zeng et al., 2001).

Differential Sensitivity for Frequency

Historically, two paradigms have been used to measure differential sensitivity for frequency (Δf). The first paradigm is a frequency modulation (FM) technique where tones are modulated at a low rate of 2 to 4 Hz, with the listener's task being to detect the change in modulation rate. The second paradigm has used a successive steady tone technique where the pulsed tones differ in frequency by a small amount and the listener's task is to indicate whether the first or second tone is higher in frequency. The minimal amount of frequency change that a listener can detect between the successive tones is the DL value.

The seminal study by Shower and Biddulph (1931) was the first to successfully delineate the ear's differential sensitivity (DL) for frequency. By using the FM technique, they overcame the technological limitation of that era, which was the production of transient clicks that resulted from turning a pure-tone signal on and

off. Subsequent investigations (Harris, 1952; Moore, 1973; Wier, Jesteadt, & Green, 1977) using the successive pulsed tone technique were not in agreement with the Shower and Biddulph 1931 study. The main reason for the difference between the data of Shower and Biddulph and the more recent investigation of Wier et al. (1977) is attributed to the different test paradigms, with the FM technique producing a stimulus with a more complex acoustic spectrum, resulting in a different form of phase-locking for neural coding than the successive pulsed tone technique (Moore, 1998; Gelfand, 2009). In addition to the measurement paradigm, the parameters of sensation level (SL) and tone duration will affect the absolute DL value (Moore, 1973; Wier et al., 1977)

The most recent investigations (Moore, 1973; Wier et al., 1977) have shown smaller DL values at low and mid frequencies (<2000 Hz) and larger DL values at higher frequencies (>2000 Hz) (Figure 2–6). The Wier et al. (1977) data at 40 dB SL shows the DL value at 200 to 800 Hz to be approximately 1.0 to 1.5 Hz and at 1000 to 2000 Hz to be approximately 2 to 3 Hz. As illustrated in Figure 2–6, above 1000 Hz, the DL value increases to 16 to 18 Hz in the region of 4000 Hz and rapidly increases above 4000 Hz to 67 to 70 Hz in the region of 8000 Hz. These data are interpreted to be consistent with the VIIIth nerve's phase-locking characteristics for frequency, which decrease about 1000 to 2000 Hz and is absent above 4000 to 5000 Hz (Moore, 2012), with place information being responsible for frequency perception above 5000 Hz. Naturally, place and phase-locking mechanisms do not operate exclusively of one another, with the place mechanism being in operation over the range of human hearing with phase-locking resulting in better frequency discrimination up to 4000 to 5000 Hz (Moore, 1993). With all that is known about human DL for frequency performance, it is surprising and unfortunate that a clinically useful DL test for frequency has not been clinically developed for auditory processing.

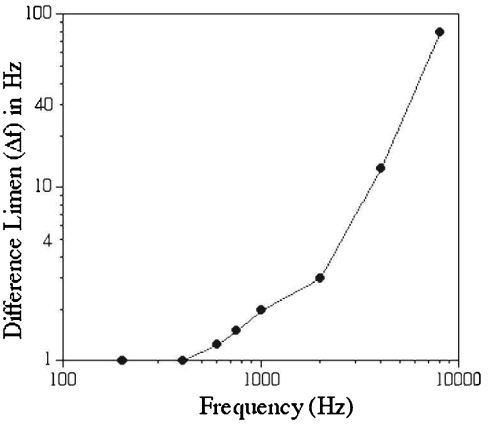

Figure 2–6. Frequency difference limen (Δf) at a sensation level of 40 dB, from Wier et al. (1977).

Differential Sensitivity for Intensity

Historically the main methods to measure differential sensitivity for intensity (ΔI) have been modulation detection, increment detection, and intensity discrimination between successive pure tones (Moore, 1998). Using an adaptive two-alternative forced-choice technique (2AFC), the modulation detection paradigm would present an unmodulated interval and amplitude modulated (AM) interval to the listener, whose task would be to indicate which interval contained the AM signal. The increment detection

(continuous pedestal) paradigm using 2AFC would have a continuous tone occurring during both intervals, with one interval containing an increment superimposed on the continuous tone, much like the classic Short Increment Sensitivity Index (SISI) test (Jerger, Shedd, & Harford, 1959). It is important to note the SISI is a test of recruitment, and the ΔI is not explored. The listener's task is to indicate which interval contains the increment. For the intensity discrimination between successive pure tones paradigm, the listener is presented two successive tones/pulses (gated pedestal), with one tone being more intense than the other and with each tone assigned to a separate interval. Again, the listener's task is to identify the interval that contains the more intense tone.

In the seminal study on differential sensitivity for intensity, Riesz (1928) (like Shower and Biddulph in 1931) overcame the transient click problem of that era that resulted from turning pure tones off and on by using AM tones, where two pure tones that differed slightly in frequency were presented simultaneously. For example, if a 1000-Hz and a 1003-Hz tone were presented simultaneously, the resulting tone would modulate (beat) at a rate equal to the frequency difference of the two stimuli, which in this case would be 3 Hz, which Riesz determined to be the optimum modulation rate to study DLs for intensity. Reisz's listeners adjusted the intensity of the AM tone until they could detect the beats for a broad range of frequencies (35–10,000 Hz) presented at an equally broad range of SLs (0–100 dB). Weber's law predicts that the differential sensitivity fraction/ratio (ΔI/I) will be constant irrespective of stimulus level or frequency. Riesz (1928), however, demonstrated that the ΔI/I had an intensity effect particularly at low SLs, where the fraction was reduced as intensity increased. At moderate and high SLs, Riesz's data came close to ap-proximating the prediction of Weber's law; the approximation is referred to as the "near miss" to Weber's law. Subsequent investigations (Florentine, Buus, & Mason, 1987; Jesteadt, Wier, & Green, 1977; Moore & Raab, 1974; Viemeister & Bacon, 1988) have likewise supported the near miss to Weber's law for the SL parameter.

In addition to an intensity effect for ΔI/I, Riesz's (1928) data reflected a frequency effect; the ΔI/I was inversely related to frequency up to 1000 Hz (i.e., ΔI/I was reduced as frequency increased). Above 1000 Hz, Riesz's data were consistent with the prediction of Weber's law. Subsequent investigations (Jesteadt et al., 1977; Schacknow & Raab, 1973), however, did not support Riesz's findings. Specifically, Jesteadt et al. (1977) did not report a frequency effect, showing instead that ΔI/I was constant across frequencies at any given SL. Another investigation (Florentine et al., 1987), did find, in general, a frequency effect, although there was not a strong frequency effect at analogous frequencies tested by Jesteadt et al. (1977). A compilation of results (Gelfand, 2009) suggests that Weber's law is consistent for a range of SLs, 10 to 40 dB (Rabinowitz, Lim, Braida, & Durlach, 1976) or 20 to 50 dB (Viemeister & Bacon, 1988), but shows an SL effect above and below these ranges. In short, DLs for intensity become smaller as the SL increases for mid frequency stimuli (Gelfand, 2009).

Differing intensity DL results among investigations could be attributed to the test paradigm differences. Recall that Riesz (1928) used the AM methods, whereas Jesteadt et al. (1977) used the increment

detection (continuous pedestal) method, and others (Florentine et al., 1987; Viemeister & Bacon, 1988) utilized the intensity discrimination between successive tones (gated pedestal) method. Turner, Zwislocki, and Filion (1989) compared the increment detection and successive tone methods and reported smaller intensity DLs for the increment detection method.

Profile Analysis

As reflected above, the traditional method for studying intensity discrimination was to use individual single frequencies, that is, noncomplex stimuli. Profile analysis (Green, 1988) is a contemporary paradigm that utilizes a multifrequency complex to study intensity discrimination. Typically, the stimulus comprises a 21-frequency complex, encompassing a range of frequencies, such as 300 to 3000 Hz or 200 to 5000 Hz, in which all the components are equally, logarithmically spaced along the frequency range and are of equal amplitude. In the typical 2AFC paradigm, the listener is presented with a standard (reference) stimulus of a 21-frequency complex in one interval, and in the other interval a background stimulus made up of a 20-frequency complex with a single frequency being greater in intensity than the other component frequencies (Figure 2–7). Naturally, the listener's task is to identify the interval that contains the signal/target frequency that is contained within the multifrequency background complex. The frequency of the target signal frequency is varied on each trial. In order to ensure that the listener is discriminating spectral shape rather than the overall spectrum level of the two stimuli, a roving standard procedure is used whereby the intensity of the standard (reference) stimulus and target signal are randomly varied for each presentation.

The intensity discrimination threshold derived from a profile analysis task appears to be better than comparable DLs for single frequency paradigms. The profile analysis thresholds are, however, affected by the frequency location of the increment component signal within the multifrequency background complex. When the frequency of the signal increment component is in the middle of the multifrequency complex (500–2000 Hz), the thresholds are smaller than when the increment frequency is closer to each end of the frequency range. The profile analysis thresholds are better when the range of frequencies that comprise the background stimuli widen and the number of frequency components within the range increase (Green, 1993). Conversely, the profile analysis threshold becomes poorer when components that are near in frequency to the target frequency are added (Green, 1993).

Loudness

While the physical measure of sound is the decibel scale, the subjective measure (psychological attribute) of sound is loudness. Although there are similarities between the two measurements, such that loudness increases when intensity increases, they do not increase at the same rate, which is illustrated by loudness contour curves, which are the common way to equate intensity and loudness. These contour curves plot the intensity of different frequencies as they are compared with the loudness of a 1000-Hz reference

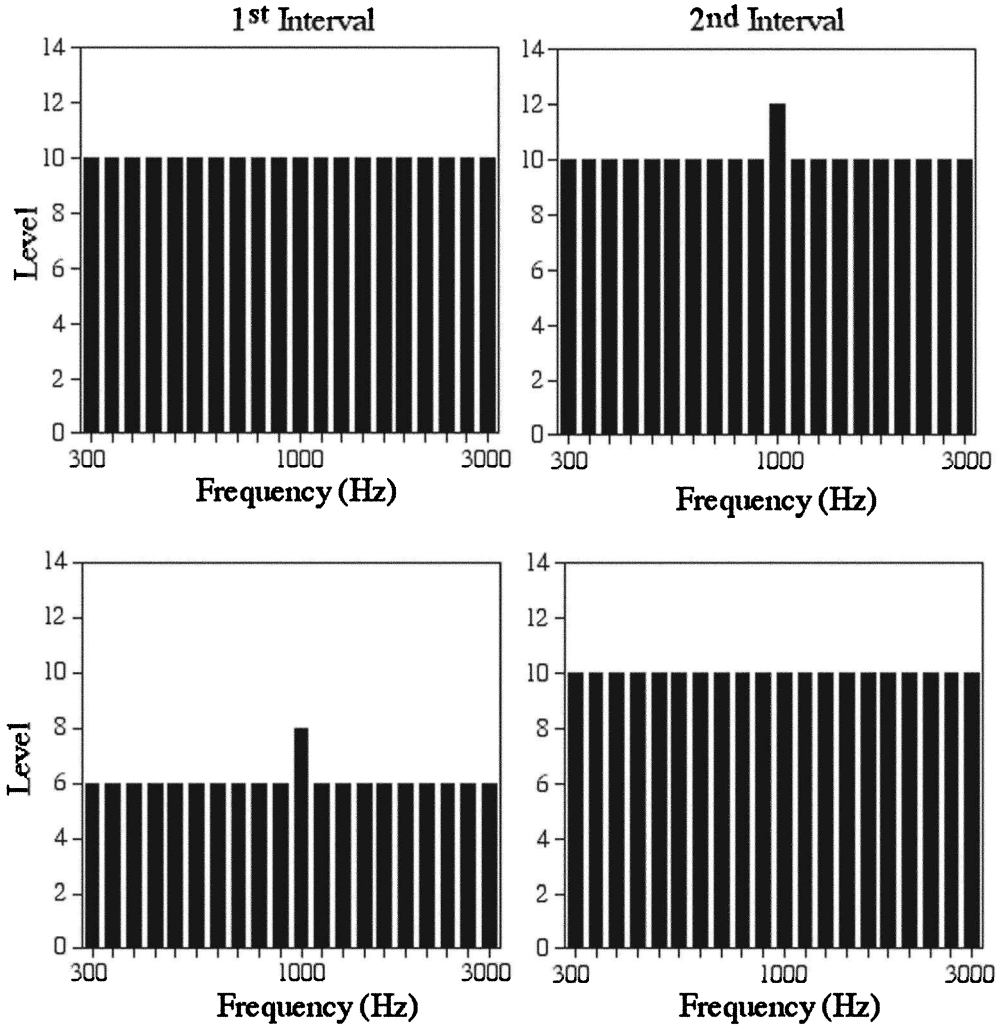

Figure 2–7. Profile analysis using a two–interval forced choice procedure with roving levels. The listener is to indicate which interval contains the signal level that differs from the background frequencies.

tone so as to be perceived as equal in loudness to the reference tone.

Fletcher and Munson (1933) established loudness contour curves by asking listeners to match the loudness of tones of varying frequencies to the loudness of a 1000-Hz reference tone set at varying sound pressure levels (SPLs). Figure 2–8 displays an example of loudness contour curves. Examination of these loudness contour curves shows that the sensation of loudness grows faster in the low frequencies than it does in the high frequencies. Notice how the rate of loudness grows steeper for low frequencies as the intensity of the reference decreases. Stated differently, more energy is required to reach the same loudness level as

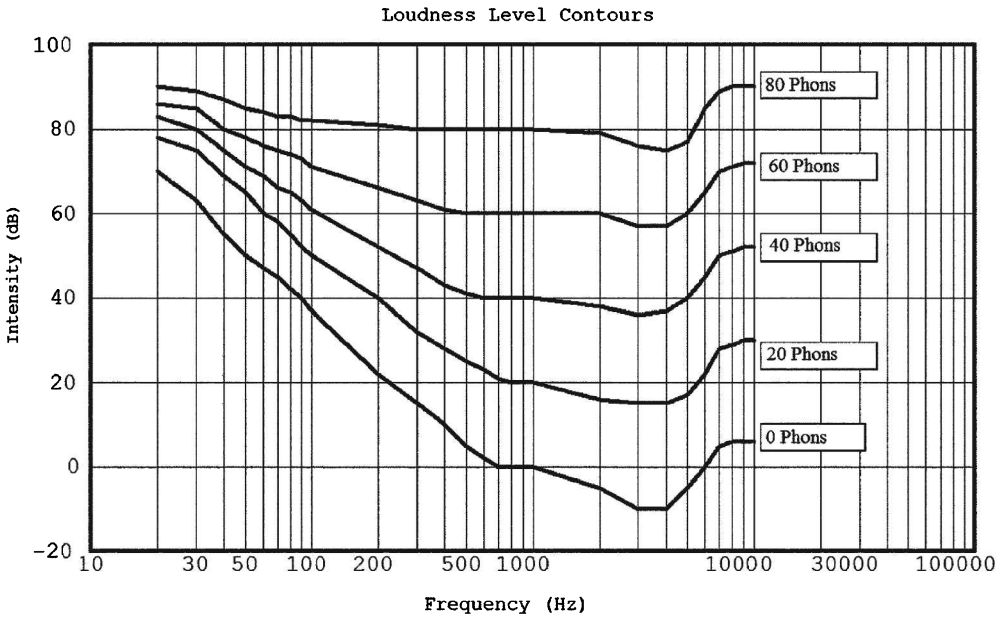

Figure 2–8. Equal loudness contours based upon the data of Fletcher and Munson (1933). Frequencies are depicted along the *x*-axis and the perceived loudness intensity is along the *y*-axis. Redrawn with permission from Harvey Fletcher, *Journal of the Acoustical Society of America, 5,* 82. Copyright 1933, Acoustical Society of America.

frequency decreases. The rate of loudness growth remains relatively flat across the mid-frequencies; however, an increase in the growth of loudness is noted in the extreme high frequencies. Note that the loudness contour curve at 0 dB equals the minimum audible field (MAF) threshold curve. Equal loudness contours may be used to ensure that tests that utilize two or more different frequencies do not provide a loudness cue, making sure that the differing frequencies are of equal loudness—for example, as in the Frequency (Pitch) Patterns Test (Musiek & Pinheiro, 1987).

The phon is the unit of loudness level most commonly used and is referenced to a 1000-Hz tone at 40 dB SPL. As illustrated in Figure 2–8, a tone with a loudness of 40 phons would match the loudness of a 40 dB SPL tone at 1000 Hz, and a tone with a loudness of 20 phons would match the loudness of a 20 dB SPL tone at 1000 Hz. Thus, all of the tones (frequencies) on a particular phon curve (equal loudness contour) are considered to be equal in loudness to the reference SPL level at 1000 Hz for that particular loudness contour.

Another way to depict loudness is the sone scale, which provides a comparison in loudness between two stimuli (Figure 2–9). Like the phon scale, the sone scale uses a 1000-Hz tone at 40 dB SPL as the reference. Thus, one sone equals the loudness level of 40 phons; two sones are twice the loudness of one sone; and three sones are three times the loudness of one sone. When loudness is expressed in sones, frequency is not rel-

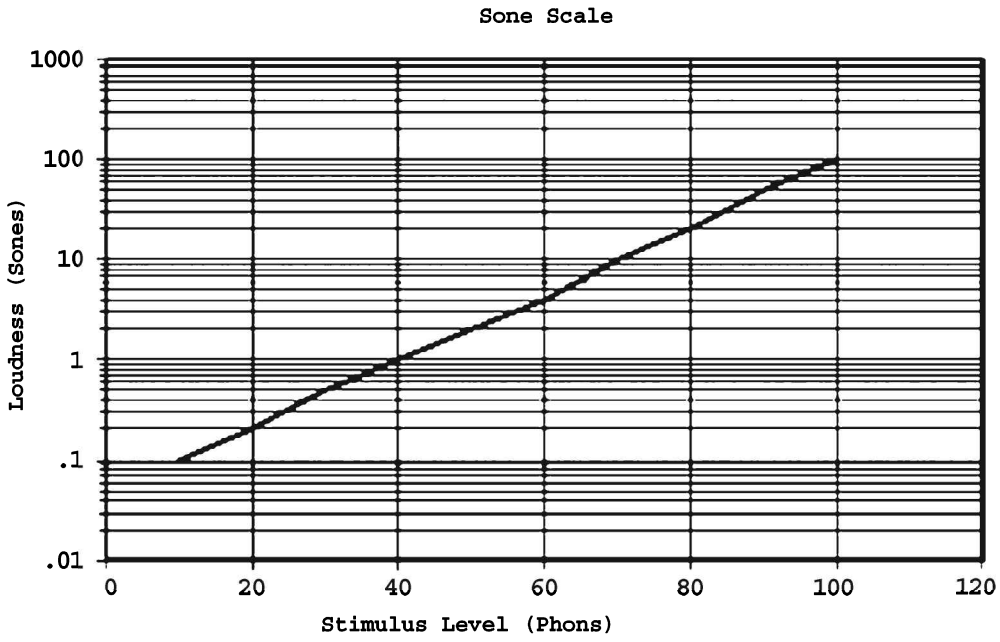

Figure 2–9. Sone scale with loudness in sones depicted as a function of stimulus level in phons such that 40 dB SPL or 40 phons equal one sone. A 10 dB increase in level (to 50 dB SPL) is equal to a doubling in loudness (2 sones). Another 10 dB increase in level (to 60 dB) is equal to another doubling in loudness (4 sones).

evant because the reference is the phon, which represents the same loudness level across frequencies.

Factors Affecting Loudness Perception

Duration also affects loudness perception: The perceived loudness of a stimulus changes as the duration of the tone increases. Similar to absolute threshold and acoustic reflex threshold, loudness perception is affected by temporal integration (i.e., summation; see Temporal Effects section). Specifically, using a loudness balance paradigm with a long (500–1000 msec) reference tone and test tones that vary (10–640 msec) in duration, Richards (1977) demonstrated that loudness perception increases up to approximately 80 msec. A small amount of additional loudness integration does occur beyond the 80 msec "critical duration." The 80 msec critical duration value has, however, been somewhat variable among studies (Gelfand, 2009). The SL for the loudness matching paradigm has been shown to affect temporal integration for loudness, with SLs between 20 and 50 dB demonstrating the maximum temporal integration.

Another loudness duration effect is loudness adaptation, which is the perceived decrease in the loudness of a long, steady tone that is fixed in intensity (Gelfand, 2009). For example, a reference tone is presented and a test tone is matched in

loudness to that reference. The reference tone is presented for a few minutes, followed by the test tone, which is matched in loudness to the reference tone. The next consecutive loudness match will show that the test tone reference tone loudness match was at a lower dB level than the original loudness match, thus reflecting a loudness adaptation effect. Hellman, Mikiewicz, and Scharf (1997) found that loudness adaptation increases as the stimulus frequency increases and the SL decreases. Furthermore, maximum loudness adaptation occurs within three minutes of the stimulus onset.

Like duration, bandwidth affects loudness perception. If the loudness of two tones is compared, they will be perceived as the same loudness as long as they remain within the same critical bandwidth. Once they become more than a critical bandwidth apart, a noticeable increase in loudness will occur (Gelfand, 2009). Suppose a listener is presented with two pairs (sets) of tones that are close in frequency, all at the same intensity level. One set of tones is designated as the reference tones and the other set as the test tones. The listener's task is to compare the loudness of the test tones with the reference tones. The distance between the pair of comparison tones is gradually increased. The listener will perceive the two sets of tones as equal in loudness as long as they are a critical bandwidth apart. Once the distance between the test tones exceeds the critical bandwidth while the reference tones remain constant, a significant increase in loudness perception will occur as the listener compares the test tone complex with the reference tone complex. Further, a loudness summation effect has been reported to occur when more tones differing in critical bandwidth are added to the test tone complex (Florentine, Buus, & Bonding, 1978; Gelfand, 2009).

Pitch

The psychological attribute of frequency is pitch. High-frequency tones stimulating the basal end of the cochlea are perceived as high pitched, and low-frequency tones stimulating the apical end of the cochlea are perceived as low pitched. The basic unit of pitch is the mel, which is referenced to a 1000-Hz tone presented at 40 dB SPL and considered to be 1000 mels in value (Stevens & Volkmann, 1940; Stevens, Volkmann, & Newman, 1937). Thus, a 1000-Hz tone at 40 dB SPL has a loudness level of 40 phons or one sone, and a pitch level of 1000 mels. A comparison frequency that is considered to be double the pitch of 1000 mels would have a pitch of 2000 mels (Figure 2–10).

As with loudness and intensity, pitch and frequency do not increase at the same rate: pitch perception increases at a slower rate than frequency. For example, a 3000 Hz tone will have a pitch of 2000 mels. Thus, the mel scale is a condensed/compressed scale when compared with the physical parameter of frequency, although there is a close relationship between any given pitch on the mel scale and the corresponding point of maximum displacement on the basilar membrane (Humes, 1994). To appreciate further the compressed relationship between frequency and the mel scale, the 0 to 20,000 Hz range of human hearing is compressed into 3500 mels, as shown in Figure 2–10.

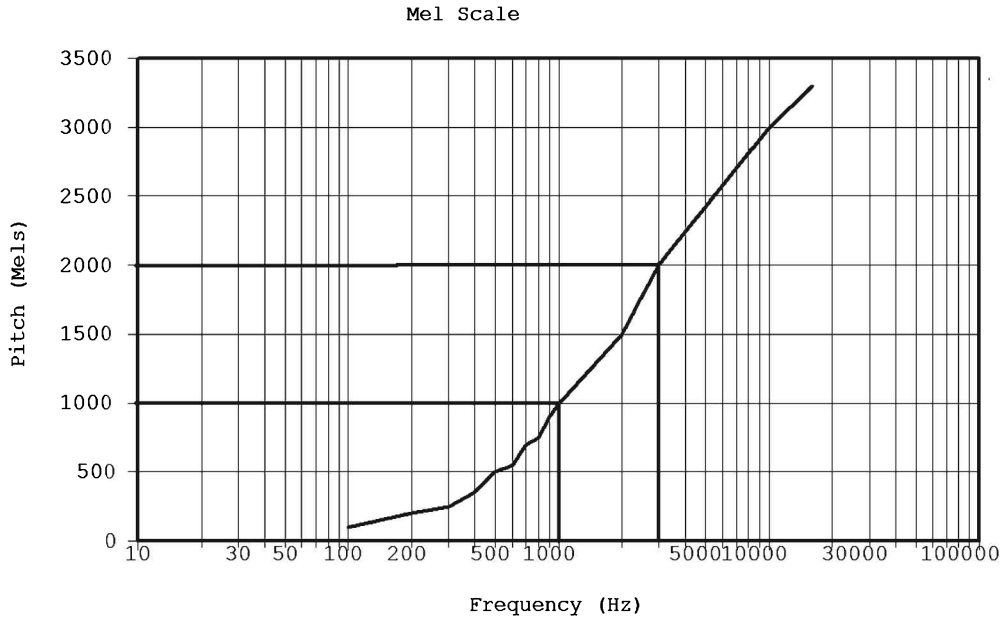

Figure 2–10. A mel scale. In the low frequencies, pitch and frequency grow at a similar rate; however, as frequency is increased, pitch grows at a slower rate. A tripling of frequency from 1000 to 3000 Hz only doubles the pitch from 1000 to 2000 mels. Redrawn from the *Journal of Psychology* with permission of the University of Illinois Press.

Pitch Theories

There are two theories of pitch perception: the place theory and the temporal theory. The place theory assumes that the perceived pitch of a stimulus corresponds to the point of maximum displacement along the basilar membrane. The temporal theory implies that the perceived pitch of a stimulus is related to the temporal pattern of neuron firing (phase-locking) produced by the frequency of the stimulus. The tonotopic organization of the cochlea and the close correspondence between pitch and basilar membrane displacement obviously play roles in our perception of pitch, and support the place theory of pitch perception. The temporal theory of pitch perception is supported by the "missing fundamental" phenomenon, which is also known as "periodicity pitch," "residue pitch," and "virtual pitch" (Gelfand, 2009). Illustrating the missing fundamental phenomenon, listeners match the pitch of the complex tone to the fundamental frequency even when the complex tone comprises only the harmonics of the fundamental frequency but is devoid of the fundamental frequency itself. For example, a complex tone made up of a series of pure tones such as 500, 700, and 900 Hz will be perceived by the listener to have a pitch equal to 200 Hz, which is the fundamental frequency of this series of pure tones.

Although no energy is present at 200 Hz, the listener will perceive the pitch of

the complex stimulus as 200 Hz. Another way to demonstrate the missing fundamental phenomenon would be to present a high-frequency carrier tone that is pulsed at a slow rate. In this demonstration, the listener will choose a tone with the frequency matching the period of the pulse rate (Thurlow & Small, 1955). For example, if a 4000 Hz tone is interrupted every 25 msec, the listener will match the pitch of that tone to a frequency of 400 Hz, as 25 msec is the period of a 400-Hz tone. Since the addition of appropriate ipsilateral masking to a multifrequency complex tone will not mask the pitch of the fundamental frequency, a place theory cannot explain the missing fundamental phenomenon. Thus, what role do the two theories play in pitch perception? A temporal mechanism, that is, VIIIth nerve phase-locking, appears to be responsible for low-frequency pitch perception, and a place mechanism, tonotopic organization of the cochlea, is responsible for high-frequency pitch perception, with both mechanisms operating for mid-frequencies (Humes, 1994; Moore, 1998).

Nonsimultaneous or Temporal Masking

Nonsimultaneous masking refers to a change in detection threshold produced by a test paradigm where the masker and the test signal (probe) do not overlap in time. This psychoacoustic test paradigm is referred to as temporal masking, which takes one of two forms, backward or forward. Backward masking refers to a test sequence where the test signal (probe) precedes the masker (i.e., the masking effect occurs backward in time). Forward masking refers to a test sequence where the masker precedes the test signal (Figure 2–11). In both backward and forward masking paradigms, there is a time separation/delay between the probe and masker. In backward masking, the probe is terminated before the onset of the masker. In forward masking, the masker is terminated before the onset of the probe. Forward masking appears to have been more thoroughly investigated than backward masking, possibly because the backward masking effect

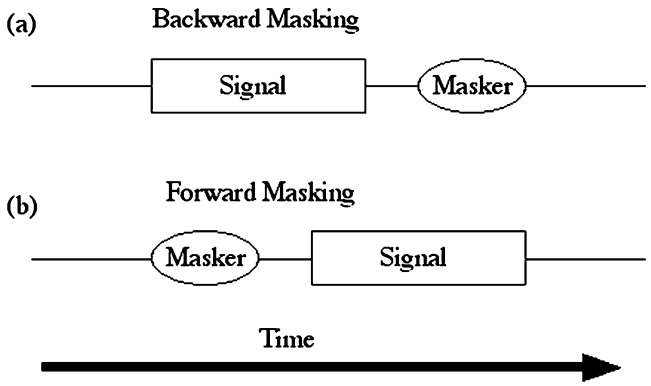

Figure 2–11. (a) Backward masking, where the masker trails the signal, and (b) forward masking, where the masker leads the signal.

is dependent upon the amount of pretest practice the listeners have received (Moore, 1998), as well as attention to task plus the cognitive abilities of the listener (Kidd, 2002; Wright, 1998). Furthermore, minimal to no backward masking effect has been reported in listeners who have received considerable pretest instruction (Oxenham & Moore, 1994).

In comparing the reported backward and forward masking data, Gelfand (2009) offered several observations. First, backward masking provides more masking of the probe tone than does forward masking. Second, ipsilateral masking (probe and masker in the same ear) provides more masking than contralateral masking (probe and masker in opposite ears). Third, shorter delays between the probe and the masker result in greater masking effects. Fourth, the closer the probe and masker are in frequency, the greater the masking effect. In addition, the combination of backward and forward masking in a test paradigm provides a greater masking effect than the simple sum of the backward and forward masking obtained separately (Bilger, 1959; Elliott, 1969).

There is a precipitous reduction in the backward masking effect as the gap between the probe and the masker increases from 0 to 15 msec. Although the backward masking effect can still be seen when the gap is as great as 100 msec (Elliott, 1967), there is very little further reduction in the backward masking effect, as the gap between the probe and the masker is extended from 15 to 100 msec. Similarly, there is a reduction in the forward masking effect, although less precipitous, as the gap between the masker and probe increases from 0 to 25 msec, with a minimal forward masking effect occurring between 25 and 100 msec. In addition to the delay/gap parameter, temporal masking effects are influenced by the duration and intensity of the masker. Specifically, the forward masking effect increases for increments in masker duration up to approximately 20 msec (Moore, 1998), although some studies (Kidd & Feth, 1982; Zwicker, 1984) have reported an effect for masker duration of up to 200 msec. Similar masker duration effects have not been reported for backward masking (Moore, 1998). Increments in the intensity of the masker do not result in equivalent increments in the forward masking effect; roughly, a 10 dB increment in the masker will result in only a 3 dB increment in the masker effect (Gelfand, 2009; Moore, 1998). Comparable intensity effects for backward masking do not appear to have been thoroughly investigated.

Although the exact physiology underlying nonsimultaneous temporal masking is not well understood, it is hypothesized that temporal masking effects may have both peripheral and central components (Duifhuis, 1973; Gelfand, 2009; Moore, 1998). Specifically, the precipitous reduction in temporal masking as the gap between the probe and the masker increases from 0 to 15 to 25 msec has been viewed by some (Gelfand, 2009; Moore, 1998) to represent the peripheral component of temporal masking. Forward masking is thought to be primarily peripheral due to the continued response of the basilar membrane overlapping the response of the probe after the termination of the masker (Gelfand, 2002; Moore; 1997). Also underlying forward masking is the similarity between forward masking characteristics and the short-term adaptation/fatigue characteristics of the VIIIth nerve. Stated differently, the VIIIth nerve's responsiveness

to the signal is reduced because of the short-term adaptation/fatigue produced by the masker (Kidd, 2002; Gelfand, 2009). Arguing against a dominant peripheral source for forward masking is the data from cochlear implant listeners (Chatterjee, 1999; Shannon, 1990), which approximate those from noncochlear implant listeners (Kidd, 2002). In other words, listeners devoid of the cochlea–VIIIth nerve connection produce forward masking patterns akin to listeners with a normal cochlea–VIIIth nerve connection. These data directly question whether the periphery is the predominant source of forward masking. Although it is hypothesized that backward masking, the least understood of the two temporal masking types, is predominantly a centrally dominated phenomenon (Gelfand, 2009; Kidd, 2002), the hypothesis appears to be based more on speculation than research reports. The speculation may be derived from the previously mentioned observation (Kidd, 2002; Wright, 1998) that backward masking is significantly affected by attention factors and cognitive abilities.

Temporal Effects

Temporal Integration

A signal parameter that affects hearing sensitivity for tones is signal duration. Tones with durations of 200 to 300 msec will produce the lowest absolute thresholds. As the duration of a tone is increased from 30 to 300 msec, the absolute threshold will improve by 10 dB (i.e., a 10-fold change in duration results in a 10 dB increment in sensitivity). Conversely, a 10-fold decrease in duration (from 300 to 30 msec) results in a 10 dB reduction in sensitivity. Tonal durations greater than 300 msec do not result in an improvement in absolute threshold. This duration effect is referred to as temporal integration/summation, suggesting that the ear integrates the total stimulus energy over time, which can be described by a constant (i.e., threshold × pulse duration = constant). An alternative to the classic temporal integration model is the multiple look model, which states that the auditory system does not perform an integration function but that the absolute threshold improves because longer stimulus duration provides more opportunity for the auditory system to sample the stimulus (Viemeister & Wakefield, 1991).

Temporal Resolution

The auditory system is required to discriminate small timing differences when processing speech. These differences can be changes over time in the envelope of the signal or changes in the AM of the signal. The process of detecting these quick timing changes is referred to as temporal resolution and can be interrupted by hearing impairment, aging, maturational delay, and possibly CAPD.

Gap Detection

A common method used to assess temporal resolution is to establish a gap detection threshold (GDT). The GDT is the smallest amount of silence between two signals (a sinusoid, broadband noise or a narrowband noise) that a listener can detect. Alternatively, the GDT paradigm may utilize two or more signal pairs with one signal burst containing the silent

gap. In the previous paradigm, the listeners' task was to indicate whether they heard two successive stimuli or one signal. In the latter paradigm, the listeners' task is to indicate which of the stimulus pairs contain the gap. Another paradigm offered by a new gap detection test (Musiek et al., 2005) utilizes a series of broadband noise segments that contain 0 to 3 gaps per segment, with the gaps varying in duration. In any particular noise segment, the location and duration of the individual gaps are randomized (Figure 2–12b).

In the simplistic GDT paradigm illustrated in Figure 2–12a, a narrowband noise signal is broken down into an initial segment followed by a silent gap followed by a final segment. The initial and final narrowband signals are referred to as markers. The length of the gap or time interval between the markers is then adjusted until the listener's GDT (the level just above where the listener cannot hear the gap) is established. At gap intervals below the GDT, the listener will perceive a constant narrowband noise even though a silent gap is present.

When the same signal (narrowband noise) is used as the initial marker (before the gap) as well as the final marker (after the gap), the test is considered to be a within-channel GDT paradigm. According to the hypothesis that gap processing occurs centrally, the same perceptual channel is used to process the signal when it is presented in a within-channel

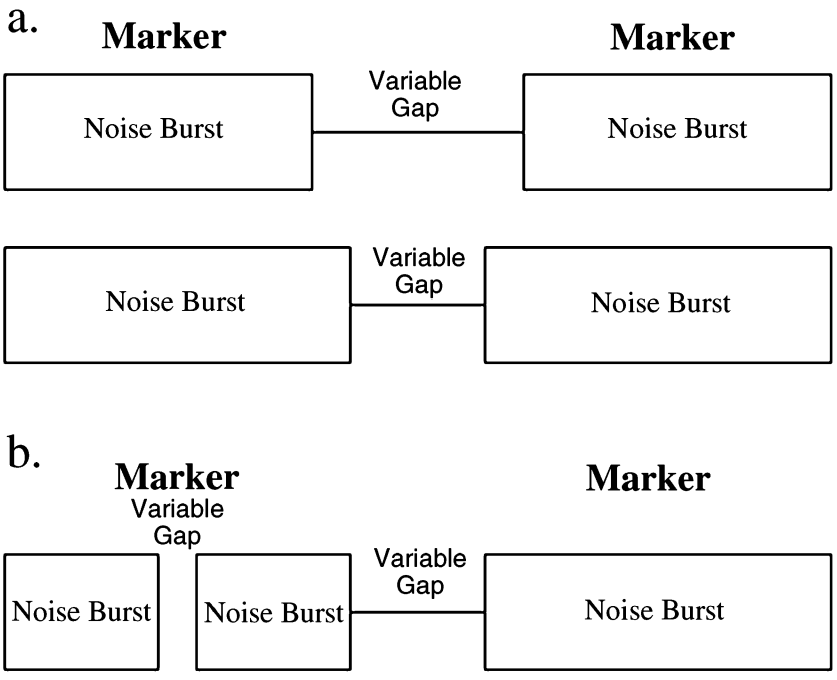

Figure 2–12. Gap detection threshold (GDT) paradigms. (a) The size of the gap between the two bands of noise (markers) is varied, with the smallest gap that a listener can detect being the GDT. (b) The size of the gap between the two bands of noise (markers) is varied as is the size of the gap contained within a marker (Musiek et al., 2005).

paradigm. A within-channel GDT paradigm would be a monotic or diotic presentation of the marker-gap-marker test sequence, with markers that are within a half octave of each other (Formby & Forrest, 1991). For example, the test sequence would begin with an initial 1000 Hz narrowband noise followed by a gap followed by a final 1000-Hz narrowband noise. An across-channel GDT paradigm could involve a dichotic presentation of the marker-gap-marker sequence with similar frequency markers, or a diotic/monaural presentation, but with dissimilar frequency markers (more than half an octave apart). For the across-channel paradigm, the test sequence would start with a 1000-Hz narrowband noise followed by a gap and then terminate with a 2000-Hz narrowband noise. Generally, the within-channel paradigm produces smaller GDTs, on the order of 2 to 24 msec, than those of the across-channel paradigm, which are on the order of 14 to 50 msec (Formby, Gerber, Sherlock, & Madger, 1998; Grose, Hall, & Buss, 2001; Lister, Besing, & Koehnke 2002; Lister, Koehnke, & Besing, 2000). According to the GDT central hypothesis, this type of test sequence is referred to as across-channel because two processing channels are being utilized to accommodate the different inputs (Phillips, Taylor, Hall, Carr, & Mossip, 1997). One processing channel is used for the first marker and a second processing channel is used for the second marker. Since the GDT is similar when the test paradigm is presented dichotically or monaurally with dissimilar frequency markers, it has been suggested that the channels may be centrally located within the auditory system (Formby, Gerber, Sherlock, & Madger, 1998; Phillips & Hall, 2000). In addition, the across-channel paradigm is thought to access higher order cortical auditory function than the within-channel paradigm, thus possibly tapping cortical mechanism that are involved in speech perception (Formby et al., 1998; Phillips et al., 1997).

Hearing Loss and Aging Effects on GDT

GDTs have been found to be similar for listeners with hearing loss and listeners with normal hearing (Lister et al., 2000). This finding lends support to the theory that the perceptual channels for processing gap detection are centrally located, because damage to the peripheral system (hearing loss) does not appear to affect the GDT. Conversely, there are studies that show either a larger GDT when a sensorineural hearing loss (SNHL) is present (DeFilippo & Snell, 1986; Florentine & Buus, 1984; Grose, Eddins, & Hall, 1989) or no difference in GDTs between normal and SNHL listeners (Gordon-Salant & Fitzgibbons, 1999; Hall, Grose, & Buss, 1998; Moore, Peters, & Glasberg, 1992).

Strouse, Ashmead, Ohde, and Grantham (1998) found that there are age-related differences in temporal processing. Older listeners, without SNHL, were found to have higher GDTs, which would appear to be an indication of an aging effect in the central auditory system. In a similar investigation, Snell and Frisina (2000) utilized GDTs as one method to measure age-related differences in temporal processing. They found that during adulthood, changes in auditory processing take place, as reflected in larger GDTs for the older group of subjects without SNHL. These results suggest that in the absence of peripheral hearing loss, aging of the auditory system affects temporal processing, which in turn affects speech perceptional abilities.

In summary, it is known that SNHL, aging, and maturation can affect the ability to discriminate fine temporal differences in a signal, which can affect performance on a GDT task. As the ability to detect fine differences in a signal is important when processing a speech signal, poor performance on a GDT task suggests an inability to hear the subtle acoustic changes in a speech signal that may result in speech perception difficulties. This would be especially true in the presence of background noise, where the fluctuations in the noise can obscure the fluctuations in the speech signal. See Chapter 15 for discussion of temporal processing and Chapter 18 for discussion of aging and the central auditory nervous system.

Temporal Modulation Transfer Function

Another way to study temporal resolution is to determine the temporal modulation transfer function (TMTF), which associates the depth of AM to the modulation frequency. Specifically, a broadband noise of a constant spectrum level is sinusoidally amplitude modulated (SAM; Figure 2–13). In a typical, 2AFC paradigm, the listener is presented with a standard (reference) stimulus of a nonmodulated broadband noise in one interval and in the other interval, the SAM broadband noise that is the test stimulus. The listener's task is to identify the modulated signal. The modulation rate of the signal is varied and the listener's threshold for detection of the modulated signal is determined. The faster the SAM rate, the closer in time the modulations occur, thus measuring temporal resolution. The TMTFs are measured according to the modulation depth, which is the depth (height) of the amplitude modulation (see Figure 2–13), with the TMTF being the point at which the listener can detect the smallest amplitude change in the modulated signal as compared with the nonmodulated signal. The TMTF can be depicted as a percent of modulation depth or expressed in decibels.

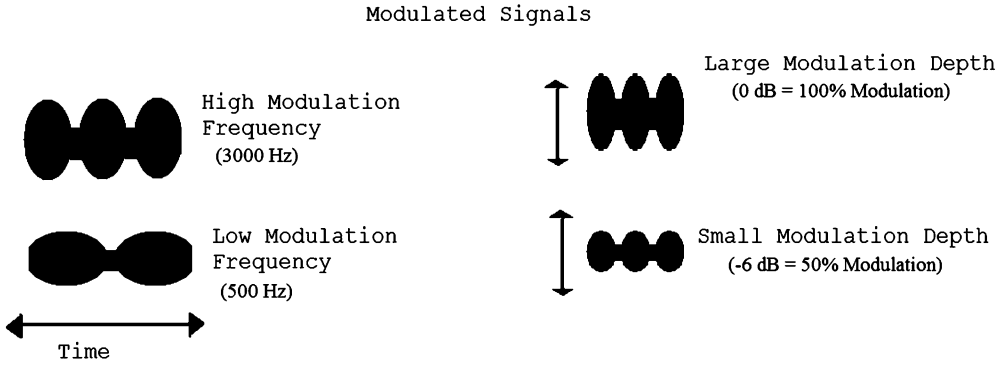

Figure 2–13. Temporal modulated transfer function (TMTF) stimuli. The illustration on the left shows two stimuli of differing modulated frequencies, with the one on the top having a higher modulation rate than the one on the bottom. The illustration on the right demonstrates modulation depth, with the one on the top having a larger modulation depth than the one on the bottom.

Measured in this way, 0 dB would equal 100% modulation. As the modulation percentage decreases (i.e., smaller depth of modulation), the dB level becomes more negative (see Figure 2–13). For example, 50% modulation is equal to −6 dB and 25% modulation is equal to −12 dB (Gelfand, 2009). The sensitivity of the human auditory system is highest for modulation frequencies in the 2 to 50 Hz range and precipitously declines above 100 Hz (Bacon & Viemeister, 1985).

Masking Level Difference

The masking level difference (MLD) is a psychoacoustic phenomenon in which detection or recognition of a monaurally or binaurally presented signal is improved in the presence of a binaurally competing noise. This improvement results from the auditory system's use of subtle binaural phase and amplitude level differences between simultaneously presented signals or masking noises. In two seminal articles, Hirsh (1948) and Licklider (1948) showed that the normal auditory system takes advantage of subtle, interaural time differences in the detection and/or recognition of binaurally presented acoustic stimuli, producing a release from masking. This release from masking phenomenon is referred to as the MLD value.

The MLD represents an advantage in detection or recognition of the binaurally phase-altered condition in reference to the non-phase altered condition (monaural or binaural reference). Specifically, the signal or noise in one ear is adjusted in phase from 0° to 180° relative to the signal or noise in the other ear, whereas the other signal or noise remains in phase interaurally. This results not only in a separation of starting phase between the two ears, but also an increase in perceptual loudness level due to the addition of waveform amplitudes that overlap (coincide) during the binaural correlation process.

Listeners with normal auditory brainstem function demonstrate a release from masking under MLD conditions, while listeners with certain types of auditory pathology do not demonstrate a comparable masking release. The relevant literature indicates that the MLD mechanism is located centrally, but its function can be affected by the status of the peripheral sense organ. For example, investigators have demonstrated that individuals with long-standing unilateral conductive hearing loss have reduced MLD performance, with MLD recovery occurring after the resolution of the conductive hearing loss (Hall & Grose, 1993; Wilmington, Gray, & Jahrsdoerfer, 1994). Cullen and Thompson (1974) reported equivalent MLD values for a group of normal hearing subjects and four clinical subjects who had undergone temporal lobe resections, suggesting that a bilaterally intact auditory reception cortex is not necessary for a release from masking to occur. Cullen and Thompson concluded that the MLD phenomenon is the result of two-ear interaction at a subthalamic level. Subsequent studies (Lynn, Gilroy, Taylor, & Leisea, 1981; Olsen & Noffsinger, 1976; Olsen, Noffsinger, & Carhart, 1976; Quaranta & Cervellera, 1977) have shown that clinical subjects with brainstem lesions do not produce the characteristic release from masking that is reflected in the MLD measure, whereas lesions of the subcortex and cortex do not affect the MLD values. Thus, the expectation is that the results of the auditory brainstem response (ABR)

and the MLD should coincide (Hannley, Jerger, & Rivera, 1983) because the MLD and some waves of the ABR are generated within the same or adjacent anatomical structures. There have been subsequent reports, however, that do not confirm this expectation (Hurley, Hurley, & Berlin, 2002; Levine et al., 1994). A possible reason for this discrepancy is that the MLD may be dependent upon phase-locking by individual neurons to *some portion* of the stimulus, whereas the ABR is dependent not only on phase-locking but having all the neurons phase-locking to the *same phase* of the stimulus (i.e., group synchrony) (Levine et al., 1994). Collectively, these and other results suggest that cross-correlation processes at the mid-brainstem level are responsible for the MLD.

Basic Characteristics of MLD

The MLD is generally computed in two ways: (1) the percentage improvement in the detection or recognition of an auditory signal in reference to the nonphase altered condition, or (2) the decibel improvement in the detection of an auditory signal in reference to a nonphase altered condition. Conventional MLD nomenclature uses S as the signal designation, N as the noise designation, m as the monaural designation, o as the binaural homophasic designation, and π as the binaural antiphasic designation (Figure 2–14). The most common MLD conditions are: (1) SoNo, in which both the signal and the noise are in phase interaurally; (2) SπNo, in which the signal is 180° out of phase interaurally and

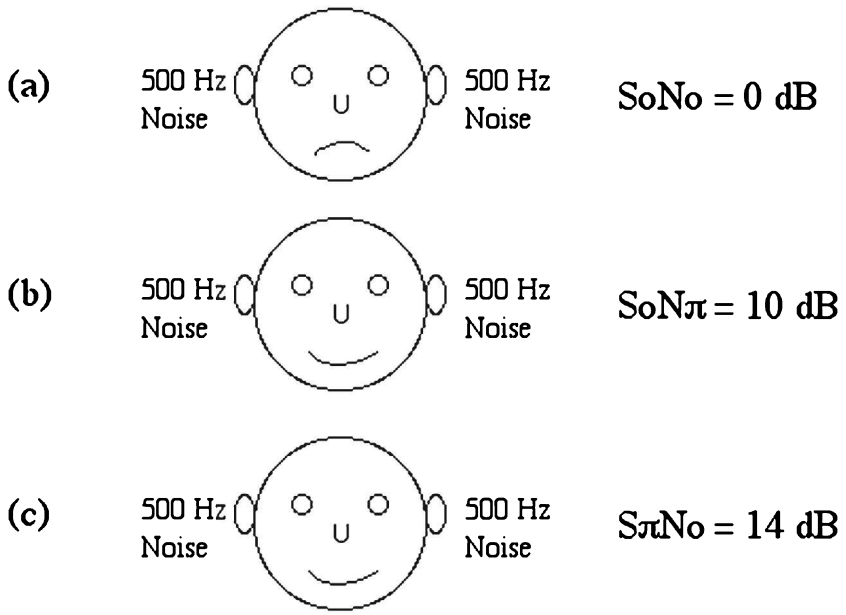

Figure 2–14. Masking level difference (MLD) for three typical conditions with the signal being a 500 Hz pure tone and the masker being a narrow band of noise center at 500 Hz. The values for the SoNπ and SπNo conditions are mean values taken from the data of Hurley et al. (2002).

the noise is in phase interaurally; and (3) SoNπ, in which the noise is 180° out of phase interaurally while the signal is in phase interaurally. In the monaural MLD paradigm, the largest MLD value is obtained for the SmNo (6–9 dB) condition followed by the SmNπ condition (3–6 dB), both referenced to SmNm. For the binaural paradigm, the largest MLD value occurs at SπNo (13–15 dB), followed by SoNπ (10–13 dB) (Gelfant, 2004; Hurley et al., 2002; Jeffress, 1972; Olsen, Noffsinger & Carhart, 1976). One of the major variables that affect the magnitude of the MLD is the stimulus test frequency.

Several studies (Flanagan & Watson, 1966; Hirsh, 1948; Hirsh & Burgeat, 1958) have shown that the largest MLD values occur at 300 Hz, decreasing above and below this frequency. The MLD values are minimal above 1500 Hz because of the auditory system's inability to neurally transmit high frequencies effectively. Release from masking seen for stimuli that contain frequencies above 1500 Hz most likely is accounted for by interaural differences in level. Thus, the MLD is primarily a low-frequency phenomenon.

Another important parameter is the bandwidth of the binaurally competing noise. The magnitude of the MLD value appears to asymptote when the noise reaches a 40 to 50 dB spectrum level (Dolan, 1968; Townsend, 1969). MLDs at low noise intensities, albeit reduced, may be the result of internal noise interacting with low-intensity external noise, producing an effective masker (Soderquist & Lindsey, 1970). This internal noise is the low-frequency jitter produced by the auditory system. A narrowband noise yields much larger MLDs for the SπNo condition than a wideband noise as long as the narrowband is not smaller than the critical band of the test signal it masks. Narrower bandwidths of noise, however, do not change the detection or recognition performance for the SoNo condition (Jeffress, 1972). Variables that do not appear to significantly affect the magnitude of the MLD are age, gender, and central auditory dysfunction above the brainstem.

Theories of MLD Function

There are two theoretical models of MLD function: the lateralization-vector (L-V) theory (Jeffress, 1972) and the equalization and cancellation (EC) theory (Durlach, 1972). Simplistically presented, the L-V theory states that a central process, primarily correlation processes within the central nervous system are responsible for the MLD phenomenon. The L-V theory is based on two mechanisms that need to work sequentially: (1) a peripheral mechanism to preserve and transmit the signal and (2) a central mechanism to compare temporal information from both ears. The central mechanism converts differences in time of arrival to differences in place by neural summation, reads the arriving impulses from the two ears, and then neurally delays the impulses from the ear that leads in time. This model hypothesizes that the MLD advantage in detection is provided by a lateral shift in the auditory image to one ear produced by differences in both time and level. In short, the L-V model is based on the premise that the phase difference between the ears results in time of arrival difference at the central processor, which results in the release from masking effect.

The EC model is a nonphysiological electrical analogue. The EC model postulates that the stimuli to the two ears are normally adjusted to approximate

equality (equalization) and then, when the signal is altered 180° in phase interaurally, the waveform in one ear is subtracted from the waveform in the other ear, resulting in a cancellation effect. The result of this subtraction is the perception of the signal alone. A pivotal component in this model is the EC factor, which is the ratio between the binaural input relative to one of the monaural inputs. Thus, the EC factors describe the change in the signal-to-noise ratios and therefore the change in the masked threshold produced by binaural processing. In the EC model, processing of binaural stimuli takes place in three basic components: (1) two band-pass filters representing the two ears, (2) an EC mechanism that receives the binaurally processed signal, and (3) a decision device that receives either the monaural signals directly from the band-pass filters or the binaural signal from the EC mechanism. Ideally, the EC mechanism improves the signal-to-noise ratio by transforming the total signal received at one input relative to the total signal received at the other input in such a way that the masking components become exactly the same in both channels (the E process), and then the signal is subtracted in one channel from that in the other (the C process). If these operations are perfectly performed, the masking signal is completely eliminated.

Binaural Hearing

The old adage that two ears are better than one is certainly true in that there are more sensitive binaural DLs for both frequency and intensity relative to monaural DLs. Likewise, the binaural absolute threshold for pure tones and spondaic words is approximately 2 to 3 dB better than a monaural threshold due to binaural summation (Gelfand, 2001). In addition, binaural summation results in binaurally presented sound being perceived at twice the loudness level as monaurally presented sound (Marks, 1978; Figure 2–15). Furthermore, binaural speech intelligibility is, on the average, better than monaural speech intelligibility, particularly in the presence of background noise (Moore, 1998). In short, binaural listening provides advantages in hearing sensitivity, loudness perception, general speech perception, and speech perception in adverse listening conditions.

Binaural Fusion and Binaural Beats

Two interesting binaural phenomena observed under headphones and involving the central auditory system are binaural fusion and binaural beats. Binaural fusion is the sensation of hearing a fused auditory signal at midline. For example, if two recordings of word lists are created by using a high-pass filter and a low-pass filter and the modified lists are presented dichotically, the listener's word identification performance for the modified word lists will be similar to identification of the original (i.e., unfiltered) word list (Bornstein, Wilson, & Combron, 1994). The human auditory system combines the high-pass information in one ear and the low-pass information in the other ear to derive a *fused* word. In this scenario, the stimuli are the same words with different filtering characteristics. If completely different stimuli are presented to each ear, there will be no fusion in the midline, as signals must be similar for the fusion effect to occur (Cherry &

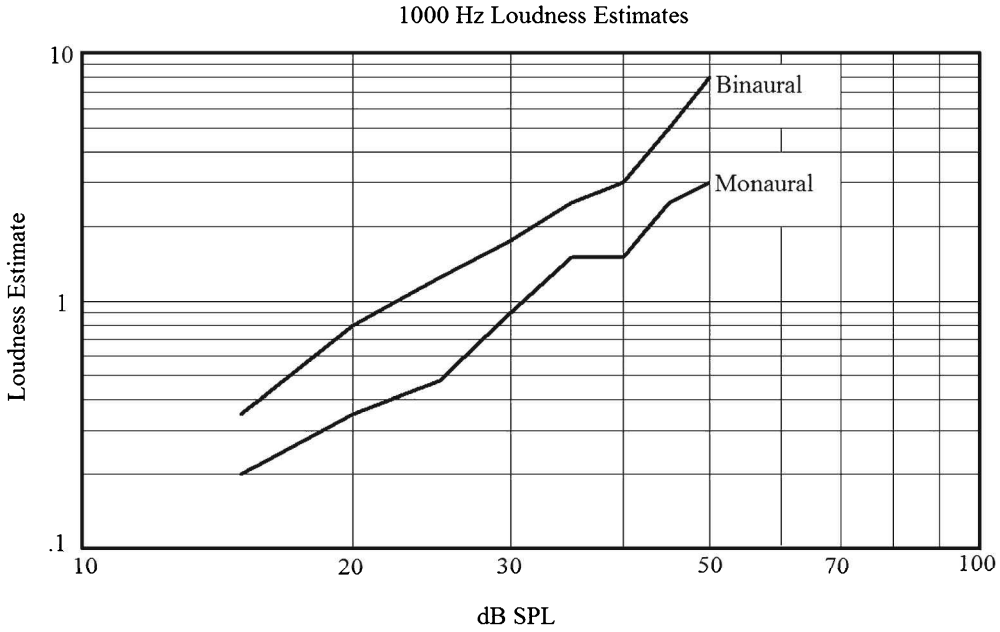

Figure 2–15. Loudness magnitude estimates for 1000 Hz based on data from Marks (1978). The lower line on the graph shows average loudness magnitude estimates for monaural listening, while the upper line on the graph shows loudness magnitude estimates for binaural listening. Redrawn with permission from L. E. Marks, *Journal of the Acoustical Society of America, 64,* 107–113. Copyright 1978, Acoustical Society of America.

Sayers, 1956). The fusion phenomenon is frequency sensitive in that if different high-frequency tones are presented to the ears of a listener, the listener will report hearing separate tones; however, if a low-frequency tone is superimposed over both high-frequency tones, the listener will report hearing a fused auditory signal (Leaky, Sayers, & Cherry, 1958). The probable site of a "fusion mechanism" (Cherry & Sayers, 1956) is within the central auditory system, where a cross-correlation analysis takes place. In fact, one of the first tests of central auditory function utilized a binaural fusion paradigm (Matzker, 1957).

Another interesting phenomenon, which occurs with binaural fusion, is binaural beats. Beats occur when two stimuli, close in frequency, are presented simultaneously to one ear, resulting in a waxing and waning perception (Gelfand, 2009). Beats will also occur when tones close in frequency are presented dichotically to each ear. For example, if a 600-Hz tone is presented to the right ear and a 603 Hz tone is presented to the left ear, the listener will perceive a 600-Hz tone at midline that waxes and wanes in loudness at a rate of three per second. The binaural beat phenomenon will occur for two frequencies that are 2 to 10 Hz apart and may be perceived until the frequency separation is approximately 20 Hz apart. After this point, the stimuli are perceived as two separate tones later-

alized to each individual ear. The binaural beat phenomenon is optimal for frequencies between 300 and 600 Hz (Licklider, Webster, & Hedlun, 1950). Like binaural fusion, the binaural beat phenomenon involves processing within the central auditory system, with the superior olive complex being implicated as the control center (Wernick & Starr, 1966)

Directional Hearing

One of the advantages of being a two-eared listener is the ability to locate a sound source in the everyday listening environment. Localization ability is based on two parameters of a signal that become modified through travel from a sound source to each ear. The two parameters are time of arrival differences and intensity differences between the ears. If the signal is presented to the right side of the head, it has to travel farther to reach the left ear, compared with the right ear, resulting in an interaural time difference (ITD) and an interaural intensity difference (IID). Neither of these interaural differences is consciously perceived by the listener, who hears a fused single sound.

ITD varies as a function of the azimuth (angle) relative to the head position (Figure 2–16). ITDs increase from zero when the sound source is either directly in front of the listener or directly behind the listener (0° or 180° azimuth), to a maximum when the sound source is to either side (90° azimuth) of the

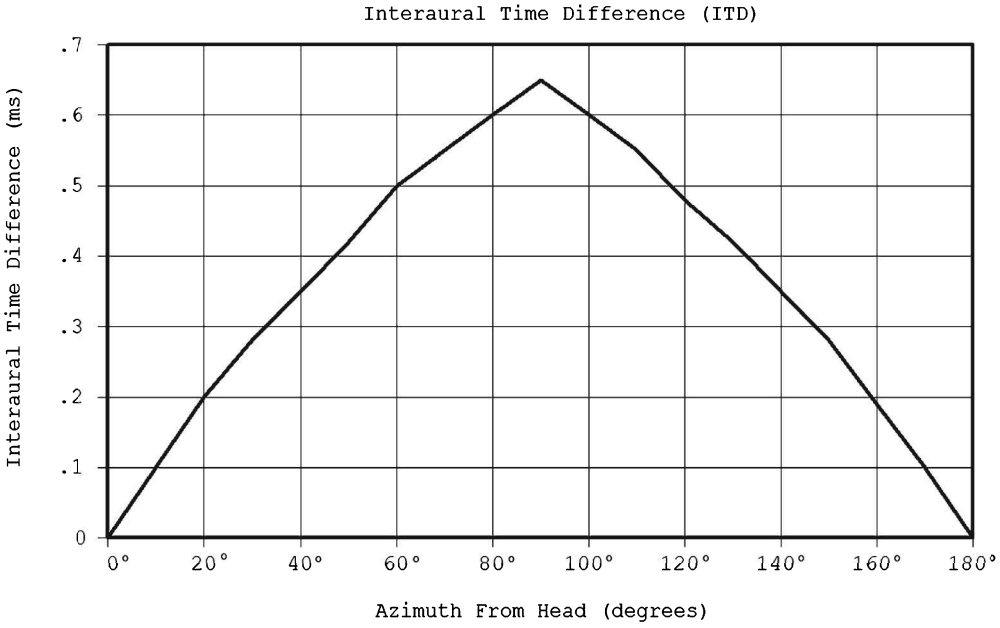

Figure 2–16. Interaural time differences (ITDs) as a function of azimuth from the head (Fedderson et al., 1957). Note that there is no ITD between ears when the signal is directly in front (0° azimuth) or behind (180° azimuth) the listener. The largest ITD occurs when the signal is presented directly to the opposite ear (90° azimuth). Redrawn with permission from W. E. Feddersen, *Journal of the Acoustical Society of America, 29*, 988. Copyright 1957, Acoustical Society of America.

listener (Feddersen, Sandel, Teas, & Jeffress, 1957). The ITD and IID effects are frequency specific. The wavelengths of low-frequency tones are long enough to bend around the head, creating an ITD between the ears that provides a localization clue. For high-frequency tones, the IID serves as the localization cue because the wavelength of high-frequency tones is too short to bend around the head; therefore, a "head shadow" effect occurs, resulting in an IID between ears (Figure 2–17). The IID is negligible at low frequencies but can be as large as 20 dB at high frequencies (Fedderson et al., 1957). In addition to ITD and IID, spectral changes in the stimulus brought about by the folds of the pinna coupled with reflections from the head and torso provide localization cues. These pinna-induced spectral changes assist the listener in locating an elevated sound source, in front/back discrimination, and in monaural localization (Middlebrooks, 1992). Listeners' localization abilities are most accurate below 1000 Hz and above 4000 Hz, whereas most listener localization errors occur between 2000 and 4000 Hz. Lastly, localization is better for complex stimuli than for pure tones (Stevens & Newman, 1936).

Directional hearing also involves the ability to identify small changes in position of the sound source (i.e., the minimum audible angle). Listeners are able to identify the smallest change in location of a sound source when the frequency is below 1500 Hz and above 2000 Hz

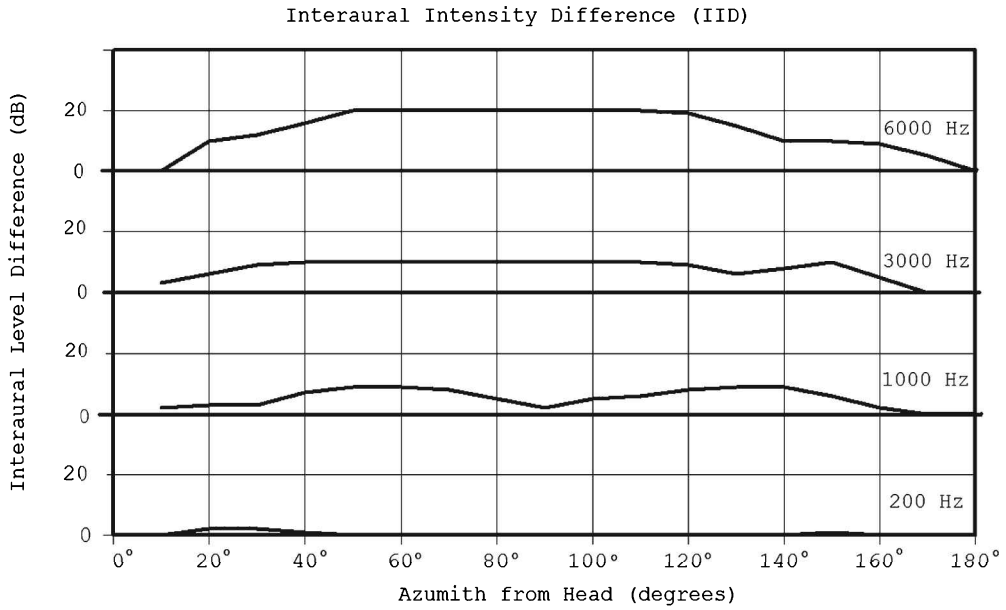

Figure 2–17. Interaural intensity differences (IIDs) as measured by frequency (Fedderson et al., 1957). IID is negligible between ears at 200 Hz. As the frequency becomes higher, the amount of IID between ears becomes larger, increasing to approximately 20 dB at 6000 Hz. Redrawn with permission from W. E. Feddersen, *Journal of the Acoustical Society of America, 29*, 988. Copyright 1957, Acoustical Society of America.

(Mills, 1958, 1972). Between 1500 Hz and 2000 Hz, listeners demonstrate the poorest ability to discriminate a change in the sound source location. Listeners are able to discriminate 1° to 2° changes in azimuth when a sound source is located at a 0° azimuth or directly in front of them because small changes in location result in large ITDs (Mills, 1958). The poorest discrimination of azimuth degree change in the minimal audible angle occurs when the sound source is located to either side (90° and 270° azimuth) of the listener because the IID remains the same despite large changes in sound source location (Gelfand, 2009). This lateral head area is designated the "cone of confusion," reflecting the poor discrimination of the minimal audible angle changes (Gelfand, 2009). Despite the fact that there is a large area in which listeners have difficulty discriminating fine changes in the direction of a sound source, listeners are able to compensate with head movement that constantly changes the direction of the cone of confusion, thus minimizing its effect (Moore, 1998).

Lateralization

A psychoacoustic phenomenon related to localization is lateralization. The major difference between localization and lateralization paradigms is that the former uses a sound field to deliver test stimuli, while the latter utilizes earphones to deliver the test stimuli. Binaural presentations of stimuli that are equal in frequency and intensity and have no significant interaural temporal differences will result in a fused perception, with the listener hearing only one sound in the midline. A change in interaural intensity will result in the well-known Stenger effect, with the listener perceiving one sound that is lateralized to the ear receiving the greater intensity. Binaural presentation of two stimuli equal in intensity but differing in frequency will result in the listener perceiving two stimuli. In the lateralization paradigm, the use of earphones allows one to vary independently the IID and ITD; however, in a localization paradigm, IID and ITD cannot be separated due to sound field presentation.

A common paradigm to study IID and ITD utilizes a 2AFC design with successive presentations of binaural stimuli that have an interaural difference (i.e., IID or ITD). One stimulus will be the same at each ear, while the other stimulus will have an IID or ITD. The listener indicates if the second stimulus was different than the first stimulus or if the second stimulus changed location relative to the first stimulus. Using the first paradigm, Yost (1974) studied phase discrimination (ITD) by presenting a reference stimulus that had an interaural phase difference (θ), creating lateralization to one side of the head followed by presentation of a test stimulus that had varying amounts of phase differences ($\Delta\theta$) larger than the reference stimulus ($\theta + \Delta\theta$). Yost's findings are illustrated in Figure 2–18a. Note that interaural phase discrimination was the best at 0° and 360°, which corresponds to zero phase differences between the ears. The poorest interaural phase discriminations were seen at 180°, where the test signals lateralized to one side. Phase difference ($\Delta\theta$) discrimination was essentially the same for frequencies up to 900 Hz, but much poorer at 2000 Hz. These findings suggest that interaural phase appears to be an important cue for low-frequency lateralization, but not for higher frequency lateralization, a finding consistent with the role of timing cues for localization.

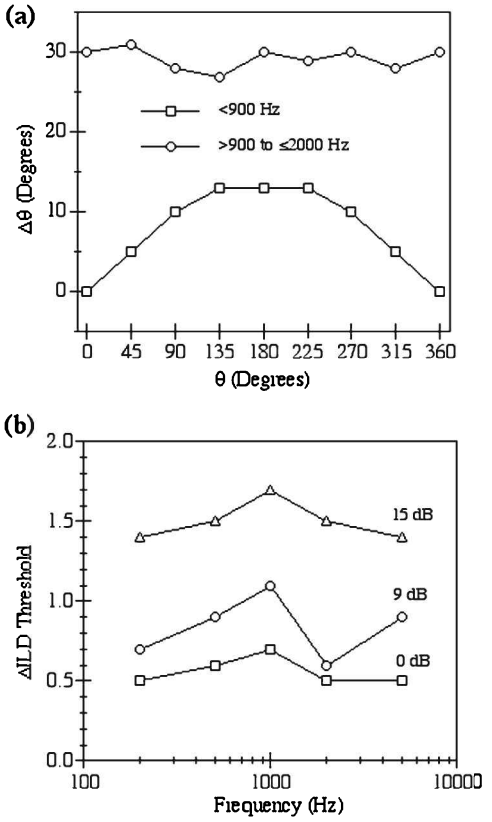

Figure 2–18. (a) The amount of interaural phase change ($\Delta\theta$) needed to discriminate a lateralization change from a reference (θ). Adapted and redrawn with permission from W. A. Yost, *Journal of the Acoustical Society of America, 83*, 1846. Copyright 1988, Acoustical Society of America. (b) Interaural intensity difference (IID) thresholds as a function of frequency from 200 to 5000 Hz. Redrawn with permission from W. A. Yost, *Journal of the Acoustical Society of America, 55*, 1299. Copyright 1974, Acoustical Society of America.

Figure 2–18b depicts IID discrimination as a function of frequency from 200 to 5000 Hz. IID is rather constant across frequency for a particular lateralization site, with a slight increase at 1000 Hz. The 0, 9, and 15 dB designations in Figure 2–18b are the IID values used to produce lateralization sites at the midline, halfway between the midline and the left ear, and the left ear, respectively. Clearly, the midline lateralization site produced the best IID discrimination, with the poorest IID discrimination occurring at the left ear lateralization site. Although lateralization research suggests that ITD is the main clue for low frequencies, interaural time also seems to provide the binaural system cues for complex high-frequency sounds that may be modulated by a low-frequency component (Yost, 2000). For example, consider three stimuli, a 250-Hz tone, a 3000-Hz tone, and 3500-Hz tone that are AM by a 250-Hz tone. Listeners will be able to detect an ITD for the 250-Hz tone, but not for the 3000-Hz tone. On the other hand, listeners will detect an ITD for the 3500-Hz tone that is AM by a 250-Hz tone (Gelfand, 2009). In summary, lateralization research indicates that IID is the cue for high frequencies, and ITD is the cue for low frequencies and for complex stimuli that consist of a high-frequency carrier that undergoes low-frequency repetition.

With the advent of virtual reality technology, a sound field environment can be simulated accurately by using each individual listener's head-related transfer function (HRTF) for each ear to create stimuli containing the necessary perceptual cues that can be well controlled through earphone presentation. Due to individual differences in skull, torso, and pinna, the HRTF must be determined for each listener so that the amplitude and phase spectra contain the cues unique to the listener in order for an earphone virtual reality test paradigm to be accurate. A number of investigations comparing individualized versus nonindividualized HRTFs to assess simulated

localization performance demonstrated poorer performance when listeners used other than their own HRTF in the virtual reality experiment (Middlebrooks, 1999; Møller, Sorensen, Jensen & Hammershoi, 1996; Wenzel, Arruda, Kistler, & Wightman, 1993). At least one study comparing sound field localization and earphone simulated localization reported correlation coefficients of 0.97 to 0.98 between the actual sound location and the subject's sound field judgments, and correlations of 0.83 to 0.96 between the actual sound location and the subject's simulated virtual reality earphone judgments (Wightman, Kistler, & Perkins, 1987). See Chapter 16 for additional discussion of binaural listening and clinical measurement of binaural fusion, localization, lateralization, MLD, and other measures of binaural interaction.

Proposed Psychoacoustic Tests for CAPD

Our purpose in this section is to propose extending the work of Zeng et al. (2001) on the psychoacoustic study of auditory neuropathy to the psychoacoustic study of CAPD. In this section, we propose test paradigms that are based on some of the psychoacoustic procedures previously reviewed. In our deliberations, we considered only procedures that could be easily adapted to compact disc delivery and are believed to have a central auditory aspect to a subject's test performance (Pickles, 2012). Our construction of the proposed test procedures takes into account paradigms that could be delivered in a clinic test environment and time limitations that may operate in a clinical setting. Furthermore, we have reviewed some of the cochlear implant literature to get a better grasp of the clinical practicality of the proposed psychoacoustic tests. Although we provide test protocols in the test descriptions, these protocols are more for illustrative purposes to aid the reader in understanding the possible clinical application of each psychoacoustic test. Although other specific psychophysical test procedures could be used for each test, we have chosen to use a three-alternative, forced-choice procedure that would result in a 70.7% correct response (Levitt, 1971, 1978; Zeng et al., 2001; Zeng, Kong, Michalewski, & Starr, 2005). Clearly, the clinical application of each of the proposed psychoacoustic test would need considerable development, standardization, and normalization. Last, in proposing the following psychoacoustic tests for central auditory assessment, we gave no consideration to how each test would fit into the construction of a "test battery" that would attempt to assess various aspects of auditory behavior.

Temporal Modulation Transfer Function

The first proposed psychoacoustic test is based on TMTF. Our paradigm follows the work of Bacon and Viemeister (1985) and Won, Drennan, Nie, Jameyson, and Rubinstein (2011). The test paradigm could utilize a two-interval, two-adaptive forced-choice procedure or the method of constant stimuli (Fraser & McKay, 2012) to measure modulation detection thresholds (MDTs). In brief, the stimuli would be 2 sec in duration. One of the two 1 sec observation intervals would consist of AM wideband noise, and the other 1 sec observation interval would

consist of continuous wideband noise. For the modulated stimuli, amplitude modulation would be applied to the wideband noise carrier. Both the modulated and unmodulated signals would be gated on and off with 10 msec linear ramps, then concatenated with no gap between the two signals. Five to seven different modulation frequencies would be tested (10, 50, 75, 100, 150, 200, and 300 Hz) depending upon further test development, which could suggest that fewer than five modulation frequencies are needed. Stimuli would be presented at a conversational level of 65 dB HL. During one of the two 1-sec observation intervals, the carrier would be sinusoidally amplitude modulated. The subjects would be instructed to choose the interval that contains the modulated noise. Visual feedback of the correct answer would be given after each presentation. A two-down, one-up adaptive procedure could be used to measure the modulation depth threshold, converging on 70.7% (Levitt, 1971), starting with a modulation depth of 100% and decreasing in steps of 4 dB from the first to the fourth reversal, and 2 dB for the next 10 reversals. For each tracking run, the final 10 reversals would be averaged to obtain the MDT for that tracking history. MDTs in dB relative to 100% modulation would be obtained. First, subjects would complete all modulation frequencies in random order, and then the subjects would repeat a new set of modulation frequencies with a newly created random order. Generally, six tracking runs, which generated a total of 60 reversals, would be conducted to determine the average thresholds for each modulation frequency. If subjects could not complete the six tracking runs for some modulation frequencies due to time and scheduling constraints, the mean across the multiple tracking runs (generally three, four, or five) would be used to compute the average thresholds when six tracking runs are done.

Monaural Intensity Discrimination and Monaural Frequency Discrimination

Differential sensitivity is an extremely valuable measure of the auditory system's ability to process spectrally complex signals. These auditory measures may be important in accessing many clinical populations such as those with hearing impairment, CAPD, auditory neuropathy spectrum disorder, and so on. Thus, our second proposed psychoacoustic test is a version of a monaural intensity discrimination task (IDT) and frequency discrimination task (FDT). Thresholds are obtained using a three-interval, two-alternative forced-choice protocol. In this procedure, the listener is presented with three signals in series. Two identical signals, in either frequency or intensity, serve as the standards, and a third signal that varies in either frequency or intensity serves as the target tone. The target tone is presented with equal *a priori* probability for interval placement within the three intervals. The interstimulus interval is 400 msec, with 1000 Hz being the standard test frequency, as the ear is very sensitive to 1000-Hz signals and has been shown to produce some of the lowest discrimination thresholds in comparison with higher frequency test signals (Jesteadt & Sims, 1975; Moore, Ferguson, Halliday, & Riley, 2008). The standard 1000-Hz tone will have a linear rise/fall time of 5 msec and total duration of 300 msec and will be presented at 75 dB SPL. A two-down, one-up adaptive

procedure as described by Levitt (1971), tracking the 70.7% correct response level, will be employed. With this procedure, intensity is reduced after two consecutive correct responses or increased following one incorrect response. A reversal is the trial response that meets the criteria for a change in the direction of the adaptive procedure that increases or decreases the distance of the variable and target signal. The test will be terminated after 20 reversals, and the threshold is taken as the average of the last 10 reversals.

For the IDT, both the standard and the variable tone will be 1000 Hz, with the variable 1000 Hz tone being changed in intensity. The initial intensity difference of the variable and standard tones will initially be 5 dB. Subsequently, the variable tone will be adaptively changed in steps of 2 or 1 dB. For the FDT, a 1000 Hz tone will again be the standard, with the variable tone being adjusted in frequency. The initial frequency difference between the standard and variable tone will be 100 Hz (i.e., 1000 Hz as the standard tone and 900 or 1100 Hz as the variable tone). The frequency steps for the variable tone will adaptively change in steps of 10, 5, or 1 Hz. Visual feedback will be given after each presentation.

Simultaneous, Forward, and Backward Masking

The third proposed psychoacoustic test is simultaneous, forward, and backward masking utilizing a brief stimulus tone measured in the presence of masking temporally presented simultaneously, or in a forward or backward masking task. Recall that in BM the test signal (probe) precedes the masker and is terminated before the onset of the masker, whereas in FM the masker precedes the test signal and is terminated before the onset of the probe. In short, for both backward and forward masking paradigms, there is a time separation between the probe and masker. This forward and backward masking procedure is based on the works of Wright et al. (1977) and Moore, Cowan, Riley, Edmondson-Jones, and Ferguson (2011).

The target tone is 1000 Hz, 20 msec in duration, with a 10 msec rise and fall time. The level of the tone is adapted to obtain threshold and will range from 20 to 100 dB SPL. The band-pass masking noise will be 600 to1400 Hz, with 300 msec duration and 10 msec rise and fall time and presented at 40 dB SPL spectrum level. Again, the test tone will be presented immediately before (forward masking), immediately after (backward masking), or at approximately 150 msec after the 300 msec band-pass noise has begun (simultaneous masking). Each interval will be separated by an interstimulus interval of 400 msec.

Participants will be tested using a three-interval, two-alternative forced-choice procedure in which they must identify which interval contains the target or tone. The interval containing the target tone will be chosen randomly with *a priori* probability. A two-down, one-up adaptive procedure could be used to measure the threshold, converging on 70.7% (Levitt, 1971). With this procedure, intensity is reduced after two consecutive correct responses or increased following one incorrect response. The signal level will initially decrease by 10 dB, with subsequent step-size adaptively decreased in steps of 5, 2, and 1 dB. Thresholds will be obtained for each masking condition separately, with the test terminated after 20 reversals, and the threshold taken as

the average of the last 10 reversals. Visual feedback of the correct answer would be given after each presentation.

jnd's in Binaural Interaural Time Difference and Binaural Interaural Intensity Differences

Ear asymmetries or ear weaknesses are often seen in behavioral tests of central auditory processing, even though there is symmetrical normal peripheral hearing. Interaural asymmetries or imbalances may not be evident in monaural tests but may be evident as a degradation of binaural performance using appropriate test protocols. Thus, our fourth proposed set of tests are jnd's for binaural interaural time differences (BITDs) and binaural interaural intensity differences (BIIDs), which would investigate binaural hearing abilities. These procedures are based on works by Hawkins and Wightman (1980) and Koehnke, Culotta, Hawley, and Colburn (1995), who investigated binaural performance in normal and hearing-impaired listeners.

Again a three-interval, two-alternative forced-choice procedure will be employed. Two binaural signals that are identical signals in terms of intensity and time are the standard tones. The target signal is a binaural stimulus that consists of one monaural standard tone and one that differs in either intensity or phase. The interval containing the target stimuli will be chosen randomly with *a priori* probability, and the target will be imposed on the right and left ears with *a priori* probability. Listeners will indicate which interval contains the target, or dichotic presentation.

The standard stimuli for the BITD and BIID tests will be one-third-octave band noise centered at 1000 Hz, presented at 75 dB SPL for a duration of 300 msec, with a rise/fall time of 15 msec. For the BIID dichotic intensity experiment, the intensity range will be 10 dB. The step-size will initially change in a 5 dB step and then adaptively change from 1 to .5 dB attenuation steps. For the BITD dichotic procedure, the initial offset difference will begin at 1000-μsec increments and then adaptively change to a step-size increment of 100 μsec. Again, the target will be imposed randomly on right and left ears. The test will be terminated after 20 reversals and the threshold taken as the average of the last 10 reversals.

Summary

The goals of this chapter were to provide a brief review of classic and contemporary psychoacoustic methods and phenomena, indirectly highlight aspects of psychoacoustics that might apply to CAPD diagnosis, and leave the reader wondering why basic psychoacoustic profiles of listeners with CAPD have yet to be created, as psychoacoustic tests are not contaminated and or limited by specific language impairment (Nickisch & Massinger, 2009). High precision in measuring human test performance is one of the basic foundations of "evidence-based audiology" (Bess, 1995; Cox, 2005). In order to exact this precision, however, each examiner must understand that any given behavioral response to an auditory test stimulus could be a true indication of the listener's perception, a guess on the part of the listener, or a change in the listener's response criterion (bias) (see Silman, Silverman, & Emmer, 2000). Cou-

pling sound measurement methods with psychoacoustic profiling should lead to a better understanding of the perceptual deficit(s) that listeners with CAPD experience and better define the heterogeneous profiles seen in CAPD. Technological advances such as virtual source auditory testing (Besing & Koehnke, 1995; Koehnke & Besing, 1996) need to be paired with psychoacoustics to study CAPD. Lastly, before psychoacoustic *profiling* can be used to guide customized treatment for CAPD, additional psychoacoustic data from listeners with confirmed CAPD are needed.

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CHAPTER 3

DEVELOPMENT OF THE CENTRAL AUDITORY NERVOUS SYSTEM

JOS J. EGGERMONT

Introduction

Human auditory maturation can be studied at three levels, that is, the structural, the functional, and the behavioral. Structural aspects are currently more and more studied by neuroimaging methods that visualize the density of gray and white matter (magnetic resonance imaging [MRI]) and can trace fiber tracts through the diffusion of water along or perpendicular to them (diffusion tensor imaging [DTI]). These methods provide similar but much less detailed information compared with histology performed on the brains of deceased infants and children; however, they provide access to the living brain and allow longitudinal studies. Behavioral studies may quantify auditory discrimination and perception as a function of age. Ideally, structural, functional, and behavioral methods of assessment should give similar time lines of auditory maturation, but in practice there is not a perfect correspondence between the results of the different approaches.

Functional imaging methods quantify the brain's use of either oxygen or metabolites such as glucose (e.g., positron emission tomography [PET]) or changes in the amount of oxygenated blood (i.e., functional MRI [fMRI], blood oxygen level dependent [BOLD] response). These imaging methods provide accurate localization but lack temporal resolution. The

temporal aspects of neural function can be assessed by auditory evoked potentials (AEPs) or auditory magnetic fields (AMFs) that quantify the degree of neural synchrony underlying these signals with great temporal precision. For low-noise recordings, AEPs and AMFs also allow the localization of the distributed dipole density in the brain and of their equivalent current source dipoles. The latter represent the location and strength of the center of gravity of all the synchronized activity within a prespecified brain region and latency range. These centers of gravity compare favorably with those obtained by fMRI, as we have shown with simultaneous recordings (Scarff et al., 2004).

The auditory system matures rostral from the periphery and is characterized by early maturation of the auditory brainstem and the brainstem reticular activating system (RAS) pathways, followed by a later-onset and very extended maturation of thalamocortical and intracortical connections (Moore & Linthicum, 2007). The specific lemniscal (tonotopic) and extralemniscal (nontonotopic) auditory pathways mature at different rates than the nonspecific RAS, which activates the synapses on the pyramidal cell dendrites in cortical layer I in the first half-year after birth. This provides a bit of a paradox, since it allows activation of auditory cortex well before maturation of its main thalamocortical input in layer IV. Maturation of cortical cells and axons occurs initially in layer I, then is followed by layers IV to VI, and then again upward to the superficial layers II to III (Moore & Guan, 2001). The parallel processing in the RAS, lemniscal, and extralemniscal pathways may offer ultimately a top-down influence upon processing of auditory information (Kral & Eggermont, 2007).

Neural Correlates of the Discrimination System Maturation

Within 30 days of term birth, the peripheral human auditory system is fully developed (Eggermont, Ponton, Coupland, & Winkelaar, 1991). In the first 1.5 years of life, the brainstem and midbrain become fully mature. Thalamocortical maturation, however, may take up to two decades, as does the corpus callosum (CC) (Yakovlev & Lecours, 1967). These estimates are based on findings from histology, functional imaging, and auditory evoked responses. Yet, behaviorally, infants appear to be capable of amazing auditory discriminatory and memory feats. This puzzle has been addressed in a recent review paper by Eggermont and Moore (2012), of which I include only the main aspects in this chapter.

In particular, infants below the age of about six months can discriminate individual speech phonemes in both their native language and in languages to which they have not been exposed (Trehub, 1976). Electrophysiological testing reveals that infants exhibit AEPs arising both in the brainstem and in the forebrain. These behavioral and physiological findings are in basic agreement with histological and imaging studies showing early and rapid maturation of auditory structures in the brainstem and in some elements of cortex. In this section, we will review the auditory brainstem response (ABR), the middle latency response (MLR), the early-maturing AEP components, and the mismatch response (MMR), as well as the structures that are their presumed generators (Table 3–1).

Table 3–1. Structural, Electrophysiological, and Behavioral Correlates in Maturation

Age	Structural	Electrophysiological	Behavioral
<6 mos	Layer I cortical axons are mature Acoustic radiation myelination starts	ABR wave I latency mature ABR waves III-V, MLR, P2 and MMR present but latencies are immature	Discrimination of (speech) sounds, detection of change
6 mos–5 yrs	Brainstem axons are mature Acoustic radiation myelination mature Axonal neurofilaments in cortical layers IV, V and VI mature Cortical synaptic density peaks	ABR, MLR, P2, N2, MMN, and T-complex are mature P1 present but latency immature	Onset and development of perceptual language and general auditory perception
5–12 yrs	Axonal neurofilaments in cortical layers II and III are mature Decrease in cortical synaptic density	P1 matures, N1 emerges but amplitude remains immature	Processing of masked and degraded speech improves
>12 yrs	Cortical axons are all mature Temporofrontal language-related nerve tracts mature	Asymptotic maturation of N1	Speech understanding in reverberation and background noise matures

Brainstem Maturation

The auditory brainstem forms a pathway consisting of axonal tracts interrupted by synapses in nuclei, as shown in Figure 3–1. The brainstem auditory nuclei and axonal pathways are formed very early in development and are identifiable by the 8th week of embryonic life. The first step in axonal maturation in the brainstem pathway is the formation of the neurofilaments that are part of the inner framework of the axon. Immunostained neurofilaments are first observed in brainstem auditory axons at the 16th fetal week and become more numerous by the 22nd fetal week (Moore, Guan, & Shi, 1997). By the 27th to 29th weeks, the beginning of the third trimester, the stained axonal pathways have an adultlike configuration. The next stage of axonal development is the formation of myelin sheaths in the cochlear nerve, acoustic stria, lateral lemniscus,

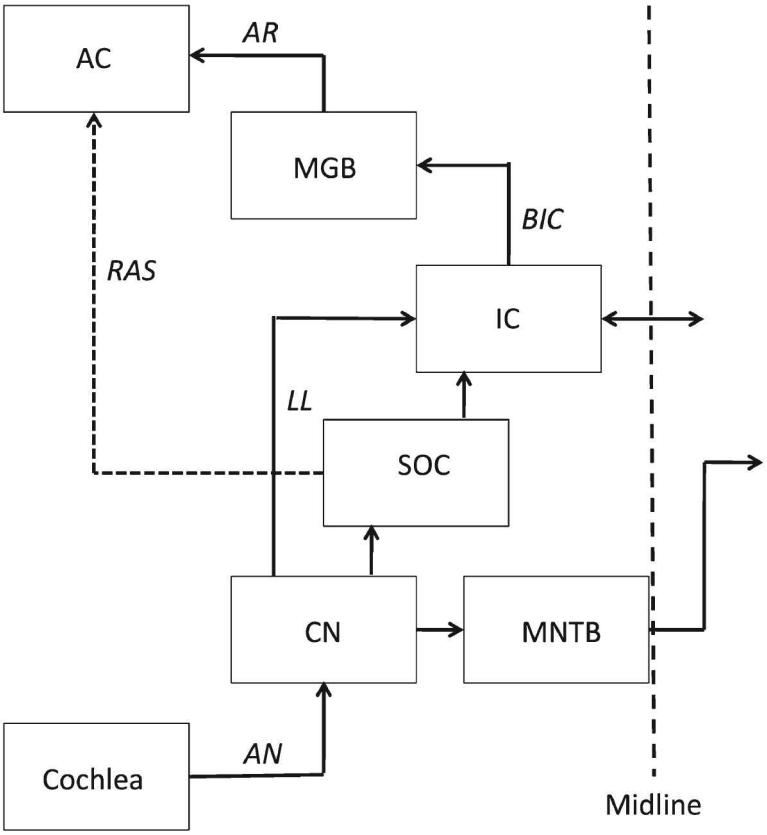

Figure 3–1. Schematic representation of the left hemisphere of the human auditory system, with cellular structures labeled inside the boxes and axonal pathways in italics. Auditory nerve (*AN*) fibers carrying activity from the cochlea enter the cochlear nuclei (CN) and terminate on its various cell groups. Projections from the bushy cells in the CN pass through the acoustic stria to innervate the nuclei of the superior olivary complex (SOC). Other projections from these cells interact with the contralateral SOC via the medial nucleus of the trapezoid body (MNTB) for sound localization. Axons from the stellate cells, octopus cells, and fusiform cells in the CN continue through the lateral lemniscus (*LL*) to end in the inferior colliculus (IC) on both sides (only one side drawn). Collicular projections pass through the brachium of the inferior colliculus (*BIC*) to reach the auditory part of the thalamus, the medial geniculate (MG). From the thalamus, projections pass through the auditory radiation (*AR*) to the auditory cortex (AC) of the temporal lobe. In an alternative pathway, collaterals from lemniscal axons enter the reticular core of the brainstem, from which arises the reticular activating system (*RAS*) pathway to layer I of the cortex.

and brachium of the inferior colliculus by the 27th to 29th week. Myelin density then steadily increases to at least 1 year postnatal in histological investigations (Moore, Perazzo, & Braun, 1995). MRI studies (Sano, Kaga, Kuan, Ino, & Mima, 2007) found that axons in the cochlear nucleus and superior olivary complex showed changes in myelin intensity from 3 to 13 postnatal weeks; the lateral lemniscus showed this from 3 to 8 corrected postnatal weeks. The inferior colliculus showed a more extended period of intensity change from 2 to 39 corrected postnatal weeks. The onset of these changes lagged considerably behind the observed histological changes, indicating less sensitivity of MRI to early stages of myelin formation. However, MRI detected continued maturation in the inferior colliculus after the lower brainstem appeared mature. Thus, histological techniques allow visualization of early myelin in small numbers of neurons, but imaging can track the rate and continued course of maturational changes in myelin density.

In preterm babies, auditory evoked responses can be obtained from about 25 weeks conceptional age (CA) on: Specifically long-latency evoked responses are very clear at that time (Weitzman & Graziani, 1968). At 27 weeks CA, the first ABRs have been obtained (Starr, Amlie, Martin, & Sanders, 1977). This presents another little puzzle: Why are the long latency responses earlier detectable? Does this bear any relation to the very early discrimination prowess of infants? The solution may reside with the RAS that can forward brainstem activity via slowly conducting fibers to layer I of the auditory cortex. This would result in very long latencies for the AEPs, as is observed. Since the late cortical responses are based on synchronous long-duration postsynaptic potentials, they sum much better to make up a compound response than do the short-duration biphasic action potentials in the auditory nerve and brainstem tracts that make up the ABR. So the absence of an ABR does not mean absence of neural brainstem activity.

A typical adult ABR consists of a sequence of up to seven vertex-positive waves, separated by negative valleys. The ABR can be reliably recorded in premature infants from the 28th to 29th weeks CA. Pasman, Rotteveel, de Graaf, Maassen, and Notermans (1991) found that at 30 to 35 weeks CA, the ABR vertex-ipsilateral mastoid wave I, the negative wave following wave II, wave V, and the vertex contralateral mastoid recorded waves II and V were the most consistently present, with detection rates of 87 to 100%. At ages 35 weeks CA and older, waves I, III, and V at both sides were clearly present (Ponton, Eggermont, Coupland, & Winkelaar, 1992).

Clearly, ABR maturation will be affected by cochlear maturation. Although the cochlea has a generally adult appearance by the end of the second trimester, investigations utilizing distortion product otoacoustic emissions and cochlear traveling wave delay measurements indicate that full cochlear maturity is not achieved until a few weeks before term birth, with an additional 3 to 6 months required for maturation of the inner hair cell ribbon synapses mediating frequencies above 6000 Hz (Eggermont, Brown, Ponton, & Kimberley, 1996; Eggermont, Ponton, Coupland, & Winkelaar, 1991). Thus, just as in the output of the cochlea, represented by wave I, all subsequent ABR generators show a cochlear place-dependent maturation, characterized by its most sensitive frequency (Ponton,

Eggermont, Coupland, & Winkelaar, 1992). For all practical purposes, the maturational process underlying the I to V interval (i.e., the central conduction time) reaches adult values 58 to 87 weeks after the first recordings at 35 weeks CA, which is about 1 to 1.5 years postterm.

The maturational time period of the I to V interval, reflecting activity in brainstem structures up to the level of the lateral lemniscus, is only slightly faster than that of the MLR. The MLR components that are most clearly detectable in infants are the P0–Na waves. Because intracranial recordings from the surface of the human brainstem matched the P0 peak to a postsynaptic potential at the inferior colliculus (Hashimoto, Ishiyama, Yoshimoto, & Nemoto, 1981), it seems likely that the P0–Na waves reflect transmission in the brachial pathway from the inferior colliculus to the thalamus (see Figure 3–1). The P0–Na waves are barely detectable at the 25th to 27th fetal weeks but are fairly well defined by the 33rd fetal week and are more pronounced by the time of term birth. The Na peak latency decreases steadily from about 28 ms at the 30th fetal week to around 20 ms at term (Pasman et al., 1991; Rotteveel, Colon, Notermans, Stoelinga, & Visco, 1985; Rotteveel, Stegeman, de Graaf, Colon, & Visco, 1987). By the third postnatal month, the Na peak achieves a latency of about 18 ms, a value that remains unchanged throughout childhood, teen years, and adulthood (Kraus, Smith, Reed, Stein, & Cartee, 1985). This latency suggests a cortical origin of the Na-Pa complex, albeit that a gerbil animal model allowed recording of responses analogous to Na and Pa putatively generated in the thalamus (Kraus, Smith, McGee, Stein, & Cartee, 1987). The much larger distance from the scalp to the thalamus in humans, and the fact that the dendrite orientation in the medial geniculate body (MGB) is random, makes scalp recording of evoked potentials generated by this structure largely impossible (Eggermont, 2007). The Pa with a latency of about 30 ms is clearly of primary auditory cortical origin and generated in layers III/IV (Ponton & Eggermont, 2007).

Neurofilament formation increases the intra-axonal diameter, whereas myelin formation, occurring on the outer surface of the axons, provides electric insulation of the axon. These two structural processes jointly determine axonal conduction velocity. The synchrony in the development of myelin in cochlear nerve and brainstem implies that rapidly conducted axonal potentials appear at about the same time in pathways from the cochlea to the inferior colliculus and in the collicular projection to the medial geniculate body. Potentially then, the onset of rapidly conducted action potentials should occur at much the same time along the entire pathway from cochlea to the thalamus.

If age-related latency shifts are, in fact, due to progressive myelination of the auditory tract, there should be a general correspondence between the process of myelination and the time course of ABR latency changes during the perinatal period. In studies of premature infants, the observed rate of decrease in interwave latencies is quite large between the 28th and 40th weeks (Ponton et al., 1992; Starr et al., 1977). In postnatal infants, the values of the interwave intervals decline more slowly and approach adult values by 1 to 2 years of age (Jiang, Zheng, Sun, & Liu, 1991; Mochizuki, Go, Ohkubo, & Motomura, 1983; Ponton et al., 1992). It thus appears that the changes in myelin density and axonal velocity run generally parallel, with rapid change from 30 to 40 weeks of gestation and slower

change through the first and second postnatal years (see Table 3–1). Similar early and rapid maturation of latencies is observed in the P0–Na complex generated in the upper brainstem and primary auditory cortex.

With regard to conduction velocity, increasing myelin thickness should mean faster conduction, causing decreasing ABR peak latencies. It has been shown that the ABR intervals that reflect only axonal conduction time (I–II and III–IV) have an adultlike conduction time at term birth (Moore, Ponton, Eggermont, Wu, & Huang, 1996). However, at the time of term birth, adultlike conduction time may not mean adultlike conduction velocity, because the brainstem is growing and thus the auditory pathway is still lengthening. For instance, the distance between cochlear nucleus and the upper contralateral lateral lemniscus increases from 12 mm at 20 weeks CA to about 36 mm at 90 weeks CA (about 1 year of age), and to about 41 mm in the adult. In the same period the conduction velocity in this tract increases from 6 m/s to about 35 m/s (adult), which compensates nearly perfectly for the increase in path length (Moore et al., 1996). It thus appears that increasing myelin thickness, by compensating for increasing pathway length, keeps the conduction time constant in the first year of life. Synaptic changes, therefore, appear to be responsible for late stage maturation in brainstem conduction time.

Early-Maturing Cortical Potentials (Fields) and Their Generators

The basic structure of the auditory cortex matures rapidly in the perinatal period. Cortical neurons enlarge their dendrites during these months, and cortical thickness and lamination are adult-like by age 1 to 2 (Moore & Guan, 2001). In contrast to cortical layers II to VI, which are made up of tightly packed cell bodies, layer I consists mostly of axons and dendrites. The latter are made up by the apical dendrites of pyramidal cells in layers II, III, and V that extend into layer I. In the months before and after term birth, axonal maturation in cortex is confined to layer I. The Cajal-Retzius (C-R) cells that are intrinsic to layer I (Marin-Padilla & Marin-Padilla, 1982) employ the excitatory transmitter glutamate (del Rio, Martinez, Fonseca, Auladell, & Soriano, 1995). Neurofilament staining of human cortex indicates that C-R axons progressively develop their capacity for action potential conduction; at the 22nd fetal week only a small number of axons are stained, whereas at 4.5 postnatal months a prominent population is visible (Moore & Guan, 2001). The excitatory influence of this system is likely modulated by a population of intrinsic layer I inhibitory neurons (Imamoto, Karasawa, Isomura, & Nagatsu, 1994).

In addition to C-R cell intrinsic layer I axons, the pyramidal cell dendrites in layer I are also innervated by afferent axons coming from lower levels of the nervous system. Around the time of birth, these very thin axons enter the cortex from below and form branches within layer I that run parallel to the surface for distances up to several millimeters. These axons are present as early as the 7th gestational week (Marin-Padilla & Marin-Padilla, 1982) and are presumed to come from the earliest developing part of the central nervous system, the RAS (see Figure 3–1). As with the intrinsic C-R axons, neurofilament staining shows an increasing number of mature axons from the time of term birth to at least

4.5 months postnatal (Moore & Guan, 2001). The organization of this system, with its long tangential axons contacting the dendrites of large numbers of cortical neurons, would promote broad activation of the neurons in auditory cortex. However, these thin and very lightly myelinated RAS axons must have a very slow conduction velocity and therefore could contribute only to long-latency evoked potentials.

After the first half-year of life (i.e., between 4.5 months and one year of age), there is a complete disappearance of the layer of C-R axons (Moore & Guan, 2001). The GABAergic intrinsic cells in layer I, however, remain present into adulthood (Winer & Larue, 1989). During this same period, the number of RAS axons in layer I is markedly reduced. By one year of age, along with regression of the C-R and RAS systems, a number of relatively thick spiral axons are observed running upward to reach and enter layer I (Moore & Guan, 2001). These ascending axons in human cortex are projections from the medial (magnocellular) division of the MGB (Hashikawa, Molinari, Rausell, & Jones, 1995), and thus would provide the first source of input from the thalamus to the cortex. Because this system of input travels through the main auditory pathway to the thalamus, instead of through the thin-axon RAS pathway, it has the potential to generate shorter latency layer I–evoked potentials. Because it arises from a nontonotopically organized part of the MGB, these axons are part of the extralemniscal pathway.

At the same time, during the first postnatal year, there is a gradual onset of development of lemniscal thalamocortical projections to deeper layers of cortex. This so-called acoustic radiation (see Figure 3–1) is a large bundle of myelinated axons running from the MGB to the auditory cortex (Bürgel et al., 2006). By one month after birth, some neurofilament maturation is evident in axons of the acoustic radiation (Moore & Guan, 2001). This filament maturation is followed by increased myelination in the radiation from 3–4 months postnatal, as detected by histological staining (Kinney, Brody, Kloman, & Gilles, 1988) and structural MRI (Pujol et al., 2006). This gradually developing "bottom-up" mode of stimulation of auditory cortex neurons likely interacts with their "top-down" activation by layer I axons, with resulting modification of the early-maturing cortical potentials.

Functional MRI studies show maturation of the cortical response to auditory stimuli in infants with increasing age. By three months of age, all infants demonstrate a BOLD response to speech stimuli (Dehaene-Lambertz, Dehaene, & Hertz-Pannier, 2002). The bilateral activation in the superior temporal plane was greater on the left, with the highest level of response in the primary auditory area. There was also activity in higher level auditory areas located in the superior temporal and angular gyri. Thus, overall, the pattern of cortical regional activation evoked in infants via layer I stimulation is remarkably similar to the pattern seen in older children and adults.

In perinatal infants, brainstem potentials are not the only sign of auditory activity in the central nervous system, given that cortical potentials can be recorded as well. Cortical potentials present in infants include both the longest-latency AEPs and the MMR. Both of these types of potentials undergo dramatic changes across the first years of postnatal life.

Presentation of a novel (oddball or deviant) stimulus produces a larger AEP compared with that for a frequent

stimulus just preceding it. The difference between the oddball and frequent AEP is in adults called the mismatch negativity (MMN). The presence of an MMN is considered an indicator of preattentive detection of stimulus change, be it acoustical, phonetic, or contextual (Näätänen, 2001). One has to realize that the MMN is never recorded as such from the scalp; it is a construct designed by the investigator and is obtained by subtracting two nonsimultaneously recorded AEPs. Thus, in interpreting the MMN, one should always inspect the individually recorded AEPs, or their magnetic equivalent fields that are at the basis of this construct.

The MNN is related to the suppression of activity of the frequent stimulus as a result of its (quasi-) periodic presentation. In infants, a particular maturational AEP sequence, deviating from that in adults, is observed. The predominantly negative cortical AEP waveform as observed in adults is dominantly positive in neonates and infants, and to avoid confusion, it is often called the MMR. The suppression of the frequent-stimulus AEP is clearly present in 3-month-old infants and can form the basis for finding an MMR in infants and even in fetuses as young as 28 weeks gestational age (Draganova et al., 2005; Draganova, Eswaran, Murphy, Lowery, & Preissl, 2007). Infants of 8 months of age still showed slow positive MMR to /da/-/ta/ phoneme contrasts (Pang et al., 1998), whereas using gap duration as the contrast, a transition to an adultlike MMN at 6 months of age was observed (Trainor et al., 2003).

He, Hotson, and Trainor (2007) investigated the emergence of discriminative responses to pitch by recording two-, three-, and four-month-old infants' AEP responses to frequent and infrequent pitch changes in piano tones. In all age groups, the infants' responses to deviant tones were significantly different from the responses to the frequent tones, suggesting that the two tones were processed as different.

The above findings all suggest that auditory cortex metabolism and electrical or magnetic evoked activity is related to the behavioral change detection capabilities of infants. Since MMRs are derived from AEPs, this also implies that the AEPs are detectable at an early age. Early studies (Barnet, Ohlrich, Weiss, & Shanks, 1975; Ohlrich, Barnet, Weiss, & Shanks, 1978; Pasman et al., 1991; Rotteveel et al., 1987) show that in preterm babies at 24 weeks, the AEP is dominated by a large negative wave with a peak latency of 200 ms, followed by a positive peak at 600 ms that by 30 weeks is reduced to a latency approaching 300 ms and increases in amplitude. By the time of term birth, this positive peak has a latency of about 250 ms and dominates the response because the earlier negative wave has nearly completely disappeared.

This dominance of positive components continues until about age five months, when a new negative peak with latency around 400 begins to increase in amplitude and reduce in latency. Even by age five years, this sequence of positive (100 ms)–negative (200 ms)–positive (~350 ms) peaks (Figure 3–2) is still very much the standard morphology for AEPs recorded at the vertex (Pang & Taylor, 2000; Ponton, Eggermont, Kwong, & Don, 2000). Barnet et al. (1975) showed that during the first three years of life the latencies of the three dominant waves called P2, N2, and P3 decreased linearly with log age. Eggermont (1988) showed that an exponential decrease with age (time constant of ~40 weeks) comprehensively described the changes in P2 and N2 latency and was interpretable in biological terms (Ponton & Eggermont,

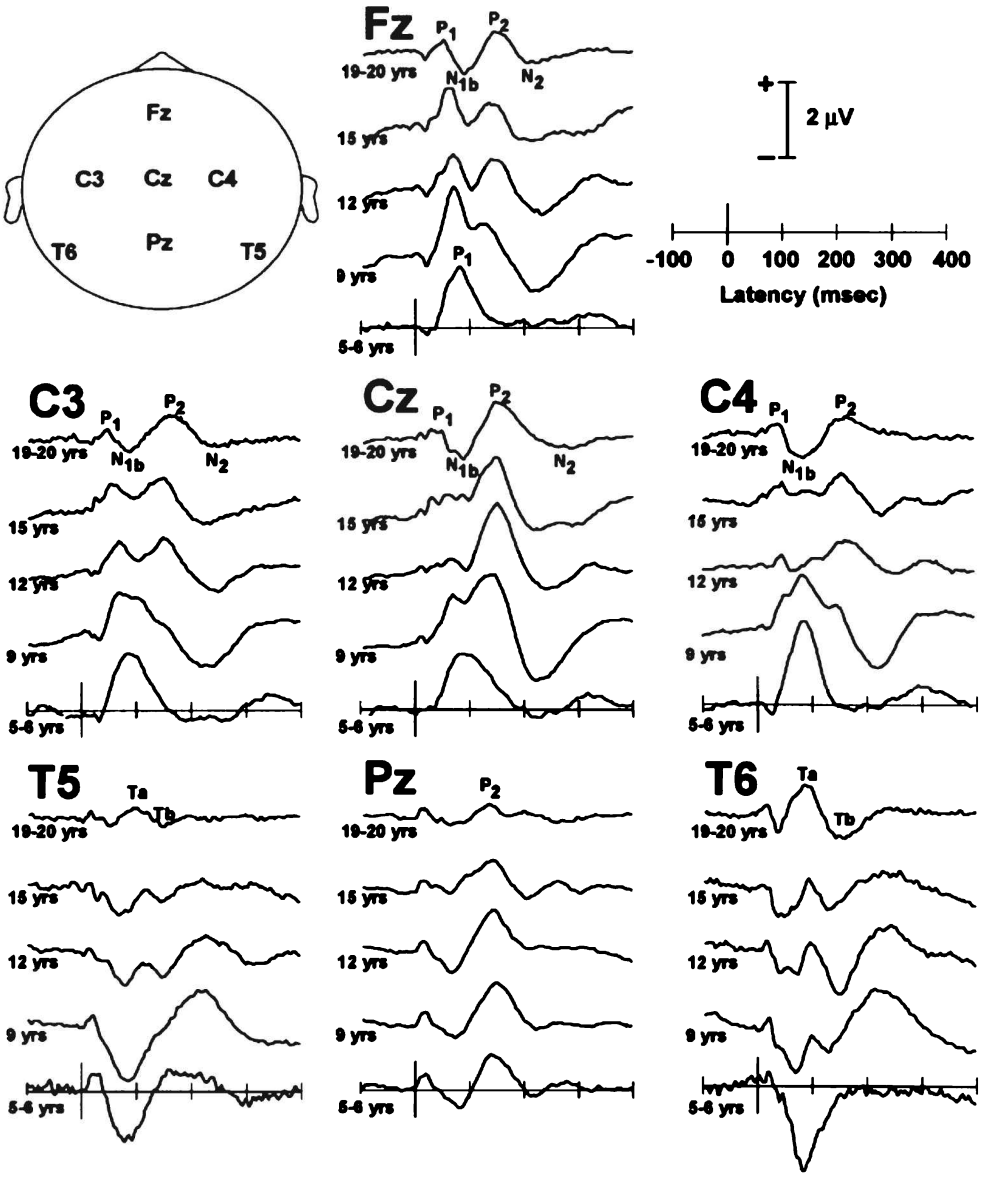

Figure 3–2. Age-dependent morphology of the AEPs for different recording sites. Note the late (~9 years) appearance of N1 in the Fz and Cz recordings [Reprinted from *Clinical neurophysiology*, Vol. 111, Ponton, C. W., Eggermont, J. J., Kwong, B., and Don, M., Maturation of human central auditory system activity: Evidence from multichannel evoked potentials (pp. 220–236). Copyright 2000, with permission from Elsevier]

2007). This time constant is only slightly longer than that of the maturation of the wave I to V latency difference in the ABR, suggesting that the maturation of this early cortical activity is either limited by that of the auditory brainstem or "co-tuned" to it. Since the RAS pathway originates as an offshoot of the brainstem

pathway, getting its auditory information from collaterals of lateral lemniscus axons, brainstem pathway activity would definitely be a synchronizing factor.

We now consider the very early maturation of the MMR as an indicator of change detection. What neural structures are used to accomplish this and what underlies the change from the slow positive MMR to the faster negative-positive MMR at around four to eight months of age depending on the acoustical change contrast? In two-month-old sleeping infants, the AEPs to frequent stimuli are large and positive and the response to the deviant is larger and also positive, yielding a positive MMR. In awake two-month-old infants, the AEPs to frequent and deviant stimuli are both smaller but still positive, with the latter showing a negative rebound at very long latencies (600–800 ms) (Friederici, Friedrich, & Weber, 2002). Positive MMRs, resulting from positive AEPs, suggest a depolarization site (sink) in layer IV, the entrance of thalamocortical activation, which is likely the earliest specific or lemniscal auditory input to the cortex.

Myelination of the acoustic radiation begins only at three months (Kinney et al., 1998), so if these unmyelinated fibers are functional, the results could be long-latency AEPs and MMRs. Alternatively, there could be an active hyperpolarization site (source) in layer I, where the axons are clearly mature (Eggermont, 2007). At four months of age, the AEPs are still broad positive peaks for both frequent and deviant stimuli, but the differences are that the AEP to the deviant is smaller for short latencies and larger for long latencies. Thus, the MMR has become a biphasic negative-positive wave, at least for frontal and central electrodes (He et al., 2007). In the late preteen years, the MMR becomes determined by the difference between the now dominant negative N1 components of the AEP for the frequent and the deviant condition (Gomot, Giard, Roux, Barthélémy, & Bruneau, 2000). The "real" MMN, then, is clearly the result of changes in a superficial late depolarization (sink) in layer I and/or upper layer II (Javitt, Steinschneider, Schroeder, & Arezzo, 1996).

As discussed earlier, cortical neurons are strongly driven during the perinatal months by the excitatory, C-R cell system in layer I. The rapid decrease of the C-R cell population in the middle of the first year of life ushers in a period in which GABAergic cells in layer I may exert a stronger influence. By age one year, thalamic (MGB) axons are beginning to form the more mature excitatory innervation of layer I. During this same period, from three to four months of age, the acoustic radiation has begun to myelinate, a process that continues until four or five years of age. Thus, from about three months of age, some synchronized activation of layer IV synapses should be expected. A set of mechanisms that could accommodate the various MMRs and AEPs observed in the preterm and postnatal period is presented by Eggermont and Moore (2012).

Neural Correlates of the Perceptual System Maturation

The transition from infancy to preschool years is characterized by rapid neural and cognitive development. Brain volume increases by 25% between birth and about 4 years of age (Courchesne et al., 2000), and this increase is almost entirely

due to expansion of the neocortex. Auditory cortical thickness more than doubles between birth and age 1 year (Moore & Guan, 2001), whereas cortical synaptic density doubles from birth to age 6 years (Huttenlocher & Dabholkar, 1997). Because conscious perception is based on cortical function, cognitive growth during this time is also rapid (see Table 3–1). Children's expressive vocabulary progresses from only a few words at 1 year to full sentences by 3 years of age. Between 6 months to 5 years, changes in speech perception occur that result in a bias toward the native language, such that discrimination of nonnative language sounds is strongly reduced (Werker & Tees, 1984). This coincides with the emergence of a differential cortical response to native and nonnative language contrasts in the MMN (Näätänen et al., 1997). The child's perceptual ability continues to mature gradually during school-age years. Between 5 and 12 years, perception of degraded speech and speech in noise gradually improves (Eisenberg, Shannon, Martinez, Wogonski, & Boothroyd, 2000; Elliott, 1979). Children's consonant identification abilities reach adultlike levels of performance at about age 15 in reverberation-only and noise-only listening conditions, but identification in the reverberation-plus-noise listening condition does not mature until the late teenage years (Johnson, 2000).

Late-Maturing Generators of Cortical Potentials

The basic cytoarchitecture and laminar organization of auditory cortex, as shown in Nissl stains, is adultlike by the end of the first or second year of life (Moore & Guan, 2001), yet the pattern of evoked cortical potentials still changes radically across childhood and teen years (see Figure 3–2). A possible explanation lies in the fact that the various systems of axons driving the cortex have a long maturational time course.

Thalamocortical axons have a typical termination pattern that consists of a dense innervation of layer IV, with sparser horizontal collaterals to layer VI and short vertical collaterals extending into the deepest part of layer III (Hashikawa et al., 1995). Neurofilament immunostaining (Moore & Guan, 2001) demonstrated small numbers of filament-stained axons traversing layers IV, V, and VI at ages 1 and 2 years. By age 3, the typical pattern of large endings in layer IV and short collaterals into the deepest part of layer III is evident. By age 5, mature thalamocortical axons fill the deeper cortical layers (Moore & Guan, 2001). Myelination of these thalamocortical axons occurs progressively from the 3rd to 4th postnatal months until about 4 to 5 years of age. Thus, in the perceptual maturation phase, the same area of temporal cortex is being activated as in the perinatal period, but the method of activation is different. In contrast to activation by the diffuse horizontal layer I system, the thalamocortical input forms a vertical, columnar, tonotopic system of connections. The course of maturation of the thalamocortical axons innervating core, belt, and parabelt is identical across the time period from 3 to 6 months to 4 to 5 years (Moore & Guan, 2001).

A second stage of cortical maturation occurs in later childhood. By age 5 to 6, mature thalamocortical axons fill the deeper cortical layers, but axons in layers II and III still show little sign of stainable neurofilaments (Moore & Guan, 2001). However, by age 11 or 12, the density

of mature axons in the upper layers has become equivalent to that of an adult. Studies in primates have shown that layers II and III are the source and target of many of the association axons running between the core and belt auditory cortex (Rauschecker, Tian, Pons, & Mishkin, 1997), as well as between belt and parabelt cortex (Hackett, Stepniewska, & Kaas, 1998). In addition, most of neurons in the CC connecting the auditory cortex with the corresponding area in the contralateral hemisphere arise and terminate in layers II and III (Hackett, Stepniewska, & Kaas, 1999). Thus, maturation of layer II and III axons significantly broadens the scope of intracortical processing, both within and between hemispheres.

DTI (diffusion tensor imaging) has become a powerful technique to explore the structural basis of brain development (Hüppi & Dubois, 2006). Water diffuses preferably along myelinated axon tracts. Parameters that allow an assessment thereof are the average diffusivity (D_{av}) and the fractional anisotropy (FA); the latter is based on water diffusion differences in the two directions. These parameters are assessed by DTI and calculated in each voxel of the image. D_{av} is the mean of the parallel and perpendicular diffusion values. During white matter development, D_{av} reflects premyelination changes in axonal width and axon neurofilaments, and in myelination. The increase in white matter FA values during development also takes place in two steps. The first increase again takes place before the histologic appearance of myelin. The second, more sustained, increase in anisotropy is associated with the histologic appearance of myelin and its maturation. This two-stage increase in white matter anisotropy occurs at different rates (and thus different time constants) for different brain areas (Hüppi & Dubois, 2006).

In the thalamus of subjects between 5 and 30 years old, single exponential maturation time courses were found both for D_{av} and for FA, with time constants in the range of 5.6 years and 8.9 years, respectively. The 90% maturation milestone was reached between 13 and 20 years (Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008). In the temporal and frontal regions of cortex, some myelinated white matter is seen relatively early, but quantitatively relevant changes are observed only after the fifth month. Half of the children (0–39 months old) in a study by Pujol et al. (2006) showed a myelinated white matter content of 10% by the age of 18 months in language-related areas. A total of 90% of children met this myelination criterion for language-related regions only after 35 months. White matter relative volume changes showed a maturation time constant of about 30 months. This suggests that temporal cortex myelination is adult-like at about 7 to 8 years, comparable to the maturation of white matter in the thalamus and around the time that the N1 component of the AEP appears. This estimate was smaller by a factor of about 2 than that obtained from a cohort of 88 volunteers between 3 and 30 years old (Paus et al., 2001; Pfefferbaum et al., 1994). This is to be expected given the nonoverlapping and very different age groups, the larger the age span in the population, and the longer the estimated time constant in the case of prolonged maturation. Maturation of FA with a time constant equal to 29 months was found in a group ranging in age from neonates to 10-year-old children for the superior longitudinal fasciculus (SLF), the major temporal-frontal tract that plays an

important role in language. This maturation time course parallels that of myelination, which also occurs in the first two years of life (Zhang et al., 2007). In contrast, Lebel et al. (2008) described maturation in the SLF for subjects between 5 and 30 years old as much slower and very similar both for D_{av} and FA, with time constants of 7.5 years and 9.6 years, respectively, again similar to the findings for the thalamus. The 90% milestone was reached between 13 and 20 years. These contrasting findings, albeit obtained in nonoverlapping age groups, still suggest two very distinct stages of maturation (Table 3–2). In a study comprising 241 neonates and young children up to 8.5 years using T2-weighted MRI, Su, Kuan, Kaga, Sano, and Mima (2008) observed myelination changes in auditory cortex to progress with a time constant of 5.5 years. However, in children 5 years of age and older, no significant differences from adults were detectable.

Functional Measures of Cortical Function

Across early childhood, there is increasing prominence of short-latency over longer-latency positive cortical AEP components, which may be related to the newly maturing system of thalamocortical connections. The middle latency components typically mature earlier than

Table 3–2. Maturation Along the Auditory System

Structure	Test	Maturity	Authors
Auditory nerve	ABR wave I	3–6 mo	Eggermont et al. (1991)
Brainstem	ABR–click	2 yr	Eggermont (1988)
Brainstem	ABR–mid freq	2 yr	Ponton et al. (1992)
Thalamus	DTI	20 yr	Mukherjee et al. (2001)
Thalamus	DTI	15–20 yr	Lebel et al. (2008)
Acoustic radiation	histology	4–5 yr	Kinney et al. (1988)
Corpus Callosum	DTI	2 yr	Ding et al. (2008)
Auditory Cortex	MRI	12–16 yr	Su et al. (2008)
Temporal cortex	DTI	7–8 yr	Pujol et al. (2006)
SLF	DTI		Zhang et al. (2007)
SLF	DTI	13–20 yr	Lebel et al. (2008)
T complex, P2, Pa, Pb	AEP	1.5–2 yr	Eggermont (1988)
N2	AEP	5 yr	Ponton et al. (2000)
P1, N1, TP200	AEP	15–20 yr	Ponton et al. (2002)

the long-latency AEPs, consisting of the P1–N1–P2–N2 complex with mature latencies of about 50, 100, 150, and 200 ms. The adult waveform P1–N1–P2–N2 complex (see Figure 3–2) is achieved between 14 and 16 years of age (Pasman, Rotteveel, Maassen, & Visco, 1999; Ponton et al., 2000).

P1 can be distinguished at 30 weeks CA as a broad wave with a latency of 80 to 100 msec (Pasman et al., 1991). Over time, its peak latency gradually shortens to reach an adult level of 50 ms, and during that time the amplitude decreases. The late appearance of N1 is partly due to its greater sensitivity to the interstimulus interval (ISI) in children than in adults. Therefore, most studies using ISIs shorter than 1 sec do not observe N1 reliably before age 9 (Ponton et al., 2000), but with a longer ISI, N1 becomes visible from age 6 on (Gilley, Sharma, Dorman, & Martin, 2005). Although the latency changes for P1 and N1 peaks are similar, the maturational changes in magnitude are opposite; P1 magnitude decreases while N1 increases with increasing age. Because N1 emerges in the AEP at about 9 to 10 years of age, when the neural generators producing the P1 peak are essentially adultlike, and given the partial temporal overlap and common tangential dipole orientation of these two components, it is possible that the magnitude and latency changes of the maturing N1 peak are superimposed on those of P1 (Ponton & Eggermont, 2001). During early maturation, it is difficult to clearly identify a component as N2 when the N1 is not yet visible. However, their maturational trajectories are different, as shown by the N1/N2 amplitude ratio increase with age. The N2 is mature much earlier (time constant 2 years) than the N1 (time constant >4 years), as shown by Ponton, Eggermont, Khosla, Kwong, and Don (2002).

Results of equivalent current dipole source modeling (Ponton et al., 2002) demonstrated that the three orthogonal dipole components of the sources in each auditory cortex hemisphere isolate three distinct sets of AEP components. The MLR peaks Pa and Pb are best represented by the sagittal (anteroposterior) oriented dipole sources; the "classic" P1–N1–P2–N2 sequence is isolated to the tangential (to the scalp) oriented sources that are perpendicular to the superior surface of the temporal lobe; and the T-complex peaks Ta and Tb, together with the TP200, are represented in the radial (perpendicular to the scalp) oriented dipole sources. The grouping of AEP components isolated in each orthogonal dipole remained the same across a 5- to 20-year age span. This suggests that the orientations of the AEP generators are essentially adultlike by 5 years of age. Note, however, that Albrecht, Suchodoletz, and Uwer (2000) attributed the emergence of the N1/P2 complex in adolescence (in children older than 8 years) to a change of the tangential dipole source potential. However, one has to be careful to use the term "N1/P2 complex," as P2 matures early (reaching adult values by as early as 2–3 years of age), while the N1 follows a much longer developmental time course, extending into adolescence (Ponton et al., 2000).

Based on a global measure of similarity between an individual's AEP waveforms, Bishop, Hardiman, Uwer, and von Suchodoletz (2007) distinguished three developmental periods: 5 to 12 years, 13 to 16 years, and adulthood. There was a fairly sharp change in the AEP waveform at Fz around 12 years of age, when N1–P2 was fully mature. Ponton et al.

(2000, 2002) distinguished three maturation groups of potentials: one group reaching maturity at age 5 to 6 and consisting of the MLR components Pa and Pb, the AEP component P2, and the T-complex, recorded from temporal electrodes. The temporally recorded Ta/Tb complex, which is distinct from N1, is already mature at age 5 to 6 (Pang & Taylor, 2000; Tonnquist-Uhlen, Ponton, Eggermont, Kwong & Don, 2003). Because of its radially oriented dipole, it is likely generated in BA22. A second group that was also relatively fast to mature (time constant 2 years) was represented by N2 only. A third group was characterized by a slower pattern of maturation (time constants of 4–9 years) and included the AEP components P1, N1, and TP200, the long-latency component following the T-complex. The observed latency differences combined with the differences in maturation rate indicate that P2 is not identical to TP200. The results also demonstrated the independence of the T-complex components, represented in the radial dipoles, from the P1, N1, and P2 components, contained in the tangentially oriented dipole sources.

The work of Moore and Guan (2001) on the structural maturation of the human auditory cortex and the sink-source description of synaptic activation (Eggermont, 2007) has led to a straightforward interpretation of the cortical layer of origin of the scalp-recorded AEPs. From the polarity of the AEP components, one has to conclude that scalp-positive components such as the P1 and P2 are generated in the lemniscal input layers (lower III–IV) of auditory cortex. P1 is likely mature by age 12 (Ponton & Eggermont, 2001), comparable to the time of maturation of the thalamus, as shown in DTI (see Table 3–2). The P1 maturation was estimated from cochlear implant cases that failed to develop an N1. As most scalp-positive evoked potential components originate from excitatory synaptic activity in lower layer III and layer IV, most of these (e.g., the MLRs) indeed appear fully mature with respect to latency and amplitude by that age. The pedunculopontine tegmental nucleus, a cholinergic subdivision of the reticular formation that receives auditory input, may be significant for generation of both the human P1 (Harrison, Woolf, & Buchwald, 1990) and P2 (Näätänen & Picton, 1987). An overview of the estimated ages of maturity is given in Table 3–2.

In contrast to P1 and P2, the scalp-negative components must originate from excitatory inputs in more superficial layers (II, upper III). This is based on the location of the activated excitatory synapses that generate these components in animals (Eggermont, 2007; Eggermont & Ponton, 2002). Several studies have suggested that the development of the N1 component may relate to the formation of functioning synaptic connections within the upper layers of the auditory cortex (Ponton et al., 2000, 2002), with the synaptic activation (sinks) in older infants and children concentrated in the deeper layers of the cortex (causing a surface positive wave) and not extending fully into the upper layer parts of the dendritic trees (which would generate a surface negative wave). Because of this prolonged maturation of superficial cortical layers, it is not surprising that for stimulus repetition rates of ≥1 Hz, N1 cannot be recorded below the age of 8 to 9 years. The maturation of N1 extends well into adolescence and appears to be associated with activity originating from the newly mature upper layer II synaptic activity.

However, this explanation of late detectability of N1 does not at first sight fit with the early emerging responses in

the same latency range recorded from the lateral scalp, the so-called T-complex (Tonnquist-Uhlen et al., 2003), which is likely also generated in auditory association regions of the cortex (Ponton et al., 2002). The T-complex consists of two surface negative waves Na and Tb, even in infants, and with latencies in adulthood similar to N1. Picton and Taylor (2007) speculated that the infant auditory response generated in the supratemporal plane contains a large positive wave with peak latency between 100 and 200 msec that obscures an also present smaller negative N1 component. However, the maturation of the T-complex is very different from that of N1, so it is unlikely that the underlying sources would be similar. As mentioned by Tonnquist-Uhlen et al. (2003), the Tb component of the T-complex does not show further maturational changes after 5 years of age, and latency and amplitude are statistically similar to those of P2, a component that has been ascribed to RAS activation of cortex. This was similar for electrodes T4 and T6 (both contralateral to the stimulus ear). AEPs at T6 are a faithful inversion of those at C4. Whereas the overall morphology on T4 is different from a time inverted C4, the probability that Tb is the inverse of P2 remains.

A very interesting issue is the relationship between the protracted maturation of the N1 and the development of higher cognitive skills during childhood. The N1 emerges after children have normally acquired basic skills of verbal language, whereas language acquisition is heavily contingent on auditory sensory processing (see Table 3–1). This is consistent with the view that the N1 does not index the perception of the sound features but reflects facilitative or integrative processes, sound detection, and orienting (for review, see Näätänen, 2001).

Structural and Functional Maturation of the Corpus Callosum

The CC, the largest white matter tract in the brain, connects the left and right cerebral hemispheres. Callosal fibers are important for motor and sensory integration, attention, memory, and general cognitive functioning (reviewed by Bamiou et al., 2007]). Callosal axons in the auditory system originate from pyramidal neurons in layer III, and to a much lesser extent from the infragranular layers V and VI, and layer I (Code & Winer, 1986). Large-diameter fibers with fast conduction times dominate the posterior part of the auditory area of the CC. In contrast, the anterior part of the splenium and the isthmus, which connect auditory association areas, contain smaller-diameter fibres (Aboitiz, Scheibel, & Zaidel, 1992). The development and myelination of the CC is ongoing during the first two decades of life (Yakovlev & Lecours, 1967). Overall growth of the CC during maturation is related to increased myelination of the axons (Luders, Thompson, & Toga, 2010).

Barnea-Goraly et al. (2005) in a DTI study found overlapping changes in FA and white matter density with age in the body of the CC, which contains fibers important for connecting motor, sensory, and auditory cortices. Kim et al. (2006) showed that compared with the normal control group, the FA values of children with language impairments was significantly reduced in the genu of the CC. Ding et al. (2008) showed that the FA in the genu of the CC increased until 5 years of age and remained nearly constant thereafter; it showed a significant increase from 0 to 2 years versus 2 to 5 years, whereas there was no difference in the other age groups. FA in the splenium

of the CC (sCC) values showed no significant changes after 2 years of age. Deoni et al. (2011) presented a quantitative study of myelination in healthy human infants, aged from 3 to 11 months. Using a new myelin-specific MRI technique, they reported a spatiotemporal pattern of myelination beginning in the cerebellum, pons, and internal capsule; proceeding caudocranially from the sCC and optic radiations (at 3–4 months); to the occipital and parietal lobes (at 4–6 months); and then to the genu of the CC and frontal and temporal lobes (at 6–8 months).

Interhemispheric difference in cortical response maturation was reflected in the very early maturation of the T-complex in the left hemisphere (at age 3) and much later (at age 7–8) in the right hemisphere (Pang & Taylor, 2000). The maturation at age 3 follows that of the myelination of the thalamocortical radiation that matures around 3 to 4 years of age (Yakovlev & Lecours, 1967). From dipole source modeling (Ponton et al., 2002), one obtained the impression that the contralateral dipole (right hemisphere) matured earlier and faster than the ipsilateral one. Whereas N1 could be obtained at electrode C4 in some children in the age group of 5 to 6 years old with a latency around 138 ms, it was never seen at C3 (ipsilateral) before age 7 (Ponton et al., 2000). In the dipole representation, the first signs of N1 appeared contralaterally around age 9 and at age 10 ipsilaterally. For N1 the contralateral dipole reached maturity with a time constant of ~4 years, whereas the maturation of the ipsilateral dipole had a time constant about twice as long (Ponton et al., 2002). Thus, the activation of the ipsilateral hemisphere occurred approximately one year later than that for the contralateral one and matured at approximately half the rate thereof. Part of this maturational difference between hemispheres may reflect the late maturation of the CC, whose cross-sectional area continues to grow well into the second decade of life (Yakovlev & Lecours, 1967)

Effects of Environmental Noise on the Developing Brain

Development in Abnormal Acoustic Environments

The newborn auditory system in laboratory animals shows a well-described time period of acute sensibility to external sounds such that it may permanently shape the central auditory nervous system. This is the so-called critical period, the time and length of which differs for specific sound features (de Villers-Sidani & Merzenich, 2011). Critical periods can be prevented from closing under stimulation with continuous noise (Chang & Merzenich, 2003). Neural processing, as a result of aberrant sound stimulation in the critical period, can permanently be changed and may detrimentally affect maturation and adult auditory processing. For instance, stimulation with noise bursts disrupts primary auditory cortex both in neonates (Zhang, Bao, & Merzenich, 2002) and adults (Zhou & Merzenich, 2012).

The critical period, in general, is a time period when the best neural representation of the world is selected from among the many competing inputs that affect the maturing nervous system. The growth and function of lateral inhibitory circuits may be important for terminating the critical period. The difficultly of this problem is highlighted by the fact that the closure of the early critical period may be dependent on the input received (Chang & Mer-

zenich, 2003; Zhang et al., 2001). Moreover, specific types of auditory experience can result in the critical period remaining open in some parts of A1, but being closed in others (de Villers-Sidani, Simpson, Lu, Lin, & Merzenich, 2008), further emphasizing the fact that critical periods are controlled by sensory inputs.

It is not exactly known whether there are similar critical periods in human auditory development, but from the cochlear implant literature one may derive critical periods for the necessity of auditory stimulation for binaural hearing (<2 years of unilateral hearing, i.e., one cochlear implant; Gordon, Salloum, Toor, van Hoesel, & Papsin, 2012); for the development of certain auditory evoked response components (i.e., N1; <3 years of deafness under the age of 6; Ponton & Eggermont, 2001); and for normal language development (Svirsky, Robbins, Kirk, Pisoni, & Miyamoto, 2000). The development of the CC may play a role here. Conductive hearing loss in children is a major determinant of language delay and may potentially cause long-lasting deficits.

Noise in the Neonatal Intensive Care Unit

The human cochlea is fully developed by 24 weeks of gestation. A blink startle response can first be elicited (acoustically) at 24 to 25 weeks and is constantly present at 28 weeks. Hearing thresholds are 40 dB at 27 to 28 weeks and reach the adult threshold of 13.5 dB by 42 weeks of gestation (Birnholz & Benacerrah, 1983). Early born preterm children typically end up in the neonatal intensive care unit (NICU), and quite often they show signs of auditory neuropathy and sensorineural hearing loss; however, even in case they do not, they may have other neurological problems from which they only very slowly recover (Marlow, Wolke, Bracewell, & Samara, 2005). It has been said (Brown, 2009) that early birth disrupts the normal sequence of sensory development; tactile and balance first, followed by auditory and finally visual sensations. In the NICU), the auditory sense may be forcefully stimulated, largely because of the continuous presence of noise, albeit of relatively low level. This has been attributed to various disorders in later life such as attention deficit and hyperactivity disorder (ADHD) and CAPD.

A busy NICU is by default a noisy environment. Noise is also present in the confines of an isolette or incubator. A big issue is the so far largely unknown effect of prolonged noise exposure in the NICU on the neonatal brain. Whereas it has been established that this does not cause hearing loss, it may still have profound effects on hearing, as animal studies suggest. In neonatal and adult animals, band-pass noise exposure leads to contracting tonotopic maps surrounded by expanding tonotopic maps (Pienkowski & Eggermont, 2012). This refers mostly to critical periods in animals, but potential extrapolations can be drawn that pertain to human auditory development.

Even in the absence of specific central nervous system injuries, preterm birth, per se, may be responsible for the delay observed in the formation of sulci in extremely low gestational age newborns when imaged at term-equivalent age (Ajayi-Obe, Saeed, Cowan, Rutherford, & Edwards, 2000; Kapellou et al., 2006). Furthermore, MRI studies assessing brain maturation in preterm infants ranging from 23 to 36 gestational weeks have demonstrated a reduction in total cerebral volume and that of specific structures, like the hippocampus, when evaluated at term-equivalent age. Moreover, this

reduction seems to persist during childhood and adolescence (Constable et al., 2008), generally correlating with neuropsychological development (Beauchamp et al., 2008).

Recent studies indicate that excessive exposure of the premature infant to noise can result in a variety of physiological disturbances that may have long-term effects on his or her nervous system. One example of a potential long-term consequence may be ADHD. Although ADHD is not fully understood, it has been determined that there is a possible sensorineural disturbance due to hypoxia that results in the diagnosis (Barkley, 1998). Because environmental stressors such as noise in the NICU can result in bradycardia and hypoxia, one cannot rule out the potential link between premature exposure to noise above 50 dB SPL, the hourly limit recommended by the Standards for Newborn ICU Design (2002), and sensorineural disorganization resulting in ADHD. However, in light of hearing thresholds of about 40 dB in 27 to 28 week CA (Birnholz & Benacerrah, 1983), this seems somewhat contradictory.

According to Bremmer, Byers, and Kiehl (2003), sensory development in utero follows a sequence starting with touch followed by movement, sense of position, and balance; smell and taste; hearing; and finally vision. This sequence is not altered by preterm birth; however, stimulation of a particular sensory system outside the natural developmental sequence may cause detrimental long-term effects. For example, unusually early sensory experience in a later developing system such as hearing could interfere with the functioning of an earlier-developing sensory system such as movement and balance. Preterm infants are typically exposed to enhanced auditory stimulation and reduced vestibular stimulation at a time in development when they would normally experience low-pass filtered auditory stimulation and regular vestibular stimulation from the mother's movements (Brown, 2009). This information would suggest that limiting noise levels in the NICU could enhance the development of those sensory systems that normally manifest themselves before hearing emerges. It is thus possible, but not really demonstrated, that an early-developed auditory system exerts an inhibitory modulation on some parts of the somatosensory system. This is even more important for extremely prematurely born infants, who are exposed to the extrauterine environment in the NICU for longer than older newborns of the same term-corrected age. However, since hearing thresholds are very high at CA <27 weeks, this seems unlikely. Several studies of long-term outcomes in NICU graduates cite speech and language problems (Marlow et al., 2005; Stjernqvist & Svenningsen, 1999; Kern & Gayraud, 2007). However, few have specifically linked them with noise levels.

Environmental Sound and the Fetus/Preterm Infant

The fetal environment acts as a low-pass filter (Gerhardt & Abrams, 1996); therefore, fetal noise exposure from a given source is different from the exposure presented to a preterm baby by the same source in air. A second confounding factor in determining the effects of fetal noise exposure is the ontogeny of the ear. Even though the mid-frequency range of the human cochlea develops first (Eggermont et al., 1991; Ponton et al., 1992), the fetal ear receives low-

frequency stimuli first. The consequence of a loud-noise exposure delivered via a low-pass filtered and attenuated noise environment may not be the typical high-frequency "notch" seen in adults. Instead, noise-induced hearing loss to the fetal ear may later on be revealed as changes to low- and mid-frequency stimuli. The same noise exposure that causes only a temporary threshold shift in the adult may cause more permanent damage to the developing ear (Ohlemiller, 2008). Continued noise exposure to the fetus could be expected to cause increased damage, over time.

To explore this, Lalande, Hétu, and Lambert (1986) examined 131 children whose mothers had worked, while pregnant with that child, in noise conditions ranging from 65 to 95 dBA. Results showed a three-fold increase in the risk of high-frequency hearing loss in the children whose mothers were exposed to noise in the range between a $L_{Aeq,9m}$ (average noise dose for the 9-month pregnancy) of 85 to 95 dB. Furthermore, there was a significant increase in the risk of the child's hearing loss at a frequency of 4000 Hz when these exposures involved a strong component of low-frequency noise. This emphasized the sensitivity of the 4 kHz region in the cochlea to noise in general but seems to contradict the effects of low-pass filtering in utero (see above).

Summary of Structural and Functional Correlations

A summary of the endpoints of the maturation process (based on data and references shown in Table 3–2) are shown in Figure 3–3.

Structural and Functional Brain Correlates of Discrimination

Discrimination, as a behavioral process, is the ability to recognize the differences between auditory stimuli, and involves two subsystems: (1) analysis of the physical parameters of the stimulus is performed by the brainstem; and (2) attention to and awareness of the stimulus is mediated by the RAS (and later thalamic) input into the layer I system. Discriminative ability and both subsystems are operational in the months before and after term birth.

Brainstem nuclei perform a detailed analysis of stimulus physical parameters, following the spectral analysis performed by the cochlea. Histologic studies show rapid development of the brainstem pathway, including cells and axons, in the months before and after term birth. Brainstem development occurs synchronously from cochlear nerve to thalamus. Neurofilament development is an autonomous process, but myelination is driven by axonal activity. Imaging studies confirm the rapid maturation of myelin in the early postnatal months. The ABR biphasic waves reflect activity in myelinated pathways of the lower brainstem. ABR development, with onset early in third trimester and rapid postnatal maturation, mirrors the anatomical development of the brainstem, with shortening latency reflecting the effect of increasing myelination on conduction velocity. The developmental pattern of P0-Na mirrors that of the myelination of the brachium of the inferior colliculus, in being present at birth and before, with significant maturation by age 3 months. Once established, there should be no change in the functioning of the brainstem pathway over the life span.

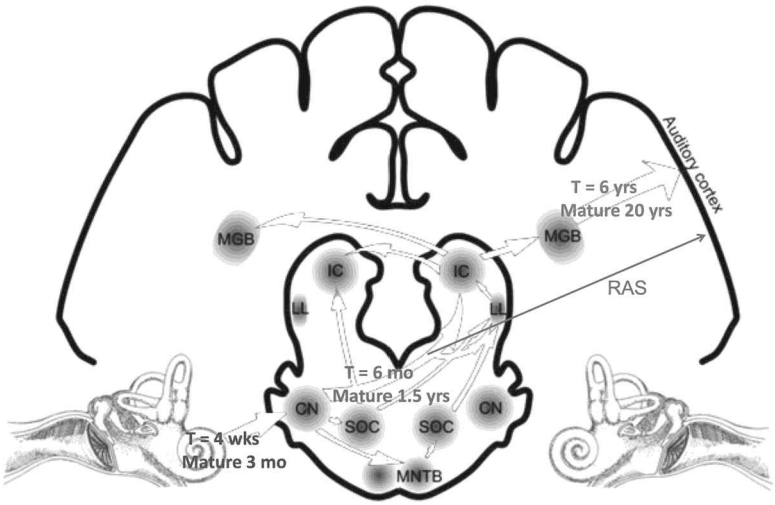

Figure 3–3. Anatomical and functional maturation periods of the human auditory system. The fastest maturing system is the cochlea and auditory nerve with a time constant of 4 weeks and reaches maturation at approximately 3 months. The brainstem up to and including the fibers into the MGB mature with a time constant of approximately 6 months and reach maturity at approximately 1.5 years of age. This also includes the maturation of the RAS pathway that innervates cortical layer I. The thalamus, the auditory radiation, and the cortex do not appear to be mature until approximately 20 years of age [Partially reprinted from *Brain Research Reviews*, Vol. 56, Kral, A., & Eggermont, J .J., What's to lose and what's to learn: Development under auditory deprivation, cochlear implants and limits of cortical plasticity (pp. 259–269). Copyright 2007]

Awareness is a function of cortex. At birth and in the first months of life, the only specific pathway to cortex is the RAS system to layer I. RAS input consists of very thin, slowly conducting axons, which implies generation of long-latency potentials. Within layer I, activity is magnified by the intrinsic C-R cell axon system. At birth and in the first months of life, the only cortical axons capable of conduction are the RAS and C-R axons in layer I. Both RAS and C-R axons undergo rapid maturation from the prenatal period to around 6 months of age. RAS and C-R axons form a very nonspecific projection, with each axon running for millimeters and contacting a large number of dendrites, producing top-down stimulation of neurons in the deeper cortical layers. Functional MRI confirms that layer I activates all areas of the auditory cortex, with adultlike activation by 3 months of age. During the latter half of the first year of life, axons resembling primate MGB projections reach layer 1. The P2 and derived MMR are both present at birth and mature rapidly after birth. The time constants of maturation of P2 and MMR are the same as that of ABR wave V. Age 6 months is an anatomical turning point, the time when the C-R system disappears, RAS input appears

reduced, and thalamic input to layer I arrives. Age 6 months (4–8 months) is a physiological turning point at which the MMR turns from positive to negative, and P2 latency begins to shorten to its adult value. Age 6 months is also a behavioral turning point, the time when, for instance, universal discrimination of language sounds begins to regress. The layer I arousal system remains functional across the life span, albeit much reduced compared with neonates. The MMN and P2 are robustly present in late-onset (postlingual) deafness, probably because they mature very early.

Structural and Functional Brain Correlates of Perception

Perception connects a stimulus to its meaning or significance. From the anatomical point of view, it is a function of the cortex, driven initially by bottom-up stimulation from the thalamus and continuing with intracortical input into superficial layers. Both perception and these cortical connections begin to develop slowly in the second half of the first year of life and continue into late childhood to adult years. This phase is characterized by thalamocortical input and forms the biological substrate of perception and the acquisition of language.

Filament maturation in the acoustic radiation, the proximal part of the pathway from thalamus to auditory cortex, begins shortly after birth. Myelination of the axons in the acoustic radiation begins at around 4 months postnatal and continues to about 4 years. Because of its straight medial-to-lateral course, the acoustic radiation will not generate a far-field potential. Filament maturation of the distal thalamic axons into the deeper cortical layers is an extended process, occurring progressively from the later part of the first year of life to age 5 to 6 (Moore & Guan, 2001). The onset of thalamocortical input into the cortex from about 6 months of age coincides with the regression of the layer I axonal system and the decline in universal speech sound discrimination. The Pa–Nb complex is not present at birth but is mature by age 5. It may result from excitatory (Pa) and inhibitory (Nb) PSPs (postsynaptic potentials) in layer IV. Pa–Nb is unaffected by late-onset deafness, possibly because the thalamic input to layer IV is relatively mature by age 2 to 3 years. P1 is likely generated by cortical deep layers activated by thalamocortical axons, and thus should reflect perceptual processing. Because P1 is a deep-layer cortical potential, it should not appear until after 6 months of age. However, the evoked potential component usually labeled P1 is a mix of the middle latency component Pb and the "real" P1, which results from the output of the cholinergic pedunculopontine tegmental. P1 is well developed by age 5 to 6, which is a good fit with the histology. As shown by deprivation studies, maturation of P1 is dependent on afferent activity, quantified by the time in sound. This implies that myelination is a factor, but it may also reflect synaptic maturation. Since P1 reflects a dipole field, it requires synchronous activation of fast-responding dendritic synapses in layer III/IV with return current in the apical dendrite (Eggermont & Moore, 2012).

Histologic studies show that input into cortical layers II–III matures between age 6 and 12 years. All input to layers II to III is from other cortical areas. Thus, maturation of layers II and III axons significantly broadens the scope of cortical processing, both within and between

hemispheres, and provides a basis for changes in perception in later childhood and beyond (Moore & Guan, 2001). Excitatory synapses in the upper layers generate the negative scalp-recorded N1 wave that matures gradually from age 6 to 12. The N1 wave partly overlaps P1 and thus partly masks it, accounting for the P1 decrease in latency past 6 years of age (Ponton et al., 2000).

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CHAPTER 4

NEUROLOGICAL SUBSTRATE OF CENTRAL AUDITORY PROCESSING DISORDER

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Central auditory processing disorder (CAPD) is seen in both children and adults, including the elderly. Central auditory processing deficits and CAPD have been linked to a number of different etiological bases, including those comprising the focus of this chapter—frank neurological lesions or compromise of the central auditory nervous system (CANS). Although the incidence of children with CAPD resulting from neurological abnormalities is considerably lower than the incidence of children with CAPD and learning problems in the absence of an identifiable underlying neuropathology, some of this latter group of children also present neurological issues underlying their auditory problems. It must be emphasized that the origins of CAPD in children are neurobiological—that is, whether the source of CANS dysfunction is benign (neuromaturational or neuroanatomical) or the result of frank neurological lesion or compromise, the underlying source of the resulting central auditory deficits and CAPD originate in the central nervous system (Bellis, Chermak, Weihing, & Musiek, in press; Chermak, Bellis, & Musiek, 2007; Chermak & Musiek, 2011). Following a brief overview of causation in adults, we turn our attention to the neurological origins of CAPD in children.

Acquired Causes of CAPD in Adults

Acquired causes of CAPD in adults include: neurological lesions or compromise of the CANS, including neo-

plasms; neurodegenerative processes (e.g., multiple sclerosis, Alzheimer's); head trauma; impaired cerebral circulation (e.g., strokes); aging, including central aspects of presbycusis; noise exposure; and exposure to neurotoxic chemicals or heavy metals (e.g., styrene, lead, mercury). Although the peripheral effects of presbycusis are well known, recent research has documented age-related decline in central auditory function. Age-related decline in temporal processing has been observed beginning in the fourth decade of life (Kumar, 2011). Gates, Feeney, and Mills (2008) reported that central auditory function declines more than pure tone sensitivity or outer hair cell function from the seventh decade and beyond. Moreover, even a modest decline in peripheral sensitivity reduces the degree to which central auditory structures (e.g., bilateral superior temporal gyri, thalamus, brainstem) are activated (as seen in functional [f]MRI) when listening to complex sentences, and a linear relationship between hearing acuity and gray matter volume in the primary auditory cortex, suggesting atrophy, has been reported (Peele et al., 2011). (See Musiek, Baran, Shinn, & Jones, 2012 for discussion and case presentations of etiologies underlying CAPD in adults, as well as Chapter 15, Volume 2 of this Handbook.)

Etiology of CAPD in Children

A number of years ago, we proposed three bases for CAPD in children who also presented with learning problems (Musiek, Gollegly, & Ross, 1985). We noted that there is a small percentage of children with CAPD and related learning difficulties whose auditory problems arise from neurological disorders. Etiologies may include seizure disorder (e.g., Laudau Kleffner syndrome), traumatic brain injury, neoplasms, neurodegenerative disorders, neurotoxicity, cerebrovascular accidents, metabolic disorders, and genetic disorders across a wide variety of sites of lesion at all levels of the CANS (Bamiou, Musiek, & Luxon, 2001; Musiek & Chermak, 2009). The two other bases of CAPD in children are neuromaturational lag and neuroanatomical abnormalities. We briefly outline these two other bases of pediatric CAPD before focusing on the population of children with central auditory deficits or diagnosed CAPD with neurological involvement.

Neuromaturational Lag and Neuroanatomical Abnormalities

The two other bases of CAPD in children are neuromaturational lag and neuroanatomical abnormalities. Children diagnosed with CAPD with a presumed underlying neuromaturational basis present central auditory systems that appear to be maturing more slowly than seen in normal children, often secondary to auditory deprivation and/or delayed myelin maturation in the subcortex, cortex, and/or corpus callosum (Bamiou et al., 2001; Baran & Musiek, 1999; Musiek, Baran, & Pinheiro, 1994; Musiek, Gollegly, & Baran, 1984). These delays, at least theoretically, can result in decreased performance on central auditory tests and related hearing difficulties and are likely related to the long maturational course of the CANS (Geidd et al., 1996; Musiek et al., 1984; Yakovlev & LeCours, 1967).

The third source of CAPD in children, neuroanatomical abnormalities, results from abnormal development of the higher auditory areas yielding abnormal anatomy (Boscariol et al., 2009, 2010, 2011; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990). In contrast to CAPD resulting from neurological disorders, which may be congenital or acquired, neuroanatomical abnormalities are congenital and they are not frank neurological disorders. These neuroanatomical abnormalities include ectopic areas (misplaced, small nests of normal cells) and polymicrogyri (underdeveloped gyri in greater number than seen in normal brains) of the cortex, and other anatomical variances. The ectopic areas and polymicrogyri have been noted in children with learning problems (mostly in the left hemisphere and in the auditory cortex) by close examination of the brain postmortem (Boscariol et al., 2009, 2010, 2011; Galaburda et al.,1985; Galaburda & Kemper, 1978). In addition, the planum temporal, which is typically larger on the left side of the brain in normal children, has been shown to be symmetrical or larger on the right side in children with dyslexia (Galaburda et al., 1985; Hynd et al., 1990). Other anatomical regions of the brain in children with learning problems have been shown to be smaller (suggesting that these areas are underdeveloped) relative to normal controls. Interestingly, the areas that are abnormally small are the isthmus of the corpus callosum and the insula, which are both auditory responsive areas (Musiek & Baran, 2007). The other brain area that has been shown to be reduced in size in these children is the genu of the corpus callosum, which contains primarily fibers of the frontal lobe (Hynd et al., 1990). Based on the aforementioned, it is reasonable to assume that children with learning problems may have neurological, maturational, or anatomical variances that influence central auditory function. In fact, Banai, Abrams, and Kraus (2007) concluded that as many as 40% of children with learning disabilities have abnormal speech-elicited auditory brainstem responses and that these individuals are also likely to exhibit abnormal cortical processing, although the researchers were not able to answer the question of causality underlying the abnormal auditory physiological function.

Whether the source of CANS dysfunction is benign (maturational or neuroanatomical) or the result of frank neurological lesion or compromise, the underlying source of the resulting central auditory deficits and CAPD is *neurobiological,* originating in the central nervous system. Indeed, there are strong similarities between genetically induced lesions of the brain and those that are created by direct damage to the brain, and both are associated with deficits in central auditory processing, learning, and language, among others (see Szalkowski & Fitch, 2011). In this chapter, we focus on the effects of neurological abnormalities on the performance of children on various behavioral and electrophysiological central auditory tests and procedures. We begin by examining the nature of CAPD.

Nature of CAPD

CAPD results from deficits in the perceptual processing of auditory stimuli in the CANS. These neurobiological deficits

manifest primarily (if not solely) in the auditory modality and are often observable as abnormal amplitude, latency, and/or morphology of one or more of the auditory evoked potentials (AEPs). Abnormal neurophysiologic representation of auditory stimuli underlying CAPD may include interhemispheric transfer deficits, which may be due to a developmental delay in myelination, lack of appropriate hemispheric lateralization, atypical hemispheric asymmetries, imprecise synchrony of neural firing, and decreased central inhibition (Jerger, Thibodeau, Martin, et al., 2002; Kraus, McGee, Carrell, Zecker, Nicol, & Koch, 1996; Moncrieff, Jerger, Wambacq, Greenwald, & Black, 2004; Musiek et al., 1994). These underlying deficits result in difficulties in one or more auditory processes, including sound localization and lateralization, auditory discrimination, auditory pattern recognition, temporal aspects of audition (e.g., temporal resolution [temporal gap detection], temporal integration, temporal ordering, and temporal masking), auditory performance in competing signals [including dichotic listening], and auditory performance with degraded acoustic signals) (ASHA, 2005). CAPD is diagnosed on the basis of performance on a battery of sensitive, efficient, and well-normed behavioral and electrophysiological measures of central auditory function (AAA, 2010). CAPD in children frequently is associated with (or may lead to) difficulties in listening, learning, oral and written language (including reading and spelling), and in other academic areas and social functions. These comorbidities arise from the complexities of brain organization and function in which a shared neurophysiologic substrate supports brain networks that are nonmodular, temporally coupled, and synchronized (Chermak, Bellis, & Musiek, 2007). Networks are integrated (versus segregated) and are organized to subserve process-specific (e.g., language) rather than domain-specific (e.g., auditory or visual) processing (Buschman & Miller, 2007; Price, Thierry, & Griffiths, 2005).

The unfortunate increase in the number of children diagnosed with CAPD following traumatic brain injury due to accidents (e.g., contact sports, bicycle riding without helmets, motor vehicles) (Langlois, Rutland-Brown, & Wald, 2006) has led to a deeper appreciation for the neurological origins of CAPD in children (Musiek & Chermak, 2009). The authors undertook this review of the neurological basis of CAPD in children to underscore this relationship. Following a review of selected aspects of neuroanatomy, we provide a summary of key published reports documenting central auditory processing deficits in children stemming from neuroanatomical abnormalities, genetic abnormalities, congenital brain lesions and perinatal events, brain trauma, mass lesions, epilepsy, cerebrovascular disease, and neurodegenerative disorders. In some studies, the patients were diagnosed with CAPD. In other studies, central auditory deficits suggestive of CAPD were documented; however, a specific CAPD diagnosis was not made. Since a number of etiologies and precipitating events impact multiple areas of the brain (e.g., mass lesions, neurodegenerative disorders), sections of the review are organized by anatomical site and others are organized by etiology. Moreover, given the impact of these precipitating events across multiple brain regions, it is likely the central auditory deficits documented in the studies reviewed below coexist with deficits in other systems, including language, attention , and learn-

ing. It is not the authors' intent in this review to differentiate comorbid deficits or potential confounds (which would be of greatest potential impact on behavioral tests) resulting from insults that extended beyond the central auditory system. Following the literature review, the authors discuss implications for clinical practice and future research.

[redacted]lected Aspects of [redacted]tional Neuroanatomy

In analyzing the effects of various neurological disorders on central auditory tests in children, it is critical to understand the anatomy of the CANS. Though the functional anatomy of the CANS evolves across the life span, there is well-established anatomical information regarding the locus of brain areas actively involved in auditory processing. Knowledge of these areas is critical to correlating the site or sites of the anatomical lesion to performance on central auditory tests. If auditory areas of the brain are damaged, one should expect that this dysfunction is reflected in a patient's performance on central auditory tests with documented sensitivity to central auditory system function. If in fact the central auditory system is compromised, the degree to which this deviancy is reflected by a given test is a direct measure of the test's sensitivity. Obviously other factors can enter into this correlation between test result and anatomical site of lesion. Brain plasticity, patient age, duration since the lesion occurred, size and precise location of the lesion, and other factors can all influence the relationship between test results and neurology.

Key Neuroanatomical Factors Influencing Central Auditory Tests and Electrophysiological Procedures

Key functional anatomy is discussed relative to central auditory behavioral tests and electrophysiologic procedures. Most behavioral central tests are affected by brainstem compromise. If the site of involvement is the cochlear nuclei and/or the mid- to lower pons, the performance deficit generally will be ipsilateral to the site or bilateral (Jerger & Jerger, 1974). Involvement of the upper pons and more rostral areas often yield a performance deficit in the ear contralateral to the lesion. Cortical lesions demonstrate a contralateral effect on central auditory behavioral tests unless the corpus callosum is involved (Jerger & Jerger, 1974; Musiek, Kibbe, & Baran, 1984).

The corpus callosum, the largest commissure in the brain, is responsible for transfer of information from one hemisphere to the other. The corpus callosum is anatomically segmented, with the posterior 1/5 of the callosum—the splenium—composed of visual fibers mostly from the occipital lobe. Anterior to the splenium is the isthmus (sometimes called the sulcus), which is a thinned segment considered the auditory segment of the corpus callosum. Anterior to the isthmus is the trunk or body where fibers from the parietal lobe (motor and somatosensory) and frontal lobe cross. The genu, perhaps the largest and most anterior region of the corpus callosum, is the site where fibers from the frontal lobe and olfactory system cross. The corpus callosum is most significant, because as studies of split brain patients have shown, severe left ear deficits are seen for verbal report on dichotic listening

tests and auditory pattern perception is reduced bilaterally when the corpus callosum is sectioned in split brain patients. However, central auditory tests that do not require interhemispheric interaction, such as monaural low-redundancy speech tests, show essentially no effect of commissurotomy (Musiek & Baran, 2007). Central auditory findings in a 14-year-old boy with a temporal-parietal tumor involving callosal and cortical fibers are presented in Figure 4–1.

In cases of corpus callosum involvement, a left ear deficit is observed when a verbal response is required from the patient because transcallosal transfer from the right to the left hemisphere is required for a verbal response in most people (Jerger & Jerger, 1974). Two types of tests, however, may not follow this performance pattern: masking level differences (a measure of binaural interaction mediated in the brainstem) and auditory pattern perception. Typically,

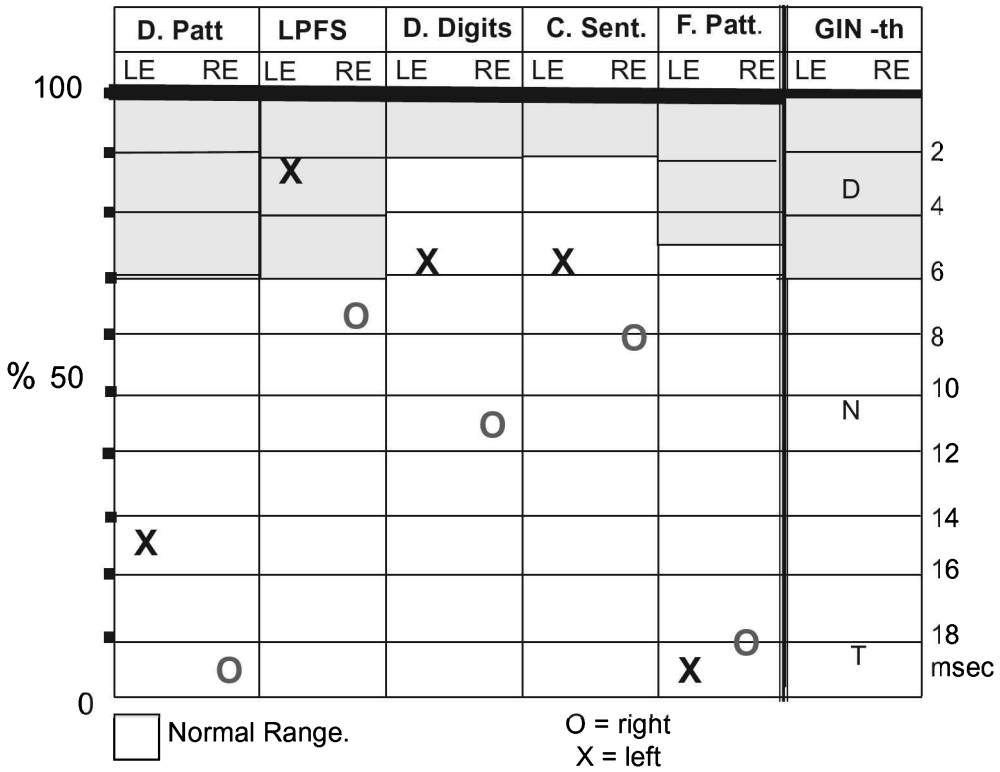

Figure 4–1. Fourteen-year-old patient who had a temporal-parietal tumor for which he was being followed neurologically. The lesion affected the superior temporal gyrus and the inferior parietal lobe superior to the sylvian fissure in the left hemisphere. Both the white matter (callossal fibers) and gray matter were compromised. Note that many of the central tests showed bilateral deficits. These findings were likely related to callosum involvement that caused left ear deficits and left hemisphere cortex involvement which resulted in right ear deficits. This is an example of bilateral involvement from one lesion involving callosal and cortical fibers. Peripheral hearing was within the normal range bilaterally.

abnormal masking level differences are observed bilaterally in the presence of brainstem involvement because this test requires an interaction between the two ears; hence, laterality effects typically are not observed. For auditory pattern perception, in most instances, but certainly not all, bilateral deficits are noted if either hemisphere or the corpus callosum is involved. This is likely related to each hemisphere coding an aspect of the pattern, with these aspects summed by the interaction of the hemispheres with the corpus callosum, yielding optimum perception. It is theorized that the right hemisphere codes the acoustic contour of the pattern and the pitches, and the left hemisphere sequences and linguistically labels the stimulus (Musiek, Pinheiro, & Wilson, 1980).

There also are several key anatomical factors underlying AEP outcomes. Abnormal auditory brainstem responses (ABRs) usually are seen ipsilateral (to the stimulated ear) or bilaterally when the brainstem is compromised. This is perhaps, in part, related to the fact that the generator sites of the first five waves do not extend rostrally beyond the upper pons (lateral lemniscus); hence, many fibers involved in a laterally positioned lesion are ipsilateral (Moller, 2000). This is likely not the only reason there are very few contralateral findings on ABR; however, it is certainly a factor. Other AEPs have more cortically located generators, such as the middle and late potentials (N1, P2), and are influenced by lesions in these regions. If multiple electrodes are used and distributed over each hemisphere, the electrodes closest to the lesion site will yield waveforms that are compromised, which is referred to as the "electrode effect" (Musiek & Lee, 1999). At times, the deficit noted in patients with cortical lesions will not necessarily align with a particular electrode site but with a particular ear. That is, the abnormality is seen in the evoked potential upon stimulation of a particular ear—hence the term "ear effect." Ear effects are not as site specific as electrode effects and need to be interpreted with caution (Musiek, Charette, Kelly, Lee, & Musiek, 1999; Musiek & Lee, 1999)

Mass Lesions, Degenerative and Vascular Disorders, and Disorders Related to Surgical Interventions

The Brainstem

Mass lesions, degenerative disorders, vascular disorders, and disorders related to surgical interventions involving auditory regions of the brain may result in compromised central auditory function. It is logical to begin this discussion at the brainstem level and proceed rostrally.

One of the most sensitive audiological tests for brainstem involvement is the ABR. Abnormal central conduction time measurements (I–III, III–V, I–V interwave intervals) indicate central auditory dysfunction, although an abnormal I–III measure also may result from an auditory nerve disorder. Rotteveel and colleagues demonstrated abnormal ABRs based on extended central conduction times in 11 of 14 children with tumors of the brainstem (mean age 9.5 years) (Rotteveel, Colon, Hombergen, Stoelinga, & Lippens, 1985). Davis and colleagues reported similar ABR results (5/6 abnormal) in a slightly smaller pediatric population with

tumors of the brainstem (Davis, Aminoff, & Berg, 1985). Goldie et al. reported that 21 of 31 children (ages 1–16 years) with brainstem tumors presented abnormal ABRs, a slightly lower *hit rate* compared with the previous studies (Goldie, van Eyes, & Baram, 1987). However, in the Goldie et al. study, only two ABR indices were employed (I–V interwave interval and a V–I amplitude ratio). Kalamanchey and colleagues reported on 14 childhood cases of brainstem involvement (Kalmanchey, Skripeczky, Miriszlai, & Schuler, 1991). Although this research group did not report statistics on the sensitivity of the ABR, they too presented waveform findings that documented central conduction time abnormalities. In a review article on ABR and posterior fossa (brainstem) tumors, Kalamanchey (1997) noted that it is well accepted that ABR is highly sensitive to posterior fossa brainstem tumors in children and that the ABR is of value to monitor change in the course of brainstem tumors and degenerative disorders of the brainstem.

Electrophysiologic tests are not the only type of assessment tool that is sensitive to childhood brainstem disorders. Behavioral central auditory tests also have been shown to be sensitive to this pediatric population. In one study, all five children (6–8 years of age) with auditory brainstem lesions showed abnormal findings on the Pediatric Speech Intelligibility (PSI) test (Jerger, Johnson, & Loiselle, 1988). In a case report of a 12-year-old who suffered a hemorrhage of the right inferior colliculus secondary to trauma, three tests of dichotic listening all were abnormal and revealed the classic contralateral ear deficit, despite normal hearing sensitivity bilaterally. An auditory pattern perception test also was abnormal in the contralateral ear. Tests of localization yielded errors only in the contralateral hemifield. These findings are consistent with damage to one inferior colliculus (Champoux, Paiement, Mercier, Lepore, Lassonde, & Gagné, 2007).

A number of neurodegenerative diseases can affect central auditory function as measured at the brainstem. These diseases, in general, damage myelin and/or synaptic transmission along the brainstem pathway. Davis et al. (1985) reported ABR results from 17 children (1–18 years) with various degenerative diseases including Leigh and Alexander disease, leukodystrophy, Friedreich ataxia, galactosemia, and others. Fourteen of the 17 pediatric patients presented abnormal ABRs. Interestingly, nine of the 14 patients with deviant ABRs demonstrated an absent wave V in at least one ear. Finding an absent wave V in the presence of at least one of the earlier waves is considered a powerful indicator of brainstem involvement (Musiek, Shinn, & Jirsa, 2007). It is important to note that the ABR results in children with brainstem involvement are highly similar to the results reported in adults with brainstem involvement. The sensitivity of the ABR procedure and the findings of increased central conduction times and severely attenuated or absent late waves seen in children with brainstem involvement mirror that seen in adults with brainstem involvement.

The Cerebrum

AEPs utilizing the N1 and P2 responses have been shown to be compromised in disorders that affect the auditory areas of the cerebrum (Musiek & Lee, 1999). Due to the effects of sleep, long maturational course, and other factors on AEPs, these

late potentials have not been utilized as frequently as the ABR in children. Fifteen children with vascular lesions of the left hemisphere (ages 8–18 years) were tested using the N1, P2 responses for both tonal and speech stimuli. Although sensitivity of the AEP was not computed, both the tonal and speech procedures yielded AEPs that were significantly delayed for the group with lesions compared with a control group (Papanicolaou, DiScenna, Gillespie, & Aram, 1990). Though this study's main aim was to look at language engagement for the left hemisphere, differential diagnostic information was obvious when comparing the two pediatric groups.

Vascular lesions in children have been evaluated using behavioral central auditory tests. Dichotic listening was employed by Bulgheroni and associates to evaluate 16 children (ages 8–14 years) who suffered congenital vascular disorders (Bulgheroni, Nichelli, Erbetta, Bagnasco, & Riva, 2004). The subjects were divided into left (n = 10) and right (n = 6) hemisphere lesions. A strong right ear advantage was seen in five of six children with right hemisphere lesions, consistent with the well-known lesion effect. In those with left hemisphere lesions, four revealed a right ear and five presented a left ear advantage. The right ear advantage is not consistent with the classic lesion effect; however, this finding could have resulted from undetected corpus callosum involvement, as discussed earlier. Jerger et al. (1988) reported on five children with cortical/thalamic involvement (see description above of these cases under *Brainstem*). All five children presented abnormal performance on the PSI competing message test. The children with left hemispheric lesions yielded bilateral ear deficits, which again may be attributable to corpus callosum involvement, as highlighted in a follow-up paper (Jerger & Zeller, 1989). It is important to note that in the Jerger et al. study, a group of children with suspected CAPD related to learning problems performed similarly to the children with neurologically confirmed problems. Similarly, CAPD and abnormalities of the interhemispheric pathway also were reported along with working memory deficits and congenital aniridia in a child with PAX 6 mutation, as discussed below under *Genetics* (Bamiou et al., 2007). Also discussed below under *Traumatic Brain Injury*, corpus callosum lesions have been reported in 20% of children sustaining moderate to severe (Mendelsohn, Levin, Harward, & Bruce, 1992).

Congenital and Early Infancy Events

Several studies have examined central auditory processing in children with hemiplegia, including children with spastic cerebral palsy/hemiplegic cerebral palsy, due to prenatal and perinatal incidents, including cerebral hemorrhage and lack of oxygen, as well as febrile illness during infancy. Most studies report a left ear advantage for dichotic digits, words, or syllables in children with congenital left hemisphere lesions and a right ear advantage for children with congenital right hemisphere lesions, consistent with the classic lesion effect (Brizzolara, Pecini, Brovedani, Ferretti, Cipriani, & Cioni, 2002; Isaacs, Christie, Vargha-Khadem, & Mishkin, 1996). Hugdahl and Carlsson (1994) examined directed dichotic listening in children with left and right hemiplegia as documented by computer tomography (CT) scans showing left or right hemisphere anomalies, with

no signs of epilepsy and normal hearing sensitivity. The left hemiplegia group (n = 18) showed a consistent left ear advantage, and the right hemiplegia group (n = 13) showed a consistent right ear advantage for consonant-vowel nonsense syllables, independent of attentional instruction. In contrast, normal controls (n = 19) showed the expected right ear advantage during the forced-right attention instruction and a small left-ear advantage during the forced-left instruction. Hugdahl and Carlsson (1994) concluded that lateralized damage to the motor system also alters perceptual performance and that dichotic listening is related to temporal lobe function in both cerebral hemispheres. Brizzolara et al. (2002) investigated cerebral lateralization using dichotic words in 26 children with congenital hemiplegia (lesion occurring before the end of the neonatal period) with normal hearing sensitivity, absence of mental retardation, and absence of treatment-resistant epilepsy (in the few cases who had presented seizures, the seizures were controlled by therapy). The children's lesions were documented with magnetic resonance imaging (MRI), which is more sensitive to and provides greater spatial resolution of brain lesions than CT. Although their findings were consistent with those already summarized, they noted that language reorganized in the right hemisphere only when the left lesions involved cortical-subcortical regions impinging on the left temporal lobe.

Traumatic Brain Injury

Even minor head injuries can cause sensory deficits, often impacting both the peripheral and central auditory systems (Hall, Huangfu, Gennarelli, Dolinskas, Olson, & Berry, 1983). CAPD has been reported in more than 50% of pediatric patients with traumatic brain injury (Flood, Dumas, & Haley, 2005). Traumatic brain injury (TBI), like other disorders that affect the CANS, such as strokes, tumors, and degenerative and developmental disorders, compromises the higher auditory system (Meyers, Roberts, Bayless, Volkert, & Evitts, 2002). Despite a unique onset and evolution of problems, TBI bears many similarities to other CANS disorders (Musiek & Chermak, 2009).

Corpus callosal tissue often is involved in TBI due to rapid, nonsynchronized acceleration of the hemispheres, which results in torquing and shearing of corpus callosal fiber, which are tightly attached to each of the hemispheres (Wilde et al., 2006). Corpus callosum lesions have been reported in 20% of children sustaining moderate to severe TBI (Mendelsohn et al., 1992). Benavidez et al. (1999) correlated MRI area measurements in 51 right-handed children with TBI and compared the TBI group's dichotic listening performance (and a number of motor, tactile, and visual functions) with that of 16 right-handed control children. They reported that posterior regions of the corpus callosum, the area of the corpus callosum known to mediate interhemispheric processing of auditory and language events, were more vulnerable to TBI, possibly due to diffuse axonal injury. Consistent with findings in adults, they found an increased right ear advantage (i.e., evidence of lateralized left hemisphere efficiency relative to the right hemisphere) for consonant-vowel nonsense syllables that was associated with callosal atrophy. Since there was no increased laterality asymmetry for motor and tactile tests, Benavidez et al. (1999)

concluded that the increased lateralization in the verbal dichotic measure was not due to global cognitive deficit.

The ABR has long been used in the neurophysiological examination of children with severe head injury (Chiappa & Ropper, 1982). The ABR obtained in comatose children with TBI presents strong predictive value regarding long-term outcomes (Blancafort, Marco, Poch Puig, Garcia, Nogues, & Berenguer, 1995). Hattiangadi, Pillion, Blomine, Christensen, Trovato, and Speedie (2005) described a 12-year-old child with auditory agnosia following a nonpenetrating TBI. MRI performed 10 days post-injury revealed edema, primarily frontal, subcortical, and commissural. ABR measurements were normal, indicating normal structure and function of the auditory pathways at the brainstem; however, the middle latency response (MLR) was absent for stimulation of both ears, suggesting bilateral lesions of auditory radiations and/or bilateral lesions in the auditory cortex (Scherg & von Cramon, 1986). Behavioral central auditory tests revealed essentially normal sound localization; however, dichotic digit performance was depressed bilaterally, with a significant left ear advantage, as well as frequency discrimination deficits, poor gap detection, and deficits in duration pattern recognition for both hummed and labeled conditions. Frequency discrimination deficits precluded administration of a frequency patterns test.

Epilepsy/Landau-Kleffner Syndrome

Given the possibility of sensory and cognitive deterioration due to seizure activity and associated brain damage, behavioral and electrophysiological measures of the central auditory system have been obtained in children with epilepsy. Studies generally confirm cerebral dysfunction secondary to epileptic activity. Turkdogan, Us, and Akyuz (2003) recorded AEPs in 50 epileptic children, 32 with MRI abnormalities and 18 with normal MRI, and 21 normal children. N2 and P3 latencies were significantly longer in children with epilepsy relative to the control children. No significant differences in N2 and P3 mean latencies or P3 mean amplitudes were seen between epileptic children with structural abnormalities and those with normal MRIs.

Boatman et al. (2008) administered a comprehensive battery of behavioral central auditory tests, as well as ABR and cortical AEPs to 14 right-handed children aged 7.5 to 11 years with normal hearing sensitivity: seven normal controls and seven with benign rolandic epilepsy, a common form of childhood epilepsy that accounts for 14 to 20% of cases (Bouman, Bovenkerk, Westendorp, & Brouwer, 1997). Seizures in benign rolandic epilepsy typically begin between 3 and 12 years of age and resolve spontaneously by age 15 to 18 years (Holmes, 1993). Although these children typically perform normally on global measures of language and cognition, they tend to experience difficulties processing spoken language in the presence of background noise despite normal hearing sensitivity (Staden, Isaacs, Boyd, Brandl, & Neville, 1998). All seven children with epilepsy in the Boatman et al. (2008) study demonstrated age-appropriate auditory continuous attention (i.e., vigilance) deficits on at least one test of central auditory function (speech recognition in noise). The finding of central auditory dysfunction in the presence of age-appropriate

attention is consistent with a growing literature that recognizes that higher order cognitive abilities can impact central auditory performance; however, cognitive deficits do not cause CAPD (Bellis et al., in press; Weihing, Bellis, Chermak, & Musiek, in press). Five (71%) of the children with epilepsy exhibited deficits on two or more tests (including dichotic listening and filtered speech recognition). A statistically significant difference between the two groups was seen only for speech recognition in noise. Only one child with epilepsy demonstrated an age-appropriate right ear advantage for dichotic listening. Six children with epilepsy presented either an atypically large right ear advantage or an atypical left ear advantage. No difference between groups was seen for the ABR, indicating normal auditory transmission up to the level of the inferior colliculus. No significant differences were seen between groups for N100 response latencies or amplitudes for tones or speech, or for MMN (mismatch negativity) response latencies or amplitudes for tones. The MMN was present for all normal controls; however, MMN responses for speech were prolonged in four children with epilepsy and absent in the three other children with epilepsy, precluding statistical comparisons. Those with no MMN to speech presented the poorest speech recognition in noise performance. Although the neural generators of the N100 response include the primary auditory cortex (Scherg & von Cramon, 1986), nonprimary auditory areas located on the lateral superior temporal gyrus are the neural generators of the MMN (Naatanen, 1990).

Landau-Kleffner syndrome (LKS) is a rare, childhood neurological disorder characterized by the sudden or gradual development of aphasia and an abnormal electroencephalogram (EEG). Typically, children with LKS develop normally until about ages 3 to 7 years, at which time, for no apparent reason, they lose their language skills, especially auditory comprehension, and present auditory agnosia for environmental sounds. Clinical seizures do not occur in all children; however, EEG recordings reveal paroxysmal unilateral or bilateral spike-and-wave discharges, predominantly over the temporal regions (Wioland, Rudolf, & Metz-Lutz, 2001). The language disorder is thought to result from the epileptic discharges in critical language areas of the temporal lobe. The prognosis is varied: Some children experience a permanent, severe language disorder, whereas others may regain much of their language abilities over months or years. Seizures generally disappear some time between age 12 years and adulthood (Paquier, Van Dongen, & Loonen, 1992). Most children present a permanent one-ear extinction on dichotic listening tests contralateral to the temporal cortex that had been the site of the epileptic focus (Metz-Lutz et al., 1997; Plaza, Rigoard, Chevrie-Muller, Cohen, & Picard, 2001; Wioland et al., 2001).

To explore the hypothesis of functional hemispheric reorganization following epilepsy with aphasia, Metz-Lutz et al. (1997) conducted a long-term follow-up study of four children with LKS using dichotic listening tasks. The four children were tested after they had completely recovered from epilepsy. Despite the absence of seizures, these children's language performance continued to fall below age-appropriate norms. Consistent with the contralateral effect seen in adults with brain lesions, all four children presented dichotic extinction contralateral to the side where spike-and-wave

discharges predominated during the active phase of the epilepsy. This finding suggests a permanent unilateral dysfunction in the temporal cortex, a conclusion consistent with positron emission tomography (PET) scan studies conducted during the late recovery phase of the LKS, which showed a unilateral hypometabolism in the temporal cortex, which had been the site of hypermetabolic activity during the active phase of the LKS. Wioland et al. (2001) corroborated the linkage between hypometabolism and unilateral dichotic extinction, reporting a unilateral voltage reduction of the late AEPs (particularly N1c) over the temporal areas that had previously been the site of epileptic discharges in five LKS patients who had completely recovered from epileptic activity for several years. The reduction in N1c (thought to originate in associative cortexes) was seen contralateral to dichotic ear extinction, which persisted at the time of the AEP recordings. By contrast, early (brainstem) and middle latency AEPs were normal.

Genetics

Most developmental disorders result from a complex combination of genetic susceptibilities and environmental risk factors. Dyslexia and specific language impairment are known to have a significant genetic component (Bishop, 2009). Shared genetic influences may underlie comorbidity (i.e., pleiotropy, where a gene or combination of genes influence more than one behavioral phenotype) (Willcutt, Pennington, Olson, & DeFries, 2007). Many genes have been identified as "generalist" genes that have broad effects on the brain and development (Kovas & Plomin, 2006). For example, FOXP2 is a transcription factor important for modulating the plasticity of neural circuits in the developing brain (Groszer et al., 2008). Genes affecting central auditory processing might have pleiotropic effects on other behaviors and vice versa.

Although genetic studies of CAPD are in early stages, an accumulating literature is documenting the genetic basis of specific auditory processes (e.g., dichotic listening) (Morrell et al., 2007), as well as the genetic basis of CAPD in children (Addis et al., 2002; Bamiou et al., 2004, 2007). Morell et al. (2007) reported that 73% of the variation in dichotic listening is due to genetic differences—a magnitude comparable to well-known inherited traits such as type 1 diabetes and height. Peretz, Cummings, and Dube (2007) confirmed an hereditary component in nine large families presenting congenital amusia or tone deafness.

Perhaps the most comprehensive report detailing CAPD in the presence of a genetic mutation is that of Bamiou et al. (2007), who documented central auditory processing deficits indicative of reduced auditory interhemispheric transfer in 11 children with PAX6 mutations and MR images documenting abnormalities of the interhemispheric pathway. PAX6 mutations are characterized by developmental aniridia and abnormalities of brain structures responsible for interhemispheric information transfer, including an absent or hypoplastic anterior commissure and a smaller corpus callosum (Pandya & Seltzer, 1986; Sisodiya et al., 2001).

All children (both the 11 with PAX6 mutations and the 11 matched healthy controls) in the Bamiou et al. (2007) report presented normal hearing sensitivity, tympanograms, and transient-evoked otoacoustic emissions. Ten of 11 children with PAX6 mutations presented

deficits on central auditory tests: seven presented abnormal results in the left ear for dichotic digits; eight presented abnormal results for frequency patterns; 10 presented abnormal findings for duration patterns; all 11 presented normal gaps-in-noise performance. Compared with controls, children with PAX6 mutations exhibited a smaller anterior commissure ($n = 7$) and a smaller corpus callosum ($n = 4$). Overall, the MR images were completely normal in only one of the PAX6 children. Brain volumetry to measure the corpus callosum in nine of the children with PAX6 mutation revealed significantly smaller area of the corpus callosum compared with nine randomly chosen age-matched healthy controls. The MRI findings were generally consistent with those seen in adults with PAX6 mutations (notwithstanding the presence, albeit smaller, of anterior commissure in the children and the more often reported finding of complete absence of the anterior commissure in adults—a difference that may reflect age-related degeneration of interhemispheric structures). The behavioral test results also are consistent with the major findings in adults with PAX6 mutations and with performance of patients with sectioned corpus callosum and anterior commissure in whom severely decreased left ear performance is seen for dichotic listening, bilaterally reduced performance for duration and frequency patterns, and normal gaps-in-noise performance. Not surprisingly the children with PAX6 mutations exhibited difficulties on central auditory tests that depend on interhemispheric transfer of information (i.e., dichotics and pattern tests), but not on the gaps-in-noise measure, which does not rely on interhemispheric transfer, thereby demonstrating the anatomical basis (corpus callosum) of the PAX6 mutation. Parental responses to a questionnaire indicated children with PAX6 mutations experienced more difficulty localizing sound and understanding speech in noise. These reported difficulties can be explained by interhemispheric transfer deficiencies (Lessard et al., 2002; Sisodiya et al., 2001). Bamiou et al.'s work demonstrating the genetic basis of anatomical abnormalities that present as central auditory deficits corroborates the neurobiological basis of CAPD.

Based on linkage analysis, Addis et al. (2002) postulated that a mutation of a single gene on chromosome 12 underlies the auditory processing difficulties and language impairments observed in three generations of a family. All affected members displayed abnormal MMN for syllable duration discrimination compared with gender- and age-matched controls, with the two 5- to 6-year-old affected children showing no MMN. Symptoms of CAPD also have been documented in X-linked adrenoleukodystrophy (X-ALD), a rare metabolic disorder characterized by demyelination. Difficulties understanding speech in noise despite normal hearing sensitivity and academic difficulties are the most frequently reported initial symptoms (Moser, Smith, Watkins, Powers, & Moser, 2001).

Discussion and Implications

This review of the neurological causes of CAPD, which we focused almost exclusively on children to emphasize the neurobiological origins of CAPD in children, raised several interesting issues and presented a number of important implications. Clearly demonstrated is the effi-

ciency of central auditory behavioral tests and electrophysiological procedures in evaluating pediatric patients with known or suspected neurological involvement. The types of central auditory deficits experienced by children with neurological involvement (e.g., speech recognition difficulties in everyday listening environments) place them at risk for academic difficulties. Multidisciplinary evaluation is crucial given the complex clinical profiles and frequent comorbidities across this population. Comprehensive testing of auditory functions using behavioral and electrophysiological methods is important to identify children who can benefit from interventions, including assistive listening devices and environmental modifications to improve signal-to-noise levels in the classroom. The range of therapeutic approaches, including bottom-up auditory training and top-down language-based and cognitive-based therapies, may prove useful as well (Chermak & Musiek, 2007).

Historically, children have been the majority of individuals diagnosed with CAPD and provided treatment; however, CAPD was first diagnosed in adult patients with lesions of the CANS (Bocca, Calearo, & Cassinari, 1954). Most central auditory processing tests were developed to probe CANS dysfunction secondary to confirmed pathology, rather than to evaluate auditory processing in young children (Chermak et al., 2007). Our review demonstrates, however, that patterns of performance deficits seen in children with documented CANS lesions are comparable to those patterns seen in adult patients with documented CANS lesions. In fact, the deficit patterns in central auditory test battery performance in children with auditory-related complaints but with no identifiable lesions of the CANS and no apparent prenatal or perinatal disease, injury, or exposure-related explanation for their CAPD mirror those patterns seen in pediatric and adult populations with circumscribed disorders of the CANS, and these deficit patterns correlate with neuroimaging results (Boscariol et al., 2009, 2010, 2011; Jerger et al., 1988; Musiek, Gollegly, & Baran, 1984). Similarly, performance patterns are observed on central auditory tests during maturation and aging that reflect the time course of functional and structural age-related changes in the central nervous system, particularly the corpus callosum, in normal populations (Bellis & Wilber, 2001). These common patterns indicate that lesion studies of adult (or pediatric) patients can serve to approximate a gold standard for CAPD in children with no identifiable CANS lesion. If certain test patterns have been demonstrated to have good sensitivity and specificity in adults with confirmed CANS lesions (Bamiou et al., 2006; Blaettner, Scherg, & von Cramon, 1989; Musiek, Baran, & Pinheiro, 1990, 1992), then one may presume with a high degree of likelihood that the same pattern of test results, when observed in a child or an older adult undergoing testing for central auditory dysfunction, confirms CAPD in that child or older adult. As is the clinical standard in many disciplines (e.g., cognitive neuroscience, neuropsychology), it is appropriate to extrapolate from documented brain-behavior relationships in individuals with known CANS involvement to infer CANS dysfunction in pediatric and geriatric patients displaying similar performance patterns on similar measures (Kolb & Whishaw, 2008; Musiek, Bellis, & Chermak, 2005).

Our review provides compelling evidence to counter the argument asserted

by some that damage to the brain in adults may not be the same as a "developmental disorder" of the CANS in children. Clearly, tests standardized on individuals with documented brain lesions—children and adults—provide important information on functional auditory abilities in affected persons of all ages and implicate auditory brain regions despite the absence of documented neurological involvement. Although some might argue that an absolute gold standard is not possible given the heterogeneity of disorders affecting the CANS, it is clear that test efficiency measured on patients with well-defined lesions of the CANS provides an important guide for establishing the validity of central auditory diagnostic tests (Jerger & Jerger, 1974; Musiek et al., 2005). Recent studies have confirmed that involvement of auditory regions (e.g., perisylvian polymicrogyria, cystic lesions in the left sylvian fissure) in children leads to performance deficits and patterns on central auditory tests comparable to those of patients with documented CANS lesions (Boscariol, 2009, 2010, 2011; Grindle, O'Reilly, Morlet, & Finden, 2010), and there is considerable evidence that upon autopsy many individuals with CAPD exhibit neuroanatomical abnormalities in auditory areas of the central nervous system (ASHA, 2005). The Boscariol et al. reports (2009, 210, 2011) may offer the strongest evidence to date that structural changes in an area of the brain associated with auditory and language processing can lead to changes in auditory processing and, therefore, in language and learning as well. Whether the source of CANS dysfunction is benign or the result of neurological lesion or compromise, the underlying source of the resulting CAPD is neurobiological, originating in the central nervous system.

All regions of the CANS must be assessed in children referred for central auditory evaluation, including the brainstem ascending pathway, auditory regions of the subcortex, interhemispheric pathways, and the auditory cortex. In order to assess these anatomical regions, AEPs, electroacoustic procedures, and central auditory behavioral tests should be employed following a thorough audiological evaluation of the peripheral auditory system. Restricting assessment to only a selected anatomical region and/or not examining multiple central auditory processes compromises the efficiency (i.e., sensitivity and specificity) of the assessment and may fail to provide crucial information to support intervention. Valuable information is obtained from the case history as well, helping to uncover the potential etiological basis for the disorder, as well as the functional impact of the disorder on the individual's communicative, vocational, and/or academic performance. If multiple brain areas are involved as a result of a brain lesion, and one of the areas is auditory, as documented by performance deficits on a sensitive and efficient test battery, supplemented by neuroimaging studies as appropriate, then an auditory disorder is part of this child's deficit complex. The authors reject the assertion by some that CAPD can only be diagnosed in the absence of other modality deficits (Musiek et al., 2005). In these cases, the auditory disorder coexists with other brain dysfunctions, which may include visual and somatosensory problems. Recognizing the potential for multiple system involvement in children with neurological problems is key to rehabilitation and management. For example, a child with Waardenburg syndrome has many nonauditory as well as auditory problems;

to ignore the auditory component would be catastrophic. Because of the nature of neurological disorders, as well as developmental disorders, multiple areas of the brain often are involved, leading to comorbid diagnoses. Multidisciplinary assessment is necessary to accurately differentially diagnose and appropriately treat these disorders (Musiek & Chermak, 2007). Confirmation of a neurological etiology requires the collaborative efforts of physicians, audiologists, radiologists, and others. Understanding the status of the central nervous system is critical to planning and implementing effective intervention to minimize difficulties in listening, language, learning, reading, and in other academic and social areas.

Intervention for the neurologically involved child with CAPD also requires a multidisciplinary team approach, often involving audiologists, speech-language pathologists, psychologists, physical therapists, occupational therapists, educators, and families (who often are aware of important issues about which the patient may not be aware) to ensure a comprehensive approach to target the range of issues. Identifying CAPD in children with other disabilities (e.g., visual disability in children with PAX6 mutations) presents management implications, particularly in the classroom setting. Given the known interactions among sensory, cognitive, and language systems, intervention for CAPD with a neurological component can promote improved cognitive and social functioning in the home, school, and social settings. It is important to emphasize that CAPD varies in severity along a continuum—with some children demonstrating less extensive involvement of the CANS, as reflected in mild deficits on central auditory measures, as might be seen in those with learning problems, to more significant CANS involvement, as seen for example in the case of a stroke, as revealed in more profound deficits on central auditory measures secondary to extensive damage to the auditory cortex. Clearly, the probability of treatment success may be inversely related to the patient's degree of deficit, particularly in the neurologically involved patient (Musiek & Chermak, 2008).

One cautionary note must be offered regarding the limitations of the traditional method of inferring brain function from examination of patients with brain lesions. Inferring function from lesion studies is limited by the absence of discrete anatomical modules underlying a particular brain function (Musiek et al., 2005). Moreover, even if modularity controlled auditory processing, brain damage is not typically limited to the boundaries presumed to underlie the functional modules (Heinslus, Bogousslavsky, & Van Melle, 1998). Furthermore, significant anatomical differences across individuals, as well as the plasticity of regions changing function in response to damage in other regions of the brain (Raineteau & Schwab, 2001) pose limitations to the lesion method of inferring brain function. Electrophysiological methods (e.g., evoked potentials, magnetoencephalography) and brain imaging methods for studying brain function (e.g., fMRI, PET, single-photon emission computed tomography [SPECT], and diffusion tensor imaging [DTI]) have already provided new insights into brain function, although they too present limitations. For example, evoked potentials and magnetoencephalography present poor spatial resolution, and fMRI, PET, SPECT, and DTI present poor temporal resolution. Clearly, the use of both tools provides the complementary strengths to overcome

their respective weaknesses (Rorden & Karnath, 2004).

Finally, it is important to emphasize that audiologists' primary purpose in evaluating children with learning problems and associated auditory symptoms is to determine if they have an auditory disorder. Although audiologists are concerned about learning, language, attention, and other coexisting problems, our charge is to evaluate central auditory function (AAA, 2010). Other members of the professional team are responsible for evaluation of these other comorbid deficits. Central auditory test findings in individuals with documented disorders of the CANS provide a valid model upon which to rely in interpreting central auditory test findings in children.

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CHAPTER 5

AN INTRODUCTION TO CENTRAL AUDITORY NEUROSCIENCE

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Introduction

All of the conscious and unconscious operations executed on any auditory sensation we have probably involve activity in the central auditory nervous system (CANS). The list of those operations—which might include generation of the perceptual event, resolving it in spectrum and time and space, differentiating it from other auditory sensations, recognition and identification of the event, and so on—can appear daunting in itself. So too can the plethora of empirical articles on the central auditory underpinnings of those functions, and the theoretical frameworks in which those articles are cast. Nevertheless, if we are ever to have a penetrating comprehension of central auditory processing, some attempt must be made to examine the relation between the subjective experience of hearing and the neural machinery that mediates it—and both of those in the context of a firm understanding of the stimulus. Fortunately, we are becoming increasingly aware that the anatomy and physiology of the central auditory system follows specifiable principles or rules, as does human auditory perception. In at least some instances, it has become possible to show that there is an orderly mapping of auditory function visualized in the neurophysiology with that seen psychophysically.

This chapter provides an overview of central auditory neuroscience, from auditory nerve to auditory cortex, and points to some psychophysical corollar-

ies of central auditory function as they emerge. The chapter is composed with two goals in mind. One is to provide the reader with a brief, stand-alone summary of auditory neuroscience and its perceptual correlates, and enough referencing that the reader can leave the chapter with at least a qualitative comprehension of principles of auditory neuroscience and a means of tracking it further. The second goal is to provide a foundation for the rest of this book. Ultimately, a knowledge of central auditory neuroscience must embrace normal function, dysfunction, development, and aging. To that end, principles of central auditory neuroscience need to be described in a way that enables comprehension of the level of the system and the fashion in which pathological and developmental processes operate to exert their effects.

These are tall orders, and the best that a single chapter can do is provide an outline. Many of the references cited below are review articles or recent empirical ones that address important conceptual issues. What follows begins with an account of the auditory nerve, because it is the physiology and connectivity of the auditory nerve that sets some of the resolution and organization of information available to the brain. We then work our way through the cochlear nuclei, the superior olivary complex, the auditory midbrain, and then the thalamus and cortex. At each of those stations, we will explore identifiable neural circuits and their functions. Having laid out the afferent auditory system in this serial, anatomical way, we will then take a functional approach, first, by devoting some time to neuroplasticity, and second, by exploring temporal aspects of central auditory processing. Both of these are topics of intense current investigation in their own rights, and both have been implicated in the genesis of central auditory processing disorder (CAPD) or the remediation of it.

The Auditory Nerve

The afferent auditory nerve is made up of spiral ganglion neurons whose dendrites contact the inner hair cells of the cochlea and whose axons depart the cochlea through the internal auditory meatus, ultimately to contact cells of the cochlear nuclei. The cochlea can be construed as a linear array of filters, with those for high frequencies arising in the basal turn, and those for low frequencies being located in the apical turns. This tonotopic organization is a consequence of the strictly mechanical properties of the basilar membrane; in particular, its stiffness and width gradients from base to apex. The selectivity and sensitivity of the mechanical tuning at any given point is shaped by the passive mechanics of the basilar membrane at that site and by the active contribution of the outer hair cells (Dallos, 1992). The latter serve both to increase the sensitivity of the response and to enhance the frequency tuning (Ruggero & Rich, 1991). A rough metaphor might be that the outer hair cells serve as amplifiers, with the outer hair cell shortening and lengthening responses linked to upward and downward motions of the basilar membrane, respectively. It is in this way that the outer hair cells' contractile response adds to the passive mechanical response. The outer hair cells' mechanical response is driven by the outer hair cells' electrical one, which is in turn proportional to the basilar membrane's mechanical response. The outer

hair cell contribution is decidedly nonlinear. It is greatest for frequencies at or near the one to which that site is most sensitive, with the result that the active process adds to the sensitivity of the mechanical response in a frequency selective way.

Each spiral ganglion cell receives its input from a single inner hair cell, and the neurotransmitter is almost certainly glutamate (Pickles, 2008). There are no (neural) longitudinal interactions between cochlear nerve cells (Spoendlin, 1967), which consequently function independently, each driven entirely by its single input. In this sense, cochlear nerve cells "inherit" their properties from a unitary source. Their frequency tuning is slave to that of the inner hair cell they innervate, and therefore to that of the basilar membrane at that site (Narayan, Temchin, Recio, & Ruggero, 1998; Sellick, Patuzzi, & Johnstone, 1982). Each nerve cell is thus characterized by a characteristic frequency (CF) to which it is most sensitive and narrowly tuned. The auditory nerve, then, can be thought of as an array of fibers across which are mapped the frequency filters of the cochlea: Which frequencies are present in the stimulus will be expressed in which neurons of the array are activated. It is, of course, the independence of these frequency channels that permits frequency-specific hearing losses after local cochlear damage. It is due to the fact that cochlear pathology typically affects outer hair cells (and so the fine frequency tuning of the transduction process) that the consequence of outer hair cell damage is not merely a loss of sensitivity to sound, but also an impaired ability of the cochlea to execute a spectral decomposition of the sound: There is a "blurred" mapping of the sound energy through the impaired sector of the cochlea onto the auditory nerve array.

The spike discharge patterns of cochlear neurons are determined in large part by the physiology of the hair cell/afferent fiber synapse (Ruggero, 1992). For low frequency stimuli, spike discharges in cochlear neurons are phase-locked to upward motions of the basilar membrane. This is because it is upward motions of the membrane that depolarize the inner hair cell and thus elicit neurotransmitter release; downward motions of the basilar membrane hyperpolarize the hair cell and thus prevent neurotansmitter release. Phase-locking is strongest at very low frequencies but is statistically detectable in responses to frequencies as high as 2.5 to 3 kHz or so. Accordingly, in addition to the tonotopic ("place") code above, a second representation of sound frequency resides in the timing of action potentials in the nerve cell array activated by the sound: The intervals between spikes are informative about periodicities in the stimulus. Precisely how pitch (the subjective correlate of stimulus frequency) is recovered from interspike intervals by the brain is unclear, but there is little doubt that sounds that produce pitch percepts also evoke responses whose interspike intervals are directly related to the temporal stucture of the stimulus and thus also to the subjective pitch (Cariani & Delgutte, 1996). This is true even of sounds that contain no energy, or no special concentration of energy, at the pitch frequency; this is made possible, of course, by the fact that the time waveform of the stimulus has a periodicity at the pitch frequency.

At higher frequencies, the biophysical coupling of basilar membrane motion and inner hair cell neurotransmitter release is not synchronized on such a fine-grained moment-by-moment basis, and the result is a steady depolarization

of the hair cell for as long as the stimulus is present. It follows that for as long as the stimulus is present, there is continuous release of neurotransmitter by the hair cell and thus a continuous train of action potentials generated in the auditory nerve fiber. In practice, tone- or noise-burst stimuli evoke a highly time-locked onset response, which then adapts to a steady firing rate (Ruggero, 1992). The firing rate itself is determined by the relation between the spectral content of the stimulus and the nerve cell's frequency-intensity response ranges. Most cochlear nerve cells have monotonic, saturating spike rate-intensity functions, although they vary in the extent to which firing rates saturate at high stimulus levels. In general, then, one can conceptualize the activity of each cochlear nerve cell as indicating the presence, amplitude, and timing of stimulus energy within its narrow frequency passband.

Cochlear Nucleus

The cochlear nucleus has three broad nuclei: a ventral division (VCN), which is divisible into anteroventral (AVCN) and posteroventral (PVCN) nuclei, and a dorsal nucleus (DCN). Each axon of the auditory nerve bifurcates as it enters the cochlear nucleus, with one branch innervating AVCN, and the second branch innervating PVCN en route to DCN (Rhode, 1991). This constitutes an early expression of parallel processing, that is, a divergent projection upon multiple target nuclei that are then able to execute separate operations on the same input simultaneously. In each of these nuclei, the entering auditory nerve axons terminate in an orderly tonotopic fashion, so that a sheet of neurons comes to be innervated by each cochlear site in each of the three divisions of the nucleus. Thus, the AVCN, PVCN, and DCN contain relatively complete representations of the cochlear partition, and stimulus processing continues to be executed on a frequency-by-frequency basis.

Functional specialization within the cochlear nucleus has been studied extensively (see Rhode, 1991 and Rhode & Greenberg, 1992 for review). One striking specialization occurs in the AVCN. So-called spherical bushy cells of the AVCN are cells with relatively poorly developed dendritic trees and cell membranes that have a low input resistance. In the low frequency region of the AVCN, individual bushy cells are innervated directly by small numbers of auditory nerve axons via elaborate synapses termed "endbulbs of Held." The presynaptic terminal is large and wraps partially around the postsynaptic cell body. There are numerous synaptic contacts made via this specialization, with the result that the low input resistance of the postsynaptic cell is met by a large synaptic current. In turn, this means that the postsynaptic cell's behavior becomes dominated by a very small number of auditory nerve inputs (as few as 1–4; Rhode, 1991). In particular, the timing of postsynaptic spikes tends faithfully to reflect that of the auditory nerve input ("spike-in/spike-out" transmission). Low frequency cochlear output is often phase-locked, and this synaptic specialization preserves the temporal organization of the input and therefore information about the phase of the stimulus at the eardrum. In turn, the cells of the medial superior olive (MSO) derive their inputs bilaterally from spherical bushy cells of the VCN (Cant, 1991) and are thus able

to compare the timing of phase-locked spikes arriving from the two VCNs. This comparison is informative about the relative phases of the stimuli at the two ears, which is important in sound localization (see below). A second group of AVCN cells, the "globular bushy cells," also receive inputs from the auditory nerve via endbulb contacts; the globular bushy cells have large axons (and therefore high axonal conduction velocities) and project upon nuclei of the trapezoid body.

A quite different specialization is seen in some cells of the PVCN. "Octopus cells" (multipolar neurons) have large, relatively unbranched dendrites that appear to be oriented somewhat across the tonotopic array of input fibers, with the result that they receive input from relatively broad cochlear segments. Physiological studies show these neurons to have broader frequency tuning curves than other cells of the VCN, which is consistent with the anatomical organization of their inputs. The discharge patterns evoked by tone-burst stimuli in these neurons are often dominated by a very precisely timed onset transient; the jitter in (standard deviation of) first spike latency can be as low as a few tens of microseconds, which is again consistent with a highly convergent input (Rhode, 1991), because variability in first spike timing declines with increases in the number of inputs. The phase-locking of spikes by some of these neurons is among the most temporally precise of any cells in the central auditory system, including the auditory nerve. The axonal outputs of these cells are directed toward the periolivary nuclei and the contralateral ventral nucleus of the lateral lemniscus (Schwartz, 1992).

The output neurons of the DCN are fusiform cells. They receive input from the auditory nerve and from local inhibitory circuitry. They direct their outputs to the contralateral auditory midbrain, especially the inferior colliculus. DCN cells are excited by a narrow range of tone frequency-intensity conjunctions ("response area"), but they also are inhibited by tones falling in domains that flank the excitatory one. It is possible to categorize DCN cells according to the organization of these inhibitory response areas (Young & Brownell, 1976), and subsequent studies have offered evidence on which morphological cell types are characterized by various patterns of inhibitory inputs and on the organization of the connectivity between the cell types (Rhode & Greenberg, 1992). For the present purposes, there are at least two new physiological response properties that emerge from this circuitry. One is that because the inhibitory response areas flank the excitatory one at the cell's CF, the cell can develop a marked sensitivity to stimulus bandwidth. It is thus not simply the presence of stimulus energy within the excitatory response area, but also the distribution of energy across the excitatory and inhibitory response area(s) that determines discharge rates in these cells. A second feature to emerge from the inhibitory circuitry in at least some DCN cells is the presence of an inhibitory input at CF. Often the excitatory input at CF is the more sensitive, but the presence of both means that these cells have a nonmonotonic spike rate-intensity function for CF (and nearby) tones.

Neurons with nonmonotonic rate responses to tone pulses or other signals are increasingly common at more rostral sites in the auditory pathway, and the apparent "tuning" to stimulus level has been incorporated into accounts of the central representation of sound intensity. The responses of central neurons

also become increasingly dominated by sound onsets (Phillips, Hall, & Boehnke, 2002). Because responses to variations in stimulus level are typically studied without also covarying stimulus rise-time, it is important to determine independently whether the responses to differences in sound level are actually driven by the plateau level of the sound or by the dynamics of sound pressure change at sound onset (Heil, 1997). In practice, it is often the latter (Heil, 1997; Phillips, Hall, Guo, & Burkhard, 2001). For sustained responses, this issue may be moot because the sustained response enables a separation of the "code" for ongoing stimulus level from the response to the onset transient. Nevertheless, the striking dominance of onset responses seen rostrally, and the rethinking of exactly which stimulus parameter is encoded in the rate-response function, raises new questions about the contribution of forebrain and hindbrain mechanisms to the mediation of perceived loudness.

[redacted]perior Olivary Complex

The superior olivary complex (SOC) is a bilateral structure that contains a number of separable cell groups distinguished by their cytoarchitecture, connectivity, and function. For the purposes of this chapter, two cell groups are important: the medial (MSO) and lateral (LSO) superior olivary nuclei. Their particular significance derives from the fact that these nuclei are crucially involved in the neural encoding of the binaural cues for the spatial location of sounds. Both nuclei are tonotopically organized, and neurons of both nuclei ultimately derive their inputs from both ears. These two features of their inputs confer on the SOC the ability to execute interaural stimulus comparisons, and to do so on a frequency-by-frequency basis. The frequency specificity of the interaural disparity coding is important. Interaural time (ITD) and level (ILD) differences are the principal binaural cues used in sound localization. They derive from the travel time of sound around the head and the acoustic shadow cast by head and pinnae, respectively. The magnitudes of the cues vary with the size of the receiver's head, the azimuth of the sound source, and the spectral composition of the sound itself. For spectrally rich sources, each frequency component generates its own ITD and ILD, and the distribution of these provides a wealth of information about source azimuth—but this wealth is usable only if the nervous system encodes the interaural disparities at each frequency independently. It is because the nervous system does indeed encode interaural disparities in a frequency-specific way that localization performance is so much more acute for broadband sources than for very narrowband ones.

The MSO receives direct projections from the AVCN bilaterally (Cant, 1991). Interestingly, it is often the case that the particular AVCN cells that contribute to this innervation are the same ones that receive endbulb innervation from the auditory nerve (Oertel, Wu, & Hirsch, 1988) and that thus carry very faithful information about the phase of the stimulus at the ears. In turn, MSO cells are able to execute a temporal coincidence detection on the spike trains arriving from the two cochlear nuclei, and through that mechanism, encode the relative phases of sounds at the two ears. That is, the phase-locked spike trains evoked in

the cochlear nerve are conferred upon the bushy cell axons, and the recipient MSO cells can compare the timing of spikes from the two sides. What results is a neural sensitivity to interaural phase expressed as a cyclical relation of MSO cell spike count to interaural phase, with the period of the cycle being equal to the period of the sound at the two ears (Yin & Chan, 1990).

In principle, coincidence detection could be mediated using exclusively excitatory mechanisms. In practice, however, it seems likely that the neural encoding of ITDs also relies on glycine-mediated inhibition (Brand, Behrend, Marquardt, McAlpine, & Grothe, 2002). If one imagines the afferent spike trains from either AVCN as constituting phase-locked half-periods of excitation, then the inhibition expresses itself as half-periods of inhibition inserted between the excitatory half-periods. The "insertion" is probably achieved through inhibitory neuron satellites of the MSO, activated in parallel by the AVCN input. Variations in ITD will now cause shifts in the relative timing of excitation/inhibition from one side and excitation/inhibition from the other side and will do so on a cycle-by-cycle basis. The result is that the modulation of spike rate evoked by variations in ITD is deeper than could be achieved by excitatory mechanisms alone. The greater the modulation of spike rate by the stimulus manipulation, the more salient is the neural signal.

In practice, the neural path length from the contralateral ear is usually the longer, so that in order for the spikes from the two AVCNs to arrive synchronously at the MSO, the phase of the stimulus at the contralateral ear must precede that at the ipsilateral ear; that is, the interaural stimulus timing difference offsets the neural travel time difference from the two ears. This has the consequence that the relation of spike rate to ITD is not only cyclical; spike rates are greatest for ITDs favoring the contralateral ear, minimal for ITDs favoring the ipsilateral ear, and the functions are disposed such their steepest slopes are associated with very small ITDs (McAlpine, Jiang, & Palmer, 2001). It seems to be the case that the ITDs associated with peak responses are systematically longer for cells with lower CFs (which, of course, have longer periods). The result is that the steep portion of the spike count versus ITD function is usually associated with very small ITDs (McAlpine et al., 2001) and therefore with azimuths near the midsagittal plane. This point is illustrated in Figure 5–1, which shows idealized spike count versus ITD functions for cells of relatively low, medium, and high CFs. Note that spike count is a cyclical function of ITD for each of the neurons and that because peak spike rates are associated with longer ITDs in the lower-CF cells, the steep portion of the function is associated with relatively small ITDs. The absolute range of ITDs that an individual will encounter depends on head size, source location, and source frequency, but small ITDs are the ones most likely to occur naturally (shaded area in Figure 5–1). Now, it is the steep part of the function in which neural firing rate most unambiguously specifies ITD, and, through the azimuth-ITD relation, source location. This may be one reason why free-field sound localization acuity is greater for sources near the midline (Middlebrooks & Green, 1991; Phillips & Brugge, 1985).

ITD coding is usually studied at the neuron's CF, but the phenomena seen in responses to CF tones extend to those to off-CF frequencies. One fascinating

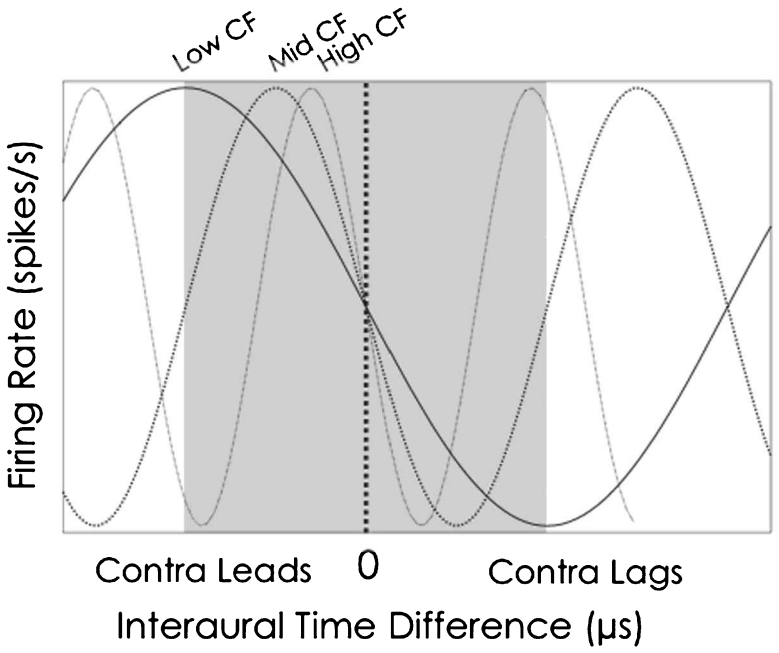

Figure 5–1. Idealized spike rate-vs.-ITD functions for neurons of relatively low, medium, and high CF. Shaded area indicates the range of ITDs associated with near-midline azimuths. Each of the three neurons shows a cyclical relation of spike count to ITD, which is expected of a coincidence-detector mechanism working on two phase-locked inputs. Note that for each neuron, spike rates are high for ITDs that favor the contralateral ear. This reflects the fact that the neural path length is longer from the contralateral ear than is the path length from the ipsilateral ear. Importantly, irrespective of CF, the steep part of the function is always centered at small ITDs.

concept to emerge from studies of ITD coding at different frequencies within a neuron's effective range is that of "characteristic delay" (see Yin & Kuwada, 1983 for studies of this at the level of the inferior colliculus). For many cells, the ITD functions obtained with different frequencies coincide at a single ITD. When the coincidence is at a peak in the delay functions, then the coincidence is at an ITD favoring the contralateral ear; when the characteristic delay occurs at the trough in the delay functions, the characteristic ITD favors the ipsilateral ear. The net result of this is that the ITDs evoking the most vigorous responses, and often also the most highly differentiated responses, are associated with contralateral azimuths.

A slightly different way of thinking about ITD coding emerges from the work of McAlpine et al. (2001). They described the distribution of "best delays" (best ITDs), rather than characteristic delays, seen in responses to noise stimuli of guinea pig midbrain cells. They found that best ITDs, expressed in units of time, were inversely related to CF (i.e., larger

for low-CF cells). Expressed in units of phase, however, best delays were nearly independent of CF, and very close to 45 degrees of ITD, favoring the contralateral ear. This has since been confirmed in gerbils (Brand et al., 2002) and cats (Hancock & Delgutte, 2004). It has the consequence that one can conceptualize the noise delay function as being broadly tuned, with the best ITD favoring the contralateral ear, and with the steep portion of the function centered at zero ITD. Interestingly, the contralateral bias in the functions, and therefore the fact that the steep part of the ITD function is centered over zero ITD, cannot solely be due to the greater length of the axonal pathway from the contralateral ear. If the inhibitory action of glycine is blocked with strychnine, then the ITD function becomes not only shallower in amplitude (see above), but displaced so the peak of the function is now at zero ITD rather than 45 degrees contralateral (Brand et al., 2002). This is an important point. It has the consequence that over the range of ITDs that the animal will encounter in nature, there is little modulation of cell firing rate in the glycine-treated animal, but massive modulation in the normal animal. The ITD coding circuitry thus seems specifically to have evolved in a way that maximizes sensitivity to ITDs near zero.

The axonal outputs of the MSO project most heavily on the ipsilateral dorsal nucleus of the lateral lemniscus (DNLL) and central nucleus of the inferior colliculus (ICC), so the contralateral bias in the representation of spatial information developed in the MSO is conferred on higher centers.

Neurons of the LSO receive an excitatory input from the VCN of the same side. From the contralateral side, the VCN projects upon the medial nucleus of the trapezoid body (MNTB) adjacent to the LSO. This connection is excitatory. In turn, the MNTB projects upon the LSO, and this connection is inhibitory. The excitation exerted on both the LSO and MNTB by projections from the cochlear nucleus is mediated by an excitatory amino acid neurotransmitter, and the inhibition exerted on the LSO by the MNTB is mediated by glycine (Wu & Kelly, 1992). Both the inputs to LSO cells are intensity dependent, with the strength of the excitatory and inhibitory drives being monotonically related to peripheral stimulus level. This means that the spike output of LSO cells is a sensitive function of the relative levels of the stimuli at the two ears, that is, a sensitive function of ILD. The inputs and the outputs of the LSO are responses sustained for the stimulus duration. Studied with transient stimuli, however, LSO cells (and therefore their inputs) display a familiar latency-intensity relation, with first-spike latencies inversely related to stimulus level. Because the responses to transients (e.g., clicks) are also transient, LSO cells are susceptible to a time-intensity trading, such that the effects of an ILD favoring one ear can be offset by an ITD favoring the opposite ear.

The general form of ILD sensitivity in the LSO is a sigmoidal relation of spike count to ILD, with high spike rates associated with ILDs favoring the ipsilateral ear, and the steep portion of the spike count function associated with ILDs near zero. Studied with virtual space technology, many LSO cells have spatial receptive fields occupying the ipsilateral acoustic hemifield, and medial borders near the midline (Tollin & Yin, 2002). The axonal outputs of the LSO are directed most heavily to the contralateral DNLL

and ICC. These connections are excitatory, and because they are crossed, they have the consequence of conferring on those centers a contralateral bias in the representation of ILDs, paralleling the one offered by the MSO for ITDs.

Figure 5–2 shows idealized spike count versus ILD functions as might be obtained from neurons rostral to the LSO; it illustrates their general form after the decussation. Spike rates are high when the ILD significantly favors the contralateral ear, and near zero for ILDs favoring the ipsilateral ear. The steep portions of the functions are associated with small ILDs. The absolute values of naturally occurring ILDs varies with head size, sound source frequency, and source azimuth, but it is always true that the slope of the function relating ILD size to source azimuth is steepest for near-midline azimuths (Irvine, 1987). This means that the neural code (spike rate) for the stimulus information (ILD size) is most unambiguous for cue values that themselves most precisely specify source azimuth (Phillips & Brugge, 1985). If psychophysical acuity for sound localization reflects the precision of stimulus information "mapped through" the neural code, then it is no surprise that behavioral acuity for source location is greatest at the midline.

Some cells of the MSO, the LSO, and some of the surrounding cell groups of

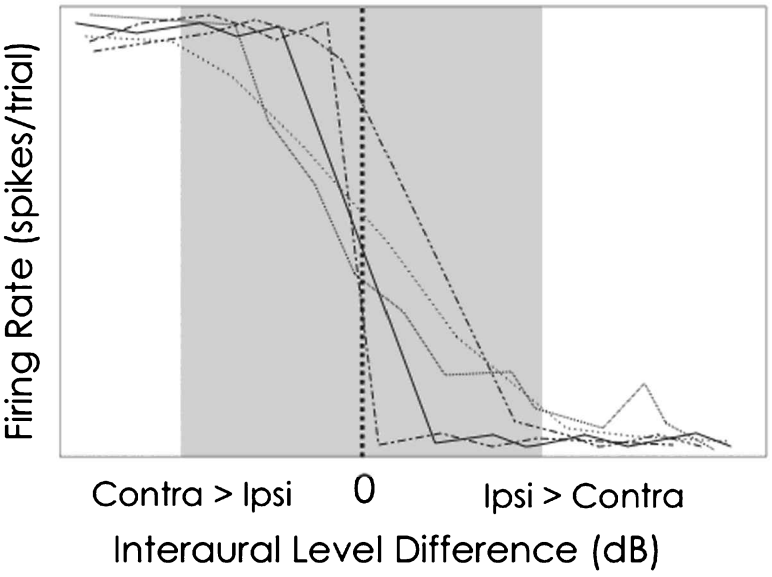

Figure 5–2. Idealized spike rate-versus-ILD functions. Note that spike rates are high, and relatively undifferentiated, for ILDs significantly favoring the contralateral ear, and that they are low, and undifferentiated, for ILDs significantly favoring the ipsilateral ear. This means that spike rate is the most unambiguous indicator of ILD (and therefore of source azimuth) for small ILDs (shaded area). Shaded area also indicates the range of ILDs associated with near-midline azimuths.

the olivary complex are those whose axons form the last leg of the descending, efferent auditory system (Warr, 1992; see also Spoendlin, 1967), that is, they are olivocochlear neurons. The olivocochlear neurons are typically divided into lateral and medial systems. The lateral one consists mostly of fine axons from cells in or near the LSO; these ultimately project toward the inner hair cells of the ipsilateral ear, where they terminate on the afferent nerve endings serving the inner hair cells. The medial pathway arises from larger cells outside the MSOs. Their large, myelinated axons generally cross the midline to innervate the contralateral ear. These fibers innervate the base of the outer hair cells directly. The olivocochlear system, perhaps especially that serving the outer hair cells, may ultimately be shown to have many effects on cochlear sensitivity. Let us consider one with a clear psychophysical expression. Focused auditory attention can operate selectively within the frequency domain (Scharf, Quigley, Aoki, Peachey, & Reeves, 1987). It can manifest, for example, as better detection thresholds for threshold-level tone pips of an attended frequency than for tone pips of nonattended frequencies when presented against a noise background. There is a clear "tuning" of the phenomenon, which is perhaps what we would expect of a system working in a frequency-specific fashion. Recent evidence from neurological case studies, specifically in patients who have had their olivocochlear axons cut, indicates that activity of the olivocochlear pathways may serve to dampen sensitivity in nonattended frequency channels, thus giving a sensitivity advantage to the attended channel (Scharf, Magnan, Collet, Ulmer, & Chays, 1994). Note that in this case, olivocochlear activity does not "add" in some way to sensitivity in the attended channel but, rather, it dampens sensitivity in the nonattended ones.

Inferior Colliculus

The central nucleus of the ICC is a mandatory synaptic station for auditory sensory information ascending beyond the auditory midbrain. It is a laminated, tonotopically organized structure, surrounded by pericentral (ICP) and external nuclei (ICX). ICP and ICX also receive auditory input, but their functions are less well known, save for the fact that ICX is multimodal and is likely involved in the neural circuitry mediating head and pinna movements. The ICC receives significant projections from the LSO bilaterally, from the ipsilateral MSO, and from the DNLL bilaterally. The projections onto the ICC are highly differentiated, not only in the sense that they respect the tonotopy developed more caudally, but in the sense that there are regional differences in anatomical convergence patterns within the ICC. In turn, this means that there are regional differences in neuronal physiology as well. Thus, one caudal, predominantly high-frequency region of the ICC receives inputs jointly from the contralateral LSO and the contralateral DCN, and neurons in that region of the ICC thus "inherit" the properties of those afferent sources (e.g., sensitivity to interaural level differences, and a nonmonotonic spike rate-versus-intensity profile; Oliver, Beckius, Bishop, & Kuwada, 1997; Semple & Aitkin, 1981).

As mentioned above, the crossed projection from the LSO is excitatory; the

projection from the ipsilateral LSO is, however, inhibitory and probably mediated by glycine (Glendenning, Baker, Hutson, & Masterton, 1992). Note that this arrangement preserves the contralaterality of spatial representation in the LSOs' projections onto the ICC. The DNLL projections onto the ICC appear to follow a similar functional pattern. In this case, it is the crossed projection that is inhibitory, likely mediated by gamma amino-butyric acid (Shneiderman, Oliver, & Henkel, 1988). If we recall that the MSO and LSO inputs to the DNLL confer a distinctly contralateral bias in the spatial representation there, then the fact that the crossed DNLL projection to the ICC is inhibitory would again function to preserve the contralaterality of spatial representation in the recipient ICC.

The further question is whether this inhibitory circuitry in some way does more than simply "preserve" the contralaterality of spatial representation in the ICC. Does it enhance spatial coding? There is evidence that interfering with DNLL function unilaterally interferes with the neural coding of interaural level (Li & Kelly, 1992a) and time differences (Kidd & Kelly, 1996) in the contralateral ICC; the spike rate-versus-interaural disparity functions less precisely indicate the disparity size or sign in the presence of DNLL inactivation. What is less clear is whether the spike rate-versus-disparity functions in the ICC of intact animals are any "sharper" or more precise than those seen in the nuclei providing their input. That is, does the inhibitory circuitry improve the precision of the code above that seen in the SOC, or does it maintain the precision of SOC coding in the face of converging inputs from additional sources that would otherwise degrade the code in the ICC? We do not yet have good answers to this question.

A second way of thinking about this general issue is to ask whether spatial coding in the ICC has acquired some qualitatively new feature (i.e., one not seen more caudally), rather than having sharpened a preexisting neural code. In this regard, Spitzer and Semple (1993, 1998) studied the responses of brainstem auditory neurons to dynamic interaural phase differences, that is, stimuli that provide information about source *movement* rather than sound source *location*. They showed that the response rate of ICC neurons evoked by a given interaural phase disparity depended on the immediately preceding stimulus-response history; in contrast, MSO response rates more closely reflected the instantaneous interaural delay, irrespective of the stimulus history. These findings suggest that there is an emergence of sensitivity to dynamic stimulus features at the level of the ICC; it remains to determine exactly what circuitry is responsible for this emergence.

[redacted]ial Geniculate Body [redacted]d Auditory Cortex

The medial geniculate body contains a parvocellular ventral nucleus (MGv) in which neurons are arranged in sheet-like layers, each deriving its input from a single cochlear place, and arranged tonotopically. The MGv is surrounded by a number of other nuclei, the most notable of which are the magnocellular, medial division (MGm) and the dorsal division (MGd). There is some evidence of a parallelism in the pathways linking

the midbrain and thalamus such that ICC preferentially projects upon MGv through the brachium of the ICC, while the nuclei surrounding the ICC project upon the nuclei abutting the MGv (Calford & Aitkin, 1983; Winer, 1992). This parallel organization involving the parvocellular and magnocellular divisions of the medial geniculate body should not be confused with the "magno" and "parvo" streams of processing in primate vision. In the primate visual system, the magno and parvo streams arise in the retina, extend into the lateral geniculate body, and then to the striate cortex and beyond (Livingstone & Hubel, 1988); they are largely independent pathways, each sampling the sensory epithelium, but with different stimulus feature selectivities (color versus luminance, sensitivity to motion or not, contrast sensitivity, different periodic temporal responses), by neurons with different receptive field sizes and adaptive properties (transient and sustained). In the auditory system, there is little evidence for magnocellular and parvocellular "streams" of processing in this sense of the term. And although there are certainly auditory system neurons whose responses are dominated by "transient" or "sustained" discharge patterns (Phillips et al., 2002), these neurons are not organized into streams in the way that they may be in vision. Accordingly, allusions to magnocellular and parvocellular "streams" of processing in audition (e.g., Stein, 2001) or to "transient subsystems" (Galaburda, Menard, & Rosen, 1994) are to be interpreted skeptically.

Neurons in MGv are narrowly tuned to tone frequency, have short response latencies, and show the familiar patterns of binaural input and interaction (Clarey, Barone, & Imig, 1992). Cells of the MGm and MGd tend more often to be broadly tuned to frequency, and those of the MGd particularly often have longer latencies and show irregular, habituating responses to acoustic stimulation. Some of them are multimodal.

The auditory cortex is a broad area of the temporal cortex that is responsive to acoustic stimulation. It is divisible into a number of separable "fields" or territories, based on cytoarchitecture, and/or the presence or absence of a tonotopic map, and/or patterns of connectivity with the thalamus and other cortical fields. Some of the fields are tonotopically organized, and these typically receive thalamic input from MGv, with the densest such projection serving the primary auditory cortex (AI; Winer, 1992). The nontonotopic fields receive thalamic afferents more heavily loaded in favor of the MGm and MGd. The MGm projection is perhaps particularly interesting, because it targets almost all of the cortical auditory fields, and in this way reflects its own input, which is very convergent even at the level of individual neurons. In cats, the primary field is surrounded by other tonotopic fields, which typically take their names from their spatial position in relation to AI (e.g., anterior field, posterior field) and the probably nontonotopic field AII, which appears to contain broadly tuned neurons (Reale & Imig, 1980). In primates, AI abuts a near-mirror image rostral field, and these are flanked by so-called "lateral belt" fields, at least some of which are also tonotopic (Merzenich & Brugge, 1973).

The thalamocortical connections in the auditory system are largely reciprocal (Winer, 1992). There is a dense and quite orderly projection from deep layer V and layer VI back to the thalamus,

and another back to the ICC. These projections form a "loop" between the midbrain, thalamus, and cortex, and this loop may be a medium through which the cortex is able to "select" or modulate the relative strengths of inputs across the tonotopic (or other) representational dimension (Suga, Yan, & Zhang, 1997; Yan & Suga, 1996; Zhang, Suga, & Yan 1997; see also below). The cortex is also the origin of a longer descending, efferent auditory pathway that ultimately reaches the cochlea (Warr, 1992).

In the tonotopic fields, and we shall deal mostly with AI because it is the most well studied, the tonotopic organization is expressed in the form of roughly parallel, strip-like assemblies of neurons, each deriving its input from a particular cochlear site, and thus a convergent input from the sheet of MGv neurons representing the same cochlear place. These "iso-CF" strips of cells span most of the cortical layers, and the strips themselves are spatially arrayed according to CF, forming the familiar tonotopic "map." Borders between adjacent tonotopic fields are typically marked by a reversal in the tonotopic sequence of neural CFs, so that the tonotopic maps of adjacent fields are somewhat mirror images of each other.

Within AI's tonotopic map, there are patches of tissue distinguished by their cells' other neurophysiological properties. The majority of cortical auditory neurons are binaurally influenced (Semple & Kitzes, 1993), and there are local territories or patches of cortical tissue dominated by cells expressing one or other form of binaural interaction (Imig & Adrian, 1977). Some patches of tissue contain cells with predominantly "suppressive" binaural interactions (typically, though not always, reflecting an excitatory input from the contralateral ear and an inhibitory one from the ipsilateral ear). Others contain cells with predominantly "summative" binaural interactions (often reflecting a net excitatory input from each ear). At least some of these patches are elongated and are oriented orthogonal to the iso-CF lines of the tonotopic map. It is likely that cells with suppressive binaural interactions are the neurons that have free-field spatial receptive fields occupying the contralateral acoustic hemifield, with the azimuthal location of the receptive field's medial border being determined by the relative sensitivities and strengths of the contralateral excitatory and ipsilateral inhibitory inputs (Middlebrooks & Pettigrew, 1981; Rajan, Aitkin, Irvine, & McKay, 1990; Samson, Clarey, Barone, & Imig, 1993). The neurons with summative binaural interactions are likely the same neurons that have spatial receptive fields centered on the midline, or which are omnidirectional.

The corticocortical connectivities of "suppressive" and "summative" binaural patches may differ (see Hackett & Phillips, 2006 for review). Cells with suppressive binaural interactions have connections that tend to be restricted to cortical targets in the same cerebral hemisphere (Imig & Reale, 1981), while cells with summative interactions tend to have stronger callosal connectivity (Imig & Brugge, 1978). There is good reason to link "suppressive" binaural interactions with free-field spatial selectivity for the contralateral auditory hemifield, and "summative" binaural interactions with free-field spatial selectivity for midline locations or with omnidirectionality (Clarey, Barone, & Imig, 1992; Hackett & Phillips, 2006). Taken together, these data suggest that the spatial information carried by intrahemispheric connectivity

is dominated by the contralateral auditory hemifield, while the spatial information carried interhemispherically is not. In this regard, unilateral lesions of the auditory cortex in animals produces sound localization deficits that are restricted to sources in the contralateral auditory hemifield, and there is an auditory "neglect" syndrome in man characterized by inattentiveness to (or "extinction" for) sources contralateral to parietal cortical damage (Phillips, 2001).

In man, there are further psychophysical expressions of this hemifield-specific organization. There is growing evidence that spatial processing in man is mediated by two perceptual channels, each with hemifield azimuthal tuning (left or right), and medial borders that overlap at the midline (Boehnke & Phillips, 1999; Phillips & Hall, 2005; see also Stecker, Harrington, & Middlebrooks, 2005 for a neurophysiological counterpart). Spatial processing of sources near the midline likely depends on the outputs of both perceptual channels, while spatial processing of sources deep in one or other auditory hemifield likely is dominated by one or other perceptual channel. This organization of processing may contribute to the perception of speech in noisy free-field environments. If a listener's task is to repeat speech presented concurrently with a noise masker, then the perceptual benefit of a 90-degree separation of speech and noise is close to 1.3 dB when the speech and noise are located in the same acoustic hemifield, and closer to 8.6 dB when the speech and noise are on opposite sides of the midline (Phillips, Vigneault-MacLean, Hall, & Boehnke, 2003). It is thus not the absolute separation of speech and noise alone that offers the perceptual advantage, but rather the extent to which the speech and noise fall into separate spatial channels and are thus available for selective scrutiny. This phenomenon may well extend to include other forms of spatial competition, for example, cocktail party paradigms. The extent to which the phenomenon reflects specifically binaural processing, or the signal to noise advantage for the speech at the ear nearer the speech (after Hirsh, 1950), is unclear. (This is not a small point. It is true that most auditory cortical neurons are binaurally influenced, and that interaural cues contribute to the azimuthal location of receptive field medial borders. However, the pinnae can become highly directional receivers for high-frequency sources, and this spatial selectivity is superimposed on neural spatial selectivity that would otherwise arise from binaural processing of ILDs due to head shadows alone. The pinna directionality could potentially saturate the ILD coding system for favored frequencies, so that although neurons in the cortex may be binaurally influenced, their responses may become dominated by events at one—the contralateral—ear. This effect would presumably be most marked for high frequencies and for sources located in the acoustical axis of the pinna.)

A second "patchy" organization of the primary auditory cortex is based on the frequency-level "response areas" or response ranges of the neurons therein. Neurons with strongly nonmonotonic spike count-versus-level functions tend to be located toward the middle of the iso-CF strips (Phillips, Orman, Musicant, & Wilson, 1985; Phillips, Semple, Calford, & Kitzes, 1994) in patches that do not respect the boundaries of the "binaural" ones. Recall that these are neurons with strong inhibitory response domains that flank the excitatory one at CF, and they

often display exceptionally narrow frequency tuning. The differential location of these neurons toward the center of AI likely underlies the narrow bandwidth tuning and poorer responses to wideband sounds seen in neural activity in the same region (e.g., Schreiner & Mendelson, 1990).

The patchy distribution of neurons according to their neurophysiologies leads us to important inferences for how we construe stimulus "representation" in the auditory cortex. Tonotopic maps are *not* representations of stimulus frequency. If it were that simple, then the pattern of excitation across the cortical mantle would be predictable from knowledge of the stimulus frequency alone, and it is not (Phillips et al., 1994). Tonotopic maps simply describe the spatial arrangement of neurons according to their CFs. The cortical *representation* of a stimulus resides in the distribution of responses in space (and time) across the cortex. Even for a stimulus as simple as a tone pulse of a specified frequency, the pattern of excitation evoked depends on the ear stimulated and the plateau level of the tone, and it takes the form of discontinuous patches of activation distributed along the relevant iso-CF strip (for low-level tones) and sometimes quite far from it (in the case of high-amplitude tones). Sometimes, the middle parts of an iso-CF strip can be devoid of activity in the presence of high-amplitude tones at that CF—because those territories are occupied by cells with nonmonotonic rate responses (Phillips et al., 1994). The fact that stimulus representation may not be in the form of neat, linear maps is in many ways functionally irrelevant. What matters is that the putative neurophysiological representation of a stimulus parameter is as differentiated as is the perceptual dimension it supports (Middlebrooks, Xu, Eddins, & Green, 1998; Stecker et al., 2005). That is, there usually must be an orderly one-to-one relation between brain state and subjective experience.

Spatial Hearing: The Mammalian Model and the Role of Head Size

Recently, neurophysiological and human psychophysical data have converged on a model of the mechanisms used by the mammalian brain to localize sound sources. Above, we saw that neurons of the superior olivary and more rostral nuclei are commonly sensitive to ILDs and ITDs. The form taken by that sensitivity might loosely be described as a "hemifield" tuning, that is, neural firing rates are high for ITDs favoring the contralateral ear, and low for ITDs favoring the ipsilateral ear (e.g., McAlpine et al., 2001). In the case of ILDs, most neurons respond strongly when the ILD favors the contralateral ear, and weakly when the ILD favors the ipsilateral ear so that the steep part of the spike rate-versus-ILD function is centered over relatively small ILDs (Phillips & Irvine, 1981). Occasionally, one sees neurons with the reverse pattern (i.e., a preference for ipsilaterally favored ILDs), or cells that display tuning to zero ILD.

The spatial selectivity of cortical neurons has now been studied extensively using free-field stimuli (Lee & Middlebrooks, 2010; Middlebrooks & Pettigrew, 1981; Rajan et al., 1990; Stecker et al., 2005). With the caveat that the free-field experiments have studied only high-CF neurons (whose spatial properties are likely to be guided by ILD sensitivity;

for example, Razak, 2011, 2012), the azimuthal tuning of cortical neurons is typically that which would be predicted from the dichotic studies: a predominance of cells responsive maximally across contralateral azimuths, and smaller proportions of cells maximally sensitive across ipsilateral azimuths or azimuths near the midline. In at least the first two of these cases, firing rates are the steepest function of azimuth for near-zero eccentricities. Stecker et al. (2005) proposed an "opponent process" ("two-channel") model of sound location mechanisms. They suggested that the azimuthal location of a sound source was encoded in the relative activity of the contralaterally and ipsilaterally tuned neural populations. Eggermont & Mossop (1998) had previously proposed a "vector sum" model of the population coding of source azimuth.

In the same year as the Stecker et al.'s neurophysiological study in cats, Phillips & Hall (2005) reported data from a selective adaptation paradigm on human psychophysical sensitivity to ILDs and ITDs. They showed exposure to highly lateralized adaptor stimuli resulted in shifts of the perceived midline (point of centrality) toward the adapted side. They explained this in terms of two perceptual channels, likely with hemifield tuning (i.e., left and right, with borders straddling the midline) such that the perceived azimuth of a source reflected the *relative* outputs of the two (left, right) channels. These psychophysical data were entirely compatible with Stecker et al.'s (2005) conclusions, but extended them to low frequencies, suggesting that McAlpine et al.'s (2001) ITD channels also had a human perceptual correlate. The psychophysical studies eventually confirmed that the human perceptual channels had hemifield tuning (Phillips, 2008; Vigneault-MacLean, Hall, & Phillips, 2007), and the model has now been extended to include a midline channel, in the ILD domain at low and high frequencies (Dingle, Hall, & Phillips, 2012), and in the ITD domain at low frequencies (Dingle, Hall, & Phillips, 2010). There is thus a very elegant similarity between the "architecture" of cortical sound localization coding strategies emerging from neurophysiological studies in animals and from studies of human perceptual performance. Independent neurophysiological studies probing the human cortical responses to ITDs, prior to the discovery of the midline channel, have been highly successful in confirming the existence of two hemifield-tuned channels (Magezi & Krumbholz, 2010; Salminen, Tiitinen, Yrttiaho, & May, 2010).

A second major development in our understanding of sound localization mechanisms comes from Heffner and Heffner (1992). Recall that the binaural cues for sound source azimuth are ITD and ILD. The magnitude of these cues depends on the frequency of the sound, the azimuth of the source, and the size of the listener's head (see Phillips, Quinlan, & Dingle, 2012). The last of these, of course, is due to the longer sound travel time between the ears, and the stronger head shadow cast by the head of large-headed animals. One might therefore expect that large mammals would have an advantage in sound localization acuity because they have larger cues with which to work. Behavioral studies of sound localization acuity typically measured the smallest angular distance between free-field speakers symmetrically disposed across the midline that would permit the animal to correctly identify which of the two speakers was activated, usually with a brief, broadband stimulus

(so, technically sound *lateralization*, rather than localization). Early studies did indeed find evidence of a relationship between head diameter and localization acuity (e.g., Heffner & Heffner, 1982). In 1992, Heffner and Heffner reported the results of a study that described the width of field of best vision (as measured by retinal ganglion cell density counts) in species of known localization acuity. They found that when width of field of best vision was partialed out, there remained *no* significant relationship between head size and sound localization acuity. On the other hand, when head size is partialed out, there remains a striking correlation between width of field of best vision and localization acuity. The range of species examined has since doubled (Heffner, 2004; Heffner, Koay, & Heffner, 2007); the striking correlation between the visual factor and sound localization acuity remains strong, and the poverty of the contribution of head size also remains. Heffner and Heffner (1992; Heffner, 2004) pose the following intriguing hypothesis. One of the functions of the auditory system may be to provide the visual system with source location information in order to direct the region of best vision to the target. If that is the case, then the narrower the width of field of best vision, the greater is the evolutionary pressure on the species to be an accurate sound localizer. Thus, species with small foveas (e.g., primates, cats) require highly accurate sound localization information to align their small foveas with the source, while animals with wide "visual streaks" (e.g., gerbils, pigs) require only crude auditory source location information to make an eye movement aligning their best vision with the source (because their widths of field of best vision are so great). To date, the only species failing to fit this pattern are subterranean (e.g., naked mole rats, pocket gophers).

The Heffner hypothesis seems very clearly to identify one of the evolutionary pressures on the development of sound localization acuity. What is less clear is the neurophysiological mechanism on which this evolutionary pressure operates. What is it about the auditory systems of animals with narrow foveas that makes them better localizers than mammals with broader regions of best vision? Phillips et al. (2012) surveyed data on this issue. They reasoned that sound localization acuity depends on the sensitivity of neural firing rate to positional differences of sounds around the midline (after Phillips & Brugge, 1985). For ITD coding, they found that the range of ITDs supporting the greatest neural sensitivity were remarkably similar in guinea pigs (McAlpine et al., 2001), gerbils (Brand et al., 2002), and cats (Hancock & Delgutte, 2004)—species with remarkably different widths of field of best vision. Much the same appeared to be true for ILD coding (Phillips et al., 2012). There seems to be little sense, then, in which the dynamic range of the population code for ITD or ILD is tailored to the vision of the species. Phillips et al. noted, however, that the slopes of neural firing rate versus ILD (and probably ITD) functions were variable in all species. Now, it is easily argued that the greatest behavioral sensitivity to change along a stimulus parameter depends on the "best" neurons coding it, that is, behavioral sensitivity to stimulus difference depends on neural sensitivity to stimulus difference. Phillips et al. (2012) therefore argued that the evolutionary pressure is not on the range of ITDs/ILDs encoded, but on the ability of the organism to base

the perceptual judgement selectively on the responses of the "best" neurons differentially encoding left from right. That is, the perceptual judgement will be most acute when it is based selectively on the responses of neurons most sensitive to the change in stimulus position across the midline, rather than on some kind of vector sum of all responses to the stimuli. This principle may be one factor involved in auditory learning (see below, and see also Chapter 22 in this volume): The acquisition of a new discrimination may exploit the "best" neural responses mediating the discrimination, and the circuits mediating that processing are then differentially reinforced or elaborated because of their behavioral relevance.

Cortical Coding of the Temporal Structures of Sounds

The fashion in which the cortex is able to encode the temporal properties of auditory stimuli is a key topic. Studied with transient stimuli, temporal jitter in single neuron mean first-spike latencies (i.e., the precision with which the cortex is able to "time-stamp" a stimulus event) can be significantly under a millisecond (Phillips & Hall, 1990). This is more than sufficient to encode the timing of the phonetically important events in speech and is comparable to that seen in the cochlear nerve. (The spectral content of the acoustic event is represented by the pattern of activity across the tonotopic array; see above, and Phillips et al., 1994; Wang, Merzenich, Beitel, & Schreiner, 1995). By contrast, the ability of cortical cells to establish a temporal code for periodic events is much poorer than that seen in the cochlear nerve; cortical neurons can entrain to periodicities up to only a few tens of hertz (Eggermont, 1991, 1994). This would be insufficient to encode glottal pulse rates on a temporal basis and is over an order of magnitude poorer than the upper limit of temporal coding of stimulus periodicities by the cochlear nerve. The poverty of cortical responses to periodic stimuli—in the face of the temporal precision of responses to transients—may reflect a synaptic depression driven by the periodicity (Eggermont, 2002). It is perhaps because of the dominance and temporal precision of transient responses in the cortex that auditory cortical lesions have different effects on the perception of speech sounds at the level of pitch and phonetics (see below).

Temporal issues are important to understanding cortical function for a second reason. The responses of cortical cells to successive stimuli are not independent, especially if the stimulus events are in close temporal proximity. Thus, the response to a specified frequency modulation (FM) depends very much on whether it occurs as an isolated event (e.g., an FM tone pulse) or as a modulation of a continuous carrier (Phillips, 1988; see also Malone & Semple, 2002 for evidence of stimulus context effects on the coding of trapezoidal ITD changes). Likewise, the response to one binaural tone can be significantly altered by the presence of a preceding "conditioning" tone (Reale & Brugge, 2000; Zhang et al., 2005; see also Brosch & Schreiner, 2000). This may be one of the reasons why laterality percepts for tone pulses of a given frequency are so markedly influenced by stimulus history at that frequency (Phillips & Hall, 2005). The mechanisms mediating these kinds of sequential interactions

are still being worked out, but likely include short-term adaptive tracking of effective stimulus level (Malone & Semple, 2002; Phillips, 1985, 1988; Phillips et al., 2002) and inhibition (Calford & Semple, 1995; Eggermont, 1991). The general point is that cortical neural responses are sensitive to the recent stimulus history.

A further issue, raised at the outset of this chapter, concerns the neural coding of the pitch of complex sounds, particularly those stimuli with no energy (or no special concentration of energy) at the pitch frequency. We have already remarked that stimuli arousing nonspectral pitch percepts must rely on a temporal coding process, and there is direct evidence that lower auditory neurons display interspike intervals related to the period of the perceived pitch frequencies of such sounds (see Cariani & Delgutte, 1996). Direct recordings from the primary auditory cortex of primates have failed to find evidence of neurons responsive to pitch frequency; rather, the responses are dominated by the relation between the spectral content of the sound and the neuron's frequency response area (Fishman, Reser, Arezzo, & Steinschneider, 1998; Schwarz & Tomlinson, 1990). However, at least some neurons in a border region between the primary field (AI) and rostral and lateral belt fields in the primate may be sensitive to nonspectral pitch and display tuning to pitch frequency (Bendor & Wang, 2005). How it is that the temporal code expressed in the auditory hindbrain is converted to a spike-rate code in pitch-tuned cortical neurons is unclear, but the location of the relevant cortical neurons near the border of the low-frequency part of AI is compatible with human brain-imaging data suggesting the existence of a peri-AI cortical region activated by temporal pitch stimuli in a perhaps homologous region (Patterson, Uppencamp, Johnsrude, & Griffiths, 2002).

Finally, the issue of temporal gap detection warrants some mention. Temporal gap detection is a behavioral measure of auditory temporal acuity. Historically, the method has provided a measure of the shortest detectable period of silence ("gap detection threshold") inserted roughly at the temporal midpoint of an otherwise homogeneous, ongoing stream of sound. Depending on the spectral content of the stimulus in which the gap is inserted, gap thresholds may be as low as 1 to 2 ms. Gap thresholds are lowest for wideband sounds (Eddins, Hall, & Grose, 1992), probably because information about the presence of the gap is being carried by more than one channel of cochlear output. This means that a central perceptual processor can recover the temporal event by temporally correlating information being transmitted roughly synchronously by separate, independent frequency channels. Studied with narrowband noises, and noise band pairs containing synchronous gaps, human gap thresholds are better for noise band pairs than for single noise bands but are independent of noise band spectral separation (Hall, Grose, & Saju, 1996). This finding is consistent with the notion that the perceptual recovery of the gap is facilitated by the synchronous transmission of the event in multiple frequency channels.

Gap detection took on new interest when it became clear that acuity for gaps bounded by different markers is poorer (sometimes by an order of magnitude or more) than is acuity for gaps bounded by identical sounds (Formby, Gerber, Sherlock, & Magder, 1998; Grose, Hall & Buss, 1999; Phillips, Taylor, Hall, Carr, & Mos-

sop, 1997). In practice, detection of gaps in the historical "within-channel" design of stimulus likely reflects the detection of a discontinuity in the time-course of activity aroused by the stimulus in the relevant perceptual channel (Figure 5–3, upper); it may reduce to the detection of the onset of the sound marking the end of the silent period (Florentine, Buus, & Geng, 1999; Oxenham, 1997; Phillips et al., 1997, 2002). By contrast, detection of the silent period in the "between-channel" paradigm may require some kind of relative timing process to be executed on the offset of activity in the perceptual channel serving the leading marker and the onset of activity in the channel serving the trailing marker (Figure 5–3, lower; Phillips et al., 1997; Phillips & Hall, 2000, 2002). Particular interest in the nature and properties of mechanisms mediating between-channel gap detection derives from the fact that gap thresholds for between-channel stimuli that model the structure of stop-consonant vowel syllables tend to average 20 to 40 ms (Phillips & Hall, 1997; Phillips & Smith, 2004). This is the range of values taken by many voice onset time phonetic boundaries, and so between-channel gap detection acuity may be a natural psychophysical parameter exploited by the speech

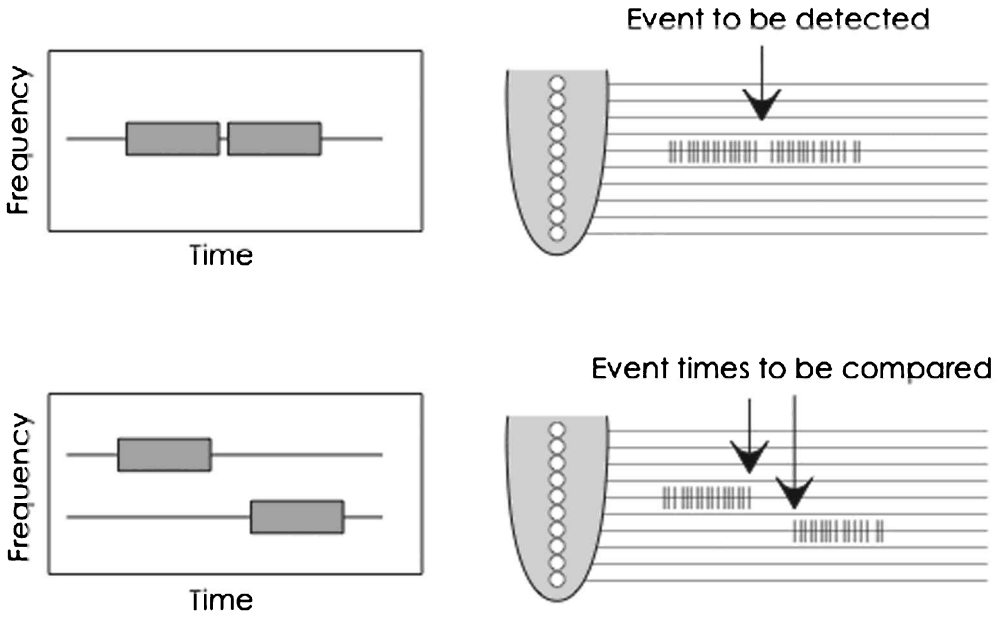

Figure 5–3. Schematic depiction of within-channel (*upper*) and between-channel (*lower*) temporal gap detection stimuli, and the perceptual operations required to perform the tasks. Left side shows the time-course of the stimuli, center shows a schematic layout of the cochlea, and right side depicts the time course of activity in the neural-perceptual channels activated by the stimuli. In within-channel gap detection, the perceptual operation is the detection of a discontinuity in the activity of the channel excited by the stimulus. In the between-channel case, detection of the silent period requires a relative timing of the offset of the leading marker and the onset of the trailing marker.

system in the generation of voice onset time phonetic boundaries (after Kuhl & Miller, 1978; Phillips & Smith, 2004). That is, the categorical phonetic boundary may be related to the categorical distinction between above- and below-threshold between-channel gaps. Elangovan and Stuart (2008) recently studied within-channel and between-channel gap thresholds (in ms) in listeners for whom they also had direct measurements of voice onset time phonetic boundaries (also in ms). They showed for the first time in the same listeners that between-channel gap thresholds were highly correlated with voice onset time phonetic boundaries, but that within-channel gap thresholds were not.

Especially in anesthetized animals, cortical neural responses tend to be dominated by brief responses time-locked to discrete stimulus events, notably stimulus onsets or amplitude increments (Phillips et al., 2002). Studied with wideband noise stimuli in within-channel paradigms, cortical neural responses to gap detection stimuli take the form of an onset response to the beginning of the leading marker, and another onset response time-locked to the beginning of the trailing marker (Eggermont, 1995, 1999). Neurophysiological "gap threshold" is thus the shortest-duration gap for which there is a detectable onset response to the trailing marker. When the gap occurs "late" in the noise signal (e.g., 500 ms after the onset of the leading marker), these neurophysiological thresholds can be as short as about 5 ms (Eggermont, 1995), and most fall in the range below 15 ms (Eggermont, 1999). If the temporal gap follows a very short leading marker (e.g., 5 ms), then neurophysiological gap thresholds are longer, averaging around 40 ms, and with a wide range (<10 to >60 ms; Eggermont, 1999). There is psychophysical evidence of a similar pattern of dependence of within-channel gap threshold on the temporal position of the silent period in some, though not all, human listeners (cf. Phillips et al., 1997, 1998; Snell & Hu, 1999). That is, gap thresholds are sometimes best (lowest) for silent periods not in close temporal proximity to the onset of the leading marker or the offset of the trailing one (i.e., when the leading or the trailing marker is very short; Snell & Hu, 1999). Interestingly, it may be that the most highly practiced listeners show the smallest effects of leading marker duration. This raises the possibility that skilled listeners are more able to rely on the activity of the subset of the neurons displaying the "best" acuity, and that one form of perceptual learning is the process of learning how to do this.

The cortical coding of between-channel gap detection stimuli has not been studied directly. Eggermont (2000) has modeled cortical responses to between-channel gap stimuli, based on direct measurement of the onset and postonset inhibitory responses driven by wideband stimuli and on the assumptions that information from different frequency-specific channels converges on broadly tuned cortical cells and that it is the activity of these cells that are the neurophysiological correlate of psychophysical performance. Certainly, the modeling effort provided a strikingly accurate neurophysiological prediction of perceptual between-channel gap detection performance (Eggermont, 2000). It is, however, an open question as to whether one needs to explain the psychophysical performance on the basis of individual neurons receiving input from both frequency (or other) channels. Perhaps all that is required are temporally

segregated responses to leading marker offset and trailing marker onset, without any special requirement that those responses come from the same neurons.

[redacted]ticity in the Central Auditory System

"Plasticity" is an umbrella term. For the present purposes, it encompasses any change in the organization of the auditory system and the behavior that the system mediates. Some of these changes are genetically programmed to proceed independently of acoustic experience (e.g., the embryological development of the auditory system); some may be activity dependent and/or experience dependent (e.g., perceptual learning) and/or dependent on "critical periods." The paragraphs that follow describe examples of different levels of plasticity.

Neonatal cochlear ablations can cause quite dramatic rewiring in the auditory brainstem. Recall that in the normal animal, the VCN projects upon the MSO bilaterally. MSO neurons have oriented dendrites, with a lateral arm receiving input from the VCN of the same side, and the medial arm receiving input from the contralateral VCN (Cant, 1991). Following a neonatal unilateral cochlear lesion, ectopic projections arise from the VCN serving the intact ear: This VCN can come to innervate both the medial and lateral dendrites of cells in both MSOs (Kitzes, Kageyama, Semple, & Kil, 1995; Russell & Moore, 1995). It is also possible for the VCN serving the intact ear to come to innervate the ipsilateral MNTB, as opposed to the contralateral one. In gerbils at least, this reorganization of connectivity occurs before the onset of hearing and may be restricted to that period; it is therefore unlikely to depend on auditory experience (Russell & Moore, 1995). There is significant shuffling of afferent connectivity to the ICC following unilateral cochlear lesions in neonates, and the reorganized connections support vigorous responses to stimulation at the intact ear, indicating that those connections are functional (Kitzes et al., 1995). In cats that receive unilateral neonatal cochlear ablations, cells of the auditory cortex ipsilateral to the intact ear are more likely to receive (especially excitatory) input from that ear than are cells in normal animals (Reale, Brugge, & Chan, 1987).

In adult rats, one can experimentally lesion the superior olivary complex. After recovery from the surgery, such animals can exhibit remarkably normal binaural input patterns among neurons in both the ICC (Sally & Kelly, 1992) and auditory cortex (Kelly & Sally, 1993). The sensitivity of ICC cells to interaural level differences is also remarkably normal in appearance in these animals (Li & Kelly, 1992b). It is not currently known if these neurons show normal sensitivity to interaural temporal parameters. The presence of any binaural input itself suggests either that substantial rewiring has taken place following the lesion or that there are other pre-existing media of binaural convergence, knowledge of which has perhaps been overshadowed by interest in the olivary nuclei. Certainly, there are many decussations of the auditory pathways (Hutson, Glendenning, & Masterton, 1991), and many or all of these are potentially capable of mediating binaural convergence. They include the corpus callosum (Kitzes & Doherty, 1994). This line of work, then, also serves to remind us that binaural interaction is a "layered"

process, occurring not just once at the olivary nuclei and then being transmitted upstream without modification; rather, it may occur repeatedly through the convergent connectivity of the afferent auditory system. (See also the description, above, of the role of the DNLL in binaural interactions.)

There has been considerable effort afforded to exploring the consequences of partial cochlear lesions in adult animals. This issue is of interest clinically, because experimental cochlear ablations in animals might simulate naturally occurring peripheral hearing loss in man. There have been compelling demonstrations that the tonotopic map in the auditory cortex is affected by frequency-restricted cochlear lesions in adult animals (Rajan, Irvine, Wise, & Heil, 1993; Robertson & Irvine, 1989; Schwaber, Garraghty, & Kaas, 1993). The reorganization takes the form of a loss of representation of the lesioned cochlear sector and an expansion of the representation of the "perilesional," adjacent cochlear sites. The reorganization is not instantaneous. Studied in acutely lesioned animals, shortly following the ablation, neurons in the affected cortical places show somewhat broad, insensitive tuning to tonal stimulation (Robertson & Irvine, 1989). Studied in chronic animals 2 to 11 months after the lesion, however, neurons in those cortical regions come to acquire more normal sensitivities, frequency tuning, and response latencies for stimuli of the "new" CFs (Rajan et al., 1993). Note that in normal animals, the vast majority of cortical cells are binaurally influenced, and the CFs of inputs from the two ears are closely matched in individual neurons; the tonotopic maps obtained for the two ears are therefore highly similar. In the cortex of animals with a unilateral, partial cochlear lesion, the tonotopic maps for the two ears are out of register in the reorganized sector. This mismatch may be a central neural correlate of binaural diplacusis, because the activity of neurons in the reorganized sector is ambiguous with respect to indicating the presence of a frequency of the normal CF at the intact ear, or a frequency of the remapped CF at the damaged ear.

It is of interest to ask whether the expanded cortical representation of the perilesional cochlear places supports superior frequency or other discriminations at frequencies represented by those perilesional cochlear sites. We can make the assumption that frequency-restricted hearing loss in man causes the same representational changes in the cortex as are seen in animals; that assumption made, frequency discrimination at the "edge" frequencies appears to be not much better than at frequencies served by the healthy cochlear sectors (Irvine, Rajan, & McDermott, 2000). Buss et al. (1998) similarly found no compelling evidence for "edge effects" in human listeners with steeply sloping hearing losses. It is possible that human frequency discrimination is already as good as the neural machinery can support, so that the provision of further cortical representational space still leaves the neural machinery at a ceiling level of performance. Irvine et al. (2000) take the general point a step further. They suggest that these cortical reorganizations following partial cochlear lesions are not appropriately viewed as "compensatory" mechanisms: Sensitivity to frequencies in the damaged cochlear region is in no way restored by the reorganization, and it is unclear in what way the "edge effects" in the reorganized cortical maps *compensate* for loss of sensitivity in the damaged frequency region

(Irvine et al., 2000). Perhaps competition for postsynaptic cortical space is the normal state of the cortex, and the edge effects are simply an expression of this, even if they are without clear positive perceptual consequences in the particular tasks examined to date, because the system is already as good as it can get.

A further form of plasticity is that associated with auditory learning. In the auditory forebrain, neurons that are narrowly tuned to frequency or amplitude, for example, those that might be found in primary auditory cortex, will undergo shifts in their CFs and even their optimal stimulus levels in response to behavioral training (Edeline, Pham, & Weinberger, 1993; Weinberger, 1997; Weinberger, Javid, & Lepan, 1993). Most of these changes appear to be shifts in selectivity within the limits of the "untrained" frequency-intensity response range. Figure 5–4 presents an idealized case. In the untrained animal, the spike count versus frequency function of a neuron shows some selectivity, and one can identify a "best" frequency to which the cell is most responsive. If the animal is then behaviorally conditioned so that an off-best-frequency stimulus takes on particular behavioral significance, then the best frequency shifts to match that of the relevant frequency. Most of the published cases are of instances in which the shift in preferred frequency remains within the limits of the effective frequency range in the untrained animal.

There is good evidence that even the excitatory frequency-intensity receptive field centered at CF itself is the envelope

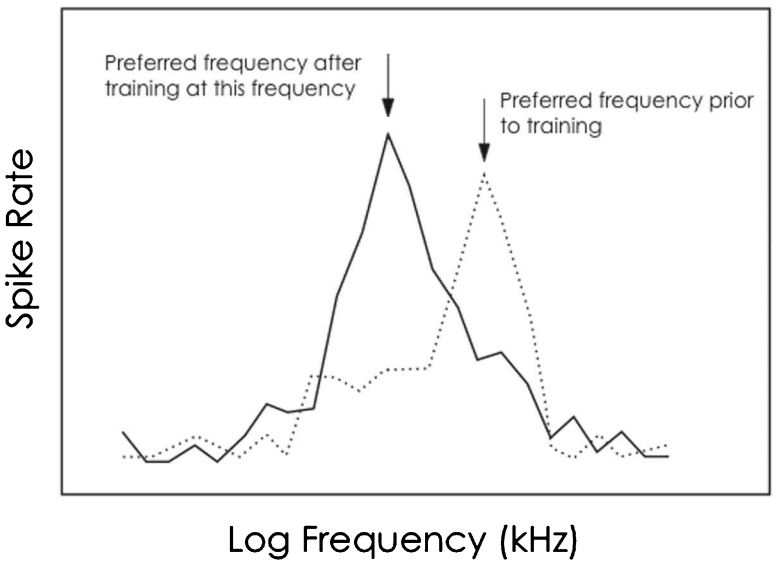

Figure 5–4. Idealized spike rate-versus-frequency functions of a single cortical neuron before and after behavioral conditioning at a single frequency within the effective response range. Note that both before and after conditioning, the neuron has a preferred frequency within its effective response range, but that the frequency preference within that range is malleable.

of convergent inputs (Phillips & Hall, 1992), so these behavioral conditioning-induced shifts might reflect a selection process exerted on the relative contributions of those inputs (after Suga et al., 1997; Zhang et al., 1997). The auditory system certainly has the machinery to do this. Recall that activity in the auditory cortex is under some degree of attentional control (e.g., Walker & King, 2011), that the auditory cortex is the origin of a massive set of direct or indirect descending projections to most of the auditory brainstem nuclei, and that these ultimately reach even the cochlea (Malmierca & Ryugo, 2011; see Phillips, Chapter 22 in this volume). These are highly topographically organized projections that complete "loops" between the cortex and the brainstem. One such loop is between the auditory cortex and the medial geniculate body; a second is formed by the ICC projecting to the thalamus, which projects to the cortex, which in turn projects back upon the midbrain. We know from electrical stimulation studies that focal cortical activity results in shifts of the tuning of ICC neurons, often toward the tuning at the site of cortical stimulation (see Suga, 2012 for detailed review). The same effects may be occurring in the cortex itself by means of horizontal connections across the cortical mantle. There is a sense in which the cortex may thus be said to self-select its inputs (Suga et al., 1997).

The factors enabling the occurrence of these effects and those determining the longevity of these changes are still being worked out. At least some of this plasticity may be enabled by cholinergic input from the nucleus basalis of the basal forebrain, because explicit pairing of electrical microstimulation of the nucleus basalis with acoustic tonal stimulation can cause these kinds of response selectivity changes, even without behavioral training (Weinberger, 1997). In this regard, exogenous acetylcholine paired with tonal stimulation also can produce stimulus-specific shifts in receptive field organization (Metherate & Weinberger, 1990). Interestingly, the cochlear lesion-induced tonotopic map reorganization appears not to depend on acetylcholine (Kamke, Brown, & Irvine, 2005), suggesting that more than one neurochemistry mediates auditory cortical plasticity.

Another expression of learning-induced plasticity has been revealed in some neurons of the secondary auditory cortical fields (Diamond & Weinberger, 1986). Neurons in these fields often have broad frequency tuning or undifferentiated, disorderly responses across the frequency domain. This broad tuning necessarily indicates a highly convergent afferent input. When a frequency within the response range is explicitly paired with a conditioning stimulus (forepaw shock), then a striking selectivity for the frequency of the behaviorally relevant stimulus can develop. It is reversed by extinction procedures. Once again, the neurons behave as if the behavioral conditioning process exercises some kind of selection process on the effective afferent input array.

Studies of learning-induced plasticity thus offer two means by which the representation of a stimulus in the cortex might be modified. Within a tonotopic field, there may be modest shifts in the stimulus selectivity of individual neurons whose frequency response ranges are close to the stimulus frequency of interest, so that the profile of activity aroused across the tonotopic field by a particular

stimulus becomes selectively stabilized or enhanced. Within a nontonotopic field containing very broadly tuned elements, behavioral conditioning can impose a selectivity on a previously undifferentiated pattern of responsiveness. Both of these expressions of plasticity may contribute to the formation of long-term "templates" for the recognition or discrimination of acoustic signals, including speech ones (after Blumstein & Stevens, 1980; Stevens, 1980). Thus, if speech perception is conceptualized as an on-line, somewhat context-dependent, matching of the acoustic signal with internal representations of phonetic spectral templates, then these learning-induced plasticities may be some of the factors that mediate template formation.

A further question concerns the effects of learning-induced plasticity on the internal organization of tonotopic (or other) cortical maps. Recanzone et al. (1993) trained primates in a frequency discrimination task and studied cortical neural physiology before and after successful training. They found expanded tonotopic representations of the test frequencies, although no significant changes in selectivity of frequency tuning, or changes in response latencies, of neurons in the reorganized area. A more recent study in cats (Brown, Irvine, & Park, 2004) showed no such change in tonotopy in animals successfully trained in a frequency discrimination task. That is, they showed changes in behavioral performance in the absence of overt changes in the neurophysiology of the primary auditory cortex. There is, of course, no special reason why the neurophysiological changes that mediate the behavioral ones need to be located in the primary auditory field. Nevertheless, these findings raise important questions about exactly what form will be taken by the plastic changes associated with auditory perceptual learning, and at what loci they will be found. We do not yet have complete answers to these questions.

Finally, there is the question of whether one should necessarily expect to see expansion of activated cortical territories in situations of successful learning. The development of proficiency at a given skill, especially one that requires the coordinated activity of many brain regions, may reflect an increase in neural processing efficiency. This could be expressed in an increased automaticity of processing and *less* cortical tissue devoted to the task, rather than more. Indeed, human brain imaging has provided an empirical precedent for this kind of hypothesis in the domain of language learning (Zhang et al., 2005). An unqualified adherence to a "bigger is better" view of brain function may therefore be unwise.

Temporal Processing and Developmental Language Disorders

Like "plasticity," "temporal processing" and variants of it like "rapid auditory temporal processing" are umbrella terms (McGettigan & Scott, 2012; Phillips, 2012). Rosen (1992) performed a valuable service in using the speech stimulus as a reminder of the various temporal grains of the stimulus and the information they carry. Thus, voice pitch information resides largely in glottal pulse rates; periodicities of this sort can be discriminated with close to tens-of-microseconds

resolution. Segmental (phonetic) information has a coarser grain (milliseconds to tens of milliseconds), and suprasegmental information (e.g., intonation contours) is expressed over even longer time scales. Stimulus dimensions with different temporal grains and time structures (e.g., transient vs. periodic) are likely encoded using different neural strategies, rendering them differentially susceptible to pathology (Phillips, 1995; Phillips & Hall, 1990). Thus, some neurological patients can show a deficit in the discrimination of phonetic information while having little trouble discriminating voice gender; others are unable to interpret prosody but retain the ability to extract the phonetic identity of a speech stimulus.

Sounds are, by their very nature, physical events distributed in time, and so there is a sense in which it is almost inevitable that a neural "sound" processing deficit is also a "temporal" processing deficit. Because the time structures of sounds come in so many forms, and because the processing of successive acoustic events is not independent for any of a number of reasons (temporal integration window width, masking time courses, adaptation, etc.), it is not particularly helpful to describe an impaired listener as having an impairment of temporal processing. Moreover, we need to distinguish the "processing of time" (that is, the establishing of a veridical neural-perceptual representation of the time structure of the stimulus) from the "time of processing" (i.e., the duration required to execute the relevant neural processes that lead to the perception of a specified acoustic event). This distinction is rarely made, and although the two processing deficits may often go hand-in-hand, in principle they need not do so. This point may be particularly relevant in studies of children, in whom the developmental improvement in perceptual performance across age may reflect changes in the "efficiency" of auditory processing in the presence of a steady adultlike temporal resolution (cf. Hill, Hartley, Glasberg, Moore, & Moore, 2004; Stuart, 2005; Stuart & Phillips, 1996).

One "rapid auditory temporal processing" deficit that has received much attention is that seen in some children with developmental language disorders. Let us use it as a medium for illustrating analytical approaches to understanding the nature of temporal processing problems. Tallal and Piercy (1973a, 1973b) showed that children with what was then called "developmental aphasia" had difficulty repeating the sequence of two brief, nonverbal sounds if the sounds were in close temporal proximity. Discrimination of the two sounds also was impaired under the same temporal contraints. Tallal and Piercy noted that if the two sounds were significantly lengthened or presented at wide intersound intervals, then repetition of the sequence, and discrimination of the elements in it, improved, often to normal. Thus, it was the conjunction of sound brevity and rapid temporal succession that was the source of the problem, and not either one of those parameters alone. The same authors went on to demonstrate that sequencing and discrimination impairments extended to stop-consonant perception, but not to the perception of steady vowels; this pointed to the possibility that the rapid sequence of events at stop consonant onset (onset burst, aspiration, formant transitions and their temporal relations) in some way exceeded the processing capacity of affected individuals. Interestingly, using synthetic speech sounds,

they showed that vowel-vowel syllables in which the duration of the first vowel approximated that of a stop consonant were subject to the same processing difficulty as were consonant-vowel syllables. By extending ("stretching") the consonant onset (formant transitions) in time, and thus providing more "time" for processing successive elements in the stream of sound, the sequencing and discrimination problems were overcome (Tallal & Piercy, 1974, 1975).

It thus seems that it was specifically not the "transitional nature" of consonant onsets that was the source of the perceptual difficulty, but the brevity of that event when it was immediately followed by another event (the vowel). Certainly the evidence presented was consistent with this view, though it would have been informative to know whether affected children had difficulty discriminating the properties of isolated frequency sweep stimuli. In any case, it is perhaps unlikely that the perceptual system tracks the successive periods in the formant transition waveform in an individuated way that results in a conscious percept of the glide direction in a speech sound; rather, the percept tends to be of a single event that has a particular quality that we label as "b," for example. This is somewhat akin to perceiving the location of a low-frequency sound source on the basis of ITDs: There is no sense in which we "hear" successive, individuated periods of the sound at each ear and their temporal relations. Rather we perceive a single event that has the quality of being "over there."

With the deficit specified in the way that Tallal and Piercy had, subsequent references to the problematic stimuli for language learning-impaired children being the "rapid formant transitions" are imprecise: Do we mean that the rate of change of frequency, on a period-by-period analysis, is too fast for the system to process? Or that the formant transition event is brief and in such close temporal proximity to other sounds that there is some interference with it being processed properly? These two questions speak to very different mechanisms, so the language with which we describe temporal processing and its disorders needs to be very precise.

Ambiguity in the use of terms arises again in the "magnocellular" theory of developmental language disorders. As mentioned earlier, the retinogeniculocortical visual pathway has at least two streams (magnocellular and parvocellular), distinguished by cell soma size and a number of physiological properties (Livingstone & Hubel, 1988). A "magnocellular theory of dyslexia" emerged from the fact that there is some evidence for a deficit selectively in the magnocellular processing stream of the visual system in dyslexics (Livingstone et al., 1991), that is, in the processing system most responsive to high stimulus temporal frequencies ("processing of time"), with the greater axonal conduction velocities ("time of processing"), and whose neurons have responses dominated by a transient component. The hypothesis has been extended to audition (Galaburda et al., 1994; Stein, 2001) because of the finding of a modest decrement in cell size in the medial geniculate body; it has done so, however, without any cautionary preamble that the auditory system does not have magnocellular and parvocellular streams of processing (or "transient subsystems") in the same sense that vision does. The magnocellular hypothesis has since evolved into a theory about large neurons and rapid processing, rather

than about specifically sensory processing per se (Stein, 2001). The intended connotations of the term "magnocellular hypothesis" have thus undergone significant change over time.

There has been recent attention on the ability of the auditory systems of language learning-impaired individuals to support the normal encoding of stimulus periodicities, especially periodic amplitude or frequency modulations of tonal or other carriers. Reasons for interest in this issue stem from the facts that the magnocellular hypothesis emphasizes "fast" stimuli and that the rate of change of stimulus events in spoken language is in the order of 2 to 50 Hz (e.g., Rosen, 1992), a range of temporal frequencies that is easily within range of the normal auditory system to encode. Note, however, that in the stream of sound that constitutes speech, it may be true that changes in envelope amplitude occur as fast as every 2 to 50 ms; but the events so spaced are spectrally different from each other (e.g., consonantal burst, onset of voicing), so there is no special burden on individual neurons to respond to all of them. This is a very different situation to that in studies of temporal frequency coding, in which the stimulus is strictly periodic (i.e., a repetition of the same waveform) and the same neurons likely contribute to the temporal response (see also Phillips, 1988). This means that studies of responses to periodic stimulus changes do not necessarily inform us about the mechanisms that encode aperiodic or discrete stimulus changes.

Stefanatos, Green, and Ratliff (1989) were among the first to provide electrophysiological evidence that the brains of developmental dysphasics were apparently less sensitive to periodic frequency modulations than were those of controls. This general point has been confirmed by others: There seems to be consistent evidence that the brains of dyslexics are poorer than those of controls at supporting electrophysiological responses to periodic FM or amplitude modulations (McAnally & Stein, 1996; Menell, McAnally, & Stein, 1999) and that dyslexics appear to be perceptually less sensitive to such modulations (Menell et al., 1999; Talcott et al., 1999; Witton et al., 1998). Interestingly, the behavioral deficits seem to be most marked at low modulation frequencies (e.g., Witton et al., 1998). Moreover, sensitivity to such modulations appears to be highly correlated with word and nonword reading performance (McAnally & Stein, 1996; Menell et al., 1999; Talcott et al., 1999; Witton et al., 1998). The relationship between sensitivity to low-frequency periodic FMs and reading performance extends to normal (i.e., unselected) children (Talcott et al., 2000; Witton et al., 1998).

These are fascinating findings in their own rights, and we have no particular wish to dispute them (but see Bishop, Carlyon, Deeks, & Bishop, 1999 and Van der Lely, Rosen, & McClelland, 1998 for evidence of developmental language deficits in the absence of concurrent —though not necessarily a history of earlier and predisposing—auditory temporal processing ones). However, in the context of the present discussion of the magnocellular hypothesis and rapid auditory temporal processing, two points warrant attention. First, periodic frequency modulations are not the same as the more discrete or singular frequency modulations that compose formant transitions in speech, so the relevance of any deficit in periodicity processing to phonology and speech perception is not immediately clear. In this regard, periodic ampli-

tude or frequency modulations are by definition not examples of "transients," despite assumptions or inferences to the contrary (e.g., Stein, 2001; Stein & Talcott, 1999; Stein & Walsh, 1997), so there is little sense in which sensitivity to such periodicities informs us about "transient" stimulus processing. Indeed, neurophysiological studies of single-cell coding of transient and periodic FMs indicate that the coding principles may be quite different (Phillips, 1988). This is an important point, because if the hypothesis is that the reading or language deficit is attributable to a "transient system" defect, then the probes of that system actually have to challenge transient coding efficiency; it is not at all clear that periodicity coding does this. It is possible that the magnocellular auditory subsystem hypothesis has confused "transient" in the sense of being concerned with the neural coding of transient stimulus features with "transient" in the sense of not responding in a sustained fashion.

Second, the impaired FM sensitivity was restricted to low FM frequencies of a tonal carrier (e.g., 2 Hz and 40 Hz, but not 240 Hz; Witton et al., 1998). Witton et al. (1998) point out that in the case of (only) the highest (240 Hz) FM rate, the distribution of energy in the stimulus exceeds a critical band, so that the discrimination of a tone with an FM from one without the FM could be based on spectral cues rather than on any tracking of the temporal variations in the stimulus. However, for the two low FM rates, a "rapid temporal processing defect" might be expected to affect responses to the higher of the two FM rates differentially. For example, if the defect in the system was in the form a temporal jitter in the neural code (after Miranda & Pichora-Fuller, 2002), then a constant level of temporal jitter would be larger in relation to the period of the high-frequency FM and so would be expected to affect performance at that FM rate differentially. In practice, however, the magnitude of the difference between dyslexic listeners' and normal listeners' FM detection thresholds was either comparable at the two low FM rates or was larger at the lowest rate (Witton et al., 1998). This may be somewhat awkward to explain for the magnocellular theory or for a rapid auditory temporal processing account of developmental language disorders.

Summary and Conclusions

Our goal in this chapter has been to provide a description of central auditory neuroscience sufficiently broad that the reader has a basis to pursue further study of the topic in its own right, and to provide a foundation for later chapters in this volume. As we worked our way through the ascending auditory sensory pathway, we saw some instances in which there are comprehensible links between the physical properties of a stimulus dimension, the neural representation of the stimulus dimension, the psychophysical correlates of that dimension, and the behavioral effects of damage to structures housing the neural representation. We have seen this in the domains of spatial hearing and sound time structure. We then explored the notion of neuroplasticity and found that term to be an umbrella one—it encompasses a form of plasticity driven by dramatic events during a critical period early in development, and also the plasticity that constitutes everyday perceptual learning in adulthood. In the

domain of auditory temporal processing and its involvement with developmental language-learning impairment, we saw another umbrella term. What, exactly, is "rapid auditory temporal processing"? Is it a reference to the efficiency or speed with which perceptual operations are performed or a reference to the time structure of the stimulus to be encoded? If the latter, do we mean "rapid" in the sense of high-frequency periodicities, or perhaps spectrally different transient events in close temporal proximity? There is good reason to believe that the mechanisms required for the perceptual timing of events in the same or different spectral (or binaural) channels are very different (Eggermont, 2000; Phillips et al., 1997), differentially relevant to speech processing (e.g., Phillips & Smith, 2004), and differentially susceptible to the effects of aging (Lister, Besing, & Koehnke, 2002; Lister, Koehnke, & Besing, 2000). Whenever a term has more than one meaning, it is crucial that the intended meaning be specified precisely. If there is a single general lesson to be learned from this chapter, it is that the most comprehensive understanding of auditory processing is going to depend on detailed, comprehensive knowledge of the stimulus, the neural representation of the stimulus, and the psychological dimensions of the percept aroused by the stimulus.

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CHAPTER 6

CENTRAL AUDITORY PROCESSING AS SEEN FROM DICHOTIC LISTENING STUDIES

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Background and Introduction

This chapter presents a selective overview of research on central auditory processing as seen from studies using dichotic listening and structural and functional magnetic resonance imaging (sMRI and fMRI), respectively, as behavioral and neuronal vehicles for the understanding underlying factors and mechanisms. The chapter is based on a series of lectures given by Kenneth Hugdahl at conferences and meetings, and overlaps somewhat with a similar chapter by Hugdahl (2012, in press). The research being reviewed has primarily been conducted in our auditory neuroscience laboratory at the University of Bergen, Norway, with variants of dichotic presentations of simple speech sounds, like consonant-vowel (CV) syllables, with different syllables being presented to the right and left ears on each trial. In the chapter we will particularly discuss how central auditory processing is shaped by the nature of the sound stimulus to be processed, and how this is systematically varied by changing parameters like voice-onset-time, and intensity and phase shifts of the dichotic stimuli.

In addition, we review research that has investigated how central auditory processing may be modulated by higher cognitive processes, like attention, working memory, and executive functions, also called cognitive control. This latter phenomenon was labeled the "forced-attention dichotic listening paradigm" by Hugdahl and Andersson in a 1984 *Cortex* paper where it was shown that instructing subjects to selectively listen to and report only the right (or left) ear stimulus in the dichotic situation had profound effects on how such relatively low-level processing stimuli like CV syllables were processed (cf. Bryden, Munhall, & Allard, 1983; see Hugdahl et al., 2009 for a more recent introduction and update to the forced-attention dichotic listening paradigm).

In a final section, we provide an overview of research in our laboratory where the paradigm has been applied to clinical disorders, including both child and developmental disorders, like dyslexia and other speech/auditory problems (e.g., Helland, Asbjornsen, Hushovd, & Hugdahl, 2008), as well as adult disorders, like auditory hallucinations in schizophrenia (Hugdahl et al., 2009, 2012).

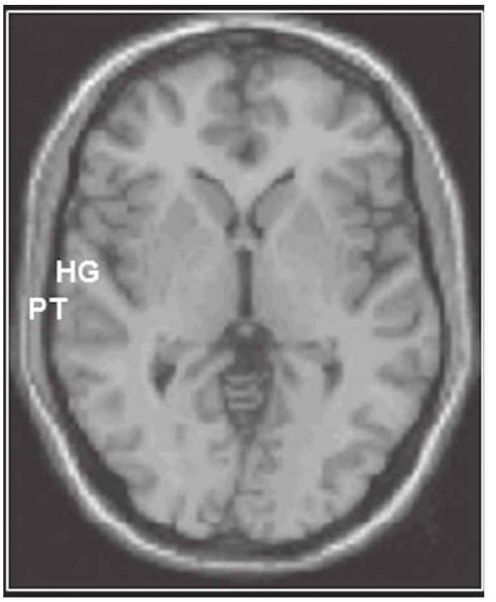

Figure 6–1. Anatomy of the Heschl's gyrus (HG) and planum temporale (PT) in the temporal lobe plan seen in a horizontal MRI section in an adult individual, revealing the typical asymmetry of these brain structures.

Brain Lateralization and Central Auditory Processing

Primary auditory cortex is located in the transverse gyrus, Heschl's gyrus in the upper posterior part of the temporal lobes (Figure 6–1), with the speech perception area, the planum temporale, just posterior to the Heschl's gyrus in the temporal plane. Heschl's gyrus is the cortical area that functionally corresponds to auditory perception and sound localization and is thus part of the primary auditory cortex. It is localized transversely across the upper part of the superior temporal gyrus in the temporal lobes, along the upper bank of the sylvian fissure. The transverse extension of Heschl's gyrus is functionally organized according to the principle of tonotopy, which means that neurons at the lateral and medial ends of the gyrus are selectively responsive to low versus high frequencies. Recent fMRI studies (e.g., DaCosta et al., 2011) have shown that the primary auditory cortex within Heschl's gyrus is organized as different tonotopic maps in humans, perpendicular to the shape of Heschl's gyrus.

What is complicating the exact demarcation of the functional specificity of dif-

ferent regions in Heschl's gyrus is the fact that its shape varies extensively across individuals (Leonard, Puranik, Kuldau, & Lombardino, 1998; Rademacher et al., 2001), with a single, duplicated, or even triplicated gyri. Although its borders are sometimes difficult to establish, it is commonly believed that the primary auditory cortex is in the most anterior, or first, gyrus in individuals with multiple gyri (Rademacher et al., 1993). When discussing the neuroanatomy of of central auditory processing, and in particular speech sound processing, it is impossible not to discuss the role of hemispheric asymmetry and laterality (see Hugdahl, 2010 and Tervaniemi & Hugdahl, 2003 for an introduction to the laterality of auditory processing and speech perception).

Asymmetry of Heschl's gyrus has been observed (e.g., Warrier et al., 2009), with larger left-to-right asymmetry and speech sound processing. Moreover, Leonard et al. (2001) found increased frequency of duplications (i.e., multiple gyri and sulci) in children with language and reading disorders. Similarly Heiervang et al. (2000) reported reduced asymmetry of cortical areas in the posterior upper bank of the temporal lobe in dyslexic children. Changes in the anatomy of Heschl's gyrus and the adjacent planum temporale has also been observed in patients with schizophrenia (Arango et al., 2012; Neckelmann et al., 2006) with reduced overall area volume and asymmetry in early psychosis patients, particularly in the left hemisphere. An interesting extension of these observations is whether schizophrenia patients with frequent auditory hallucinations, in the sense of experiencing *hearing voices*, also are the patients with the largest Heschl's gyrus volume reduction, and absence of a left-to-right asymmetry. Interestingly, Dierks et al. (1999) found that structural changes in Heschl's gyrus and adjacent areas had functional consequences in that there was a negative correlation between auditory hallucinations and reduced auditory cortex volume (see also Gavrilescu et al., 2010 for review). Finally, Murat Özgören and colleagues at the Dokuz Eylul University, Izmir, Turkey (unpublished data) have shown that asymmetry of Heschl's gyrus, with typically the left side being larger than the right side, is significantly reduced in patients with schizophrenia and that this correlated with frequency of auditory hallucinations, and affected performance on the dichotic listening task with CV syllables. Thus, it is obvious that Heschl's gyrus volume is a structural correlate to efficient central auditory processing.

Planum Temporale

An area closely related to the Heschl's gyrus, both structurally and functionally, is the planum temporale area. The planum temporale is a triangularly shaped area about 3 × 3.5 cm, extending posteriorly along the sylvian fissure from the posterior border of the Heschl's gyrus up to the descending ramus of the Sylvian fissure posteriorly (see Figure 6–1). The planum temporale typically shows a marked asymmetry, with the volume on the left side being about 30 to 35% larger than the corresponding volume on the right side (Geschwind & Levitsky, 1968).

Functionally, the planum temporale has traditionally been considered the speech perception area proper, and the left-to-right asymmetry is the structural underpinning of speech perception, overlapping anatomically with Wernicke's area. Several studies have shown that

children and adults with language and language-related problems and aberrant central auditory processing also have abnormalities of the planum temporale area (Heiervang et al., 2000; Leonard et al., 1998). More recent studies, using fMRI and related technologies, have shown, however, that the planum temporale area may not be the most critical single area in the brain for speech perception, since studies have shown increased activation in areas more ventral and inferior to the planum temporale, such as the superior temporal sulcus, when contrasting activation to speech and nonspeech auditory stimuli (Binder et al., 1997). Other studies, on the other hand (van den Noort, Specht, Rimol, Ersland, & Hugdahl, 2008) have shown peak activations in the left planum temporale to dichotic presentations of CV syllables, and Boatman (2004), in a review of direct cortical stimulation studies, showed that stimulating the planum temporale area affected phonological decoding of speech relevant material, occurring in advance of access to other language systems, such as semantics and syntax. Stimulation of Heschl's gyrus also affected phonology, but was more related to acoustic processing. Stimulation of the superior temporal sulcus and adjacent areas ventrally and more inferiorly to the planum temporale resulted in interference with the lexical-semantic interface. Thus, at present it is not clear what the exact role is of the planum temporale in central auditory processing and speech perception.

Nonhuman Studies

A clue to such an understanding could be provided from nonhuman studies, and in particular studies of anatomical asymmetry in the primate brain. In a now classic study, Gannon, Holloway, Broadfield, and Braun (1998) found an even greater asymmetry of the planum temporale area, favoring the left side, in the chimpanzee brain, compared with comparable human studies (e.g., LeMay, 1985). In a more recent overview of brain asymmetries in nonhuman species, and in particular in primate apes, Gannon (2010) notes the methodological complications and differences between laboratories, making it difficult to draw firm conclusions about whether primates have greater planum temporale asymmetry than humans, or more precisely whether such asymmetry is expressed to a larger extent in the nonhuman species.

A conclusion that can be drawn, however, from the review of existing data in the Gannon (2010) paper is that language areas in the human brain, including the planum temporale and adjacent areas "have been around for a long time" (p. 57) and that the anatomical asymmetry in the speech and language areas in the human brain has an evolutionary history in our closest ancestors down the phylogenetic tree.

Central Auditory Processing and Central Auditory Processing Disorders

The current chapter has to do with central auditory processing disorders as seen from dichotic listening studies. In this section we present an overview of what we mean by central auditory processing and central auditory processing disorders. Central auditory processing is defined as the auditory system mecha-

nisms and processes responsible for a range of behavioral phenomena, such as sound localization and lateralization, auditory discrimination, auditory pattern recognition, temporal aspects of audition (e.g., temporal masking) (ASHA, 1995). Other definitions emphasize both lower and higher levels of auditory processing and define central auditory processing as "the brain processes forming the biological substrate of . . . auditory perception, the different forms of auditory memory, as well as the attentional processes controlling for the access of auditory sensory input to conscious perception and higher forms of memory" (Näätänen, Paavilainen, Rinne, & Alho, 2007).

A commonly used experimental task for the study of central auditory processing mechanisms incorporates competing acoustic inputs, to affect different areas of the brain and processing (e.g., speech and language functions). Such a situation is particularly relevant for the present chapter, since the dichotic listening situation is tailored to study the effect of two competing acoustic inputs, with two stimulus elements being presented at the same time. A unique feature of the dichotic listening situation is that one of the stimulus elements (i.e., the right ear syllable) is perceptually a strong element that will be preferred for processing, before the left ear syllable, which is the weak element (see below for further details regarding the dichotic listening protocol). As noted by Musiek and Weihing (2011), individuals with auditory cortex lesions may sometimes show impaired performance bilaterally on the dichotic listening test, which is alleviated through commissurotomy. In this case, there is a clear reduction in left ear reports, which could be understood as release from stimulus competition, since the left ear signal cannot pass through the corpus callosum to be processed (Baran & Musiek, 1999; Musiek, Reeves, & Baran, 1985).

From a definition of central auditory processing as the mechanism involved in auditory processing, it follows that there will also be central auditory processing disorders, defined as functional auditory performance deficits (ASHA, 1995). Such disorders will affect other mental faculties related to language and reading development; thus, understanding the mechanisms, and in particular the neuroanatomical and neurophysiological mechanisms involved in central auditory processing and disorders may be important intermediate factors in understanding language- and reading-related disorders.

Dichotic Listening with CV Syllables

As introduced above, a dichotic listening situation will tap basic processes and mechanisms in central auditory processing, and could also act as a tool for diagnosis and treatment/training of individuals with central auditory processing deficits (Moncrieff & Wertz, 2008; Weihing & Musiek, 2007; see also Soveri et al., in press, 2012). In this section, we outline the general properties and processes involved in dichotic listening, as well as how to perform the test. In the next section, we review validation and reliability studies of the paradigm. We have used this paradigm during the last 30 years to study central auditory processing, speech perception, hemispheric asymmetry, and higher cognitive functions in healthy and clinical groups (see Hugdahl & Andersson, 1984 for the first dichotic listening study from our laboratory).

Dichotic presentation involves the simultaneous presentation of two different and competing auditory stimuli. This is a different situation compared with a binaural (i.e., diotic) stimulus presentation, which means that the same stimulus is presented to both ears at the same time. The dichotic stimulus presentation technique was first introduced into experimental psychology by Donald Broadbent (1958) for the study of attention and attention shifts in the auditory modality. It was Doreen Kimura (1961, 1967) who adopted the method into neuropsychology for the study of auditory processing deficits in patients with epilepsy, and subsequently for the study of hemispheric asymmetry and brain laterality (see also Kimura, 2011 for an historic overview of the dichotic listening method in clinical neuropsychology). In the dichotic listening task developed at the University of Bergen (e.g., Hugdahl & Andersson, 1984; 1986), we use pairwise presentations of CV syllables, made up of the six stop-consonants /b/, /d/, /g/, /p/, /t/, /k/ that are paired with the vowel /a/, thus constituting pairs of CV syllables of the type /ba/ - /pa/, /da/ - /ka/ and so on., using all possible combinations (also including the homophone pairs, /ba/ - /ba/, /da/ - /da/ etc.) of the six syllables (a variant originally introduced by Studdert-Kennedy & Shankweiler, 1970).

The Right Ear Advantage

The most simple variant of the CV syllables dichotic listening task is to instruct subjects to report after each syllable presentation the syllable they heard best or first. As shown in numerous replications, presenting CV syllables dichotically causes a strong perceptual effect of more correct reports for the right ear syllable compared with the left ear syllable in healthy individuals. This is called a right ear advantage (REA) and is one of the most replicated findings in experimental psychology. The REA is thus a very robust baseline phenomenon against which different auditory processing deficits can be evaluated. The REA is caused by the preponderance of the contralateral auditory pathways, and the asymmetry of speech processing, favoring the left hemisphere, and the right ear stimulus. Figure 6–2 illustrates the neuroanatomy and neurophysiology of the REA (Hugdahl, 2011; Kimura 1967, 2011).

Kimura (1967) published an influential paper regarding the underlying neuroanatomy and physiology of the REA (see also Brancucci et al., 2005 for a more recent update). According to Kimura, the REA is caused by the direct access from the right ear to the left, language-dominant, temporal lobe because of the preponderance of the contralateral audi-

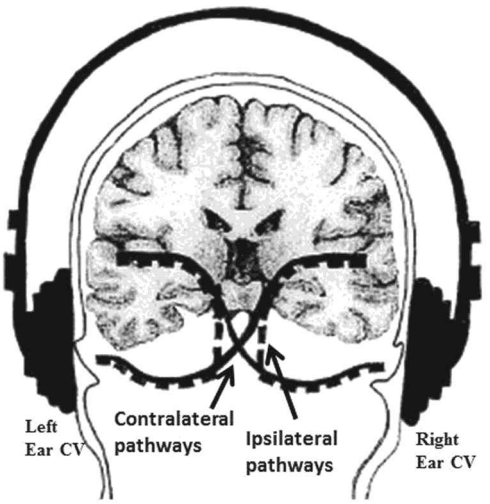

Figure 6–2. Schematic illustration of the neural pathways underlying the right ear advantage (REA) in dichotic listening.

tory fibers from the cochlear nucleus in the inner ear to the auditory cortices, while the left ear stimulus is transferred from the right, nondominant, hemisphere across the corpus callosum to the speech processing areas in the left hemisphere. In other words, the contralateral stimuli from the right and left ears have direct access to the left and right temporal cortices, respectively, due to the relative suppression of ipsilateral stimuli in the dichotic listening situation.

Although the right ear stimulus has direct access to the left hemisphere, the left ear stimulus has direct access to the right hemisphere, and since the left hemisphere is dominant over the right hemisphere for processing speech stimuli, the right ear stimulus will be preferentially processed before the left ear stimulus, thus the REA. Pollmann et al. (2002) nicely corroborated the Kimura (1967) model in a lesion study with patients with circumscribed vascular lesions to either the anterior, mid, or posterior part of the corpus callosum, who were tested with the Hugdahl version of the CV syllables dichotic listening paradigm. Since the auditory fibers cross over in the posterior third of the corpus callosum (Pandya, Karol, & Heibroun, 1971), Pollmann et al. (2002) predicted that patients with posterior lesions (preventing the signal from the left ear to be transferred to the left temporal lobe for processing) would show a stronger REA than patients with anterior lesions (leaving the posterior fibers intact for signal transfer). The results confirmed the prediction with an almost 100% REA in the patients with posterior lesions. The study by Pollmann et al. (2002) revealed another aspect of central auditory processing and speech perception, namely that CV syllables seem not to be processed at all in the right hemisphere, since almost no correct reports were observed for the left ear syllable in patients with posterior callosal lesions (see Pollmann, 2010 for an updated explanation of the REA in dichotic listening, also considering the role of attention). Figure 6–3 shows the basics of the results in the Pollmann et al. (2002) study.

Absence of an expected REA can therefore be due to a processing deficit originating in the left temporal lobe, to failure of ipsilateral suppression, or to a callosal signal transfer deficit, conceptualized as a transmission failure (Speaks, Gray, & Miller, 1975).

Extending Traditional Views of the REA

Traditionally, the CV-syllable dichotic listening paradigm has been used to assess normal and pathological lateralization for verbal stimuli, and the REA has subsequently been seen as evidence for a left hemisphere dominance or specialization for speech sound processing, or in current terminology, for a left hemisphere dominance for central auditory processing when speech sounds are involved (see Lezak, Howieson, Bilger, & Tranel, 2012 for a standard view of dichotic listening as a tool for assessing hemispheric dominance). Such a view is, however, too narrow and limited with regard to the possible applications of a dichotic listening approach (see Hugdahl et al., 2009).

First, the REA reflects cortical involvement in auditory processing (Tervaniemi & Hugdahl, 2003; van den Noort et al., 2006) and may thus be a behavioral correlate of central auditory processing disorders. Second, as described in more detail below, the REA is modulated by higher cognitive processes, like attention

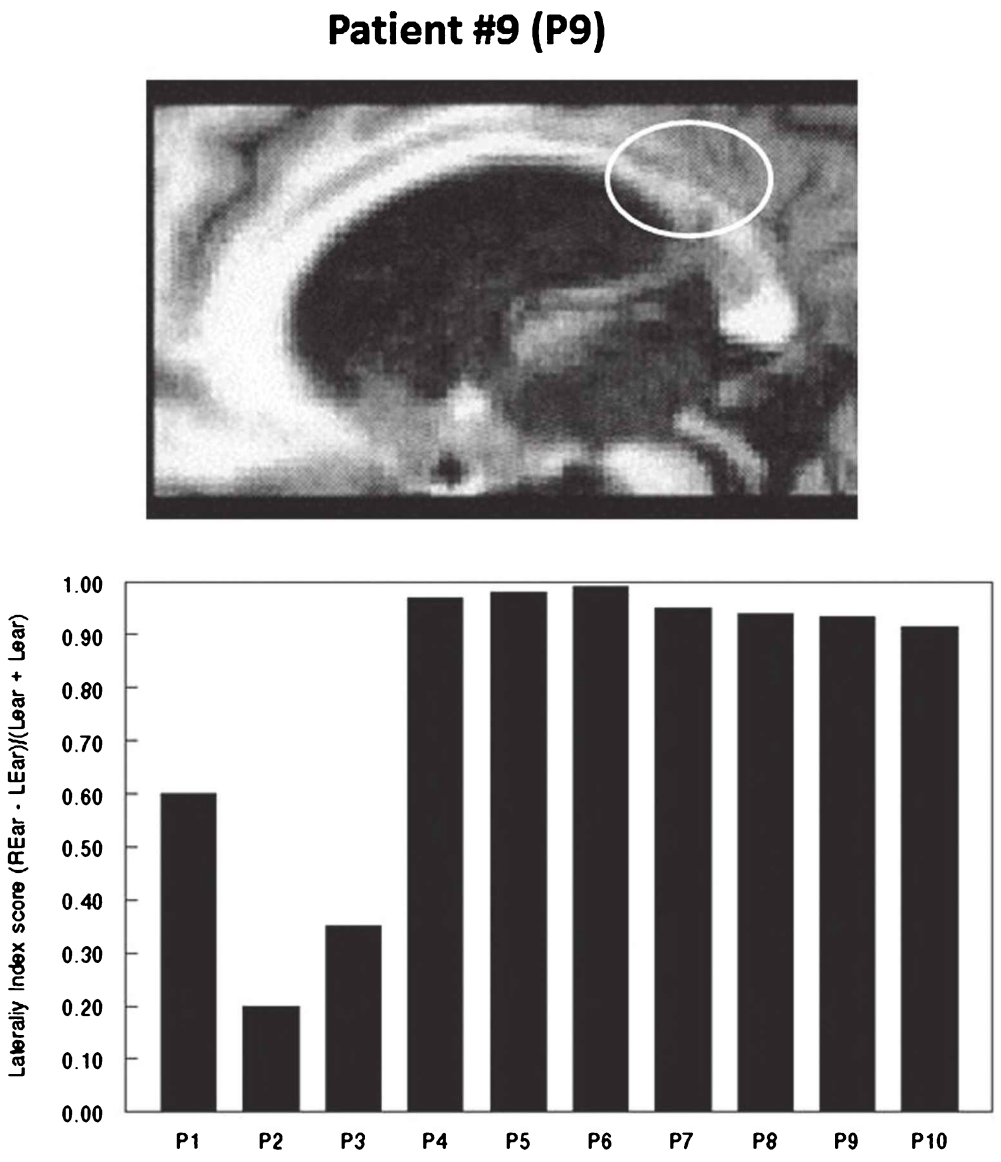

Figure 6–3. Performance on the CV syllables dichotic listening task in patients with circumscribed callosal lesions. Upper panel shows MR image of the corpus callosum in a patient (#P9) with a posterior callosal lesion. Lower panel show magnitude of the REA based on laterality index scores (1.0 = 100% REA, see formula on the *y*-axis title). The *x*-axis show the 10 patients (P1–P10) arranged from left to right according to whether their lesions were anterior (most leftward) or posterior (most rightward). Figure redrawn from data in Pollmann et al. (2002) with permission from the publisher.

and executive control, thus the dichotic listening paradigm can show the interaction of bottom-up perceptual and top-down cognitive factors in central auditory processing disorders, a topic sometimes ignored in the literature.

The University of Bergen Dichotic Listening Test and Database

Having collected normative data from more than 1800 subjects (children to adults, right- and left-handers, males and females), we have converted the paradigm into a regular neuropsychological test, with norms and structured instructions for use. The purpose for doing this has been to provide the interested researcher and/or clinician with an easy-to-use tool for assessing speech laterality and hemispheric dominance, as well as other aspects of central auditory processing (to be discussed below), for clinical and research purposes. The test comes either as a compact disc to be used on any standard PC or CD player, or as a plug-in program to the E-prime programming platform, also for use on a PC. The E-prime version has the advantage of automatic scoring of a range of response parameters not available in the CD version. On the other hand, the E-prime version requires that the E-prime programming platform be installed and that the clinician or researcher have knowledge of the E-prime programming structure. The interested clinician or researcher or clinician can get the test by sending an e-mail to hugdahl@psybp.uib.no., specifying which version is requested. A problem with earlier versions of the task was the exact timing and synchronization of the stimulus onset between the left and right channels. This was a major technical challenge in the early days with the use of reel-to-reel tape recorders. Today, with access to advanced sound processing software that runs on any standard PC or laptop, this is no longer a problem. In the last year, we have even developed the test as an iPhone app that can be downloaded for free from the App Store and run from an iPhone, iPod, or iPad. After taking the iPhone app test, the results can be submitted over the Internet to a secure server at the University of Bergen, which allows us to collect data from all over the world for a truly international database, unprecedented in neuropsychology. The iPhone app was originally suggested by Ph.D student Joseph Bless in our research group (Bless, Westerhausen, Kompus, Gudmundsen, & Hugdahl, 2011). Figure 6–4 illustrates the technical development in stimulus recording and presentation from reel-to-reel tape recorders to the use of iPhones or iPads.

Data Collected From All Over the World

Some 20 years ago we started to systematically collect data into a database, using the classic "forced-attention" CV syllables paradigm (Hugdahl & Andersson, 1986). Soon thereafter, data were submitted to the database from other research laboratories in other countries, which all used the same basic stimulus setup and procedural instructions. As of 2012, the database consisted of 1800 healthy subjects, aged 7 to 89 years, males and females, right- and left-handers, with extensive and detailed norms for different age-groups, gender, and handedness. An element of variability is entered into a database when the data are supplied from many different laboratories, with variation in equipment, and subtle differences in how the studies were performed. Traditionally, such variance would be unwanted and every effort would be taken to reduce it. We take an opposite view on such multiplicity

. . . the original equipment

. . . and the latest

Figure 6–4. Illustration of the development of the technical tools used in dichotic listening research, from the early reel-to-reel tape-recorders, to today's use of the "iDichotic" iPhone app, that can be downloaded to any iPhone, iPod, and iPad from the Apple App Store.

of data sources. That the REA effect "survives" despite the noise entered into the database is a sign of the robustness of the paradigm and the data. To have data averaged across geographic sites and disciplines is also an advantage of the Bergen dichotic database, since the database contains the natural variability that inevitably occurs in clinical settings (e.g., applying the paradigm for the study of central auditory deficits, or dyslexia).

REA Validation

The classic method of validating neuropsychological tests and paradigms is to compare performance on the test in patients with defined lesions with critical parts of the brain predicted to be necessary for performance of the function addressed by the test. When it comes to validation of speech lateralization,

the sodium amytal, or *Wada test*, after the pioneering work by Juan Wada and colleagues (Wada & Rasmussen, 1960), remains the gold standard of laterality. Sodium amytal is a barbiturate that temporarily sedates one hemisphere at a time for about 7 to 8 minutes when injected into the bloodstream to the brain. By investigating language function when sedating the left and right hemispheres separately, it is possible to determine lateralization of speech and language on an individual basis.

The results from a Wada investigation can be compared with the results from the dichotic listening investigation and the overlap between left hemisphere language localization and the frequency of the REA can be determined. A Wada test validation was made in a study in Gothenburg, Sweden (Hugdahl, Carlsson, Uvebrant, & Lundervold, 1997), where adolescent patients with epilepsy were tested with the dichotic listening CV-syllables test after having the side of speech representation determined with the Wada test. The results, shown in Figure 6–5, reveal that the dichotic listening procedure correctly classified all except one of the left hemisphere speech dominant subjects, and all of the three right hemisphere dominant subjects. Thus,

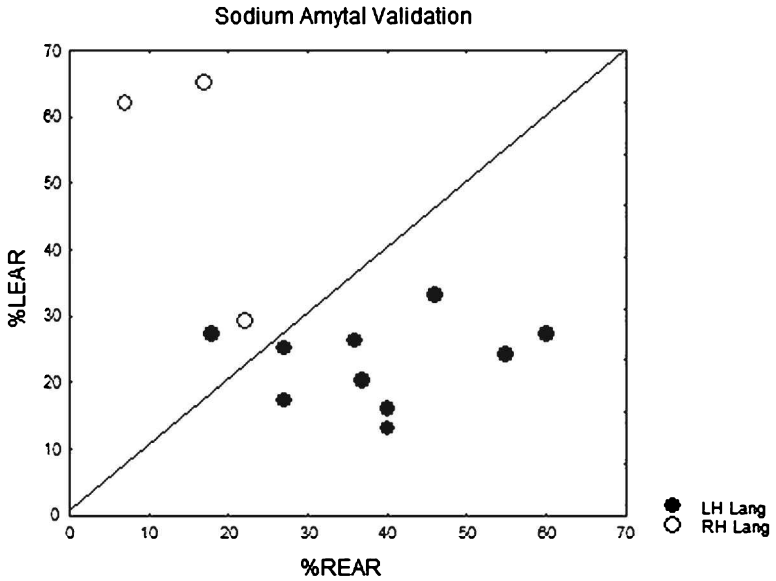

Figure 6–5. Validating performance on the dichotic listening task with performance on sodium amytal (Wada) test in 11 adolescent individuals before surgical treatment for epilepsy. Scatter plots of performance results for the right and left ear syllables, respectively. The 45-degree line is the symmetry line, all subjects below the line show a REA, all subjects above the line show a LEA. Black dots = Subjects with left hemisphere language as determined from the sodium-amytal test, open circles = Subjects with right hemisphere language as determined from the sodium amytal test. Figure redrawn from data in Hugdahl et al. 1997, with permission from the publisher.

lesion studies have corroborated the behavioral REA as a valid indicator of speech lateralization.

Neuroimaging: PET and fMRI

The REA also has been validated by applying the dichotic listening test while the subject is in the scanner. There are several imaging studies that have validated the REA. The first was a PET activation study by Hugdahl et al. (1999) that showed significantly stronger activation in the left planum temporale area compared with the homologous area in the right hemisphere to repeated presentations of CV syllables. Interestingly, the results revealed stronger activation in the right planum temporale area to short musical chords from different musical instruments. Thus, the Hugdahl et al. (1999) study revealed a double dissociation effect, a left hemisphere effect for speech stimuli and a right hemisphere effect for musical stimuli. Other fMRI studies also have shown a similar left hemisphere effect for dichotic presentations of CV syllables (e.g., dos Santos-Sequiera, Specht, Moosmann, Westerhausen, & Hugdahl , 2009; van den Noort et al., 2006; see also Tervaniemi & Hugdahl, 2003).

The REA and Subsyllabic Stimuli

Rimol, Specht, Weis, Savoy, and Hugdahl (2005) asked the question whether the imaging asymmetry observed for processing of CV syllables also could be observed at lower levels of central auditory processing using fMRI. Rimol et al. (2005) separated the consonant and the vowel components of the CV syllable and presented the isolated consonant sound only. Extracting the consonant phoneme of a CV syllable is a technically advanced matter, since the sound will resemble an isolated "click-sound." Rimol and colleagues (2005) interviewed the subjects after the study and made sure that they had perceived the sounds as consonants and vowels, respectively. The results, seen in Figure 6–6, reveal an even stronger asymmetry, favoring the left hemisphere, for an isolated consonant sound compared with the asymmetry seen to the whole CV syllable.

As seen in Figure 6–6, although the CV syllable produced an asymmetry, there was activation also in the right hemisphere, which was absent for the consonant sound. It can be questioned whether the right hemisphere activation to the CV syllable is produced by the vowel element in the syllable, with its characteristic prosodic aspect, which would resemble the right hemisphere activation observed to the musical chords in the Hugdahl et al. (1999) study. The categorical left-sided activation to an isolated consonant phoneme would indicate that the neuronal specialization for speech perception begins at the lowest possible level of central auditory processing, being present even at the level of single phoneme processing. As we see in the next section, an asymmetry for speech perception may be present even before phoneme processing, in the "gray zone" between acoustic perception and speech processing, with effects being observed for the voicing parameter in the transition from the consonant to the vowel when a syllable is spoken.

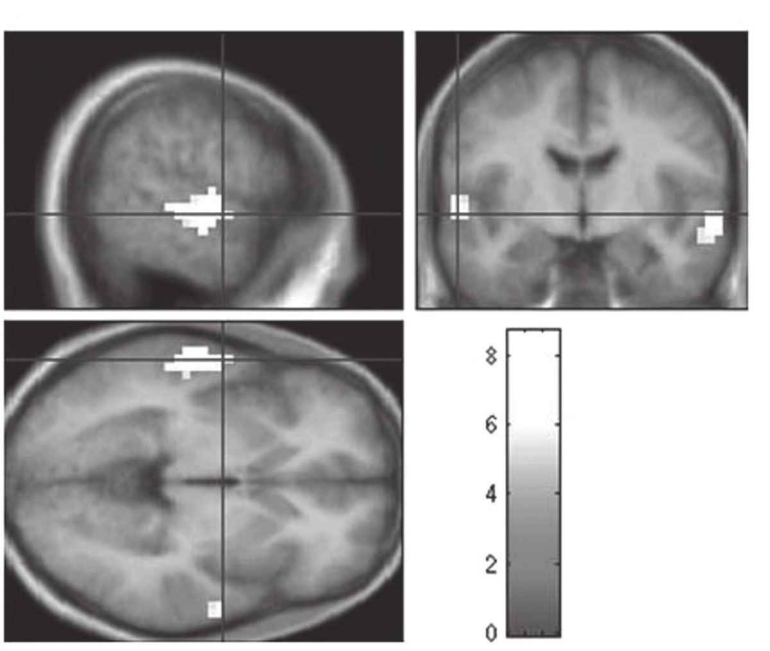

Figure 6–6. fMRI brain activation in the temporal lobes to binaural presentations of CV syllables /pa, ta, ka/ (syllabic), and isolated consonant-sounds /p, t, k/ (subsyllabic). Figure redrawn from data in Rimol et al. (2005) with permission from the publisher.

Voicing and Voice Onset Time

Voicing is a term in phonetics to denote the difference in onset time (also called voice onset time [VOT]) for the pulsing of the vocal folds following the onset of the consonant segment when pronouncing a stop-consonant syllable. Unvoiced CV syllables, like /pa/, /ta/, /ka/, have long VOT (75–80 ms) compared with voiced CV syllables, like /ba/, /da/, /ga/ (VOT = 20–25 ms).

Combining an unvoiced syllable in the right ear and a voiced syllable in the left ear of the dichotic pair produces a large REA, while the reversed combination, an unvoiced syllable in the left ear and a voiced syllable in the right ear, produces a left ear advantage (LEA; i.e., better performance to the left ear stimulus). The two conditions with equally voiced syllables, either unvoiced–unvoiced or voiced–voiced, in the left and right ears, respectively, produce intermediate sized REAs. It is frequently observed that boys lag behind girls in reading development (Kristoffersen, Simonsen, Eiesland, & Henriksen, 2012). One hypothesis to explain this lag in development may focus on the ability to identify the corresponding phonological correlates of the visual input when attempting to read. Operationally speaking, phonological decoding of graphemes when reading requires correct identification of the parameters involved in phonology, including correct identification of the voicing feature of syllables.

Differences Between Boys and Girls in Reading Development

We have studied differences between boys and girls by looking at how the various combinations of voiced and unvoiced syllables in the dichotic syllable pairs are processed by children between the age of 5 and 8 years. We had the unique opportunity to longitudinally follow a group of children between the age of 5 to 8 years who were tested each year with the standard CV syllables dichotic listening test (see Westerhausen, Helland, Ofte, & Hugdahl, 2010a). We believe that the ability to modulate the REA as a consequence of the combination of voiced and unvoiced syllables reveals a first-level manipulation of the phonetic features of speech sounds.

The ability to modulate the size and direction of the ear advantage in dichotic listening can therefore be regarded as a marker of basic phonological awareness. Alternatively, the voicing effect can be seen as a subphonetic manipulation of acoustic features of the signal (e.g., the slope of onset of the consonant segment of the syllable in its initiation). As an acoustic manipulation, the voicing effect can be seen as a prerequisite for phonetic processing. The data shown in Figure 6–7 illustrate how phonological awareness is developed in young children from the age of 5 to 8 years, and the different trajectories in development for girls and boys, with the boys seemingly lagging the girls by about two years. The data in Figure 6–7 show that both girls and boys at age 8 show an REA when the CV syllable is composed of an unvoiced syllable in the right ear and at the same time a voiced syllable in the left ear (VU combination in the figure), and LEA when a voiced syllable is presented in the right ear and an unvoiced in the left ear (UV combination in Figure 6–7).

VOT in Children at Risk for Dyslexia

We suggest that the systematic variation of the voicing parameter to elicit either

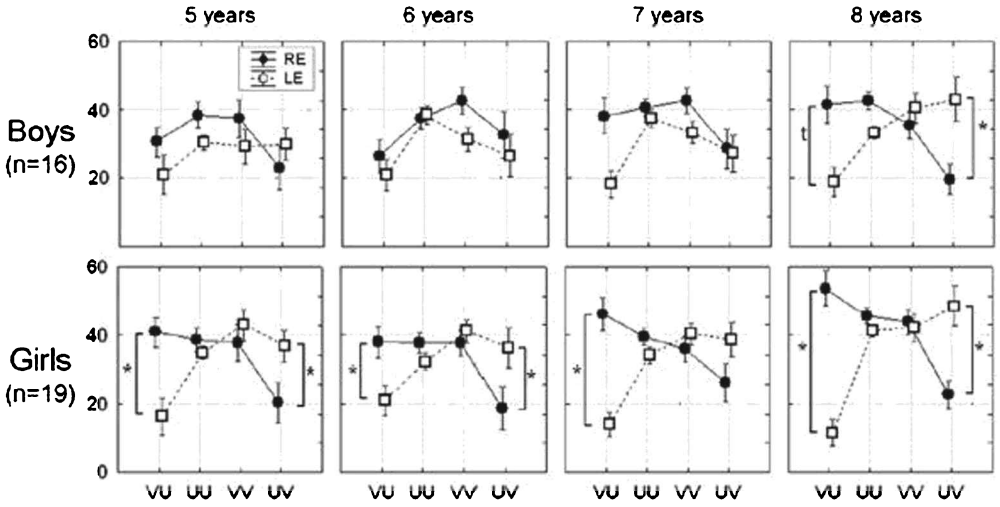

Figure 6–7. The effect of voicing and VOT on performance on the dichotic listening CV syllables task during repeated tests from the age of 5 to 8 years, and separated for girls and boys. SL = Voiced CV (Short VOT) in the left ear and Unvoiced CV (Long VOT) in the right ear, LL = Unvoiced CV (Long-Long VOT) in both ears, SS = Unvoiced CV (Short - Short VOT) in both ears, LS = Unvoiced CV (Long VOT) in the right ear and Voiced CV (Short VOT) in the right ear. RE = Right Ear, LE = Left Ear. Y-axis = % correct reports. Data from Westerhausen et al. (2010) with permission from the publisher.

a REA or a LEA may be a test of low-level aspects of central auditory processing and that failure to modulate the ear advantage in children older than 8 years may be a sign of a central auditory processing deficiency (cf. Hugdahl, 2012 in press). An example of this is seen in Figure 6–8, which shows differences in the ability to correctly identify unvoiced and voiced syllables in the right and left ears in children at risk for developing dyslexia.

The data are part of a larger study of risk factors for development for dyslexia, called "The Speak Up!" project, headed by Turid Helland, University of Bergen (see Helland, Ofte, & Hugdahl, 2006). Children at age 5 were screened with a questionnaire developed for the identification of risk factors for dyslexia, and divided into an at-risk group and a control group. The questionnaire contained questions about genetic factors in the family, health development, language, and motor development (Helland, Plante, & Hugdahl, 2011). Figure 6–8 shows that children at risk for developing dyslexia fail to show a LEA to the syllable combination with an unvoiced syllable in the left ear and voiced syllable in the right ear, as control subjects did, even at the age of 8 years. Thus, the VOT data reveal that dyslexia involves an inability of decoding the phonological structure of syllables and words, and that this may be a risk factor for dyslexia if identified at an early age. The data in Figures 6–8a and 6-8b show that failure to demonstrate an expected VOT effect in the dichotic situation appears at an early age before literacy, and that boys at risk for dyslexia fare worse than girls at risk.

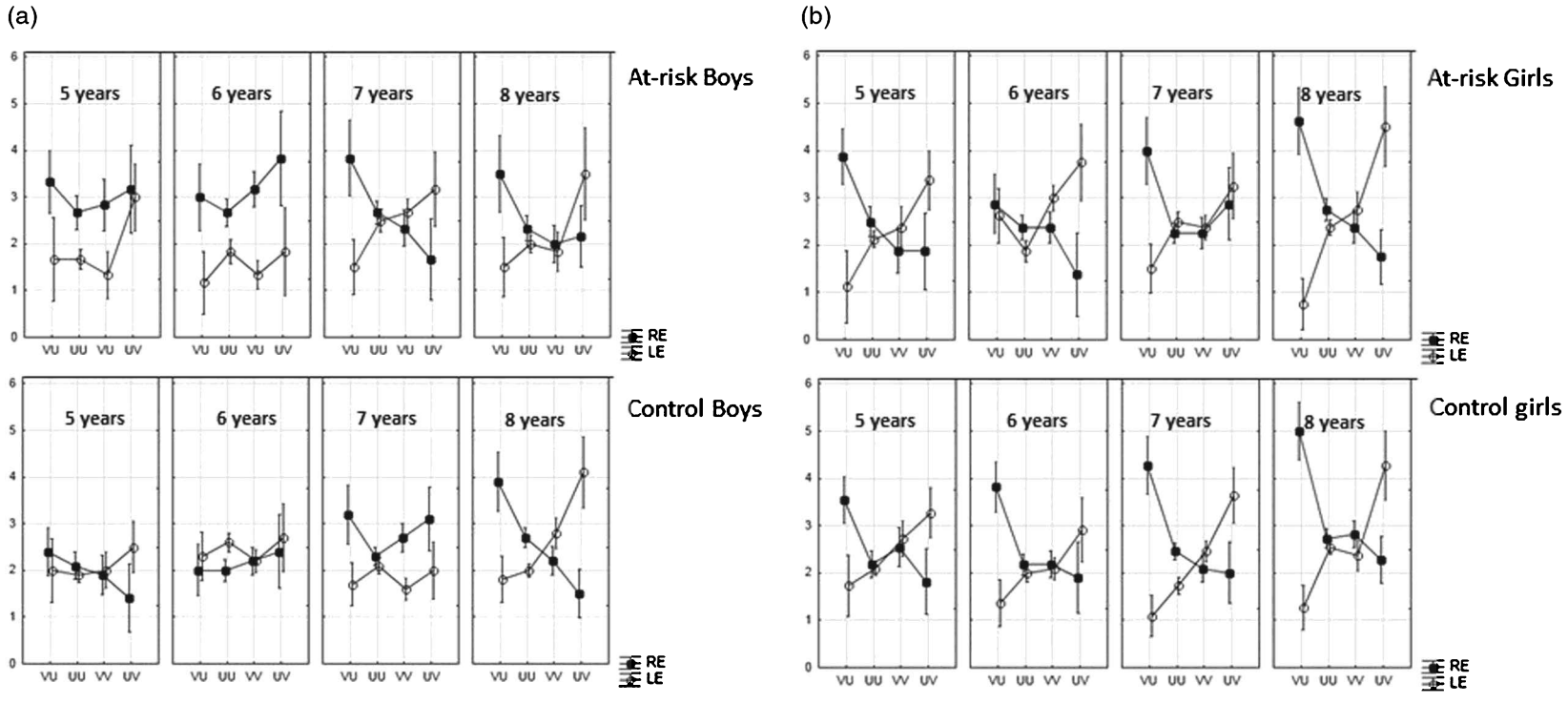

Figure 6–8. The effect of voicing and VOT on performance on the dichotic listening CV syllables task during repeated tests from the age of 5 to 8 years in children at risk for dyslexia (Figure 6–8a) and non-risk controls (Figure 6–8b), split for boys and girls. SL = Voiced CV (Short VOT) in the left ear and Unvoiced CV (Long VOT) in the right ear, LL = Unvoiced CV (Long-Long VOT) in both ears, SS = Unvoiced CV (Short - Short VOT) in both ears, LS = Unvoiced CV (Long VOT) in the right ear and Voiced CV (Short VOT) in the right ear. RE = right ear, LE = Left Ear. Data from Kristoff, the "Bergen Dyslexia Project" (Helland et al., 2006). *y*-axis = number of correct reports (3 correct reports on the average corresponds to 40%).

An analysis of variance (ANOVA) showed that the 3-way interaction of Age × VOT × Ear and the 4-way interaction of Age × VOT × Ear × Group were significant, $F(9,279) = 1.93$, $p = .048$, and $F(3,93) = 3.63$, $p = .013$, respectively. The complex interaction patterns are seen in Figure 6–8a,b, revealing different patterns on VOT modulation in boys and girls, and for at-risk and control children.

Intensity Variation and Phase Shift

Gradually varying the intensity of the signal in either the right or left ear is a different kind of bottom-up, or low-level, modulation of the REA gradually. This has been done in several studies (e.g. Hugdahl et al., 2008; Soveri et al., 2012, in press; Westerhausen et al., 2009; see also Berlin, Lowe-Bell, Cullen, Thompson, & Stafford , 1972).The typical manipulation is to increase the intensity in the right ear from 70 dB to +21 dB in steps of 3 or 6 dB at a time, and similarly for the left ear stimulus. This produces a linear increase in the number of correctly reported items from the right or left ear as the intensity is linearly increased. If the subject is instructed to attend to either the right or left ear stimulus, this will produce either a very strong bottom-up and conflict (e.g., if the intensity is increased +21 dB in the right ear and the subject is instructed to report the left ear stimulus), or a mutually reinforcing bottom-up and top-down situation if the intensity is increased in the right ear and the subject is instructed to attend to and report the right ear stimulus. As has been argued by Hugdahl (2012, in press), by systematically varying interaural intensity differences between the ears, it is possible to express a cognitive construct (the REA) in physical terms (dB), thus creating a psychophysical situation, where a complex mental concept can be operationally defined in objective dB terms. An example of intensity shifts of the right and left ear signal is seen in Figure 6–9.

Cognitive Training of the REA

Soveri et al. (2013) used intensity manipulations to study effects of training on the dichotic listening task, in particular training subjects to be able to increase left ear stimulus reports (when instructed to focus attention on the left ear stimulus). The intensity level was gradually increased for the right ear stimulus as subjects learned to increase their left ear reports on a weekly basis, thus making the task gradually more and more difficult. We now suggest that the intensity-modulation dichotic paradigm may be a valuable tool for both identification of central auditory processing disorder and subsequent training to overcome the impairment (cf. Moncrieff & Wertz, 2008; Musiek et al., 1985).

It would be interesting to study the effects of intensity variations in professional interpreters. Such individuals could be predicted to be better than monolingual individuals in coping with increasing interaural intensity differences for the right and left ear syllables, since they should have enhanced capacity for executive functioning, being able to switch between the two syllables of the dichotic pairs despite one of them being of greater intensity. As noted above, the ability of bilingual individuals to switch to a LEA in a situation with equal intensity

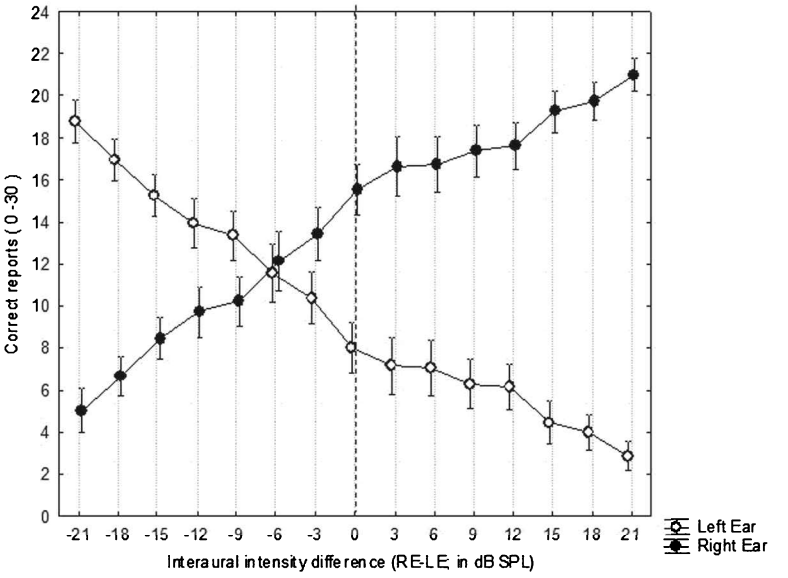

Figure 6–9. Graph of number of correct reports for the right and left ear syllable as a consequence of changing the intensity of the right or left ear signal, relative the other, in steps of 3 dB. To the right on the *x*-axis is increasing intensity in the right ear, to the left on the *x*-axis is increasing intensity in the left ear. From Hugdahl et al., 2008 with permission from the publisher.

for the two syllables in the dichotic pair was shown in a study by Soveri, Laine, Hamalainen, and Hugdahl (2011), with significantly enhanced reports from the left ear in bilingual speakers compared with monolinguals.

Phase Shifts in the Time Domain

A different kind of bottom-up manipulation of the REA involves shifting the onset of either the right or left ear syllable relative to the other, which is called phase shift manipulations. In a now classic series of studies, Berlin and colleagues (1973, 1977) showed that lagging the syllable in one ear relative to the other ear by about 15 to 30 ms had the effect of increasing or decreasing the REA, depending on which ear the lag occurred in. Specifically, if the right ear syllable trailed the left ear syllable within a 15 to 30 ms time window, the REA magnitude was increased.

A similar trend was seen when the left ear syllable trailed the right in the same time window, but the effect was attenuated compared with the trailing effect for the right ear stimulus. In order for a LEA to appear, the left ear syllable had to trail the right ear syllable up to 60 ms. Berlin (1977) explained this as a feed-forward effect, meaning the syllable that occurred as the last element in the dichotic pair was given priority for processing. This effect was, however, asymmetric, since the right ear trailing effect was more pronounced than the left ear trailing effect. Thus, the direct access for the right ear syllable in the nonlagging situation inter-

acted with the lag effect to produce a stronger effect for the right ear syllable compared with the left ear syllable.

The studies by Berlin and colleagues (presented in Berlin, 1977) on syllable intensity differences and temporal shifts were recently followed up by Murat Özgören and colleagues at the Dokuz Eylul University, in Turkey, where they put the amplitude/intensity and phase shift manipulations together, asking what would happen if the intensity difference were in conflict with the phase shift, as well as what would happen to the REA when the two manipulations acted synergistically, pulling in the same direction (Özgören, Bayazit, Oniz, & Hugdahl, 2012).

Citing from the abstract of the Özgören et al paper:

> The results showed an expected right ear advantage (REA) in the baseline (no-sound manipulation) condition. While amplitude shifts favoring right ear were found to have a greater effect on REA than phase shifts, phase shifts favoring left ear were found to have a greater effect on left ear advantage (LEA) than amplitude shifts. Furthermore, phase shifts favoring left ear had a greater effect on LEA than phase shifts favoring right ear on REA. The present results could have consequences for training of individuals with auditory and phonetic difficulties, for example, auditory processing deficits or dyslexia by designing optimal combinations of acoustic and phonetic training tool. (p. 291)

Attention Focus and Cognitive Control

A different way of manipulating the perception of the CV syllables in the dichotic pair is to selectively instruct the subject to explicitly pay attention to and report from only the right or left ear, that is, inducing a kind of top-down cognitive modulation of the REA, while keeping the stimulus parameters constant. By instructing the subjects to pay attention to and explicitly report either the right or left ear stimulus, top-down driven modulation of the bottom-up driven REA can be studied, thus examining how cognitive factors modulate a built-in perceptual REA. In this way we can study, using a dichotic input situation, how higher order cognitive factors modulate the REA and how bottom-up perceptual and top-down cognitive factors interact in central auditory processing. The attention-modulated dichotic listening task is an important extension of traditional approaches to central auditory processing and speech perception, since it provides an experimental analogue to the real-life everyday situation with more than one stimulus source at the same time, as in the well-known "cocktail party" phenomenon. It may be argued that attention is an integral part of central auditory processing, since it would be almost impossible for the brain to sort out the different sources of input whenever there is more than one source at a time, and where the individual would have to filter out irrelevant sound and focus on relevant aspects of the acoustic input.

Such a sorting or filtering mechanism requires that attention be focused on one source and that the other source appear at the same time is ignored. This is analogue to the dichotic listening situation when the subject is instructed to pay attention to the right ear syllable and to ignore the left ear syllable. As expected, in this situation the REA is increased, because the bottom-up advantage and the top-down instruction would act in concordance. However, when instructing

the subjects to focus attention on the left ear stimulus, the REA yields to a LEA. This situation can be considered a different situation with demands for cognitive inhibition of the irrelevant source and enhancement of the relevant source (Gootjes et al., 2006; Westerhausen & Hugdahl, 2010).

The "Forced-Attention" Dichotic Listening Task

The forced-attention dichotic task consists of three testing conditions. A first condition is a baseline with no specific instructions regarding focusing attention, leaving the subject free to report whichever syllable he/she prefers to report on each trial. This is called the nonforced, or NF, condition. A second condition involves the explicit instruction to focus attention to and report from the right ear. This is called a forced-right, or FR, condition. A third condition involves the explicit instruction to focus attention to and report from the left ear. This is called a forced-left, or FL, condition. The standard procedure is to present the NF condition first, and then to counterbalance the presentation of the FR and FL conditions, such that half of the subjects receive the FR condition before the FL condition, and vice versa. In clinical studies, it is advisable to counterbalance the order of the FR and FL conditions if repeated tests are performed. A manual in English with detailed instructions for how to perform the forced-attention paradigm (together with a CD of the paradigm itself) can be obtained by sending an email to the first author. Instructing the subjects to pay attention to either the right or left ear stimulus has profound effects on the REA. As seen in Figure 6–10 (based on data from 1095 subjects aged 16–89 years),the distribution of subjects differ for the right and left ear distributions, with a REA in the NF and FR conditions, and a LEA in the FL condition.

The NF condition reflects left hemisphere asymmetry for CV syllables in the absence of explicit cognitive manipulations. The FR condition shows that the REA is dramatically enhanced with the two distributions being even more separated than during the NF condition. The FR condition can be considered a nonexecutive attention condition, since the stimulus-driven baseline REA and the attention focus would act synergistically to produce an even larger REA. This is in contrast to the FL condition, where the two distributions are switched, with more subjects showing a LEA than a REA. The actual means and standard deviations (SDs) for the distributions presented in Figure 6–10 are seen in Table 6–1.

Inspection of the results in Table 6–1 reveals that not only is the magnitude of the LEA in the FL condition smaller than the corresponding magnitude of the REA in the FR condition, but the SD is also larger in the FL condition (marginally so for the FL left ear), which indicates that the task induces more variability. Increased variability in a group of suggests that the task is sensitive for individual differences. Taken together, these results indicate that different cognitive processes are at play in the FL and FR attention conditions. There have been different interpretations of the FL LEA effect—seeing it as a confounding factor for identification of a true laterality effect that should be eliminated or reduced (Bryden, Munhall, & Allard, 1983; Hiscock, Inch, & Kinsbourne, 1999; Hugdahl & Andersson, 1986) to the present view (e.g., Hugdahl et al., 2009; Westerhausen

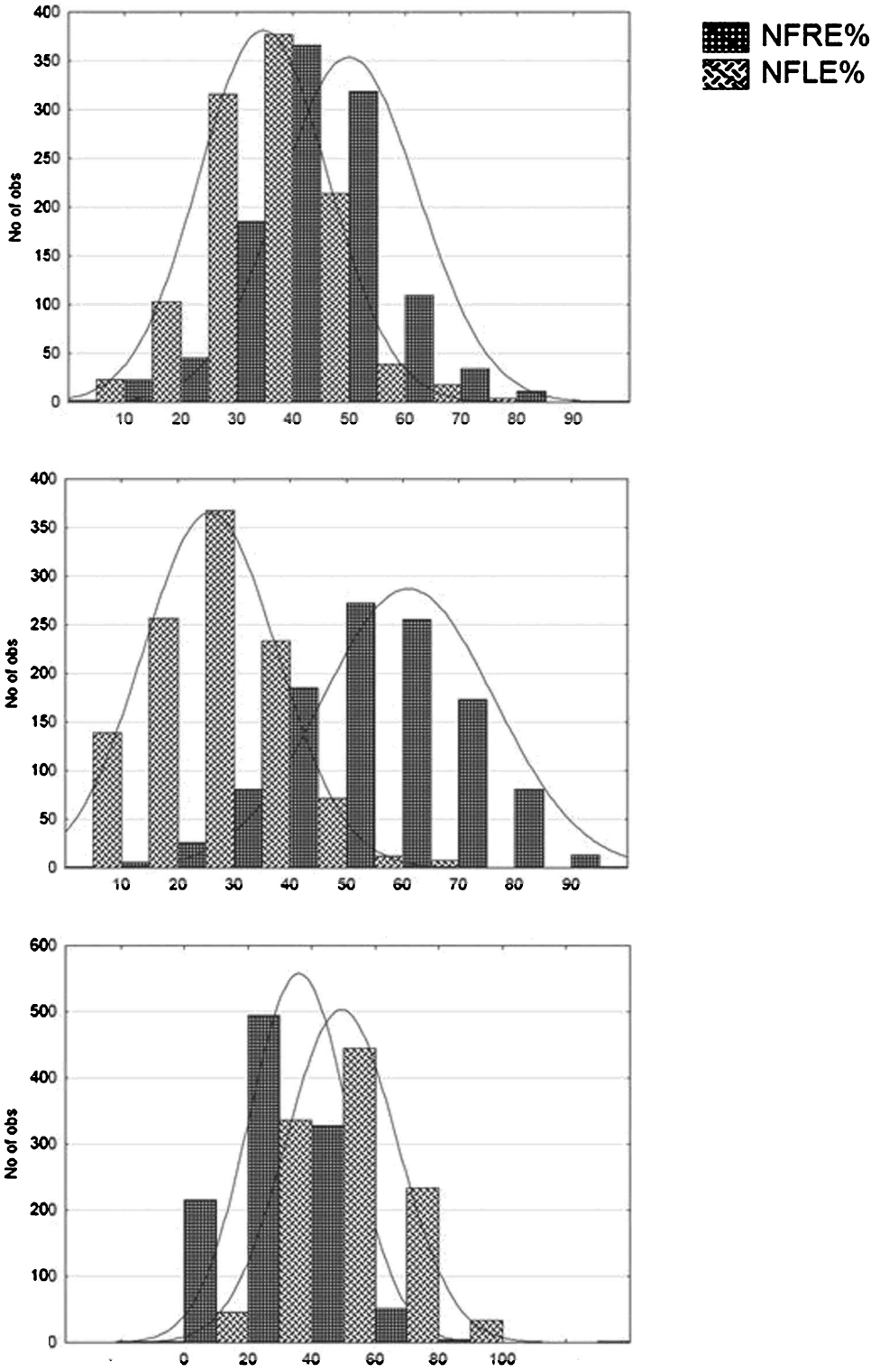

Figure 6–10. Distribution of scores from the right and left ear syllables during the nonforced (NF), forced-right (FR), and forced-left (FL) attention focus instructions. See text for further details.

Table 6–1. Means and standard deviations (SD) for % correct reports for the right (RE) and left (LE) ear, separate for the nonforced (NF), forced-right (FR), and forced-left (FL) instruction conditions. (See text for further explanations.)

	Mean	Std. Dev.
NFRE%	49,71385	12,34283
NFLE%	34,55708	11,45535
FRRE%	60,71907	15,20324
FRLE%	25,62157	11,94829
FLRE%	35,64992	15,65813
FLLE%	48,93455	17,35803

& Hugdahl, 2010) that the FL attention condition reflects an executive, cognitive control condition wherein the bottom-up REA is inhibited and attention is refocused to the left ear stimulus, which requires cognitive resources. The LEA in the FL condition is, therefore, an example of the interaction between bottom-up, stimulus-driven processing requirements and top down, instruction-driven, processing requirements, which we would argue is at the core of central auditory processing and central auditory processing deficits.

The forced-attention dichotic paradigm has been applied to a range of clinical conditions worldwide, including both adult (Dramsdahl, Westerhausen, Haavik, Hugdahl, & von Plessen, 2011; Gadea et al., 2002; Gootjes et al., 2004; Hommet et al., 2011; Johnsen, Ganagaratnam, & Asbjornsen, 2011; Kompus et al., 2011 for examples) and child disorders (Duvelleroy-Hommet et al., 1995; Hugdahl & Carlsson, 1994; von Plessen et al., 2004 for examples). In many instances, impaired top-down function inferred from the specific task of reporting the left ear stimulus when there is competition from the unattended right ear stimulus is a common feature of a central auditory deficit in both adults and children. An interesting feature of the FL effect is that it cuts across clinical diagnostic categories, ranging from language and language-related disorders to psychiatric and neurological disorders. This surprising effect speaks to a common underlying cognitive impairment that is orthogonal to clinical diagnoses that may have important consequences for treatment and training procedures in these disorders (cf. Hugdahl, in press).

Neuroanatomy and Neurophysiology of the LEA Effect

If the LEA effect in the FL processing condition reflects cognitive control and inhibition, as an integral aspect of central auditory processing, it should be of importance to try to locate the neuroanatomical and neurophysiological correlates of the behavioral effect. Thomson, Rimol, Ersland, and Hugdahl (2004) found remaining significant activations in the anterior cingulate cortex, medial middle frontal gyrus, and the inferior parietal lobule when contrasting fMRI images from the FL condition with corresponding images in the FR condition. The reversed contrast (i.e. taking the images during the FR condition and contrast with the images during the FL condition) did not leave any significant remaining activations.

Thus, it is clear from the Thomsen et al. (2004) study that the LEA effect in the FL condition has neuroanatomical correlates in cortical areas typically implicated in situations demanding cognitive control and response inhibition (e.g., Braver, Cohen, & Barch, 2002). Figure 6–11 shows unpublished data from our laboratory where images during the FR and FL conditions were contrasted with images obtained during the NF condition (which were used as baseline), in a standard fMRI box-car paradigm. The different cortical networks being activated in Figure 6–11 in the FR and FL conditions, respectively, overlap to a large degree with what Raichle (2010) labeled the dorsal attention and cognitive control networks, respectively. Thus, the different behavioral effects related to attention and cognitive control in the FR and FL conditions have their neuroanatomical and neurophysiological correlates in the parietal and frontal lobes, respectively.

Combining Intensity Manipulations and Attention Focus Paradigms

In the previous sections it was shown that the REA could be effectively modulated by changing the signal amplitude (i.e., the intensity, of either the right or left ear signal). The main finding was that the REA could withstand an interaural intensity difference of about 6 to 9 dB, favoring the left ear syllable. Moreover,

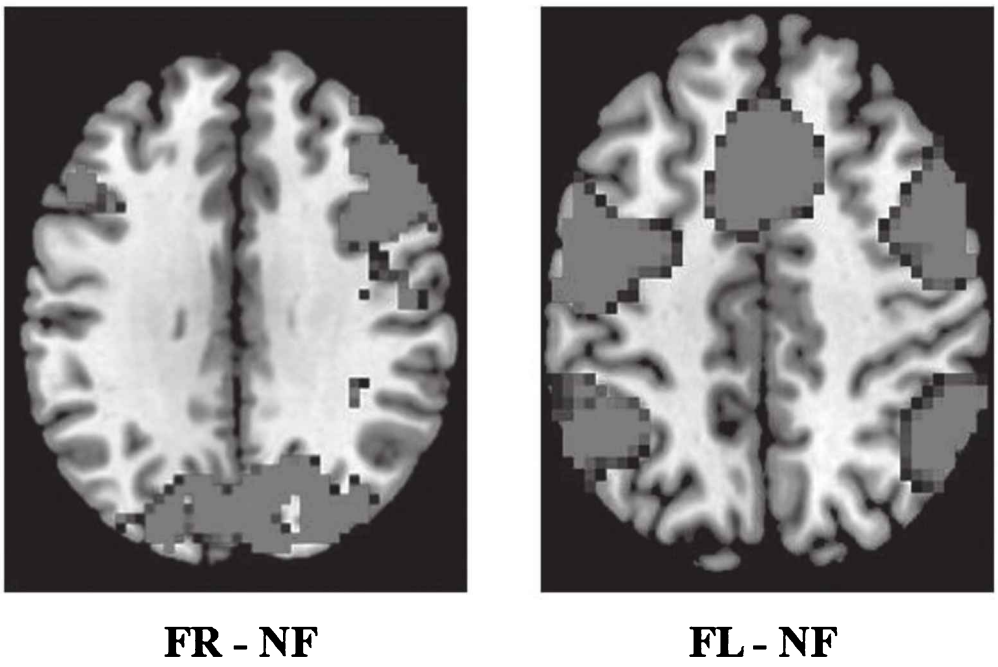

Figure 6–11. fMRI activation networks during the FR and FL attention instruction conditions, contrasted with images acquired during the non-forced, NF, condition as baseline. Note that neurological convention for orientation of the images is used, that is, left in the image is the left hemisphere and right in the image is the right hemisphere.

by instructing the subjects to actively focus attention and report the syllable presented either in the right or left ear, the REA also was effectively modulated, shifting to a LEA in healthy adult individuals when instructed to attend to and report only from the left ear. Based on these findings, one can now ask what would happen if people could use attention to "help" them either withstand an ever stronger left ear signal than in the standard situation. Similarly it could be asked whether subjects could withstand a larger intensity difference favoring the right ear signal when instructed to pay attention only to the right ear syllable. This was studied by Westerhausen et al. (2009), who systematically varied interaural intensity differences in steps of 3 dB, favoring either the right or left ear signal. The results showed the same linear modulation of the ear advantage as shown in Figure 6–9, with a strong REA and a LEA at the extreme ends, with +21 dB favoring the right or left ear signal, respectively. Interestingly, the results showed that the REA was not shifted to a significant LEA in the situation with 21 dB increase of the intensity in the left ear compared with the right ear signal, when subjects could use attention and top-down processing to withstand the effects of the bottom-up, stimulus-driven, intensity manipulation. The results also showed that in the situation where there was a 21 dB interaural difference favoring the right ear signal, and subjects were instructed to attend to and report only the left ear syllable, subjects managed to shift to a LEA. However, this was only after considerable cognitive effort, and the magnitude of the LEA at the extreme intensity level was smaller than the corresponding REA at the other extreme endpoint.

Functional MRI Studies of Combined Bottom-Up and Top-Down Modulations

Figure 6–12 (from Westerhausen et al., 2010b) shows increased cortical activation in the frontal and parietal lobes as a function of attention focus and interaural intensity differences, seen as changes in the blood oxygenation dependent (BOLD) response as measured with fMRI. The interaction of attention focus, either to the right, left, or no instruction, and interaural intensity manipulation showed that the BOLD response followed the same linear trends as was seen for the behavioral data and shown in Figure 6–9 for the NF (no instruction) condition. Thus, when the intensity was increased in one ear while the subject was instructed to pay attention to the syllable presented in the other ear, the BOLD response gradually increased in the anterior cingulate, supplementary motor, and inferior frontal gyrus in the frontal lobe, and in the inferior parietal gyrus in the parietal lobule, and in the insula. When the task became more and more difficult for the subject (as in the situation with increasing the signal intensity in one ear and being instructed to pay attention and report from the other ear), the BOLD response in frontal and parietal areas increased. In the opposite situation, when the task became easier, the BOLD response decreased in amplitude.

Thus, top-down modulations of a bottom-up driven auditory signal not only has behavioral consequences, as shown in the study by Westerhausen et al. (2009), it also parametrically modulates how the neurons in the cognitive areas in the frontal and parietal cortices respond to the challenge. These results

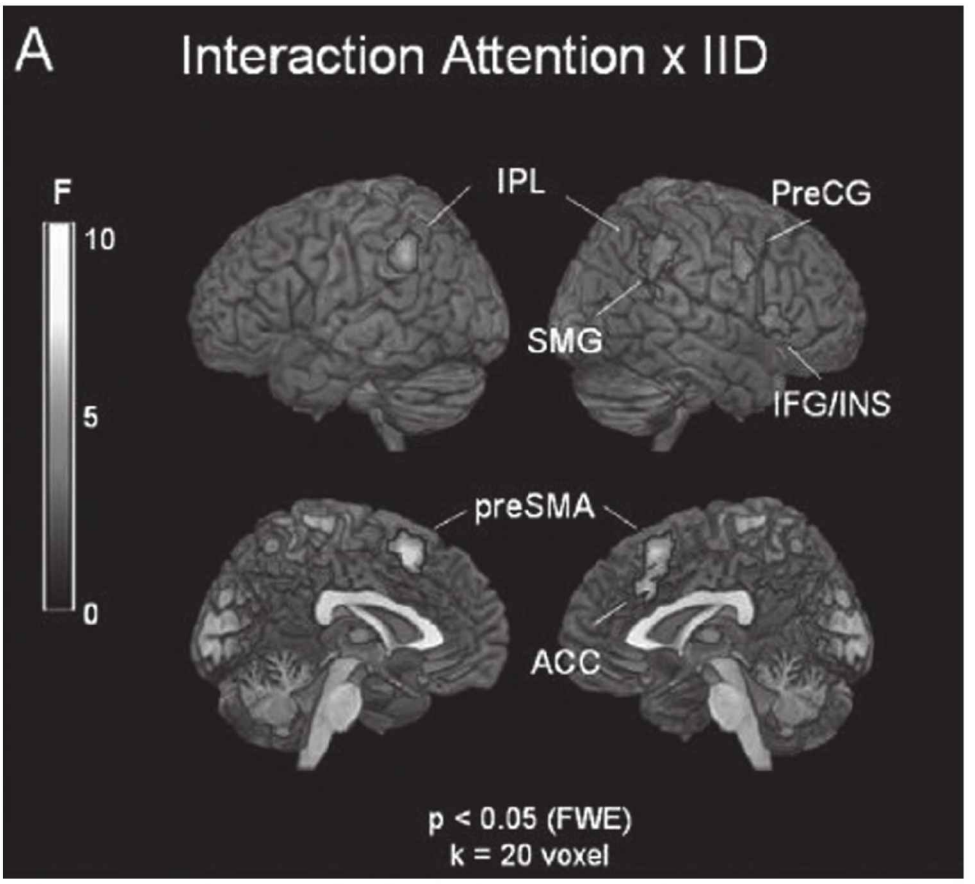

Figure 6–12. Parametric modulation of the fMRI BOLD response as a function of systematically vary the signal intensity for either the right or left ear signal, and at the same time instructing the subject to focus attention and report only the right or left ear syllable. See text for further explanations. From Westerhausen et al. (2010b), with permission from the publisher.

then further support a view of central auditory processing as the interaction of bottom-up, sensory, and top-down, cognitive factors that shape and modulate how a simple speech sound, like a CV syllable, is processed in natural surroundings, as when people are talking to each other, which requires decoding of the phonology but also correct attention and focus, and selective cognitive filtering of the signal to be processed.

Redefining Central Auditory Processing

From the review of dichotic listening studies thus far, and the demonstration of top-down cognitive control of bottom-up auditory processing seen in the dichotic listening paradigm, we would argue for a redefinition of central auditory processing. From a central auditory processing

perspective, attention focused on the left ear stimulus causes a cognitive conflict where the strong (right ear) and weak (left ear) stimulus elements are in conflict and where cognitive control mechanisms are required to resolve the processing conflict (cf. Braver et al., 2002 for a discussion of cognitive conflict and cognitive control).

One could argue that the study of central auditory processing *is* the study of the interaction between low- and high-level processing demands and how low-level perceptual processes, like the identification of a voiced stop-consonant, interacts with high-level processes, like shift of attention to the source of stimulus input, as would be seen as well in normal everyday conversation and communication. Without the ability to use higher cognitive processes and functions to inhibit or enhance lower level acoustic input, much of the flexibility and adjustability seen in human language communication would be lost.

Central auditory processing is therefore the *glue* that holds the different components together in a coherent cognitive framework. Central auditory processing deficits are deficits where one or several of the components of the interaction is not functioning, causing failure of either low-level perceptual processing or higher level cognitive modulation of the signal, or both. We would, therefore, suggest that the forced-attention dichotic listening task is well suited to tap the interactive aspects of central auditory processing that can be used both in research and clinical settings.

An advantage of the paradigm we have described is that the task is extremely simple to understand and perform, which means that it can be applied to patient groups with severe intellectual or cognitive deficits, where standard neuropsychological tests cannot be applied. A second advantage is that the different instruction conditions are very well controlled for extraneous and unwanted confounding factors. In fact, the only procedural difference between the FR and FL conditions is the word *right* versus *left*, all other parameters being constant between test conditions. In the next sections, we describe the *forced-attention* dichotic listening paradigm as it was originally called by Hugdahl and Andersson (1986) in their study of how children and adults managed to overcome the REA and show a LEA when explicitly instructed to focus attention and report only the right or left ear syllable. The forced-attention paradigm has been presented in numerous other articles and book chapters by members of our research group (in addition to Hugdahl & Andersson, 1986, see Asbjørnsen & Hugdahl, 1995; Hugdahl, 2003; Hugdahl, in press for further details).

Development Across the Life Span

It is of interest to examine the development of central auditory processing and whether there are changes in processing of the CV syllables in the different test conditions with age. Figure 6–13 shows the cross-sectional data from 400 healthy subjects (192 males and 208 females, with the number of subjects in each decade varying between 30 to 78, about equally distributed for males and females).

Since the current focus is on the interaction between bottom-up and top-down factors in central auditory processing, we have shown how this aspect of central auditory processing develops with age

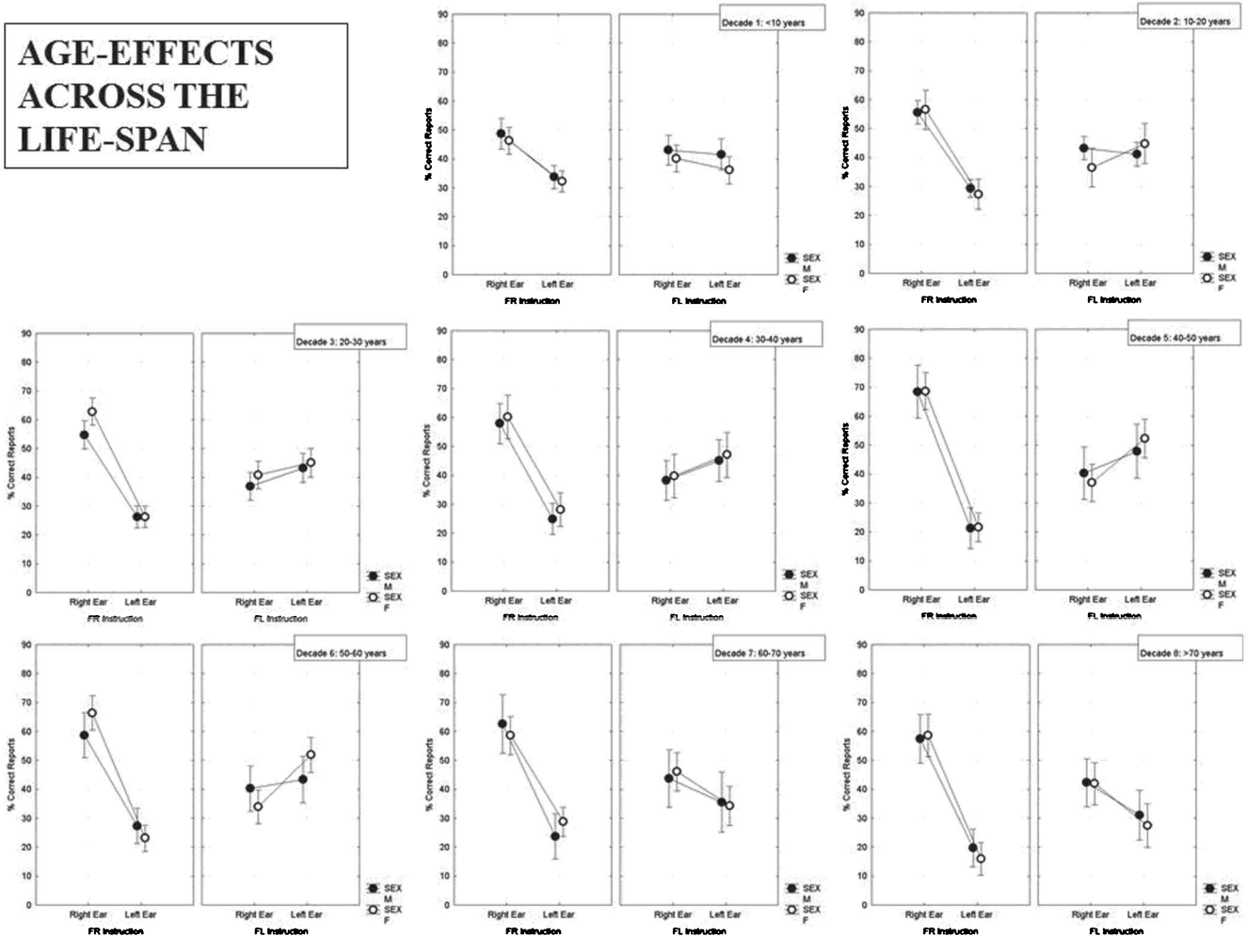

Figure 6–13. Results from 400 subjects for the FR and FL conditions, split for age across decades, that is, <10 years, 10–20 years, 2–30 years, and so forth. Black dots are right ear scores, open circles are left ear scores.

for the FR and FL instruction conditions. Looking first at the nonconflicting, FR situation, there seems to be little development with age. The REA is seen during the first decade of life and remains more or less constant with age, with a slight peak in magnitude during the fifth decade (40–50 years). It should be noted that REA is smallest during the first decade of life.

This situation is radically different for the FL condition (as seen in Figure 6–13), with both qualitative (a change in the direction of ear advantage) and quantitative developments (an increase or decrease in the magnitude of an ear advantage, without a change in the direction of the ear advantage) increasing with age. An immediate impression when eyeballing the development across decades for the FL condition is that children under the age of 10 have not developed an ability to use cognition to override the REA, and elderly individuals above the age of 60 have lost the ability to use cognition to override the REA.

In all three of these decades, there is a REA despite instructions to focus and report only the left ear stimulus. A second impression from the data in Figure 6–13 is that girls develop the skill to shift to a LEA in the FL condition before boys do, and that elderly females retain the ability at higher ages than males do. Statistical analysis of the data presented in Figure 6–13 has essentially confirmed the impressions from a visual inspection of the data. Thus, in this sense, central auditory processing is subject to developmental influences, both in youth early and older adults, with a similar impairment in using higher cognitive processes to override or overcome a built-in bottom-up REA effect. It should be noticed that the results presented in Figure 6–13 are cross-sectional and not longitudinal, which somewhat limits the conclusions that can be drawn. Presenting longitudinal data across the entire life span is, however, a daunting task, since it requires a research project that would have to go on for almost 100 years (!).

Similarly, Passow et al. (2012) applied the intensity-modulated version of the forced-attention paradigm for the study of cognitive decline in the elderly. The hypothesis was that as the task in the FL condition became more and more difficult, due to the gradual increase of the stimulus intensity in the right ear when instructed to focus attention to and report from the left ear, differences in performance on the task would gradually increase between younger and older participants. The results confirmed this with reduced ability in the elderly to use cognitive control to compensate for the increase in intensity in the right ear when instructed to report from the left ear (see also Figure 6–12 and the corresponding text about developmental effects on the REA in the various attention conditions).

The results of the Passow et al. (2012) study were that elderly individuals lacked the cognitive flexibility that was observed in the younger individuals to use cognitive control to compensate for the gradual increase in task difficulty due to increasing interaural intensity differences, assessed from the perspective of central auditory processing. In a follow-up study, Passow and colleagues (unpublished) have found that children below the age of 10 years resemble elderly individuals when it comes to the ability to use higher cognitive functions to modulate the REA when active manipulation of the relative intensity for the right and left ear syllable makes the task more and more difficult. Thus, the findings by Passow et al. in

their follow-up study essentially confirm the developmental effects described in Figure 6–12 for children aged <10 years and in particular the inability to reverse the REA to a LEA even when allowed to use attention for processing focus.

Reading Disorders and Dyslexia

Children with dyslexia and reading disorders are impaired on the CV-syllables dichotic listening task with regard to both showing a REA in the NF condition (Cohen, Hynd, & Hugdahl, 1992; Helland, Asbjornsen, Hushovd, & Hugdahl, 2008) and demonstrating a LEA in the FL condition (Hugdahl et al., 1998). Thus, children with dyslexia show impairment of central auditory processing, with regard to both asymmetry for phonology and speech processing and for cognitive control factors. The latter effect is corroborated by other studies that have shown that children with dyslexia have problems with tasks that require executive and cognitive control functions, including also working memory (Beneventi, Tonnessen, Ersland, & Hugdahl , 2010; Helland & Asbjørnsen, 2000; Jeffres & Everatt, 2004).

Does Severity of Dyslexia Matter?

Helland et al. (2008) compared two groups of 12-year-old dyslexic children with the CV-syllable dichotic listening task, testing asymmetry for central auditory processing to speech sounds. The two dyslexia groups were furthermore compared with an age, IQ, and sex-matched control group of normal reading children. The dyslexia groups in the Helland et al. (2008) study differed in severity in that some were referred to the special education unit for further clinical assessment and training. The group that did not need special education training showed an ear advantage and performance on the task similar to the control group (i.e., a REA), while the group that was referred to the special education unit did not show a REA. Helland et al. (2008) studied only the NF condition, and thus demonstrated a bottom-up effect in dyslexia. As stated in the discussion section in the Helland et al. (2008) paper, dichotic listening

> . . . is predominantly a research tool, and clinicians often find it difficult to draw clear inferences from performance on the DL test to training programmes and strategies for dyslexic children. The present study, however, points to the use of DL as a valid assessment tool in clinical work to improve differential diagnoses, particularly in relation to measures of school performance. (p. 50)

Helland et al. (2008) further wrote:

> The performance on the DL test by the two dyslexia groups may provide better insight both as to the degree of reading and writing impairment and to successful training methods. The D1 children should have no problems discriminating or distinguishing between different phonemes, showing a normal REA on the DL test, which was not distinguishable from the REA seen in the control group. This should perhaps be taken into account when designing training programs, using a bottom-up strategy to reading and writing acquisition in dyslexic children who benefit from extra teacher support. Such a strategy may, however, fail for children who still

> underachieve despite extra teacher and school support, as in the D2 group, if training in discriminating between similar phonemes and their sequencing has not taken place in advance, as is typically the case with existing training programs based on intensive auditory training (Tallal, 2006; Temple et al., 2003). (p. 50)

Dyslexia and Forced-Attention Dichotic Listening

We now present data from the same three groups for the FR and FL conditions, extending the Helland et al. (2008) study to include also the FR and FL processing conditions. Figure 6–14 shows the results for the two dyslexia groups and the control group. D1 stands for the dyslexia group that was not referred to the special education unit for training, D2 stands for the dyslexia group that was referred to the special education unit for training. The C group is the nondyslexic control group.

The results showed that the more severely affected dyslexia group (D2) was deficient in modulating the ear advantage, either during the FR or during the FL condition. The D1 group did not differ from the nondyslexic control group for any of the comparisons. This was statistically corroborated in an ANOVA, with

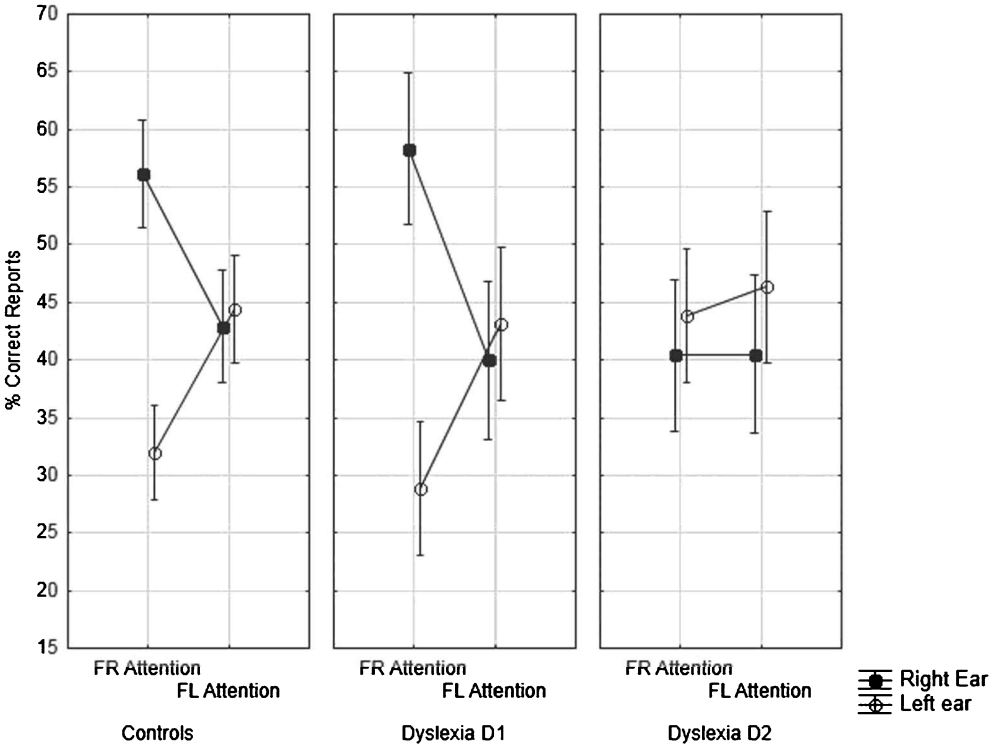

Figure 6–14. Results for two dyslexia groups, differing in severity of dyslexia, and a nondyslexic control group, during FR and FL attention instruction. The results are for 40 nondyslexic controls and 20 dyslexics in the D1 and D2 dyslexia groups, respectively. See text for further explanations.

significant differences between the D2 dyslexia group and the two other groups. The D1 dyslexia group and the control group both showed a strong REA in the FR attention focus condition, and a clear tendency toward a LEA in the FL attention focus condition. Thus, both groups showed the ability to use cognition to modulate a bottom-up, stimulus driven laterality effect for auditory processing. The results from the FR and FL instruction conditions extend the conclusions drawn by Helland et al. (2008) in that dyslexia involves auditory processing deficits related to the cracking of the phonological code and correct identification of low-level acoustic/phonetic processing features, as well as impairment of the ability for higher levels of central auditory processing, such as focus of attention on a single source in the presence of a stronger source that is competing for processing resources. The data were subjected to an ANOVA that showed that the three-way interaction of Group × Attention Focus × Ear was significant, $F(4, 154) = 2.63$, $p = .036$. As such, the forced-attention dichotic listening paradigm may prove a valuable tool in diagnostic and treatment/training situations for a range of central auditory processing disorders, also including dyslexia and other language-related disorders.

Summary and Conclusions

As argued in this chapter, combining bottom-up and top-down experimental manipulations will provide more fine-grained studies of central auditory processing, including studies of laterality and hemispheric asymmetry for speech processing and modulation of this processing by attention and other higher cognitive factors. Although the research described in this chapter is basic science, the findings could have implications for novel diagnostic and training procedures for children and adults with central auditory processing disorders. For example, Westerhausen et al. (2010b) combined the amplitude-modulation paradigm with the forced-attention paradigm and found that the REA could withstand an intensity (amplitude) increase of the left ear stimulus of about 6 to 9 dB before the ear advantage was shifted to a LEA.

In this chapter we have presented an overview and review of research on auditory central processing from the perspective of performance on the forced-attention version of the dichotic listening task or paradigm. We have for natural reasons concentrated the overview on our own research conducted at the University of Bergen, Norway; however, the forced-attention paradigm has been used in numerous studies worldwide, and in different clinical and research settings. This is important to point out when evaluating the reliability and validity of the paradigm, since it shows that the original findings are replicable not only in our own laboratory, but in other laboratories and clinics as well. Because of the wiring of the auditory pathways and the asymmetry of speech perception favoring the left hemisphere, the dichotic signal input provides an ideal experimental situation for studying the bottom-up and top-down interaction.

We are aware that dichotic listening often is viewed as a simple tool exclusively used for assessment of speech lateralization, a method that is clinically imperfect and moreover outdated by imaging and other recent techniques. As

the present chapter hopefully has shown, we do not agree with such a view, and would instead argue that because of its simplicity and *user friendliness*, the dichotic CV-syllables task is very well suited to assess both lower perceptual and higher cognitive processes in central auditory processing, including also applications to central auditory processing disorders.

A second conclusion is that top-down cognitive influences like attention and executive functions on lower level perceptual auditory factors in theories and models of central auditory processing should not be neglected in view of the data presented in this chapter. From an ecological perspective on how individuals interact in everyday life in communication and social interactions, it is obvious that both types of processing are necessary for effective communication and that the origin of the impairment may lie either in the bottom-up (i.e., perceptual or sensory processing) or top-down condition (i.e., cognitive processing) or in the interaction of the processes. Thus, different children may need different approaches to training/treatment. We suggest that future diagnostic and training/treatment regimens for auditory processing disorders involve an interactive or multidisciplinaryapproach, where all factors and processes are mapped out before training/treatment commences.

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CHAPTER 7

AUDITORY PROCESSING (DISORDER): AN INTERSECTION OF COGNITIVE, SENSORY, AND REWARD CIRCUITS

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Around the time the first edition of this book was published, the auditory processing disorder (APD) community was engaged in a lively discussion initiated by a position statement of the British Society of Audiology (BSA, 2007). The focus of this discussion was the degree to which APD could be conceptualized as a core deficit in the processing of nonspeech sounds. Our contribution to the first edition (Banai & Kraus, 2007) reflected the zeitgeist. We discussed an array of auditory characteristics associated with APD (e.g., temporal resolution, speech-in-noise perception) and their physiological correlates. We concluded that from a clinical and educational perspective there is little cause to focus primarily on the nonspeech symptoms because children with problems in this domain alone are unlikely to suffer from listening difficulties in real-life unless additional cognitive or language symptoms are present. We also suggested that brainstem processing was particularly relevant due to its impairment in populations intersecting with APD. Much of what we wrote is still true, but as with any lively scientific field, new issues have arisen that further our understanding of APD. We still maintain that auditory brainstem processing of complex sounds (cABR) offers a unique view on those issues, as supported by recent studies that suggest that cABR might help identify auditory processing deficits when standard testing fails (Billiet & Bellis, 2011). Therefore, we focus here on the more recent advances in our understanding of the neural processing of complex sounds and its relationships with perception, language, and cognition.

The first two sections of this chapter focus on two issues that are of particular importance to our understanding of APD in children. First, we briefly discuss new research on the similarities of APD and specific language impairment (SLI) and conclude that both sensory and cognitive characteristics are germane to the issues at hand. It seems that as in the case of other disorders of developmental nature, APD in children with no frank neurological deficits is best understood from a multiple risk factors perspective (Pennington & Bishop, 2009). The second section is devoted to new research on the development of auditory function that emphasizes: (1) the large heterogeneity even within the typically developing population, and (2) the intricate interplay of sensory and nonsensory influences on auditory function. Based on these two sections, we suggest that in order to understand and assess auditory processing in an ecologically viable fashion, measures that reflect those complexities are needed. In the third section we present the cABR as a potential candidate for the job, highlighting the ways in which it reflects sensory and cognitive aspects of auditory processing and their intricate interactions. A fourth section briefly deals with the potential role of reward and the interaction between auditory processing and limbic factors.

APD and SLI: Same or Different?

The field of APD has seen and continues to see many controversies surrounding almost every aspect of the issue from definition and etiology to diagnosis and remediation (see Cacace & McFarland, 2008). Of particular relevance for us is the question of overlap between APD and other learning/communication disorders in the language domain. Although this overlap has been commented on in the past (e.g., Rosen, 1999), several recent studies, which have compared profiles of children with a clinical diagnosis of APD to those with a clinical diagnosis of developmental language impairment (Dawes et al., 2009; Ferguson, Hall, Riley, & Moore, 2011; Miller & Wagstaff, 2011; Sharma, Purdy, & Kelly, 2009), make the extent of this overlap clear and impossible to ignore.

In our mind, the primary motivation for defining APD as a separate clinical entity is that some children are reported to have listening problems in the absence of other deficits that could merit a specific diagnosis of language/learning difficulty (e.g., Jerger et al., 1991). However, the data to support this idea (at least in children without obvious neurological deficits) are scant. For example, in one study (Dawes, Bishop, Sirimanna, & Bamiou, 2008), children who were referred to a specialist APD clinic were divided into groups based on their performance on standard tests of auditory processing that included both speech (the SCAN test, in which the processing of several elements of speech under nonideal listening conditions is evaluated) and nonspeech (gap detection in quiet and in noise, the identification of pitch and duration patterns) materials. The resulting groups were compared on several measures that included parental reports of presenting symptoms, the Children's Auditory Performance Scale (CHAPPS) completed by parents and teachers, and etiological measures like history of otitis media and obstetric complications and familial history. No significant differences on

any of those dependent measures were found between the groups. Furthermore, the incidence of comorbid learning difficulties also did not differ between the groups. Likewise, out of 68 children with suspected APD who were tested on a comprehensive battery of auditory tasks as well as on language, attention, and cognitive tasks, the majority of children could have been diagnosed with reading and/or language impairments as well (Sharma et al., 2009). Furthermore, in that study, the minority of children with auditory symptoms scored only within the normal range on reading and language tests so the functional significance of their auditory symptoms is not clear. Similar conclusions were reached following a large-scale population study in which the cognitive, communication, and listening profiles were compared between children defined as having typical or poor auditory processing based on a battery of auditory tasks (Moore, Ferguson, Edmondson-Jones, Ratib, & Riley, 2010).

Our discussion so far suggests that auditory processing deficits assessed with behavioral auditory tests are characteristic of only a subgroup of children with a clinical diagnosis of APD. Furthermore, the behavioral profiles of children with a clinical diagnosis of either APD or language-based learning problems such as SLI and dyslexia are remarkably similar. For example, temporal processing in the auditory and visual modalities was compared between children with APD and children with dyslexia (Dawes et al., 2009). The incidence of poor auditory processing was similar in the two groups. Likewise, no systematic group differences arose on two comprehensive comparisons of children diagnosed with specific language impairment and children diagnosed with APD (Ferguson et al., 2011; Miller & Wagstaff, 2011). For example, Ferguson and colleagues (2011) used a broad battery of audiological, speech intelligibility, intelligence, nonword repetition, phonological processing, grammar, and reading tests in addition to detailed parental questionnaires (the Children's Communication Checklist—CCC-2, CHAPPS). No significant group differences were found on any of the cognitive, language, and literacy measures and very few differences were observed on parental questionnaires. Those were restricted to listening in nonoptimal conditions and attention scales. Based on these findings, the authors concluded that a differential diagnosis of APD or SLI depends on the professional conducting the initial diagnosis and not on measurable differences between the groups (Ferguson et al., 2011). The study by Miller and Wagstaff (2011) published a few months later served to strengthen this conclusion.

Taken together, the studies reviewed above suggest that auditory perceptual deficits might be better viewed as part of a multicomponent characterization of developmental learning/language disorders rather than as a separate entity. A similar "multirisk" view was recently taken in the areas of spoken and written language impairments (Pennington & Bishop, 2009; Snowling, 2008, 2012). By this account, the etiology of developmental disorders might be too complex to be reduced to a single core deficit, because often a single weakness in one of the core components of a given skill is insufficient to cause clinical manifestation. Rather, a clinical threshold might be crossed only when several underlying weaknesses are present. Auditory processing might be one such underlying

weakness, making APD a useful concept in pointing clinicians to particular difficulties in this domain. Therefore, in the following section we review facets of typical auditory development that seem relevant to auditory problems in general.

Typical Auditory Development

It is commonly assumed that auditory function is more or less complete by the time children enter school, but studies in developmental psychoacoustics suggest that this is not the case (see Werner, 2007 for a review). Recent studies show that many auditory skills continue to develop during the primary school years, sometimes even into adolescence (e.g., Banai, Sabin, & Wright, 2011; Dawes & Bishop, 2008; Moore, Cowan, Riley, Edmondson-Jones, & Ferguson, 2011). Even the processing of sound by the auditory cortex (Sussman, Steinschneider, Gumenyuk, Grushko, & Lawson, 2008) and the auditory brainstem (Skoe, Krizman, Anderson, & Kraus, 2013) were shown to continue to mature during adolescence. Two important features highlighted by those studies that are particularly relevant for our discussion of APD are that variability within and across individuals and auditory features is much larger than previously assumed, and that the interaction of sensory (bottom-up) and nonsensory (top-down) factors cannot be ignored.

Variability Across Children Is the Rule

"Clearly the most striking difference between the adult and child data is the dramatically increased range or performance among the children" (Wightman & Allen, 1992, p. 116). This observation is true not only for the preschool children studied by Wightman and Allen, but also for older preschool (Banai & Yifat, 2011) and school-age children (Banai et al., 2011; Dawes & Bishop, 2008; Moore et al., 2011; Moore, Ferguson, Halliday, & Riley, 2008). Although in each of these studies, even some of the youngest children had adultlike performance, there were also typically developing children performing well outside this range. For example, in a study of auditory frequency discrimination, preschool children and adults were required to determine whether two tones that were presented on each trial were identical or not. More than 75% of the children had discrimination thresholds higher than those of most adults; however, the few best performing children were as good as the best performing adults (Banai & Yifat, 2011). Similar findings were reported in school-age children for frequency discrimination (Moore et al., 2008, 2011) and the detection of backward masked (Hartley, Wright, Hogan, & Moore, 2000; Moore et al., 2011), amplitude modulated (Banai, Sabin, & Wright, 2011; Moore et al., 2011) and frequency modulated tones (Banai, Sabin, & Wright, 2011; Dawes & Bishop, 2008).

This large variability at the group level often drives the conclusion that the auditory skill assessed in the children sampled is not mature. The reasons for this large variability are still not understood, but nonsensory factors are commonly implicated (Moore et al., 2008, 2010; Wightman & Allen, 1992), as discussed in the following section. Regardless, these data suggest that despite the care taken by investigators to make the tasks

as child friendly and as brief as possible, standard psychoacoustic techniques are not likely to provide good diagnostic measures because the variability in the typically developing population is such that the "normal range" is typically wide enough to encompass almost every measured score. This is especially true in young, preschool children, but also in school-age children, and sometimes even beyond 12 years of age, and could account for the observation that the profiles of auditory dysfunction in children with suspected APD are very variable (Sharma et al., 2009).

Nonsensory Factors Are Inherent Features of Psychophysical Performance

Both sensory and nonsensory factors could potentially account for the prolonged maturation of auditory function and for the large variability observed in young children. There are several reasons to consider the contribution of nonsensory functions. First, the biological maturation of the central auditory system and higher brain regions is prolonged in comparison with the maturation of the peripheral auditory pathways (Moore & Linthicum, 2007). Second, factors affecting the demands of the psychophysical tasks (e.g., working memory), are known to influence performance in both frequency discrimination (Song, Banai, Russo, & Kraus, 2006; Sutcliffe & Bishop, 2005) and gap detection tasks (Smith, Trainor, Gray, Plantinga, & Shore, 2008). It is not clear, however, how those factors influence development. Another putative source of variability and developmental change in psychophysical performance are the dynamics of performing the psychophysical task, given that psychophysical thresholds are derived from responses to a relatively long (typically a few dozen) sequence of trials. Two types of task dynamics—attention and sensitivity to the statistical structure of the stimulus sequence—were explored by different groups of researchers in a developmental context, as detailed in the following two paragraphs.

One aspect of task dynamics has to do with attention. A failure on the part of children to sufficiently attend during a portion of the trials will result in poorer observed performance. Therefore, researchers in developmental psychoacoustics often assess individual variability during task performance under the assumption that greater variability indicates poorer sustained attention. Wightman and Allen (1992) reported qualitative similarities between the psychoacoustic performance of preschool children and data from simulations designed to mimic inattention. Likewise, the frequency discrimination of younger children was in general less consistent than that of older children and adults, suggesting that poorer performance in the younger age groups derives from poorer attention (Moore et al., 2008). Furthermore, in a large sample of school-age children, performance variability was the second strongest predictor of a clinical manifestation of APD after cognitive test scores (Moore et al., 2010). Variability is not the only cause of elevated thresholds in children and cannot account for the full extent of age effects (Banai, Sabin, & Wright 2011; Banai & Yifat, 2011; Dawes & Bishop, 2008). Nevertheless, it appears to play a role in the manifestation of APD, as well as in the relationships between auditory processing and cognitive function.

The other dynamic, nonauditory, nonsensory aspect of performance considered here is anchoring, defined as the performance benefit induced by the presence of a consistently repeated reference stimulus in the sequence of trials. Decades ago, Harris (1948) recognized that participants had better discrimination thresholds when a repeated reference was used, compared with assessments in which no such repetitions occurred. He suggested that the presence of the repeated reference allowed listeners to form a reference point (an anchor). Since then, anchoring effects were recognized in frequency (Creelman & Macmillan, 1979; Nahum, Daikhin, Lubin, Cohen, & Ahissar, 2010), phase (Creelman & Macmillan, 1979), and temporal interval discrimination (Banai, Fisher, & Ganot, 2012) in adults. The concept of anchoring is relevant for our understanding of APD because anchoring is putatively induced by most standard psychoacoustic techniques. Furthermore, anchoring effects were also found to be reduced in individuals with dyslexia (Ahissar, Lubin, Putter-Katz, & Banai, 2006; Chandrasekaran, Hornickel, Skoe, Nicol, & Kraus, 2009; Oganian & Ahissar, 2012). As for children, preschool children exhibit anchoring effects in frequency discrimination (Banai & Yifat, 2011) and in verbal memory and naming tasks (Banai & Yifat, 2012), but those are smaller than those exhibited by adults and furthermore cannot account for the full extent of the age effects observed in those tasks. Nevertheless, we recently reported that anchoring explained unique variance in two important pre-reading skills: phonological awareness and letter knowledge (Banai & Yifat, 2012), suggesting that anchoring might underlie both perception and cognitive function. It therefore appears that better anchoring is associated with better reading and reading-related skills.

Interim Summary

Taken together the data reviewed in this section suggest two major conclusions relevant for APD. First, variability among children, especially during the preschool period, but also during primary school, makes psychoacoustic tasks problematic, at least as long as we do not have a better understanding of the causes of variability. Second, data on the contribution of nonsensory factors are consistent with the view that those factors might mediate the relationships between auditory processing and cognitive function and therefore might be particularly relevant for our understanding of APD. If this is the case, methods that better capture those dynamic aspects of auditory function and that yield data that are less variable between children are needed. In the remainder of this chapter, we discuss why cABR might be such a method.

[redacted]BR: A Physiological Hub of Activity

Studies of clinical populations intersecting APD, such as language-based learning disabilities and dyslexia, suggest that the cABR is relevant for our understanding and diagnosis of APD (Banai, Nicol, Zecker, & Kraus, 2005; Basu, Krishnan, & Weber-Fox, 2010; Billiet & Bellis, 2011; Chandrasekaran et al., 2009; Hornickel, Skoe, Nicol, Zecker, & Kraus, 2009; Hor-

nickel, Zecker, Bradlow, & Kraus, 2012; Rocha-Muniz, Befi-Lopes, & Schochat, 2012). Several recent reviews of that evidence are available elsewhere (e.g., Chandrasekaran & Kraus, 2012; Kraus & Hornickel, in press-a, in press-b). Reviews of the methodology are also available (Kraus, 2011; Skoe & Kraus, 2010a). Our focus here is therefore on the characteristics of the cABR that make it a good neural correlate of perceptual function —its faithful representation of the acoustic properties of complex auditory signals and its malleability to perceptual and cognitive experiences. cABRs are particularly attractive because although they capture clinically relevant variability across children, the "normal range" of the majority of the parameters derived from the physiological response is smaller than the range observed in behavioral assessments of auditory function (but see McFarland & Cacace, 2012 for an opposing view). This discussion is followed by a brief review of the perceptual and cognitive correlates of the cABR as relevant to APD.

Faithful Representation of Sound Within and Between Individuals

Auditory brainstem responses evoked by transient acoustic events (clicks) are highly reliable and consistent, leading to their routine use in hearing diagnosis (e.g., Hood, 1998; Jacobsen, 1985; Møller, 1999). Like their click-evoked counterparts, brainstem responses evoked by complex acoustic events like speech are also highly consistent within individuals over time in both adults (Song, Nicol, & Kraus, 2011) and 8- to 13-year-old children (Hornickel, Knowles, & Kraus, 2012; Russo, Nicol, Musacchia, & Kraus, 2004). Importantly for our previous discussion of behavioral variability on psychoacoustic tasks, children who are poor readers have less consistent brainstem responses —reflecting a trial-by-trial variability— than peers who are good readers (Hornickel & Kraus, 2013), which may suggest more immature neural function in children who are poor readers.

Furthermore, cABRs represent the acoustic properties of the evoking stimuli—timing, fundamental frequency, and higher harmonics—with remarkable fidelity (Galbraith, Arbagey, Branski, Comerci, & Rector, 1995; Krishnan, 2002; Russo et al., 2004; Skoe & Kraus, 2010a). For example, in the case of speech, different time frames related to the sentence, the word, and the syllable levels are captured by the ABR (Skoe & Kraus, 2010a). Modulations in the speech envelope (e.g., onsets, offsets) are represented as discrete peaks (or troughs) in the evoked response. At the level of the syllable, the pitch or the fundamental frequency of the producing source is represented in the frequency domain by the frequency-following response (FFR), a component of the response that reflects phase-locking to the periodicity of the stimulus. Fine structure (e.g., the formant that distinguishes different consonant-vowel syllables) is also represented in the time domain via subtle but systematic changes in response timing (Johnson et al., 2008), and in the frequency domain via phase and harmonic components of the response (Skoe, Nicol, & Kraus, 2011). Despite being faithful representations of the acoustic properties of complex sounds, cABRs are dynamic and perceptually relevant, as explained in the following sections.

Malleability at Different Time Scales: Online, Training-Induced, and Experience-Dependent Plasticity

cABRs are dynamic and can be modified at at least three different behaviorally relevant time scales. First the cABR is modified in response to changes in the sequence or context of evoking stimuli that occur in the time frame of seconds to minutes (Chandrasekaran et al., 2009; Skoe & Kraus, 2010b), and in relation to attentional capacities (Ruggles, Bharadwaj, & Shinn-Cunningham, 2011). A second relevant time frame is that associated with short-term experiences such as those associated with participating in a clinical training regimen (S. Anderson et al., in press; Hornickel, Zecker, et al., 2012; Russo, Hornickel, Nicol, Zecker, & Kraus, 2010; Russo, Nicol, Zecker, Hayes, & Kraus, 2005; Song, Skoe, Banai, & Kraus, 2012; Song, Skoe, Wong, & Kraus, 2008). Finally, brainstem responses are modified by long-term experiences such as those involved with learning music or a second language (Bidelman, Gandour, & Krishnan, 2011a, 2011b; Krizman, Marian, Shook, Skoe, & Kraus, 2012; Lee, Skoe, Kraus, & Ashley, 2009; Musacchia, Sams, Skoe, & Kraus, 2007; Parbery-Clark, Skoe, & Kraus, 2009; Parbery-Clark, Tierney, Strait, & Kraus, 2012; Strait, Parbery-Clark, Hittner, & Kraus, 2012); for review see Kraus and Chandrasekaran (2010).

Due to the perceptual importance of stimulus dynamics and context (see above) and to the fundamental role they play in the development of both visual and language skills due to statistical learning (see Romberg & Saffran, 2010; Thiessen, 2011), understanding how those dynamics are represented by the auditory system seems especially relevant for APD. Animal studies (Ahrens, Linden, & Sahani, 2008; Anderson, Christianson, & Linden, 2009; Antunes, Nelken, Covey, & Malmierca, 2010; Asari & Zador, 2009; Rabinowitz, Willmore, Schnupp, & King, 2011; Ulanovsky, Las, & Nelken, 2003) suggest that neurons in both subcortical and cortical auditory structures are sensitive to stimulus context and to statistical regularities. Complex ABRs in humans are likewise sensitive to online changes in stimulus dynamics, and this sensitivity is perceptually relevant (Chandrasekaran et al., 2009; Marmel, Parbery-Clark, Skoe, Nicol, & Kraus, 2011; Parbery-Clark, Strait, & Kraus, 2011; Strait, Hornickel, & Kraus, 2011) and occurs at both a local (e.g., a repetition of a note within a melody) and a global level (the melody itself) (Skoe & Kraus, 2010b). In one study (Chandrasekaran et al., 2009), speech-ABRs evoked by the same speech syllable were measured in two contexts: a repetitive context in which the same syllable was consistently presented throughout the experiment, and a variable context in which the target syllable was randomly mixed with other speech syllables. In typically developing children, pitch-related neural responses in the repetitive context condition were enhanced in comparison to responses to syllables that were presented in variable context. Also suggesting that this enhancement is behaviorally relevant, no context-related enhancement was present in the ABRs of children with dyslexia (Chandrasekaran et al., 2009), whereas musicians exhibited larger context-related ABR enhancements (Parbery-Clark et al., 2011). Furthermore, the magnitude of the neural context effect was positively correlated to indices of speech perception in noise (Chandrasekaran et al., 2009). Finally, among typically developing school-age

children, both music aptitude and literacy were positively related to the neural encoding of stimulus regularities (Strait et al., 2011).

Another time scale relevant for APD is that associated with skill enhancement following relatively short term experiences such as those induced by targeted training or therapy. Several studies in adults (Carcagno & Plack, 2011; Song et al., 2012; S. Anderson et al., in press) and children (Hornickel, Zecker, Bradlow, & Kraus, 2012; Russo et al., 2005, 2010) suggest that components of the ABR exhibit plasticity following such experiences. For example, significant improvements in speech perception in noise were observed in young, normal-hearing adults following eight weeks of training on an array of listening tasks (e.g., speech in noise, rapid speech). Those behavioral improvements were accompanied by corresponding enhancements in the neural encoding of pitch cues, particularly the fundamental frequency and the second harmonic (Skoe, Krizman, Spitzer, & Kraus, 2013; Song et al., 2012). Recent results suggest that even an aging nervous system can profit from auditory training; Anderson and colleagues (S. Anderson et al., in press) found improved neural peak timing and reduced interpeak variability in older adults after completing an auditory-based cognitive training program, with concomitant improvements in speech-in-noise perception and working memory. Importantly for APD, cABRs in children with language-related disorders and communication impairments (ASD) undergo plastic changes following relatively short-term experiences such as training (Russo et al., 2005, 2010) or the use of assistive listening devices (Hornickel, Zecker, et al., 2012). In a recent study, children with dyslexia were fitted with assistive listening devices (classroom FM systems), and the effects on their reading, phonological awareness, and ABRs were assessed after one year of use in comparison with children with dyslexia who did not use FM devices (controls). Brainstem responses in dyslexic FM users (but not in controls) became more consistent, suggesting that FM use decreases the variability of neural representation of speech sounds. Furthermore, phonological awareness and reading abilities improved significantly among FM users. Importantly for APD, the reading-related improvements were better predicted by initial neural consistency (shown to be weak in poor readers; Hornickel & Kraus, in press) than by initial reading or phonological awareness scores. Specifically, children with the least consistent neural responses prior to FM use improved the most on reading-related tasks (Hornickel, Zecker, et al., 2012).

Last, cABRs are modified by long-term experiences associated with both implicit (e.g., growing up in a particular linguistic environment) and explicit (e.g., learning music) learning processes. As an example of the effects of implicit learning, one might consider the effects of growing up speaking a tonal language, in which pitch information carries lexical meaning (e.g., Mandarin, Thai) on the neural encoding of speech. A series of studies comparing speakers of tonal languages to native English speakers suggests that long-term experience with tonal languages selectively enhance the encoding of pitch patterns that are perceptually relevant in those languages in the human brainstem (Krishnan, Bidelman, & Gandour, 2010; Krishnan, Gandour, Smalt, & Bidelman, 2010; Krishnan, Swaminathan, & Gandour, 2009). For further

details, see Krishanan and Gandour (2009). Likewise, enhanced encoding of the fundamental frequency was recently shown in bilingual compared with monolingual children (Krizman et al., 2012). Given the lack of language-related specificity in newborns (Jeng et al., 2011), these findings suggest that natural, daily experiences contribute to functional modifications of auditory processing even in the absence of explicit learning.

Music experience is a good example of the other facet of long-term explicit learning, because in contrast to native language acquisition, it (typically) involves both explicit instruction and active training. Of particular relevance in the current context is evidence that lifelong musical experience can enhance the neural encoding of sound and that those enhancements are relevant to auditory-based communication processes. Not only do musicians encode the acoustic elements of musical sounds more accurately and robustly than nonmusicians (Lee et al., 2009; Musacchia et al., 2007; Strait, Chan, Ashley, & Kraus, 2012), this enhancement transfers to the encoding of speech sounds in quiet and in noise (Musacchia et al., 2007; Parbery-Clark et al., 2009; Wong, Skoe, Russo, Dees, & Kraus, 2007) as well as emotional sounds (Strait, Kraus, Skoe, & Ashley, 2009). Furthermore, musical experience is accompanied by functional enhancements, such as better perception of speech in noise and better auditory attention (Parbery-Clark et al., 2009, 2011; Strait & Kraus, 2011; Strait, Kraus, Parbery-Clark, & Ashley, 2010; Strait, Parbery-Clark, et al., 2012). For example, comparing musicians and nonmusicians on a battery of auditory tests, it was found that musicians outperformed nonmusicians on indices of speech-in-noise perception, working memory, and frequency discrimination (Parbery-Clark et al., 2009). In line with the suggestion that neural and behavioral enhancements associated with musical experience are functionally related, it has been recently shown that neural sensitivity to stimulus context was predictive of both musical aptitude and reading skills in children (Strait et al., 2011). For recent reviews dealing with the functional and theoretical significance of music-related neural enhancements, see Kraus and Chandrasekaran (2010) and Patel (2011).

Perceptual Correlates

In the general normal hearing population, speech ABRs are correlated with speech-in-noise perception (Anderson, Skoe, Chandrasekaran, & Kraus, 2010; Anderson, Skoe, Chandrasekaran, Zecker, & Kraus, 2010; Hornickel et al., 2009; Skoe et al., 2011; Song, Skoe, Banai, & Kraus, 2011; Strait et al., 2011; Strait, Parbery-Clark et al., 2012). In particular, it appears that more robust neural representation of stimulus pitch or fundamental frequency, which is considered an important segregation cue (e.g., Darwin, Hukin, & al-Khatib, 1995), is associated with more robust speech perception in noise. For example, young adults with better speech-in-noise perception, as measured with the QuickSIN test, exhibit more robust neural encoding of the fundamental frequency of the evoking syllable in noise (2-talker and 6-talker babble) (Song, Skoe et al., 2011). In other words, individuals whose neural responses were degraded by noise to a lesser extent had better speech-in-noise perception. Similar findings were obtained in children (Anderson, Skoe, Chandrasekaran, Zecker et al., 2010) and older adults

(Anderson, Parbery-Clark, Yi, & Kraus, 2011). Another characteristic of the cABR that is related to listening-in-noise is the encoding of formant transition cues. Those are reflected both in the timing and in the phase of the ABR (Anderson, Skoe, Chandrasekaran, & Kraus, 2010; Hornickel et al., 2009; Skoe et al., 2011). For example, the subcortical differentiation of syllables containing different stop-consonants ([ba], [ga], [da]) and speech perception in noise were assessed in a group of 8- to 13-year-old children with a wide range of reading and speech perception scores. Brainstem differentiation of the three syllables was significantly correlated with speech-in-noise performance (Hornickel et al., 2009).

Cognitive Correlates

Much of the evidence for the relationships between cABRs and cognitive function comes from studies in which ABRs were compared between groups that were assumed to differ on some aspect of cognitive function (e.g., children with dyslexia, musicians, speakers of tone languages). These studies suggest robust physiological group differences between groups with different functional experiences with language (e.g., Krishnan & Gandour, 2009; Krizman et al., 2012) or music (e.g., Musacchia et al., 2007) (see above), as well as between children with and without reading/language deficits (e.g., Banai et al., 2005; Hornickel et al., 2009) and autism spectrum disorders (Russo, Nicol, Trommer, Zecker, & Kraus, 2009; Russo et al., 2008). The most obvious correlations between brainstem responses to complex stimuli and higher-level cognitive function come from studies in reading and language-impaired populations (Banai et al., 2009; Basu et al., 2010; Hornickel, Anderson, Skoe, Yi, & Kraus, 2012; Hornickel et al., 2009; Strait et al., 2011). For example, brainstem processing of frequency sweeps changing at different rates was compared between children with and without SLI (Basu et al., 2010). Responses were degraded in the children with SLI, particularly for rapid rates of presentation. Other studies compared speech ABRs between children with various diagnoses of reading and language disorders, typically showing impaired ABRs in a subgroup of the clinical sample (see Banai & Kraus, 2007 for review of those earlier works).

More direct evidence for the relationships between cognitive function and cABRs was obtained in studies in which correlational analyses were conducted in samples of children with a broad range of reading skills. In one such study, speech ABRs were assessed along with reading and phonological awareness in a group of children with a wide range of reading skills. Indices of brainstem timing and harmonic processing were correlated with reading skill, even after accounting for the effects of age, IQ, and click-ABRs (Banai et al., 2009). Similarly, stop-consonant differentiation by the brainstem was significantly correlated with phonological awareness (Hornickel et al., 2009). Although longitudinal studies are required to determine the causal directions, the above-mentioned studies certainly suggest that cABRs are sensitive to variation in reading-related skills.

We have already noted the cABRs are modulated by long term experiences. Taken together with data presented in this section, those experience-dependent modulations suggest that the relationships between cABRs and cognitive functions are strongly driven by active

processing of sound in which executive functions such as working memory and attention are used to meaningfully engage with sound. For example, more reliable brainstem encoding is associated with the enhancements in both speech-in-noise perception and working memory that are typically observed in musicians (see Kraus, Strait, & Parbery-Clark, 2012 for a more comprehensive review of the relationships between cABRs, musicianship and working memory).

Limbic Effects on Auditory Processing

In addition to the sensory and cognitive factors discussed above, studies, mostly in nonhuman species, suggest that limbic factors associated with reward and motivation are also intimately linked with auditory processing, and specifically that auditory learning is modulated by the involvement of the limbic system (David, Fritz, & Shamma, 2012; Weinberger & Bieszczad, 2011). Thus, studies of classical and operant conditioning have shown that conditioning or creating associations between stimuli and rewards or stimuli and behavioral responses yields plastic changes to the representation of sound in primary auditory cortex and that these changes are related to the function of the reward system (e.g., cholinergic activity) of the brain (see Weinberger, 2007 for a review).

Although data from human studies is sparse, available data suggest that reward and motivation have a significant impact on auditory learning in humans (Amitay, Halliday, Taylor, Sohoglu, & Moore, 2010; Laufer & Paz, 2012). Furthermore, emotional state has been shown to modulate the function of the human cortex (Kanske & Kotz, 2011), and cABRs (and those of musicians in particular) are sensitive to vocal expressions of emotion (Strait et al., 2009). Because sensory and cognitive aspects of auditory processing alone do not provide a satisfactory account of APD, limbic factors should also be considered.

Summary and Conclusions

Throughout this chapter, we presented evidence from multiple sources that auditory processing in children is variable and that standard tests of auditory function are extremely sensitive to this variability. Unfortunately, it appears that this variability compromises the use of psychophysical tests for the diagnosis of APD, especially in relatively young children. We also presented evidence that cABRs provide a "snapshot" of the sensory and cognitive processes involved in auditory processing, making it an attractive tool in APD research and for auditory assessment in general (see Shamma, 2011, who arrived at a similar conclusion based on a different line of reasoning than presented in this chapter). Specifically, we showed how cABRs might capture processes that are critically important in APD such as speech-in-noise perception and training-induced plasticity.

In addition to the evidence reviewed here for sensory and cognitive interactions in auditory processing, an increasing body of work, mainly in nonhuman animals, suggests that additional factors such as stress, reward, and other limbic factors modulate the response properties

of neurons throughout the auditory system. Those too are likely to be reflected in the cABR. Together, available evidence suggests that APD might be best viewed as a disorder stemming from a combination of vulnerabilities in sensory, cognitive, and reward processes, rather than a discrete disruption of auditory processing. Therefore, we end with the suggestion that the OPERA hypothesis (Patel, 2011), originally formulated in the context of music, might be especially useful in the conceptualization of APD. In a nutshell, this hypothesis suggests that musical expertise is beneficial for speech processing not only due to the fact that the two domains share *O*verlapping neural representations, but also because musical training activates those representations with greater *P*recision, elicits strong positive *E*motions, is highly *R*epetitive, and engages focused *A*ttention. Specifically, as auditory *P*recision and auditory *A*ttention are compromised in APD, *E*motion and *R*epetition might be critically important for its remediation.

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CHAPTER 8

THE NATURE OF CENTRAL AUDITORY PROCESSING DISORDER

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Historically, there has been much debate among professionals in many disciplines regarding the nature of central auditory processing disorder (CAPD) and the best means of diagnosing and treating it. In recent years, professionals in the fields of audiology, speech-language pathology, education, and related disciplines have witnessed a dramatic upsurge in interest regarding CAPD in both children and adults, and the demand for central auditory services has surged. As a result, it has become even more crucial that clinicians understand the fundamental principles underlying central auditory processing and its disorders.

The previous chapters in this volume have discussed the scientific bases of central auditory processing, both neurobiological and psychoacoustic, that inform our current conceptualization of CAPD, as well as etiologic factors that may underlie the disorder. Because an understanding of the fundamental nature of any disorder is critical to appropriate diagnosis and intervention, this chapter discusses key factors affecting central auditory processing and the current definition of CAPD, including how the disorder may lead to or be associated with difficulties in learning, language, communication, and related function. The topic of subprofiles of CAPD in children and adults also are explored, as are areas for future research in central auditory diagnosis and treatment.

[redacted]ors Affecting Central [redacted]ditory Processing

The way in which a disorder is defined directly informs how it should be diagnosed and treated, and by whom. In the case of CAPD, it is important to note that information to guide our conceptualizations of the disorder derives not only from the auditory neuroscience literature, but also from research in cognitive neuroscience, neuropsychology, psycholinguistics, and a host of other fields. As with any information processing in the brain, central auditory processing is complex and interactive, and an understanding of the fundamental nature of such processing is critical to the development of ecologically valid definitions for the disorder.

Bottom-Up and Top-Down Factors in Central Auditory Processing

Several factors must be taken into account when defining CAPD. First, one must recognize that although CAPD is generally considered to be a "bottom-up" deficit in fundamental auditory mechanisms, the relative influence of "top-down" factors in processing of auditory input cannot be overlooked. That is, although most current definitions of CAPD emphasize dysfunction in the central auditory nervous system (CANS), one must also consider the overarching effects of higher level, attention, cognitive, language, and related systems on fundamental sensory processing. In addition, although the auditory areas of the brainstem and cerebrum are of particular interest when considering CAPD, it is critical to recognize that parallel, distributed networks throughout the brain involving both inter- and intrahemispheric connections are activated during even the most basic sensory task. Furthermore, activity in lower level (e.g., brainstem) central auditory structures has been shown to modulate—and be modulated by—higher level cortical structures (e.g., Banai, Nicol, Stecker, & Kraus, 2005). As such, while CAPD is conceptualized as an auditory disorder, the complex and interactive nature of information (including auditory) processing leads to a great deal of heterogeneity in how the disorder presents in children and adults and also contributes to the frequently observed comorbidity of CAPD with other disorders involving attention, learning, and related functions. See Chapter 6 for additional discussion of bottom-up and top-down interactions.

Resource Allocation

A second, related factor is critical to our understanding of how disorders in central auditory processing can manifest: resource allocation. Resource allocation theory states that, during information processing, a finite store of resources is available to allocate to tasks. As such, any circumstance that requires disproportionate energy or effort to be allocated to one portion of a task leaves fewer resources for the remaining tasks. For example, the presence of a challenge to fundamental sensory input, such as peripheral hearing loss or a noisy listening environment, may create a situation in which the listener must allocate additional resources toward hearing, leading to fewer resources available for higher-order functions. As such, decreased memory for and/or comprehension of

auditory stimuli may occur when sensory input is disrupted even in the absence of primary memory, language, or related disorder (e.g., Peelle, Troiani, Wingfield, & Grossman, 2010; Pichora-Fuller, Schneider, & Daneman, 1995; Stewart & Wingfield, 2009; Wingfield, McCoy, Peelle, Tun, & Cox, 2006). Conversely, individuals with higher order attention, cognitive, or related deficits may exhibit decreased performance in tests of fundamental sensory processing due to the extra allocation of effort necessary to attend to, comprehend, or remember the stimulus (e.g., Bellis, Billiet, & Ross, 2011). That these effects occur at a neurobiological level is evidenced by the findings of Peelle, Troiani, Grossman, and Wingfield (2011), who demonstrated that even relatively mild deficits in peripheral auditory function lead to alterations in neural activity in brainstem, thalamus, and cortex. Changes in loci of neural activity also occur in normally hearing individuals when the auditory signal is degraded (Davis & Johnsrude, 2003; Harris, Dubno, Keren, Ahlstrom, & Eckert, 2009; Obleser, Wise, Dresner, & Scott, 2007) or when the auditory input is complex (e.g., Obleser, Meyer, & Friederici, 2011). Moreover, these effects can occur across modalities, so that increased effort to attend to a visual distractor may affect neurophysiologic representation of auditory stimuli, resulting from an inability to divide limited attentional resources (Wilson, Harkrider, & King, 2012). As such, one could reasonably expect that "auditory memory" or "auditory comprehension" may be compromised in an individual for whom bottom-up sensory processing, either in the peripheral or central auditory system, is deficient, due to disproportionate allocation of effort toward processing of the basic sensory stimulus.

Nonmodularity of the CANS

A third, also related, factor that must be considered when developing a definition of central auditory processing and its disorders is the degree to which CANS disorders can be expected to manifest solely in the auditory system. The topic of modality specificity as a diagnostic criterion for defining and diagnosing CAPD has enjoyed substantial attention in the literature in recent years and has given rise to two primary dissenting viewpoints. In the first, the clinical utility of a diagnosis of CAPD hinges on the ability to demonstrate that the deficit is restricted to the auditory modality (Cacace & McFarland, 2005; McFarland & Cacace, 1998). In this viewpoint, sensory processing is considered to be modality specific; therefore, analogous tests in other sensory modes (e.g., visual) should be incorporated into the diagnostic test battery in order to confirm that the deficits observed are not apparent across modalities. When deficits are found across modalities, one must infer that either sensory disorder exists in more than one modality or that the disorder is pansensory or global, rather than representing dysfunction in the CANS. This viewpoint is predicated upon the contention that sensory systems are fundamentally modular, and therefore disorders of such systems should, likewise, be specific to the modality involved.

The dissenting viewpoint contends that while some areas of the CANS do respond primarily (but not solely) to auditory stimuli, the majority of the central nervous system (CNS) is nonmodular and composed of convergent sensory tracts, multisensory neurons, and inter- and intrahemispheric connections that preclude conceptualization of any central processing disorder as entirely modality

specific (Musiek, Bellis, & Chermak, 2005). This viewpoint draws upon a large body of research demonstrating the interconnectedness among and multimodal responses in brain regions previously considered to be sensory specific (e.g., Bamiou, Musiek, & Luxon, 2003; Poremba, Saunders, Crane, Cook, Sokoloff, & Mishkin, 2003; Salvi, Lockwood, Frisina, Coad, Wack, & Frisina, 2002; see Stretfeld, 1980 and Musiek et al., 2005 for reviews). Further, this viewpoint holds that due to the uncertain neurophysiologic mechanisms underlying multimodal tests purported to be *analogous* to validated central auditory measures (Musiek et al., 2005), a more appropriate method of disentangling the relative effects of central auditory dysfunction and higher-order, global, or pansensory deficits is to make use of multidisciplinary assessments, with each area addressed by the tests (and professionals) standardized (and qualified) to do so. Finally, this perspective predicts that comorbid deficits across sensory systems may occur due to shared neuroanatomical substrates, which would be entirely consistent with the complex nature of information processing in the CNS.

Direct support for the latter viewpoint has been provided by Bellis and colleagues (Bellis et al., 2011; Bellis, Billiet, & Ross, 2008; Bellis & Ross, 2011), who demonstrated that tasks reliant on shared brain regions (e.g., corpus callosum) give rise to similar patterns of performance regardless of sensory modality but that those individuals with central auditory disorders exhibit deficits that are more pronounced in the auditory modality. Furthermore, the authors' results were consistent with the expectation, derived from information processing and resource allocation theories discussed previously, that individuals with higher order, attention-related disorders may exhibit overall performance decrements on sensory tasks, but their *patterns* of performance differ from those with CAPD. Finally, and perhaps more importantly, results of this study demonstrated that the addition of visual analogs of central auditory tests did not provide additional information pertinent to diagnosis of CAPD that was not obtained via inter- and intratest comparisons of individual performance on the auditory tests alone.

These interrelated factors—bottom-up and top-down processing, resource allocation, and nonmodularity of the CANS—help to drive our understanding and, ultimately, our definition of central auditory processing and its disorders. When taken together with the plethora of evidence demonstrating the effects of CANS dysfunction on auditory and related functions, a definition of CAPD that accurately reflects the nature of auditory processing in the CANS can be developed.

Defining CAPD

Early definitions of CAPD were rather amorphous and included processes and behaviors ranging from fundamental auditory skills to higher order functions such as linguistic analysis, memory, and use of auditorily presented information (e.g, ASHA, 1992; Kelly, 1995). Because CAPD can lead to or be associated with a variety of functional deficits in learning, language, and communication, as discussed subsequently in this chapter, professionals from a wide variety of disciplines, including speech-language pathologists, audiologists, psychologists,

and neuropsychologists, educators and educational diagnosticians, physicians, and others, began applying the label of CAPD to their patients, often on the basis of symptoms alone and without benefit of any auditory testing whatsoever. As such, CAPD became a "wastebasket term" used to describe any difficulty with auditory input or spoken language. This was a primary factor that led to the controversy surrounding the clinical utility of a diagnosis of CAPD (e.g., Cacace & McFarland, 1998; McFarland & Cacace, 1995; Rees, 1973) because the disorder, as conceptualized by many, was virtually indistinguishable from a host of other disorders with overlapping symptoms, including language disorder, attention deficit hyperactivity disorder (ADHD), and other higher order cognitive disorders, and many other impairments that may affect an individual's ability to listen to, comprehend, remember, or act upon auditory information.

In response, subsequent definitions of CAPD were more concise and emphasized the auditory-specific nature of the disorder. Thus, many of these definitions either stated or strongly implied that a diagnosis of CAPD should be made only when it could be demonstrated that the disorder is specific to the auditory modality (e.g., Cacace & McFarland, 2005; Jerger & Musiek, 2000; McFarland & Cacace, 1995). However, these definitions, too, were fraught with difficulty, as they failed to recognize the complex, nonmodular, and interactive nature of brain function, with its proliferation of shared neuroanatomical substrates, multisensory neural interfaces, convergence and divergence of sensory "tracks," and interdependence of bottom-up and top-down factors, all of which were discussed in the previous section.

An additional definition of CAPD was proposed by the British Society of Audiology (BSA, 2011). In this definition, CAPD is purported to represent a more general cognitive and/or developmental disorder rather than a bottom-up, auditory deficit per se. Much of this definition is derived from the findings of Moore and colleagues (Moore, Ferguson, Edmonson-Jones, Ratib, & Riley, 2010) in a population-based study of typical children, of whom those with lower cognitive performance (e.g., verbal memory) tended also to score lower on tests of auditory perception developed by the research team. In addition, the authors found that attention deficits tended to affect auditory test performance adversely. However, when one considers the influence of top-down factors and resource allocation on sensory processing, as discussed in the previous section, these findings are not surprising. Indeed, they are precisely what would be predicted and are entirely consistent with the contentions of Bellis with respect to the relationship between auditory performance and higher order tasks (e.g., Bellis, 2003; Bellis et al., 2011). In addition, it is important to note that the tests employed by Moore et al. (2010) were presumed by the research team to reflect central auditory processing ability; however, sensitivity and specificity of these tests to CANS dysfunction was never documented. Nonetheless, based upon their definition of CAPD, the BSA (2011) contended that CAPD is best identified via parent/caregiver and/or teacher checklists of key behaviors, rather than sensitized tests of auditory function, despite considerable evidence that such checklists are of very limited utility in differentiating children with CAPD from those with other disorders that have similar and/or overlapping symptoms (Comeaux,

Havelaar, & Bellis, 2012; Drake, Brager, Leyendecker, Preston, Shorten, & Stoos, 2006; Lam & Sanchez, 2007; Wilson et al., 2011). For additional discussion of these topics, the reader is referred to Chapter 9 in this volume.

The definition of CAPD developed by the American Speech-Language-Hearing Association Working Group on Auditory Processing Disorders (ASHA, 2005a, b) and ratified by the American Academy of Audiology Task Force on Central Auditory Processing (AAA, 2010) explicitly recognizes the auditory nature of the disorder while also taking into account information processing theory, resource allocation, and the inherent nonmodularity of the CANS. ASHA (2005a) defines central auditory processing as "the perceptual [i.e., neural] processing of auditory information in the . . . CNS and the neurobiologic activity that gives rise to the electrophysiologic auditory potentials" (p. 2). It includes the neural mechanisms that underlie a variety of auditory behaviors, including localization/lateralization, performance with degraded or competing acoustic signals, temporal aspects of audition, auditory discrimination, and auditory pattern recognition (ASHA, 1996, 2005a, 2005b; Bellis, 2003; Chermak & Musiek, 1997). As elaborated by Musiek et al. (2005), several critical aspects of the current definition and conceptualization of CAPD should be emphasized:

- CAPD is conceptualized as an *auditory* disorder of neurobiological origin. That is, in order for a diagnosis of CAPD to be made, it must be demonstrated that a deficit exists in the CANS, using tests shown to be sensitive to dysfunction in the central auditory pathways.
- Due to the interactive nature of brain function, CAPD may coexist with other disorders (e.g. ADHD, language impairment, learning disability, deficits in other modalities); however, it *is not the result of* higher level global or multimodal dysfunction. For example, children with autism or mental retardation might have difficulty with listening and/or comprehending spoken language; however, their "auditory" difficulties are attributable to a higher order, more global cognitive deficit and not to dysfunction in the CANS. Therefore, it would be inappropriate to apply the label of CAPD in these cases. With that being said, however, it should be noted that brainstem-level atypicalities in subcortical processing of speech stimuli have been implicated in some children with autism spectrum disorders (e.g., Russo, Nicol, Trommer, Zecker, & Kraus, 2009). At present, it is unclear how this finding may impact our future conceptualization of CAPD.
- Similarly, abilities such as phonological awareness and analysis, auditory synthesis, spoken language comprehension, and attention to or memory for auditory information may rely in part upon the integrity of acoustic signal processing in the CANS. However, these are higher order language- or cognitive-related abilities and are excluded from the definition of CAPD (ASHA, 2005a).
- Although the notion of *complete* modality specificity of CAPD is neurophysiologically untenable when one considers the complex nature of information processing in the brain, it is recognized that CAPD is *primarily* an auditory disorder. Therefore, individuals with CAPD

present with difficulties, documentable deficits, and complaints that are more pronounced in the auditory modality and, in some cases, auditory-modality-specific findings may be demonstrated.

These factors hold significant implications for clinicians in terms of the knowledge base required for engaging in central auditory processing service delivery, scopes of practice of the various professionals who work with individuals with central auditory dysfunction, and methods of diagnosing and treating CAPD. Several of these topics are discussed by the AAA (2010) and ASHA (2005a, 2005b) documents and are summarized below.

Knowledge Base Required for Central Auditory Service Delivery

CAPD cannot be viewed in a vacuum. Although CAPD is an auditory disorder, the nonmodularity of the CANS and the complex nature of information processing in the CNS dictate that an understanding of the disorder requires familiarity with a wide variety of scientific topics. Research pertaining directly to CAPD is not confined to the audiological literature but proliferates in numerous journals not typically read by those in the fields of hearing and speech/language. Moreover, our professional training programs generally have not addressed these areas adequately (Chermak, Traynham, Seikel, & Musiek, 1998). Many of the misconceptions that have surrounded CAPD historically may be a result of this lack of education, leading to a unitary and overly simplistic view of auditory processing.

To understand the nature of central auditory processing and its disorders and, thereby, to assess, diagnose, and treat it appropriately, one should have at least a familiarity with, if not a working knowledge of, the current literature in those areas pertaining to brain structure and function and brain-behavior relationships, including general and auditory neuroscience, cognitive psychology and neuropsychology, neurophysiology, psychoacoustics, and other topics (ASHA, 2005a, 2005b). This knowledge likely will need to be acquired through ongoing training and educational activities in addition to those obtained during the educational preparation process.

Finally, it is important to emphasize that new findings occur regularly in the clinical and scientific arenas that intersect with or underlie CAPD. As our understanding of the scientific bases of central auditory processing increases, our views of the disorder must develop accordingly. Any theory of CAPD, no matter how logical it appears on the surface or how long it has been accepted by popular consensus, cannot be accurate if it is inconsistent with the underlying scientific knowledge. A familiarity with the scientific bases of CAPD will assist clinicians in evaluating the validity of theories, popular or new diagnostic and treatment tools, and anecdotal "evidence" pertaining to diagnosis or treatment of CAPD and, thus, will result in better services to the patients who need them.

Scopes of Practice and CAPD Diagnosis, Assessment, and Intervention

Because CAPD has, in the past, often been used erroneously and inappropriately as an all-encompassing term to describe vir-

tually anyone with difficulties listening to or understanding auditory information, it is not surprising that professionals from many different disciplines have assumed responsibility for diagnosing the disorder. Thus, the label of CAPD has been applied to children and adults by audiologists, speech-language pathologists, psychologists, educators, physicians, and others. Further confounding this problem is the fact that many measures of listening, phonological awareness, language processing, and related abilities use the term *auditory processing* in their titles.

By definition, CAPD is an auditory disorder, and therefore the responsibility for diagnosing CAPD falls squarely within the audiologist's scope of practice (ASHA, 2005a, 2005b). Furthermore, the requirement for acoustical control for administration of central auditory tests and the specialized equipment necessary to diagnose CAPD require administration by audiologists who typically have the appropriate education and training. Nonetheless, a multidisciplinary approach is needed to assess fully the presenting difficulties and the functional impact of the disorder. Multidisciplinary input also is critical for identifying comorbid disorders and overlapping and/or associated deficits (i.e., differential diagnosis). In this light, the speech-language pathologist is uniquely qualified to delineate the cognitive/communicative and language difficulties that may be associated with CAPD (ASHA, 2005a, 2005b, 2005c). Psychologists and neuropsychologists delineate cognitive capacities and brain-behavior relationships and, often in conjunction with educators and/or educational diagnosticians, assess academic function across domains. Other professionals may diagnose additional disorders that may affect the individual's ability to utilize auditory information. Collectively, all of this multimodal (and multidisciplinary) information is important in assessing fully the presenting complaints of a given individual and in differentiating a CAPD from more global, higher order, or pansensory deficits that may mimic and/or coexist with CAPD. With this information in hand, intervention can be implemented to target deficit areas.

It is important at this point to differentiate among *diagnosis*, differential diagnosis, and *assessment*. Assessment may be defined as a data-gathering process that may include both formal and informal procedures to document areas of strength and weakness (ASHA, 2005a). Diagnosis, on the other hand, refers to the actual identification and classification of a specific impairment (ASHA, 2005a). In this light, then, assessment of CAPD is a multidisciplinary endeavor. Diagnosis of CAPD, on the other hand, is the responsibility of the audiologist, using acoustically controlled diagnostic test tools that have been shown to be sensitive to dysfunction in the CANS. In contrast, comprehensive intervention for CAPD may be undertaken by a team of professionals, whose composition depends upon the specific needs of the individual. As elaborated in Volume 2 of this Handbook, intervention for CAPD encompasses a variety of activities and methods and, as such, may involve audiologists, speech-language pathologists, educators, and others. Intervention, as well as assessment and differential diagnosis, most often requires a multidisciplinary effort to address the individual's presenting difficulties in a holistic and ecologically valid manner (ASHA, 2005a).

Etiology and Prevalence of CAPD in Children and Adults

It has been estimated that as many as half of all children identified with a learning disorder (or 2% to 5% of the school-age population) exhibit CAPD (e.g., Bamiou, Musiek, & Luxon, 2001; Chermak & Musiek, 1997). Prevalence figures for CAPD in the elderly population have ranged from as low as 2% to as high as 76% depending on the strictness of the criteria used for inclusion (e.g., Cooper & Gates, 1991; Golding, Carter, Mitchell, & Hood, 2004). The lack of precise prevalence figures for CAPD likely has arisen because of widely differing methods of defining and diagnosing the disorder. It is hoped that with the advent of better guidance as to appropriate methods of defining and diagnosing CAPD (e.g., AAA, 2010; ASHA 2005a), more accurate prevalence estimations will emerge.

Similarly, although the underlying etiology for central auditory dysfunction may be identified in some cases (e.g., head trauma, neurologic disorder or abnormality), in most cases, the cause of CAPD remains unknown; however, poor or inefficient neurophysiologic representation of acoustic stimuli is suspected. Imprecise temporal processing and neural synchrony, atypical hemispheric asymmetry in the neural representation of auditory (especially speech) signals, and inefficient interhemispheric transfer of auditory information have been identified in many cases of CAPD in both children and aging adults (e.g., Bellis, Nicol, & Kraus, 2000; Bellis & Wilber, 2001; Jerger et al., 2002; Kraus, McGee, Carrell, Zecker, Nicol, & Koch, 1996; see Chermak & Musiek, 2011 for review). In addition, delayed neuromaturation and neuromorphological differences have been identified as etiologies underlying CAPD (Chermak & Musiek, 2011; Musiek, Gollegly, & Ross, 1985). Chapter 4 in this volume provides an in-depth discussion of causal factors for CAPD.

Relationships Among CAPD and Language, Learning, Communication, and Related Difficulties

Not surprisingly, individuals with CAPD typically present with auditory difficulties as their primary complaint (AAA, 2010; ASHA, 2005a, 2005b; Bellis, 2003; Musiek et al., 2005). However, additional difficulties often are seen in other areas, especially in children. In some cases, and as discussed below, these additional difficulties may be related causally to central auditory deficits. In others, comorbidity of disorders may occur as a result of dysfunction in shared or closely adjacent neuroanatomical substrates. As previously discussed, even deceptively simple tasks, such as listening in noise, draw upon multiple brain regions and involve complex neurophysiologic interactions (e.g., Salvi et al., 2002).

The literature abounds with studies demonstrating, both behaviorally and electrophysiologically, relationships among central auditory deficits and disorders such as specific language impairment, learning disability, reading difficulties, and ADHD (e.g., Banai, Hornickel, Skoe, Nicol, Zecker, & Kraus, 2011; Bellis & Ferre, 1999; Billiet & Bellis, 2011;

Kraus et al., 1996; Moncrieff & Musiek, 2002; Riccio, Hynd, Cohen, & Gonzales, 1993; Riccio, Hynd, Cohen, & Molt, 1996; Tallal, Miller, & Fitch, 1993; Tillery, Katz, & Keller, 2000; Wright, Lombardino, King, Puranik, Leonard, & Merzenich, 1997). Nonetheless, these linkages should not be taken as evidence that CAPD should automatically be assumed as the underlying cause of learning, language, and related disorders. These higher order functions involve vast processes and mechanisms, only some of which may involve the auditory system.

It is important to recognize that CAPD is a heterogeneous disorder, and the relative impact of a central auditory deficit on functional abilities will be the result of the unique confluence of an individual's bottom-up and top-down processing abilities and a host of other factors (Bellis, 2003). Similarly, language, learning, and related deficits are heterogeneous. Therefore, it is difficult to draw a clear one-to-one correspondence between deficits in central auditory processes and higher order language, learning, communication, and related sequelae using large groups of subjects (ASHA, 2005a; Bellis, 2003). Indeed, the finding of a lack of correlation between fundamental auditory skills (e.g., temporal processing) and higher order, learning-related outcomes (e.g., reading) in large subject groups has led some investigators to postulate that central auditory dysfunction does not result in a meaningful disability affecting learning or related function (e.g., Cacace & McFarland, 2005; Watson & Kidd, 2002).

Other studies, however, have demonstrated that the linkages among central auditory abilities and learning and related outcomes are affected differentially by the specific nature of the central auditory deficit(s) that is(are) present and the type of learning or related difficulty with which the individual presents (e.g., Bellis, 2003; Bellis & Ferre, 1999; Cestnick & Jerger, 2000; Heath, Hogben, & Clark, 1999). Furthermore, even when a direct causal relationship cannot be established or is unlikely, the presence of an auditory deficit certainly can be expected to exacerbate academic and related difficulties by requiring that more effort be expended toward processing the incoming acoustic signal (i.e., disproportionate resource allocation as discussed above), leaving fewer resources available upstream for comprehension, retention, and other higher order functions (e.g., McCoy et al., 2005; Pichora-Fuller et al., 1995). This heterogeneity of disorders, combined with the complexity of interactions between bottom-up and top-down processing, further underscores the need for comprehensive, multidisciplinary assessment in delineating the full spectrum of difficulties exhibited by individuals with CAPD.

Subprofiling CAPD

Notwithstanding the heterogeneity of CAPD and related disorders, an accumulated body of research in auditory neuroscience as well as in neuropsychology and related fields has demonstrated that dysfunction in various brain regions or processing levels can result in relatively predictable *patterns* of deficits across functional domains. These demonstrated brain-behavior relationships have allowed us, in recent years, to begin to develop functional deficit profiles, or subprofiles, of CAPD that relate observed patterns of deficits on central auditory

tests both to neurophysiologic underpinnings and to functional language, learning, communication, and related sequelae (e.g., Bellis & Ferre, 1999; see Bellis, 2002b, 2003 for review).

When considering subprofiling of CAPD, three key caveats must be kept in mind. First, it is critical that any subprofiling method be consistent with what is known about the underlying neuroscience of the system and documented effects of CNS dysfunction on sensitized tests across domains (Bellis, 2003). As such, these theories must be dynamic and based upon solid neuroscience foundations. As the knowledge base regarding the neurophysiologic tenets of CAPD and related disorders evolves, so too must the theoretical constructs evolve. No matter how logical a subprofiling theory may seem on the surface or how well it appears to describe the functional difficulties exhibited by children and adults seen in the classroom or clinic, its validity should always be evaluated relative to the empirical evidence available in the auditory and cognitive neuroscience and related literature.

Second, subprofiling methods should never be viewed as "cookbook" methods of diagnosing or treating disorders. Perhaps the most immutable and predictable aspect of brain function is its very unpredictability. Despite the well-established documentation of certain fundamental brain-behavior relationships and deficit patterns arising from dysfunction in certain brain regions, information—including auditory—processing is too complex to assume homogeneity of functional deficits across individuals. Instead, subprofiling should be viewed as a guide to assist clinicians in identifying patterns of function across multimodal domains so that pansensory or global disorders may be ruled out and intervention efforts may be more focused and deficit specific (Bellis, 2003). At all times, however, diagnosis and intervention should be individualized and based on the unique presentation of the child or adult in question, and one should never expect each individual to fit neatly into a predetermined and circumscribed "box."

Underscoring the importance of this caveat is the study by Jutras and colleagues (Jutras, Loubert, Dupuis, Marcoux, Dumont, & Baril, 2007) in which the records of 48 children diagnosed with CAPD were evaluated with respect to compliance with two functional deficit subprofiling methods—the Buffalo Model (Katz, 1992; Stecker, 1998) and the Bellis/Ferre Model (Bellis, 2003; Bellis & Ferre, 1999). The authors found that 90% of the participants could be "classified" according to the Buffalo Model, which requires only one test for assignment to a subprofiling category. However, when attempting to classify the children according to the Bellis/Ferre Model, which requires multiple tests shown to be valid for central auditory diagnosis, combined with multidisciplinary test results, for assignment, 60% of the children remained unclassified. Unfortunately, there were several drawbacks to the authors' study. First, the authors assumed that in order for a child to be assigned to a specific subprofile, he or she must meet *every* criterion for inclusion in that subprofile, which is inconsistent with the known heterogeneity of both CAPD and learning-related disorders, as well as with the explicit recommendations of Bellis and Ferre (1999). Second, with respect to the Bellis/Ferre Model, the authors utilized test measures that were not included in that model, including unspecified speech-in-noise measures and French

versions of the Staggered Spondaic Word test (Rudmin & Normandin, 1983) and the Synthetic Sentence Identification–Ipsilateral Competing Message test (Lynch & Normandin, 1983). In fact, the only measure utilized in the Jutras et al. (2007) study that conformed to the recommendations by Bellis and Ferre (1999; Bellis, 2003), was the Frequency Patterns test (Pinheiro & Ptacek, 1971). Further, the authors' criteria for assignment to a particular subprofile under the Bellis/Ferre Model were not consistent with the criteria for that model as presented by the authors. Rather than casting doubt on the concept that performance on tests of central auditory function, along with supporting multidisciplinary test results, can be analyzed with respect to intra- and intertest patterns that conform to well-established neuropsychological and neuroscience tenets (see Bellis, 2003 for a comprehensive review), the results of Jutras et al. (2011) serve as a cautionary tale that "cookie-cutter" approaches to CAPD diagnosis should be avoided at all costs.

Third, causality should not be assumed simply because of coexistence of functional deficits, although a causal relationship may be postulated in some cases. For example, deficits in the processing of rapid spectrotemporal acoustic changes, such as those involved in speech-sound (particularly stop-consonant) discrimination, mediated by the primary auditory cortex have been postulated to contribute causally to difficulties in reading and spelling decoding of those same poorly discriminated speech sounds. It has been suggested further that these same auditory processing deficits may lead to deficits in phonological awareness, language, and articulation in much the same way that a hearing loss may cause similar difficulties in these domains (e.g., Bellis, 2002b, 2003; Bellis & Ferre, 1999; Kraus et al., 1996; Tallal et al., 1996). Treatment focused on speech-sound discrimination and speech-to-print skills, therefore, may be effective in improving the auditory component and also may have a beneficial effect on at least some of the related learning and language sequelae (e.g., Bellis, 2002b, 2002c, 2003; Sloan, 1995; Tallal et al., 1996).

In contrast, although individuals with central auditory findings indicative of deficient interhemispheric transfer of information also may exhibit increased visual-motor interhemispheric transfer time, subtle bimanual and/or bipedal deficits, and other intehemispheric difficulties (e.g., Bellis, 2002b, 2003; Bellis & Ferre, 1999; Bellis & Wilber, 2001), it would be unreasonable to assume that the auditory deficit *caused* the deficits in other sensory systems. Instead, these likely represent *associated,* comorbid deficits resulting from dysfunction in a shared neuroanatomical substrate (corpus callosum) (Bellis et al., 2008, 2011; Bellis & Wilber, 2001). In these cases, intervention may include activities that are auditory specific, as well as activities that involve interhemispheric stimulation using other modalities (e.g., Bellis, 2002b, c, 2003; Bellis & Ferre, 1999; Chermak & Musiek, 1997; Musiek & Chermak, 1995).

In still other cases, CAPD may coexist with (albeit not in a causal manner) other, valid diagnoses (e.g., ADHD, multiple sclerosis) in much the same way that hearing loss and vision loss coexist in Usher syndrome or auditory neuropathy/dyssynchrony may coexist with peripheral neuropathies in other sensory systems (Musiek et al., 2005). The presence of these comorbid conditions does not negate the existence of a central auditory deficit. It does, however, render it absolutely critical that the central

auditory deficit be verified using tests validated for this purpose to establish that the source of the auditory difficulties derives from dysfunction in the CANS beyond any exacerbating auditory effects posed by the comorbid disorder. This is important both to rule out a more global, pansensory deficit as a causal factor for reported auditory difficulties, as well as for purposes of designing appropriate intervention.

In conclusion, the use of functional deficit profiling may provide a helpful guide for clinicians in identifying neurophysiologically tenable patterns of deficits across multidisciplinary test results. This may be useful both in differential diagnosis and for purposes of designing comprehensive, deficit-specific intervention plans that address the full spectrum of difficulties exhibited by the individual. However, one should always keep in mind the complexity of the CNS and its function and never assume that any given categorization construct will describe adequately the vast range of difficulties exhibited by all individuals with CAPD.

Future Research in the Diagnosis and Treatment of CAPD

In recent years, the advent of functional neuroimaging and more advanced electrophysiologic and topographical mapping techniques have provided unique insights into neural processing of auditory stimuli. The degree to which these techniques ultimately will be transferable to the clinical arena has yet to be determined; however, these tools have advanced our understanding of central auditory processing and its disorders (see Chapters 3, 5, and 7 in this volume). By the same token, evidence indicating that neuroplasticity extends throughout the lifetime (Kolb, 1995) and that auditory training can facilitate stimulation-induced learning, thus decreasing or ameliorating central auditory dysfunction, has transformed our intervention for the disorder from a management-focused perspective to one that includes specific remediation (i.e., treatment) activities (see Chapters 1 and 7 in Volume 2 of the Handbook). Similarly, our accumulated knowledge of cognitive science and information processing directs us to include top-down strategies, as well as bottom-up stimulation activities in intervention programs (see Chapter 10 in Volume 2 of the Handbook). Together, these bottom-up treatments and top-down, central resources training improve our ability to address CAPD in a deficit-specific and individualized manner. At the same time, the robustness of time-honored and well-established tools for diagnosis of and intervention for CAPD has been demonstrated (e.g., dichotic listening tests, use of assistive listening devices and other environmental modifications), providing continued support for the use of these measures and paradigms in current practice.

As with any complex disorder involving the brain, future research will continue to impact our conceptualization, diagnosis, and treatment of CAPD. Efficient means of screening for the disorder, as well as the development of additional sensitized diagnostic measures, are needed. Validation of models of central auditory processing, including those that involve subprofiling as discussed above, will assist in delineating further the relationship among central auditory processing and learning, language, and related functions. The clinical utility of neuroimaging techniques in diagnosis

of and intervention for CAPD has yet to be determined, as well as the most efficient means to differentially diagnose CAPD and other comorbid or multimodal disorders (AAA, 2010; ASHA, 2005a). Finally, in this era of evidence-based practice, additional treatment efficacy data supporting specific programs of remediation for CAPD are needed. Nonetheless, we have come far since Myklebust's first description of children with "auditory imperception," and at present we have the tools to diagnose and treat CAPD in children and adults. (See Chapter 23 in this volume for discussion of future directions in diagnosis and treatment of CAPD and Chapters 2, 7, and 22 in Volume 2 for discussion of evidence-based practice and treatment efficacy and future directions in intervention, respectively).

Summary

The past five decades have witnessed an evolution in the definition and conceptualization of CAPD and in the methods of diagnosing, assessing, and treating the disorder. Current definitions of CAPD emphasize its auditory, neurobiologic underpinnings while recognizing the complexity of CNS function and the interactive nature of information processing. Because CAPD is an auditory disorder, the audiologist is the professional who, by education and professional scope of practice, is responsible for diagnosis; however, a multidisciplinary approach is essential to appropriate differential diagnosis and comprehensive intervention. This is especially true when CAPD coexists with or is linked, perhaps causally, to difficulties involving language, learning, communication, and related function. The development of functional deficit profiles of central auditory deficits, along with recent advances in diagnosis and treatment of CAPD, likely will result in even more efficient means of serving patients with central auditory dysfunction in the future.

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CHAPTER 9

CLINICAL AND RESEARCH ISSUES IN CENTRAL AUDITORY PROCESSING DISORDER

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Introduction

As with any clinical field, the recommended practices for evaluation of and intervention for children with central auditory processing disorder (CAPD) are dynamic, undergoing reevaluation and improvements as new research emerges. In the process of trying to maintain currency with the new advancements, clinicians should examine some of the more controversial topics that may be encountered in the literature and how these may (or may not) impact clinical practice. These controversies frequently arise from differences in perspective regarding the theoretical conceptualization of CAPD and/or how the disorder should be diagnosed and treated in the clinic. Although the recommended practices for diagnosing and providing intervention for CAPD have been developed by consensus groups with careful consideration of the merits of various positions surrounding these controversies (e.g., AAA, 2010; ASHA, 2005a, 2005b), it is important that the clinician also appreciate the research underlying the issues, so as to make better informed, evidence-based clinical decisions. To this end, we examine a number of these controversies in the present chapter with a focus on the current state of the evidence base supporting conceptualizations of and guidelines for CAPD treatment and intervention (e.g., AAA, 2010; ASHA, 2005a, 2005b). Specifically, this chapter focuses on three broad topics: (1) the degree to which CAPD should be considered an auditory-modality-specific disorder and the impact of comorbid or confounding

supramodal, cognitive, or pansensory deficits on diagnosing CAPD in the pediatric population; (2) which criteria should be used to define and diagnose the disorder; and (3) the efficacy of auditory interventions for CAPD.

Modality Specificity of CAPD and Impact of Comorbid or Confounding Disorders

CAPD in children frequently coexists with other language, learning, and related disorders (e.g., Banai, Abrams, & Kraus, 2007; Banai, Nicol, Zecker & Kraus, 2005; Bellis, 2002; Bellis, Billiet, & Ross, 2011; Bellis & Ferre, 1999; Billiet & Bellis, 2011; Hayes, Warrier, Nichol, Zecker, & Kraus, 2003; Sharma, Purdy, & Kelly, 2009). This comorbidity has led some researchers to the following, not necessarily mutually exclusive, conclusions: (1) CAPD is not an auditory disorder but, instead, is most likely one component of a more generalized cognitive and/or developmental disorder (e.g., BSA, 2010; Moore, 2011); and/or (2) for a diagnosis of CAPD to have any clinical utility, it should be viewed as an auditory-modality-specific deficit. These conclusions have led to arguments such as suggesting that performance-based CAPD evaluations be replaced with parent-report cognition related questionnaires (Moore, 2011) and that central auditory tests should be augmented by analogous testing in other, nonauditory modalities to differentiate CAPD from broader, supramodal, or pansensory issues (e.g., Cacace & McFarland, 2005). The distinctions made by these arguments hold significant implications for diagnosis and, especially, treatment of the disorder. That is, if a child is found to have a sensory processing disorder such as CAPD, whether the disorder exists alone or comorbidly with another disorder in another domain, then deficit-specific auditory interventions are indicated. In contrast, interventions for general developmental or cognitive disorders typically do not include direct auditory remediation, and if they do, that remediation is offered within the context of other treatments (e.g., language, memory) more specific to these other disorders. As such, understanding and disentangling the effects of cognition and sensory processing are critical for appropriate, effective, and efficient treatment and management of the individual child.

Is CAPD Primarily an Auditory Deficit or Should It Be Conceptualized as a General Developmental Disorder or as a Cognitive Disorder?

Certainly, any task that requires a behavioral response from the patient is susceptible to the influence of cognition, assuming that the task requires that the patient be cognizant of what is required of him or her and able and willing to comply with instructions. Perhaps for this reason, the role of patient cognition is an issue that has surfaced in regard to the behavioral assessment of a wide range of disorders. For example, attention has been paid in the literature to the overlap between cognition and speech-language deficits (ASHA, 2004; Rice, 1983; see Ebert & Kohnert, 2011 for a review), visual deficits (Lee, Kwon, Legge, & Gefrohl, 2010; Lee & Vecera, 2005), and psychological

disorders (Zdanowicz & Myslinski, 2010). This issue is compounded by the fact that cognitive measures themselves are not infallible, subject to the same confounds as any other behavioral measure (Heinrich & Schneider, 2011; Merikanto, Lahti, Castaneda, Tuulio-Henriksson, Aalto-Setala, Suvisaari, & Partonen, 2011; Yang, Lung, Jong, Hsu, & Chen, 2010). As one illustration, we note the findings of Merikanto et al. (2011) demonstrating the influence of seasonal variation in mood and behavior on cognitive test performance.

Given the nonmodularity of the central nervous system, there is no question that the influence of cognition and related functions on behavioral tests of central auditory processing should be a concern to the extent that it may cast doubt on the validity of these tests' ability to describe the auditory system's contributions to the exhibited listening difficulties. Auditory processing does not occur in isolation from cognition, nor is cognition independent of sensory-perceptual processing (e.g., auditory processing imposes bottom-up environmental constraints on attention as seen in the *cocktail party effect*; noise increases cognitive load and affects the ability to concentrate; Kujala & Brattico, 2009); musical training, which involves intensive auditory-motor training, strengthens cognitive functions (Strait, Kraus, Parbery-Clark, & Ashley, 2010). In this context, a CAPD is one in which the deficit is primarily auditory, even though there may be comorbid issues in cognitive or related functions. However, it has been argued by some that measures of central auditory processing may *solely* reflect on cognitive processing and do not indicate CANS specific functioning (Moore, Ferguson, Edmondson-Jones, Ratib, & Riley, 2010; Moore, 2011). We present here several lines of evidence that demonstrate that measures of CANS function explain variance that is unique from cognitive measures. As such, these auditory measures provide unique information that would not be obtained through the administration of cognitive tests alone. These lines of evidence include: examining statistically the overlap between cognitive and auditory processing measures, determining the degree to which auditory processing tests emerge as separate factors in factor analyses, exploring the degree to which medications that influence cognition/attention also modulate behavioral central auditory test performance, considering the percentage of children diagnosed with CAPD who also present normal performance on cognitive batteries, and reviewing CAPD diagnostic measures in which attention and memory effects are significantly reduced, such as electrophysiological measures of auditory function.

Overlap Between Cognitive Variables and Central Auditory Processing Measures

The role of cognition in the behavioral assessment of central auditory processing has been examined statistically via correlation analysis in a variety of different cognitive domains (e.g., attention, memory, IQ) and relative to a number of central auditory processes. The rationale behind these investigations is to demonstrate that variability in central auditory processing measures occurs with some degree of independence from cognitive function. Again, as noted above, we do not suggest that auditory processing occurs in isolation from cognitive function,

but rather that central auditory processing and cognition are interdependent and may even be seen as comodulatory systems, and that the shared variance between auditory processing and cognitive measures is rather modest at best. Scores attained on tasks that incorporate frequency discrimination or temporal sequencing have been shown to overlap with cognitive measures, although the degree of shared variance is low. Rosen, Cohen, and Vanniasegaram (2010) noted that tone discrimination ability shared 21% of its variance with nonverbal IQ but was unrelated to measures of attention. Auditory temporal sequencing tests have a similar degree of variance associated with cognition, although it is divided almost equally between memory and attention measures, roughly 10% each (Sharma et al., 2009).

There also exist auditory processing measures that show no significant correlation with cognitive measures. Included among these are gap detection, masking level difference, and certain speech-in-noise tasks. Both Sharma et al. (2009) and Breier et al. (2003) noted that attention and/or memory variables did not impact performance on gap detection or masking level difference tasks. Additionally, some speech-in-noise tasks, such as those included in certain subtests of the SCAN, have not been shown to be significantly correlated with memory and/or attention skills (Riccio, Cohen, Garrison, & Smith et al., 2005; Rosen et al., 2010). It should be pointed out that Rosen et al. (2010) observed that children who scored more poorly on central auditory tests had a significantly lower IQ than those who scored better on these measures. However, this significant difference was due in large part to the finding that the control group exhibited IQs that were significantly higher than average, ranging from 109 to 115 depending on the measure. Children in the poorer central auditory test performing group had near-average IQs ranging from 94 through 98.

In a recent paper, Moore et al. (2010) examined the auditory processing ability of a large sample of children (N = ~1500) ranging in age from 6 to 11 years using a *population-based approach* to understanding the disorder. Consistent with that approach, participants were not recruited on the basis of auditory, cognitive, or speech-language function, as the aim in part was to obtain findings that the authors argued would have a high degree of external validity. The authors noted that those participants who scored in the top and bottom 5% on auditory measures also tended to score the highest and lowest, respectively, on selected measures of cognition. The authors *inferred* that those children performing in the bottom 5% on the auditory measure were those who had CAPD, arguing that their sample was representative of the population and thus the prevalence of CAPD is on the order of 5%. Based in large part on this observation, the authors suggested that CAPD may be a cognitive-based disorder. However, as we elaborate below, the analysis and the conclusions drawn from this study are compromised.

First, the amount of shared variance between the cognitive measures and auditory processing measures when examined across the entire sample was extremely small, ranging from .01% to 10%, depending on the comparison being made. That many of these correlations were significant likely reflects the extremely large sample size used in the study. Second,

the authors' assumption that the bottom 5% of the sample was those who were most likely to have CAPD overlooks what is well known about childhood disorders frequently being comorbid. It is not surprising that those with very poor cognitive ability also have very poor auditory processing ability, and certainly clear conclusions regarding the validity of any one diagnosis cannot be made when the sample under study can have such a wide range of unidentified issues. Finally, and perhaps most significantly, the authors' test battery was atypical: They assumed that the auditory tasks they designed for this study were indeed appropriate for identifying the auditory deficits exhibited by children with CAPD. In fact, the auditory tasks used in this study have no demonstrated sensitivity to CAPD and were not selected from a plethora of verbal and nonverbal central auditory diagnostic tests that have been used internationally for over three decades in the diagnosis of this disorder. Many of these central auditory diagnostic tests have been shown to have considerable construct validity and sensitivity based on a multitude of publications reporting on adults, and less frequently children, with neurological lesions of the central auditory nervous system (CANS) (Bamiou et al., 2012; Chermak & Musiek, 2011; Hurley & Musiek, 1997; Musiek, Chermak, Weihing, Zappulla, & Nagle, 2011; Musiek, Gollegly, Kibbe, & Verkest-Lenz,, 1991; Musiek, Shinn, Jirsa, Bamiou, Baran, & Zaidan , 2005). If the aim of the Moore et al. study was to present an alternative perspective on CAPD, it would seem that including auditory measures with documented efficiency for diagnosis of CAPD and that are widely accepted and used tools to diagnose CAPD in children would have aided the authors in offering a more compelling argument.

Perhaps providing the most compelling data regarding the potential influence of cognitive factors on central auditory processing have been studies using brain imaging and dichotic listening tests, arguably the bedrock of central auditory behavioral tests. Based on hemodynamic data, neural regions that are associated with attention and memory are active when individuals perform dichotic binaural separation (i.e., directed attention to one ear or *forced report* of stimuli from one ear) tasks. This contrasts with dichotic binaural integration measures (i.e., reporting stimuli heard in both ears in any order or *free report*), which appear to be more reflective of true sensory perceptual asymmetries rather than cognitive processes (Hugdahl et al., 2009). This does not imply that binaural separation is driven entirely by attention, as studies in patients with confirmed neurologic involvement have shown that discrete lesions of central auditory regions contribute to reduced competing sentence performance (Musiek, 1983; Musiek et al., 2011). However, binaural separation tasks do appear to recruit areas associated with cognition to a greater degree than binaural integration tasks, as evidenced by these functional MRI studies (Cowell & Hugdahl, 2000; Hugdahl et al., 2009).

In addition to the fMRI studies, several studies have examined the relationship between central auditory processing and cognition using the same binaural separation and binaural integration measures of dichotic listening. Sharma et al. (2009) examined the relationship between binaural integration and cognitive variables (i.e., attention and memory). They observed that the dichotic

measure was significantly correlated with auditory and visual continuous performance, albeit with only 20% shared variance, with no correlation seen with memory. Importantly, the lack of a binaural separation test in this study makes it difficult to comment on the *relative* influence of cognition on binaural integration and separation tasks. Riccio et al. (2005) also examined the relationship between cognition and dichotic tasks. They examined both binaural integration (Staggered Spondaic Words [SSW]) and binaural separation (SCAN- Competing Words) tasks. The sentence recognition subtest of the CELF (Clinical Evaluation of Language Fundamentals) was used as the measure of memory, and a visual (but not an auditory) continuous performance test was applied to assess attention. No correlation was seen between the binaural separation task and attention or memory, and the only significant relationship noted for the binaural integration measure was between the right noncompeting condition of the SSW and the sentence recognition test, purportedly a measure of memory. Although this was presented by the authors as a correlation between binaural integration and memory, that the effect was noted only in the noncompeting condition and with a nondirect measure of memory makes this finding difficult to interpret.

Clearly, additional studies are needed that investigate the relationship between behavioral performance on measures of dichotic processing and cognitive ability. Nonetheless, two conclusions appear clear from the evidence available at present. First, despite the fact that binaural integration tasks typically require the listener to report a larger number of stimuli than do binaural separation tasks, binaural separation tasks appear to recruit additional brain regions that subserve executive function and attentional strategies. Second, despite this fact, binaural separation (or directed-report) tasks, nonetheless, are a valid measure of central auditory dysfunction. See Chapter 18 of Volume 1 of the Handbook for additional discussion of the relationship between cognition and central auditory processing.

To summarize, although some auditory performance measures correlate with measures of cognitive ability and other supramodal domains, for the majority of the diagnostic measures of central auditory function, this overlap generally represents a small amount of the variance between the two variables ($\leq$ 20%) (Riccio et al., 2005; Rosen et al., 2010; Sharma et al., 2009). This does not indicate that auditory processing occurs in isolation from cognitive processing, but merely that central auditory processing tests account for variability that is unique from these supramodal abilities. This likely reflects unique contributions of the CANS to processing of these stimuli. Additionally, there are many central auditory measures that demonstrate no significant relationship with cognitive and other supramodal variables (Riccio et al., 2005; Rosen et al., 2010). Moreover, correlation does not imply causation; therefore, we conclude that attention and memory issues do not *cause* CAPD in children who: (a) have been diagnosed with the disorder using central auditory tests with documented sensitivity and specificity to this disorder, and (b) have had their central auditory processing results interpreted in light of any dominating influence of potential comorbid issues (see AAA, 2010; ASHA; 2005). Notwithstanding the foregoing conclusion, it must be recognized that some degree of shared

variance between CAPD tests and supramodal measures is not unexpected, given what is known about the nonmodularity of the central nervous system. When one views the anatomical, physiological, and hemodynamic characteristics of the CANS within the context of research in neuropsychology (Lopez-Aranda, Lopez-Tellez, Navarro-Lobato, Masmudi-Martin, Gutierrez, & Khan, 2009) and cognitive neuroscience (Ciccio, Meulenbroek, & Turkstra, 2009; Gaffan, 2005; Thiebaut de Schotten, Urbanksi, Duffau, Volle, Levy, Dubois, & Bartolomeo, 2005), there is clear evidence that one should not expect memory, attention, and other cognitive functions to dissociate completely from behavioral auditory functions (see Musiek, Bellis, & Chermak, 2005 for a review). Indeed, one might even predict that attention and memory difficulties might be prevalent in the CAPD population given the increased demands placed on these domains when sensory processing is disrupted (see resource allocation and information processing theories [Pichora-Fuller, Schneider, & Daneman, 1995; Sarampalis, Edwards, Kalluri, & Hafter, 2009; Wong, Ettlinger, Sheppard, Gunasekera, & Ghar, 2010]). Elaborating on this rationale as early as 1996, Bellis (Bellis & Ferre, 1999; see Bellis, 2002 for review) argued that relatively predictable patterns of function across sensory and cognitive domains would occur in many children diagnosed with CAPD. This argument is reinforced by the observation that the frequent comorbidity of CAPD and attention deficit hyperactivity disorder (ADHD) (Chermak, Hall, & Musiek, 1999; Riccio, Hynd, Cohen, Hall, & Molt, 1994) and comorbidity between CAPD and dyslexia (Iliadou, Bamiou, Kaprinis, Kandylis, & Kaprinis, 2009) are at least partly explained on the basis of shared neural substrate and synchronized networks that underlie brain organization and processing (Chermak, Bellis, & Musiek, 2007). Brain organization and function suggest that CAPD may be best characterized not as a disorder in which deficits are auditory alone, but where difficulties in the auditory modality are *most pronounced* (Bellis, Billiet, & Ross, 2011).

Factor Analyses of Central Auditory Processing Measures

Factor analyses of central auditory processing test performance provides another means to analyze the independence of auditory processing from cognition. This statistical approach examines interrelationships among large groups of variables. Variables that are related load on the same factor, while unrelated variables load on different factors. This analysis lends itself well to the question under consideration by providing two separate predictions for the factor analysis outcome. If different auditory processing tests are all being driven by one construct, such as cognition (e.g., attention or memory), then these tests should load primarily onto a single factor that would represent the variable that is common to all of the tests. If, however, each central auditory test assesses some relatively independent aspect of CANS function, then each measure should emerge as a separate factor. To the extent that this latter scenario is observed, it can be concluded that central auditory processing measures assess disparate (auditory) abilities. Utilizing factor analysis would, therefore, be of use in determining the degree to which tests of central auditory function exhibit independence

from common cognitive or psychological variables. Undoubtedly, all behavioral tests (including behavioral tests of central auditory processing) depend to some degree on these psychological variables. However, if auditory measures provide unique information that has some degree independence from psychological function, then these auditory tests should load separately on factors that are related to the type of auditory task or region of the CANS that is recruited.

Several factor analysis studies have provided evidence supporting the independence of auditory processing from psychological variables. Schow and Chermak (1999) administered the SCAN and the SSW test (Katz, 1968) to over 300 children with normal peripheral hearing who were referred for central auditory processing assessment due to reduced classroom performance or attention issues. Subject ages ranged from 6 through 17 years, with the majority of subjects falling in the 7 through 9 years age range. The SCAN competing words, speech in noise, and filtered words subtest scores and the SSW left and right competing condition scores were examined. Results revealed two factors with eigenvalues greater than 1.0. The first factor accounted for roughly 40% of the variance and all dichotic test measures (SSW right and left competing, and SCAN competing words) loaded significantly (loadings of ~.7 or greater). The second factor accounted for roughly 20% additional variance, and all monaural low-redundancy test scores (speech in noise and filtered words) loaded significantly. That auditory processing tests that assess the same auditory process loaded together, yet separate from other auditory processes, argues against the interpretation that auditory processing performance is driven by cognitive variables.

Domitz and Schow (2000) also showed that central auditory tests that assess different auditory processes loaded onto separate factors, although with a slightly different factor structure than was found by Schow and Chermak (1999). A total of 81 children with normal peripheral hearing who ranged in age from 8 through 9 years were recruited. Roughly half of the sample presented no developmental issues, while the rest of the subjects demonstrated a learning disability, speech-language disorder, and/or a diagnosed attention issue. Tests used in the factor analysis included dichotic digits and competing sentences tests based on Musiek (1983) and Willeford & Burleigh (1994), respectively, a frequency patterns test based on Musiek and Pinheiro (1987), a monaural low redundancy test (Selective Auditory Attention Test [Cherry, 1980]), and the subtests of the SCAN included in the Schow and Chermak (1999) study. The factor analysis was performed using criteria similar to Schow and Chermak's (1999) and revealed that central auditory tests tended to load only with other tests that measured similar auditory processes. Specifically, the dichotic digits and competing sentences/words measures tended to load on a single factor (with left-ear performance loading more heavily than right ear performance); the monaural low-redundancy tests that required listening for words in ipsilateral competition loaded together; and the filtered words and frequency patterns tests each loaded alone on separate factors. Hence, four factors, each uniquely representing dichotic processing, monaural low-redundancy (ipsilateral competition), monaural low-redundancy (filtered speech), and temporal processing, were identified. As with the Schow and Chermak (1999) study, the observation that central auditory pro-

cessing tests load on factors that are process specific argues against a model that interprets central auditory test performance as being primarily cognitively driven. Were central auditory processing measures a reflection of underlying cognition, we would expect the tests to load onto one or two factors that are relatively unrelated to the central auditory process being measured. Additional studies using factor analyses that include specific tests of cognitive and related function along with diagnostic tests of central auditory processing would provide further support to this contention.

Effect of ADHD Medications on Auditory Processing Performance

Measuring central auditory function in children before and after administering medication prescribed to reduce inattention has been one research design used to examine the effect of attention on auditory processing. The logic of these studies is that if auditory processing performance measures are *relatively* uninfluenced by these medications, then these measures must be less susceptible to attention effects. Note that the underlying reasoning does not assume that CAPD tests are completely devoid of the influence attention, as this would be inconsistent with what has been previously stated regarding the nonmodularity of the central nervous system. Rather, these studies suggest that CAPD tests are not *as* affected by experimental manipulations of attention as are tests designed to specifically measure this psychological property.

Studies using this experimental design generally demonstrate that performance on diagnostic tests of central auditory function does not change when attention deficits are treated with medication. Tillery, Katz, and Keller (2000) recruited children with ADHD and normal IQ, approximately 60% of whom were also diagnosed with comorbid CAPD. Patients were administered an auditory continuous performance task (i.e., a measure of attention) and a central auditory test battery on two separate occasions: with no ADHD treatment and while taking Ritalin (methylphenidate). The order of the treatment and control conditions was randomized across subjects to control for potential learning effects in the analysis. Only the auditory continuous performance measure improved significantly while on Ritalin. Furthermore, Sutcliffe, Bishop, Houghton, and Taylor (2006) noted that children diagnosed with ADHD did not perform any differently on frequency modulation tasks when medicated; however, they noted that performance on frequency discrimination tasks changed as a function of medication. As the frequency modulation task always preceded the frequency discrimination task in this study, the latter task may have been more susceptible to subject fatigue. In addition, the longer duration frequency modulation detection stimulus could have afforded the children more time to "tune in," which may have served to minimize between condition differences. In a third study, Keith and Engineer (1991) also noted results that were dependent on the CAPD test utilized: An effect of ADHD medication was seen on dichotic and some auditory closure tests (e.g., filtered words), but not on other auditory closure tests (e.g., speech recognition in speech babble). For more of an elaboration on the effects of medication, the reader is referred to Chapter 19 in Volume 2 of the Handbook.

Overall, these findings are consistent with the proposition that attention influences performance on psychological measures of this construct more than it influences performance on CAPD measures, supporting the position that these two disorders are different clinical entities. It should be noted that we do not contend here that CAPD measures are completely devoid of attention effects: Such a contention would be inconsistent with evidence relating to the nonmodularity of the central nervous system cited in previous sections of this chapter. As stated above, we argue that a CAPD is one in which the deficit is primarily auditory, even though comorbid issues may be present. For this reason, it is generally recommended that children with ADHD who are on medication to control attention-related symptoms be tested in the medicated state for purposes of differential diagnosis of CAPD to reduce any potential negative impact of the attention disorder on auditory processing performance (AAA, 2010).

Poor Auditory Processing Performance in the Presence of Normal Cognition

When examined clinically, there is a notable percentage of patients who demonstrate central auditory processing deficits in the absence of attention, memory, and/or cognitive deficits. Indeed, many clinicians suggest that central auditory diagnostic testing only be conducted after measures of cognition, language, attention, and other possibly confounding factors have been completed (Bellis, 2003; Fandino, Connolly, Usher, Palm, & Kozak, 2011; Whitton, 2010). As these children are generally referred for some type of listening difficulty, it is important to highlight that these listening difficulties would likely be delegated as nonclinical if not for the specific assessment of auditory processing ability. Riccio et al. (1994) observed that 50% of pediatric patients being evaluated for CAPD and attention disorders showed auditory processing difficulties only. Ricco et al. (2005) found that roughly 30% of their sample was diagnosed with CAPD only, without any comorbid attention or memory disorder. Similarly, Sharma et al. (2009) noted that 30% of their subjects diagnosed with CAPD had no cormorbid attention or memory issues, though some did present with comorbid speech language issues. These findings underscore the need for multidisciplinary input in the differential diagnosis of CAPD, as recommended by current US consensus statements and guidelines (AAA, 2010; ASHA, 2005a, 2005b).

Electrophysiological Findings Are Primarily Sensory Based

Although the influence of cognition on auditory evoked and event-related potentials varies depending on the electrophysiologic measure under consideration, it is generally well accepted that brainstem evoked responses have a very large sensory component. Indeed, some of these responses are obtainable while the patient is under sedation with little to no alteration to waveform characteristics. The auditory brainstem response (ABR) can be evoked by clicks, speech, or other complex stimuli, and in a variety of different paradigms. Additionally, the middle-latency response (MLR) and obligatory cortical event-related potentials are also somewhat resistant to general cognitive

abilities, although they are potentially more susceptible to nonauditory factors, particularly if proper within-subject indices are not utilized (see below).

Click-evoked ABRs have some, although limited, utility to differentiate children who show characteristics of CAPD from those who do not. Hall and Grose (1993) compared ABRs evoked by clicks in a standard clinical protocol in children who had a significant history of otitis media (OM) but were currently asymptomatic and those who did not have this history. Results indicated that children with a history of OM showed significantly later waves III and V. As these responses were acquired shortly after the OM episode, the findings do not speak to the persistence of this deficit over time. Research has indicated, however, that OM-driven CAPD deficits tend to persist throughout adolescence and can cause long-term delays in developmental milestones when certain conditions are met (Whitton & Polley, 2011).

Two unique paradigms incorporating click-evoked ABRs have been employed in an attempt to identify children with CAPD. These paradigms are unique in that they go beyond the standard monaural protocol in quiet that is generally performed in the clinic. In the first paradigm, Delb, Strauss, Hohenberg, and Plinkert (2003) computed a binaural interaction component (BIC) in which binaural potentials were evoked at different interaural timing differences (ITD). Using a 400-microsecond ITD and designating absence of the BIC wave as indicating dysfunction, the authors found that the BIC measure had a 76% sensitivity and specificity for predicting those children who also exhibited difficulties on behavioral tests of central auditory function. In the second paradigm, Marler and Champlin (2005) used an ABR backward masking protocol to determine whether children with language-based learning impairment could be differentiated from normally developing controls. They reported that wave V latency did not differ between groups when the ABR was acquired in quiet; however, when a noise burst immediately followed the ABR stimulus, the wave V latency of the group with language-based learning impairment was significantly longer than the controls. Taken together, these findings suggest that using unconventional ABR protocols as correlates of behavioral auditory processing measures (i.e., binaural hearing and backward masking, respectively) can be useful in identifying children with CAPD and that because the ABR is generally thought of as a basic sensory measure, one can conclude that the effect is not heavily influenced by cognition.

In the past decade, the speech-evoked ABR (cABR) has emerged as a relatively reliable and valid means of identifying children with a wide range of deficits who show neural timing issues in the brainstem. Specifically, certain pediatric clinical groups seem to lack the ability to efficiently encode the acoustic characteristics of speech stimuli at the brainstem level (Banai, Abrams, & Kraus, 2007). Evidence for the utility of this measure in objectively identifying auditory contributions to clinical symptoms comes from a variety of different laboratories. For instance, Filippini and Schochat (2009) noted significant differences in cABR indices between children who were diagnosed with CAPD and those who were not. Johnson, Nicol, Zecker, and Kraus (2007) examined the cABRs of two groups of children: those with and without language-based learning

based on their performance in a backward masking task. They found that those children with learning difficulties who also showed poor psychoacoustic backward masking ability exhibited significantly delayed cABR latencies relative to those who performed well on the psychoacoustic task. Anderson, Skoe, Chandrasekaran, Zecker, and Kraus (2010) examined cABRs in children with dyslexia, noting a significant relationship between the cABR and behavioral hearing-in-noise ability even after partialing out the effects of memory. Billiet and Bellis (2011) examined performance on behavioral tests of central auditory function in two groups of children diagnosed with dyslexia: those who exhibited normal cABRs and those who did not. They found that the children with normal cABRs tended to exhibit a diagnostic pattern on central auditory tests consistent with a diagnosis of CAPD with a cortical locus of dysfunction. Conversely, those with abnormal cABRs tended not to demonstrate a cortical pattern on the behavioral central auditory tests. Billiet and Bellis concluded that the electrophysiological and behavioral measures tap different aspects of central auditory processing and different neuroanatomical substrates and that only by looking at the results from both measures in tandem can the most sensitive diagnostic battery be determined.

Another electrophysiologic measure that appears to reflect basic sensory processing of auditory stimuli is the middle latency response (MLR). Although the MLR is generally considered to be primarily sensory in nature (Jerger, Johnson, & Loiselle, 1988), it also tends to be more influenced by states of consciousness (i.e., sleep, sedation) than sensory evoked potentials that occur in earlier latency ranges. This influence is generally relatively minor, however, occurring only in specific stages of sleep (McGee, Kraus, Killion, Rosenberg, & King, 1993). Nonetheless, MLR offers some utility as an objective biomarker of CAPD. For example, children diagnosed with CAPD based on diagnostic behavioral measures have been shown to exhibit significantly smaller MLRs when compared with children who perform well on central auditory test batteries (Schochat, Musiek, Alonso, & Ogata, 2010). Additionally, Purdy, Kelly, and Davies (2002) reported significant differences between children with CAPD and controls on measures of the MLR, specifically a reduction of Nb amplitude and delayed Na latency. As with the MLR, the N1–P2 also tends to be more susceptible to nonauditory factors than brainstem potentials. Nonetheless, it still reflects important contributions from sensory regions of the central nervous system, as evidenced by its exogenous nature (Musiek, Froke, & Weihing, 2005). Using this potential, McArthur, Atkinson, and Ellis (2009) reported atypical cortical evoked responses to tones, vowels, and consonant-vowel syllables in 38% of 6- to 12-year-old children with specific language impairment or specific reading disorder.

The clinical utility of both the MLR and the N1–P2 may be enhanced in part by utilizing relative instead of absolute evoked potential indices. An absolute index is one that reflects measurement of an evoked potential under only one set of conditions. For instance, the amplitude of the MLR waveform acquired under left ear stimulation would represent an absolute index. Conversely, a relative index is one that is computed based on two or more absolute indexes (e.g., computing the difference in amplitude between the

MLR obtained with right ear stimulation minus that obtained with left ear stimulation). Relative indexes offer an advantage in partly controlling for nonauditory individual differences that may be reflected in the evoked potential recordings. For instance, when computing a relative index by subtracting the left ear evoked MLR amplitude from the right ear MLR amplitude, any nonauditory influence can be assumed to be equal for both left and right ear recordings and is subtracted out of the measurement when the relative index is computed. Accordingly, these measures have been shown to significantly reduce the within-group variability of the MLR in normal hearing children from about 7 years of age through the mid-teens (Weihing, Schochat, & Musiek, 2012). Therefore, these relative MLR measures, and their N1–P2 counterparts, could be more likely to indicate central auditory function that is less influenced by nonauditory variables.

Should Cognitive Testing and/or Nonauditory Measures Be Incorporated into Central Auditory Test Batteries to Assist Audiologists in Differential Diagnosis?

Best practice recommendations for the diagnosis of CAPD highlight the importance of incorporating knowledge of the patient's cognitive, speech-language, and related abilities into interpretation of central auditory test battery findings (AAA, 2010; ASHA, 2005a, 2005b). This recommendation is made because auditory processing does not occur separately from psychological processes, and the two can influence each other reciprocally. The goal in evaluation of central auditory processing is to determine if the deficit is primarily (or fundamentally) auditory in nature, so that the most efficient and effective intervention can be applied. Such a determination requires multidisciplinary assessments and examination of patterns of brain-behavior relationships. See Chapters 8, 18, and 20 in Volume I of the Handbook. As noted in these documents, the reasons for multidisciplinary assessment to complement central auditory testing stem from the high prevalence of comorbidity among developmental disorders in pediatric populations and the need to consider functioning across domains for purposes of differential diagnosis and development of the most appropriate, individualized intervention plan. For diagnostic central auditory testing, this means that reducing the impact of these confounds to the greatest extent possible must always be on the clinician's radar if an accurate diagnosis is to be achieved. Some have recommended incorporating cognitive tests in the central auditory evaluation, as well as utilizing visual analogs of central auditory processing tests to rule out supramodal issues. Both these suggestions are considered below.

Incorporation of Cognitive Tests in CAPD Test Batteries

An initially intuitive solution to the issue of teasing out the influence of nonauditory comorbidities would be to incorporate measures of cognition into central auditory assessment, as occurs with some frequency in the United States when screening for CAPD, although there has been no formal protocol established. For example, Bellis (2003) described a screening protocol in which cognitive

(e.g., auditory continuous performance testing to rule out attention issues; forward and backward digit span measures to assist in screening for potential memory issues), speech-language, psychoeducational (e.g., intelligence testing to screen general intelligence constructs), and related testing were reviewed to determine candidacy for central auditory evaluation. In the United Kingdom, a more direct approach has been developed by the Institute of Hearing Research Multi-Center Study of Auditory Processing (IMAP) (MRC Institute of Hearing Research). In this protocol, a standardized measure of cognition is administered to all children undergoing evaluation for CAPD to ensure that information relevant to comorbidities is available during the diagnostic session. Several issues arise, however, in either approach, which may not be apparent initially. The largest barrier, at least in the United States, is that administration of cognitive tests beyond a simple screener falls outside the scope of practice of audiologists. Furthermore, many of the more thorough cognitive batteries require a doctoral degree in psychology and/or a clinical psychology license to purchase and administer the tests (Thomas & Hersen, 2010). Failure to follow these guidelines could jeopardize the license of the practicing audiologist.

To overcome this conundrum, the AAA (2010) guidelines for diagnosing and treating CAPD recommend that information relating to cognitive and other comorbid issues be *available* to the audiologist at the time of CAPD evaluation, and/or the CAPD battery must be modified accordingly to account for any known or potential cognitive issues. The implication is that children should be evaluated for CAPD following cognitive and related assessment. Assuming this assessment has been performed, the audiologist should have sufficient information to proceed with the CAPD evaluation, taking into consideration, for example, utilization of tests that limit memory and attention load (see previous section in this chapter). Additionally, if a disorder, such as ADHD, has been diagnosed, then appropriate accommodations can be put into place before administering the CAPD battery to reduce the influence of this potential confound (see Chermak, 2007 for discussion).

Given the resources available to the audiologist with this multidisciplinary approach, it would seem unnecessary and, at least in the United States, potentially unethical for audiologists to administer cognitive measures beyond a short screener. Certainly, if such measures were not routinely given by psychologists, these cognitive tests would represent a void in assessment that needed to be filled. However, given that there is already a profession devoted to assessment of cognition and that referrals to these professionals can be made, it would seem unnecessary to incorporate diagnostic cognitive tests into CAPD batteries.

Use of Nonauditory Analogs of CAPD Tests to Rule Out Supramodal Issues

Common to most consensus statements on CAPD (AAA, 2010; ASHA, 2005a, 2005b) is the contention that although CAPD may coexist with dysfunction in other modalities due to shared neuroanatomical substrates, CAPD is not the result of supramodal or global dysfunction but, rather, is a disorder of the CANS. In addi-

tion, CAPD should manifest itself primarily, if not solely, in the auditory modality. Coexisting deficits and comorbidities highlight the importance of considering supramodal factors that could influence test results and the symptoms being exhibited by patients. To this end, Cacace and McFarland (2005) have argued for the inclusion of visual analogs of central auditory processing test measures to enhance the diagnostic specificity of CAPD assessment and the clinical utility of the diagnosis. The rationale underlying their approach seems to be that if a child demonstrates difficulties on auditory measures only, then the disorder is likely to be auditory in nature; however, if the child demonstrates difficulties on both auditory and nonauditory (e.g., visual) tests, then it would seem less likely, though not impossible, that the disorder is auditory specific. Although this approach has some merit, logistic pitfalls and issues of compliance with published best practice recommendations render this approach of questionable practical value. Some of these issues are considered below, including: The information gained from the inclusion of nonauditory analogs in central auditory test batteries is redundant with information gained from neuropsychological assessment and intratest measures of CAPD; there exist differences in the way basic sensory elements are processed in the primary auditory and visual cortices; cross-modal test equivalence for behavioral auditory and visual tasks is difficult to achieve; and there is a lack of clinically feasibly visual analogs available to audiologists.

In regard to issues of redundancy in testing, modality specificity encounters many of the same issues identified previously in this chapter. Specifically, the detection of cognitive issues that may cause supramodal deficits is within the purview of the psychologist or neuropsychologist who evaluates the patient prior to CAPD assessment. We can certainly expect that the tools used by these professionals have a sufficiently strong foundation to detect global processing issues that may be contributing to the observed auditory symptoms. If such issues are severe, then their treatment should precede CAPD assessment. Indeed, it could be that cognitive issues may be the only cause of the patient's listening difficulties and, following treatment for the cognitive issue, the auditory deficit may be resolved. In such a scenario, CAPD assessment is unnecessary.

Also related to redundancy is the finding that intratest analyses of central auditory processing tests can frequently be used to determine if the deficit is primarily auditory or if it is being driven by a supramodal issue, such as ADHD, as demonstrated by Bellis et al. (2011). Their study noted that interaural differences on dichotic tests and differences in performance based on response mode on temporal patterning tests were able to correctly differentiate children with CAPD from those with ADHD. Specifically, children with CAPD were shown to perform much better on one ear than the other when given dichotic tests, yielding a large interaural difference, while children with ADHD tended to perform poorly on both ears, yielding a small interaural difference. (See Chapter 9 of Volume 2 of this Handbook for elaboration of the mechanisms behind these interaural differences.) For temporal patterning tests, asking children to label a sequence is a significantly more demanding auditory processing task than asking children to

simply hum the sequence. In the former, both cerebral hemispheres are recruited as well as the corpus callosum, while in the latter, generally only the right hemisphere is recruited (Musiek, Pinheiro, & Wilson, 1980). The performance difference between these two response modes generally will yield a large difference for children with CAPD. For the child with ADHD, however, the task is equally difficult regardless of response mode, so the performance difference between the two conditions is small or nonexistent. As these intratest comparisons are readily available to audiologists, can be easily calculated, and can differentiate children with CAPD from those with supramodal issues, it is unclear what the audiologist would gain by utilizing visual analogs of CAPD tests.

The differences in which primary auditory and visual cortices process relevant sensory elements is a second barrier to utilizing visual analogs. In a review of the fundamental processing differences between these two systems, King and Nelken (2009) pointed out that the processing that occurs at the first levels of the visual cortex is most similar to the processing that occurs subcortically at the inferior colliculus in the auditory system. Further, the authors highlighted that learning-based plasticity tends to be more dramatic in the auditory system and that the auditory system has a relatively robust corticofugal pathway that has no equivalent of equal strength in the visual system. Put simply, the auditory system engages in a large degree of subcortical processing that does not occur in the visual system, and appears to demonstrate physiologic properties that are distinct from the latter system. These gross differences between the central auditory and visual systems make it difficult to equate performance on auditory tests and their purported visual analogs.

Perhaps related to this observation, a critical issue in trying to develop visual analogs to auditory processing tasks is to equate stimuli and tasks across the two modalities. There is, for example, no reason to expect that dichotic processing of two digits per ear is of comparable difficulty to dichoptic processing of two digits per visual field. It is entirely possible that the effort and skill required to yield a criterion performance level in one modality could be less than or greater than the effort and skill required to achieve similar levels in the other modality for a similar task. A recent study by Bellis and Ross (2011) suggests that this is indeed the case, as performance on auditory processing tasks is significantly better than performance on visual analogs of these tasks administered to normal hearing children and adults. This would seem a shortcoming for an approach that attempts to establish equivalence across two disparate modalities. There are some potential solutions to this issue, however. For instance, psychometric functions could be established for normal hearing subjects that ensure that measures in both modalities are equated for performance.

A final limitation of the analog approach is the feasibility of cross-modal testing in the audiology clinic. Although this approach has been advocated for some time (McFarland & Cacace, 1997), until recently there were no tasks designed for audiologists to perform this testing in the clinic (Bellis & Ross, 2011; Bellis et al., 2011; Lawfield, McFarland, & Cacace, 2011), and it is still unclear how audiologists would go about acquiring these stimulus materials. Furthermore, the research data obtained with these nonau-

ditory analogs do not support feasibility in a clinical setting in their current form.

In summary, inclusion of cognitive measures and/or nonauditory analogs in the diagnosis of CAPD seems inappropriate and of limited utility for one or more of the following reasons: (1) measures are not in audiologists' scope of practice, (2) measures do not generally contribute unique information when compared with central auditory tests already in use in diagnosing CAPD, and (3) difficulties exist in designing nonauditory analogs to achieve the stated goal. Again, consistent with ASHA (2005a, 2005b) and AAA (2010) recommended practices, it is our view that multidisciplinary testing, which employs standardized, validated, and efficient tests administered by various professionals trained in diagnosis of dysfunction in other sensory modalities and supramodal functions (e.g., executive function, attention, working memory), is a more appropriate approach.

Issues Pertaining to Criteria Used to Diagnose and Define CAPD

In regard to which tests should be used to diagnose and define CAPD, considerable variability in clinical practices and research designs has led to a general misunderstanding of the nature of the disorder. Although the recent AAA (2010) document sought to address many of these issues, this document is still relatively new and many of the issues it was drafted to remedy are still prevalent. Two of the more common issues that have contributed to this variability include: (1) operationally diagnosing CAPD based on self/parent/teacher report of listening difficulties alone, and (2) utilizing tests that have not been validated using the current gold standard in deciding which subject qualifies as having CAPD.

Presence of Listening Difficulties or Parent/Teacher Concern as a Criterion for "Diagnosing" CAPD

Determining (or approximating) the gold standard for CAPD is of considerable consequence for clinical practice, as well as for the conduct of research and the validity of findings. Current recommendations by AAA (2010) and ASHA (2005a, 2005b) are to utilize subjects with neurological lesions localized mainly to the CANS in determining which tests are most sensitive and specific to the disorder. Proponents of this approach argue that the method provides a clear way to operationally define *pure* forms of the disorder and shows how performance on a measure is affected by very clearly defined CANS involvement (AAA, 2010; ASHA, 2005a, 2005b). Moreover, this brain-behavior approach, which utilizes dissociations and double-dissociations of function, is the bedrock of virtually all diagnostic professions that address neurologic function. Opponents of this approach argue that many individuals who demonstrate behaviors consistent with CAPD, in particular children, do not have clearly defined neurological lesions, and so this method of validation is not satisfactory.

This disagreement has led to a range of methods by which children are determined to present with CAPD when being considered in the research literature. Common to many of these methods, however disparate the methods may be, is an attempt to define central auditory

processing ability using *performance-based* measures. Unfortunately, another approach, use of parent and teacher reports of listening difficulties for identification of children *suspected of CAPD*, remains a relatively common practice, especially in classifying research participants. When this approach is used, it is impossible to discern effects that may be attributable to true auditory difficulties versus many unknown variables, as the "suspected of CAPD diagnosis" is based on observation rather than on performance. Illustrative of this issue is a recent study by Ferguson, Hall, Riley, and Moore (2011) in which the communication, listening, cognitive, and speech perception skills of children identified as having CAPD on the basis of normal audiometry and *typical* symptoms of CAPD, as reported by parents, were examined. The authors' report of no performance differences between the group *presumed* to have CAPD based on parent report and the group with specific language impairment (who met clinical criteria for this diagnosis) demonstrates that parent report is insufficient to diagnose or differentiate children with CAPD from those with speech-language difficulties.

Moreover, studies that have examined whether screening measures can predict performance on central auditory measures generally have not shown a significant relationship between subjective report of children's listening symptoms and their actual performance on central auditory measures. Since the central auditory measures show a significant degree of construct validity in neurological models of CAPD, this lack of agreement indicates that subjective report of listening difficulties is not predictive of true CAPD. For instance, Wilson et al. (2011) found minimal correlation between questionnaires such as the Children's Auditory Performance Scale (CHAPS) and Screening Instrument for Targeting Educational Risk (SIFTER), Test of Auditory Processing Skills-Revised (TAPS-R), and performance-based diagnostic tests. Specifically, the screening measures shared, at best, only 9% of the variance with diagnostic measures, and the vast majority of the screening subscales demonstrated no significant relationship to diagnostic measures at all. Wilson et al. (2011) concluded that the CHAPS, SIFTER, and TAPS-R do not predict risk for CAPD. They suggested that these questionnaires may be used to highlight concerns about a child but not to determine whether a diagnostic central auditory processing assessment is warranted. Similar findings were reported by Drake, Brager, Leyendecker, Preston, Shorten, and Stoos (2006) and Lam and Sanchez (2007), further demonstrating that subjective report of CAPD symptoms is a poor indicator of actual performance-based auditory processing difficulties. It should be noted, however, that Iliadou and Bamiou (2012) observed a much stronger correlation between the CHAPS and diagnostic CAPD measures in a sample in which the age range of the participants was strictly limited to 11 to 12 years. They noted that the reduced variability in this age group relative to younger children may have contributed to the stronger relationship seen between observer-report and performance-based measures.

The serious limitations of the suspected of CAPD diagnosis also is corroborated by the finding that many children referred for CAPD evaluations because of listening difficulties actually perform quite well on central auditory processing measures. One would assume that if parent and teacher reports were good

predictors of auditory processing difficulties, then the CAPD hit rate (i.e., true positives) for these referrals would be much higher. For example, Wilson (personal communication) in a forthcoming study used a variety of different diagnostic criteria to determine the percentage of CAPD diagnoses. Using self-reported difficulties as a measure of central auditory dysfunction (per Ferguson et al., 2011) yielded a CAPD hit rate of 100%, whereas using sensitized performance-based measures that followed ASHA (2005a, 2005b) and AAA (2010) diagnostic criteria yielded a smaller hit rate of 71%. The latter percentage is generally consistent with the hit rates reported in other studies utilizing performance-based measures to diagnosis CAPD: Sharma et al. (2009) (72%), Iliadou and Bamiou (2012) (66%), Vanniasegaram, Cohen, and Rosen (2004) (56%), and Rosen et al. (2009) (62%). Clearly, the use of reports of listening difficulties as a diagnostic marker for CAPD leads to overidentification and overuse of the diagnostic label (i.e., higher sensitivity, but at the cost of significantly reduced specificity).

Validity of Diagnostic Tests of Central Auditory Function: The Gold Standard

Proponents of the use of patients with CANS lesions to determine test efficiency for identification of individuals, including children, with CAPD do not argue that all clinical presentations of CAPD involve neurological lesions (for a review of potential causes of CAPD in nonneurological cases, see Chermak & Musiek, 2010 and Musiek & Weihing, 2011). They do contend, however, that when considering the relative merits of a group of central auditory processing tests, we can learn which might be more advantageous for diagnosis than others by administering such measures to individuals who have known neurological involvement of the CANS. All else being equal, if one test differentiates individuals with focal CANS lesions better than another, then that test might be said to be a more valid indicator of difficulties in central auditory processing, since central auditory processing is a reflection of CANS function. Measures that have not been validated in this manner are not considered diagnostic under the guidelines set forth by AAA (2010) and ASHA (2005a, 2005b). Certainly, any measure of central auditory processing could potentially be diagnostic, but validation of the test using a clinical *gold standard* group provides a basis on which to make this claim. This requirement applies to both the specific auditory process being tested as well as to the specific stimuli and paradigm used. That is, it is not sufficient to claim a test is diagnostic because it is dichotic; one must administer the specific dichotic test to a group of individuals with documented CANS dysfunction to determine the relative utility of that specific dichotic test for diagnosis. The sensitivity and specificity of different dichotic tests can vary markedly (Musiek et al., 2011).

The current approach for approximating a gold standard may change with further developments in neuroimaging and other techniques. For example, use of fMRI (Bartel-Friedrich, Broecker, Knoergen, & Koesling, 2010) and transcranial magnetic stimulation (TMS) (Andoh & Zatorre, 2011; At, Spierer, & Clarke, 2011) in normally hearing individuals to determine processing sites for specific central auditory tests might offer one such approach to achieving a gold standard

for central auditory tests. Unfortunately, such an approach introduces the risk of inferring how pathology will manifest in the clinic by simulating it in controls. At the present time, there seems to be no better approach to validating clinical tests and determining their sensitivity and specificity than to use patients with confirmed CANS dysfunction.

A recent report by Musiek et al. (2011) demonstrates the benefits of comparing the performance on multiple central auditory tests in the same group of patients with neurological lesions of the CANS. It was noted that when comparing across individual tests, the Dichotic Digits Test (Musiek, 1983) and the Frequency Patterns Test (Musiek & Pinheiro, 1987) tended to show the best test efficiency (i.e., balance between sensitivity and specificity). Furthermore, the two-test battery comprising Dichotic Digits and Frequency Patterns, as well as the Competing Sentences Test (Willeford & Burleigh, 1994) and the Frequency Patterns two-test battery tended to show the best test efficiency of all the multiple test batteries compared in the group of adult patients studied. Musiek et al.'s findings should not be interpreted to suggest that these measures and batteries will have the same test efficiency in actual clinical CAPD populations. Their results suggest, however, that these measures and batteries will likely be better at detecting auditory difficulties that originate from the central nervous system in children and adults referred for CAPD testing because individuals with clear CANS dysfunction have difficulty performing these tests. Unfortunately, very few studies have compared performance on central auditory tests in pediatric populations. Jerger (1987) indicated sensitivity and specificity for the Pediatric Sentence Identification Test (Jerger & Jerger, 1984) that approached the efficiency of CAPD tests commonly used in adults with neurological lesions. More recently, several revealing studies have come from Boscariol and colleagues (2009, 2010, 2011), again showing similar central auditory test performance patterns and efficiencies as those typically encountered in adults.

Issues Relating to Intervention for CAPD

Our final section focuses on the efficacy of auditory interventions for children and adults diagnosed with CAPD. This topic is of considerable interest to audiologists and speech-language pathologists, families, and patients, as well as medical professionals who are often point-of-entry service providers for patients who have listening difficulties not explained by their hearing loss. Current research in auditory rehabilitation for patients with CAPD has tended to address how efficacious a given treatment is for individuals diagnosed with the disorder. These clinical studies, along with the basic research upon which the interventions are based, are described below. We restrict our consideration of the literature to auditory interventions that utilize auditory training exercises, where these exercises are defined as behavioral tasks that are auditory based and aim to strengthen the basic sensory processing of auditory information at the level of the CANS.

Animal Studies

A variety of evidence of the mechanisms underlying benefits obtained from auditory training comes from animal models. Principal among these are reports of

structural and/or physiological changes to auditory regions of the central nervous system following participation in an auditory task. For instance, Recanzone, Schreiner, and Merzenich (1993) found tonotopic reorganization of the owl monkey's auditory cortex following intensive training on a frequency discrimination task. The posttraining tonotopic gradient favored the trained frequencies used in the paradigm. Control subjects showed significantly less or no reorganization. Hassmannova, Myslivecek, and Novakova (1981) observed increases in the RNA content of cortical neurons following repetitive stimulation of the auditory cortex via tone pips, suggesting an initiation of processes through which cell division might occur. Similar increases were not seen in a control group. Bao, Chang, Woods, and Merzenich (2004) provided different auditory feedback to rats in a maze depending on their proximity to a food source. They noted that these rats showed an improved neural response to the auditory signal when compared with untrained rats.

These animal models also have been used to examine the question of mechanisms underlying efficacy of auditory training from a somewhat different perspective: How does the CANS adapt beneficially when it is challenged by an environment in which important acoustic cues are degraded? Knudsen (1988) repeatedly assessed localization ability using a behavioral task after unilaterally depriving the barn owl of sound using an ear plug. Initially, performance on this task suffered following insertion of the plug, although a gradual improvement was noted over time. This improvement was attributed to beneficial alterations in CANS organization that reflected a better match between expected object location and the new degraded cues. Removal of the plug again yielded poor performance that gradually improved over time, again reflecting a recalibration of the localization circuitry in response to the change in interaural stimulation. This finding proved to be fairly robust and was replicated in similar studies (Linkenhoker & Knudsen, 2002; Linkenhoker, von der Ohe, & Knudsen, 2005).

Auditory Training in Normally Hearing Human Subjects

One must understand how the normal system works before one can begin to explain the abnormal. A considerable body of auditory training research has been conducted in the normal hearing population, where normal hearing is typically defined as both normal peripheral hearing sensitivity and the absence of any significant disorders. Typically, these investigations examine auditory training within the context of a particular auditory processing task, such as auditory discrimination or temporal processing.

In this regard, the work of Kraus, Tremblay, and colleagues has provided some of the most convincing evidence for the effectiveness of auditory training in normal-hearing subjects. Kraus, McGee, Carrell, King, Tremblay, and Nicol (1995) and Tremblay, Kraus, McGee, Ponton, and Otis (2001) trained subjects on discrimination of consonant-vowel (CV) stimuli that varied in their spectral similarity. They indicated that most individuals exhibited behavioral and electrophysiological benefits following one week of training. Tremblay, Kraus, Carrell, and McGee (1997) showed a similar degree of benefit using a CV discrimination training task and also noted that training effects generalized to stimuli not trained in the

experiment. The finding of generalization to stimuli (and tasks) not included in the training protocol has also been reported by Delhommeau and colleagues (Delhommeau, Micheyl, & Jouvent, 2005; Delhommeau, Micheyl, Jouvent, & Collet, 2002), using a frequency discrimination task. Others have reported that improvement typically is greatest on the trained task and may not transfer or generalize between tasks (Hawkey, Amitay, & Moore 2004; Wright & Fitzgerald, 2001). The degree of generalization/transfer seen may be predicted by the subject's initial ability: Adult listeners who perform better initially demonstrate greater transfer to untrained stimuli and untrained conditions (Amitay, Hawkey, & Moore, 2005; Roth, Appelbaum, Milo, & Kishon-Rabin, 2008) and the degree of generalization depends on listeners having received a critical amount of exposure to the trained stimulus and task (Delhommeau et al., 2005; Grimault, Micheyl, Carlyon, Bacon, & Collet, 2003; Wright & Sabin, 2007).

Using a nonspeech tonal discrimination task, Jancke, Gaab, Wustenberg, Scheich, and Heinze l (2001) showed that some individuals demonstrated behavioral and electrophysiological benefits from auditory training. Individuals who benefited from training differed from those who did not in that the former group showed a decrease in activity during training in the superior temporal gyrus bilaterally on fMRI. The authors interpreted this hemodymanic finding to be consistent with "fast learning theories," which suggest that fMRI activation decreases as processing becomes more efficient. Foxton, Brown, Chambers, and Griffiths (2004) demonstrated that discriminating differences in auditory contour, or relative changes in frequency over time, relied heavily on frequency discrimination ability. Specifically, subjects who were trained in either frequency or contour discrimination showed an improved ability to discriminate auditory contours. As auditory discrimination and sequencing are thought to be components of language and reading (Wright, Bowen, & Zecker, 2000), this particular finding has obvious implications for the child diagnosed with CAPD.

Auditory Training in Children With CAPD or Auditory-Based Learning Problems

Although animal models and research with normal-hearing individuals demonstrate the mechanisms under which the normal auditory system responds to auditory training, they do not provide direct evidence of the efficacy of interventions used to treat CAPD. We consider here several studies that employ exercises that fall under the strictest definition of auditory training, as defined by ASHA (2005a), and do provide such evidence. As described in the ASHA (2005) report, auditory training includes "bottom-up treatment approaches designed to reduce or resolve the CAPD. Training activities may include but are not limited to procedures targeting intensity, frequency, and duration discrimination; phoneme discrimination and phoneme-to-grapheme skills; temporal gap discrimination; temporal ordering and sequencing; pattern recognition; localization/lateralization; and recognition of auditory information presented within a background noise or competition." Although there exists a variety of evidence for the effectiveness of computer-based auditory training (CBAT) paradigms in treating CAPD, we do not consider those in detail here as they cannot be easily categorized under

this definition of auditory training. For a review of these CBAT studies, the interested reader should refer to McArthur (2009) and Loo, Bamiou, Campbell, and Luxon (2010).

It has been recommended that auditory training for CAPD follow a processing-specific approach; that is, children with CAPD are given exercises that generally target the specific auditory process(es) identified as deficient based on central auditory tests with documented efficiency (AAA, 2010; ASHA, 2005a, 2005b; Bellis, 2002; Musiek, Weihing & Chermak, 2007). Most of the published auditory training protocols lead to improved auditory function following a schedule of 15 to 45 minutes of training, two to four times a week, for a period of 1 to 2 months. Several recent studies have provided evidence that auditory training can improve the auditory processing skills of children with CAPD or auditory-based learning problems. McArthur, Ellis, Atkinson, and Coltheart (2008) found that children with language or reading impairments who showed difficulty on frequency discrimination tasks improved on language and reading measures following auditory frequency discrimination training. By contrast, children without similar impairments who were not trained showed no sizable test-retest benefit on the frequency discrimination task. Moncrieff and Wertz (2008) had children diagnosed with significant unilateral or bilateral weakness on dichotic processing tests participate in a dichotic processing task that was similar to the dichotic interaural intensity difference training (DIID) paradigm introduced by Musiek and colleagues (Musiek & Schochat, 1998; Musiek et al, 2008; Musiek & Weihing, 2011; Weihing & Musiek, 2007). Children showed significant improvements in left ear performance on dichotic measures following the termination of training. Cameron and Dillon (2011) trained children with spatial sound separation deficits using a program (i.e., LiSN and Learn) that exercised these skills. Following participation, all children showed a much improved ability in obtaining benefits from spatial separation of speech and competition. Notable was the observation that children did not improve on trials in which spatial separation of speech and competition did not occur, suggesting that the training specifically targeted spatial separation. Schochat et al. (2010) enrolled children in auditory training targeting the difficulties they exhibited, including intensity discrimination, temporal patterning, dichotic processing, speech in noise recognition, and gap detection. The children also engaged in informal training at home on a daily basis, and this consisted of listening and language exercises. Results confirmed that the trained CAPD group, but not the untrained normal hearing controls, showed improved behavioral performance on CAPD behavioral measures, as well as greater amplitude in middle latency evoked responses following training. Murphy and Schochat (2011) enrolled children with dyslexia in a nonverbal temporal ordering training paradigm. Interestingly, results showed that children who participated in the training improved not only on CAPD measures of temporal sequencing (e.g., frequency patterns) but also on speech-language measures of phonemic awareness. This was in contrast to a second group composed of children with dyslexia who did not receive the training and did not receive similar benefits.

In summary, we are gaining a deeper understanding of how our interventions

change the brain, which in turn leads to the development of more efficient and effective interventions. We know that the success of interventions is mediated in part by cognitive (e.g., attention, memory), metacognitive, motivational, and emotional processes (Chermak, Bellis, & Musiek, 2007; Cicerone, 2012). The degree to which these factors influence outcomes of auditory interventions remains unknown. The evidence reviewed here demonstrates that auditory training is an effective treatment for central auditory processing deficits. Although clinicians and researchers might disagree as to the quantity of evidence needed to support the acceptance of scientific results, none should confuse any perceived concern about quantity of evidence with the demonstrated positive outcomes of that evidence. See Chapters 3 and 11 in Volume 2 of this Handbook for additional discussion of the efficacy of auditory training and auditory-language training.

Summary

This chapter provides the authors' perspectives on several issues encountered in the diagnosis and treatment of CAPD, with a particular emphasis on the research and evidence supporting our interpretation. There is considerable interest in the research and clinical communities in explaining the listening difficulties encountered by individuals with normal peripheral hearing. CAPD provides a model that may explain these difficulties in the context of CANS dysfunction. This model is supported by a wide range of studies that have shown that lesions of the CANS influence listening ability. Further, there is an emerging understanding of what types of dysfunction might lead to compromise of the CANS in cases that do not involve specific neurological issues (see Musiek & Weihing, 2011 for an example).

It is important that audiologists assessing individuals for CAPD understand the role that cognition and related domains play in the listening ability of their patients. As the brain is extremely nonmodular, it is certainly not surprising that CANS function can be influenced by nonauditory regions, or that listening issues can occur in the absence of CANS dysfunction. The present chapter, and the current AAA (2010) best practice document, highlight ways in which audiologists may begin to minimize or rule out the influence of these nonauditory domains by involving multidisciplinary teams, employing cognition screeners and intratest audiological comparisons, selecting tests that do not overly tax cognitive and language systems, and more. Accurate differential diagnosis depends in large part on knowledge of procedures and steps used to differentiate CAPD from confounding or comorbid issues, and the importance of this knowledge cannot be overstated.

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SECTION 2

[redacted]nostic Fundamentals

CHAPTER 10

SCREENING FOR CENTRAL AUDITORY PROCESSING DISORDER

WAYNE J. WILSON

Introduction

This chapter reviews screening for central auditory processing disorder (CAPD). After defining screening and its purpose, it considers the question "For what are we screening?" This is done primarily within the context of the three approaches to CAPD described by J. Jerger (2009): the "audiologic" approach, the "psychoeducational" approach, and the "language development" approach; and the positions on CAPD offered by the American Speech-Language-Hearing Association's (ASHA) Working Group on Auditory Processing Disorders Technical Report (ASHA, 2005; an update on ASHA, 1996), the American Academy of Audiology's (AAA) Diagnosis, Treatment and Management of Children and Adults with CAPD (AAA, 2010), and the British Society of Audiology's (BSA) Position Statement on APD (BSA, 2011a; an update on BSA, 2007), their Practice Guidance: An Overview of Current Management of Auditory Processing Disorder (APD) (BSA, 2011b), and their "white paper" (Moore, Rosen, Bamiou, Campbell & Sirimanna, 2012). A range of CAPD screening tools are then reviewed and their value considered in the above contexts.

Screening: Definition and Purpose

Wald (2008, p. 50) defines screening as the "systematic application of a test or enquiry, to identify individuals at sufficient risk for a specific disorder to benefit

from further investigation or direct preventative action, among persons who have not sought medical attention on account of symptoms of the disorder." Driscoll and McPherson (2010) note this definition implies the main purpose of screening is to benefit the individuals being examined, with the fundamental operational question asked of any screening process being, "Should this individual be referred for a diagnostic assessment?" (after Wolery, 1989). In this regard, J. M. G. Wilson and Jungner (1968) list 10 basic principles that could guide our approach to screening:

1. The condition to be screened for should be an important health problem.
2. There should be an accepted treatment for cases identified.
3. Facilities for diagnosis and treatment should be available.
4. There should be a recognizable latent (early, asymptomatic) stage in the condition.
5. There should be a suitable test to employ in screening (e.g., sensitive, specific, efficient).
6. The test should be acceptable to the population (where "appropriate" relates to the nature of the risk and the level of health education in the target population).
7. The natural history of the condition should be understood.
8. There should be an agreed policy on whom to treat as patients.
9. The cost of case finding should be nonwastefully balanced in relation to expenditure on medical care as a whole.
10. Case finding should be an ongoing process.

It is clear that CAPD and current methods of screening for CAPD do not meet all 10 of these criteria. This is due largely to ongoing debates over exactly for what it is that we are screening.

For What Are We Screening?

Despite the substantial efforts of many researchers and clinicians over many decades, we still lack *gold standards* for defining and diagnosing CAPD. Debate over this disorder continues with vigor (Cacace & McFarland, 2005; Cowan, Rosen, & Moore, 2009; Dawes & Bishop, 2009; Dillon, Cameron, Glyde, Wilson, & Tomlin, 2012; J. Jerger, 2009; McFarland & Cacace, 2009; Moore, 2006; W. J. Wilson & Arnott, 2012b; W. J. Wilson, Heine, & Harvey, 2004), with Jerger (2009, p. 10) concluding: "APD means different things to different people," and Dawes and Bishop (2009, p. 440) postulating: "APD, as currently diagnosed, is not a coherent category." Rather than being paralyzed by these controversies, this chapter considers screening for CAPD within the context of the three historical approaches to CAPD described by Jerger (2009), and the three most cited positions on CAPD: ASHA (2005), AAA (2010), and BSA (2011a, 2011b; Moore et al., 2012).

Jerger (2009, p. 6) described the three historical approaches to CAPD as being:

1. the audiologic approach, which was "built on earlier observations that persons with brain injury affecting the auditory central nervous system exhibited certain behaviors; ergo, if tests revealed the same behav-

iors, then a link to brain injury was established,"
2. the psychoeducational approach, which was "built on the premise of a set of primary auditory abilities that can be tested by appropriate techniques" without directly addressing how this relates to brain function, and
3. the language development approach, which has been built on the premise that central auditory processing ability "underlies other basic abilities such as language development and reading."

When screening for CAPD in these contexts, favoring the audiologic approach might lead us to screen for audiologic sites of lesion, favoring the psychoeducational approach might lead us to screen for deficits in auditory abilities, and favoring the language development approach might lead us to screen for language and reading deficits.

From the three historical approaches to CAPD described by Jerger (2009) have come three widely cited (but not necessarily widely accepted) positions for defining and managing CAPD: ASHA (2005), AAA (2010), and BSA (2011a, 2011b; Moore et al., 2012) (Table 10–1). Although all three positions contain elements of the audiologic, psychoeducational, and language development approaches to CAPD, these groups define and diagnose CAPD differently. In particular, ASHA (2005) and AAA (2010) require deficient processing of nonspeech and/or speech sounds that is not the result of higher order processing problems. When screening in this context, we might favor screening tools that identify persons at high risk of auditory deficits (for nonspeech and/or speech stimuli) and low risk for higher order deficits. In contrast, BSA (2011a) requires deficient processing of nonspeech and speech sounds and state that "attention is a key element of auditory processing" and "poor attention may make a major contribution to APD" (p. 5). When screening in this context, we might favor screening tools that identify persons at high risk of auditory deficits (to nonspeech and speech stimuli) resulting from a deficit in attention.

Who, When, and By Whom?

Who should we screen for CAPD, when should we screen them, and who should conduct the screening? An initial answer to these questions might best be drawn from J. M. G. Wilson and Jungner's (1968) basic principles to guide screening programs (listed above), that is, we should screen persons at risk for CAPD who have access to facilities for the diagnosis and treatment of CAPD at an affordable cost, we should screen them before they reach an age where their CAPD becomes an important health problem or at least at an age where they are most likely to benefit from intervention, and we should screen them with suitable tests applied by suitably qualified testers.

Converting J. M. G. Wilson and Jungner's (1968) recommendations into practice can be challenging. Although AAA (2010, p. 13) notes that "it is more common to find that children with a range of developmental disabilities are often screened for CAPD," they also note that "it is important that older adults with hearing complaints or significant case history information . . . be screened as well."

Table 10–1. Definitions and Diagnostic Criteria for APD According to ASHA (2005) and AAA (2010)

Definition of CAPD	Behaviors Displayed by Individuals with CAPD	Diagnostic Criteria
ASHA (2005, p. 2) states: "CAPD is a deficit in neural processing of auditory stimuli that is not due to higher order language, cognitive, or related factors." "(C)AP refers to the perceptual processing of auditory information in the CNS and the neurobiologic activity that underlies that processing and gives rise to electrophysiologic auditory potentials." "(C)AP includes the auditory mechanisms that underlie the following abilities or skills: • sound localization and lateralization; • auditory discrimination; auditory pattern recognition; • temporal aspects of audition, including • temporal integration, temporal discrimination (e.g., temporal gap detection), temporal ordering, and temporal masking; auditory performance in competing acoustic signals (including dichotic listening); and • auditory performance with degraded acoustic signals. CAPD refers to difficulties in the perceptual processing of auditory information in the CNS as demonstrated by poor performance in one or more of the above skills."	ASHA (2005, p. 5) states: "Individuals suspected of having CAPD frequently present with one or more of the following behavioral characteristics: • difficulty understanding spoken language in competing messages, noisy backgrounds, or in reverberant environments • misunderstanding messages • inconsistent or inappropriate responding • frequent requests for repetitions, saying "what" and "huh" frequently • taking longer to respond in oral communication situations • difficulty paying attention • being easily distracted • difficulty following complex auditory directions or commands • difficulty localizing sound • difficulty learning songs or nursery rhymes • poor musical and singing skills • associated reading, spelling, and learning problems. It is important to note that this list is illustrative, not exhaustive, and that these behavioral characteristics are not exclusive to CAPD . . . therefore, these behavioral characteristics are not specifically diagnostic of CAPD."	ASHA (2005) note that: • behavioral characteristics are not specifically diagnostic of CAPD (ASHA, 2005, p. 2), • demonstration of a deficit in the neural processing of auditory stimuli that is not due to higher order language, cognitive, or related factors (ASHA, 2005, p. 2), and • diagnosis of CAPD generally requires performance deficits on the order of: – ≥2 SDs below the mean on ≥2 tests, or – ≥3 SDs below the mean on one test, or – ≥2 SDs below the mean on one test when the finding is accompanied by significant functional difficulty in auditory behaviors reliant on the process assessed (ASHA, 2005, p. 10), Where the tests assess the skills listed in ASHA's (2005) definition of CAPD. AAA (2010) note the following criterion for diagnosing CAPD: • a score ≥2 SDs below the mean for ≥1 ear on ≥2 different behavioral central auditory tests (AAA, 2010, p. 22)

Definition of CAPD	Behaviors Displayed by Individuals with CAPD	Diagnostic Criteria
AAA (2010) supports the ASHA (2005) definition of CAPD. BSA (2011a, p. 3) states: • "APD is characterized by poor perception of both speech and non-speech sounds. • "APD has its origins in impaired neural function. • APD impacts on everyday life primarily through a reduced ability to listen, and so respond appropriately to sounds. • APD does not result from a failure to understand simple instructions. • APD is a collection of symptoms that usually co-occurs with other neurodevelopmental disorders". BSA (2011, p. 5) also state that: "Cognitive factors such as attention, rather than being a potential confound, may make a significant contribution to CAPD."	AAA (2010) supports the list of behaviors offered by ASHA (2005). BSA (2011a, p. 5) state that: "to define APD, it is necessary to agree upon the presenting symptom(s). Several recent studies have found that some children with Developmental APD have difficulty with speech perception. However, they appear to perform equally in quiet as in at least some forms of noise. Other studies have highlighted aspects of auditory attention (focus, concentration, distraction) and memory (for complex or multistep instructions). Still others have found problems in spatial hearing. Concerns have been consistently expressed about academic achievement, especially in relation to reading and language comprehension. However, there is no correlation between performance on auditory processing tasks and standardized measures of academic achievement. There is clearly no consensus here but, rather, a list of problems that may be due to one or several causes. The way through this may be to focus on a core symptom or symptoms; aspects of auditory perception that reflect and can be shown to contribute to the clinical presentation, and that help to add information to the overall evaluation of a child with listening difficulties."	Both ASHA (2005, p. 10) and AAA (2011, p. 22) note that inconsistencies across tests can indicate the presence of non-auditory confounds such as deficits in attention. Not explicitly stated. The BSA (2011) definition of CAPD implies a requirement to achieve a failing score on ≥2 tests, one involving non-speech sounds and one involving speech sounds. BSA (2011a, p. 6) states that: "Given the heterogeneity of the problem, one way forward is to ask why children were initially referred: the clinical presentation. Carefully constructed parent/caregiver evaluations have provided valuable and sensitive screening instruments in other developmental disorders. The development of such an instrument for listening difficulties might also lead to a gold standard. The questionnaire, or some other candidate measures (e.g., functional neuroimaging), could be used during an initial, transitional period of research, to validate direct tests, both behavioral and physiological."

Bellis (2003) argued that the absence of an appropriate tool prevents primary screening for CAPD (i.e., the mass screening of large populations). In the case of children, such primary screening is then left to parents and teachers who are best positioned to observe the child's behavior on a regular basis. A positive primary screening result would occur when the parent and/or teacher reported the child showed behaviors associated with CAPD, such as those listed by ASHA (2005) and AAA (2010) (see Table 10–1). The value of these lists is mitigated by their overlap with behaviors shown by persons with disorders other than CAPD, although any resulting increase in false positive findings may well be acceptable in the context screening. If the child fails the primary screening, then Bellis (2003) recommended a secondary screening (a more targeted screening) be conducted by a team of professionals using more formal screening tools. Her suggested team includes an audiologist, speech-language pathologist, educator, psychologist, social worker, the parents, and a physician, whose aim is to gather not only information about the child's central auditory processing, but also about the child's educational, social, speech/language, cognitive, and medical characteristics. This information is then used to "provide a picture of the child's strengths and weaknesses across domains" and to determine if there is "sufficient evidence to support the likelihood that a CAPD is present" (Bellis, 2003, p. 170). If CAPD was suspected, then referral to an audiologist for a diagnostic CAPD assessment would be appropriate. If another disorder was suspected, then a more appropriate referral would be appropriate (e.g., if a language disorder was suspected, then referral to a speech-language pathologist would be appropriate; if a cognitive disorder was suspected, then referral to a psychologist would be appropriate).

Although not disagreeing with a team approach to screening for CAPD, other authors have noted that it needs to be balanced with practical issues, including timing and cost (BSA, 2011b). In this regard, the best single tool or small group of tools to screen for CAPD continues to be sought (but has yet to be found).

The best time for screening in children would appear to be in the preschool years before CAPD becomes an important health problem and when benefit is more likely to be gained from treatment (as the auditory system is more plastic and more able to directly influence other systems in the brain). This is mitigated by the lack of screening tools, diagnostic tools, and proven CAPD treatments for this age group (W. J. Wilson & Arnott, 2012a; and appropriate chapters in Volumes 1 and 2 of this Handbook). The best time to screen adults is more difficult to determine, although findings that CANS dysfunction is a component of presbycusis (Gates, Anderson, Feeney, McCurry, & Larson, 2008; Tremblay, Billings, & Rohila, 2004) suggest screening adults in the early stages of presbyacusis may be of some value.

Finally, while the persons conducting the screening should be sufficiently qualified to perform the screening, interpret the results, and make appropriate recommendations, accessing such a person or persons within reasonable limits of time and cost remains a challenge in many regions in need of CAPD services.

How Should We Screen?

ASHA (2005) and AAA (2011) state that while there is no universally accepted method of screening for CAPD, current

screening approaches typically involve systematic observation of listening behavior and/or performance on tests of auditory function that probe auditory behaviors related to academic achievement, listening skills, and communication. AAA (2010, p. 13) also warns, however, that questionnaires "generally have poor specificity, tend to over-refer, and have not been validated." In contrast, BSA (2011a, p. 6) states that not only might a carefully constructed parent/caregiver evaluation (e.g., a questionnaire) be a sensitive screening instrument for CAPD, the development of such an instrument for listening difficulties might also lead to a gold standard for the diagnosis of CAPD (see Moore et al., 2012, for critiques of this suggestion). In this regard, higher order processing problems could be seen as potential confounds when screening for CAPD under ASHA (2005) and AAA (2010), but as potential targets when screening for CAPD under BSA (2011a, 2011b). It is therefore no surprise that a wide range of tools have been used to directly or indirectly screen for CAPD (ASHA, 2005; AAA, 2011; BSA, 2012a, 2012b).

Rather than describing all potential screening tools for CAPD, this section describes only those that:

- are specifically mentioned (but not necessarily recommended or investigated) in ASHA (2005), AAA (2010), and/or BSA (2012a, 2012b; Moore et al., 2012) as having been suggested as a screener for CAPD, and/or
- have data from subjects with and without CAPD (as determined using clearly stated diagnostic criteria) that are published in the peer reviewed, scientific literature in a form that can be used to assess its performance as a potential screening tool for CAPD (these data are discussed in the next section of this chapter).

With some exceptions, this section does not discuss electrophysiological tools or tools solely used to identify the educational, social, speech/language, cognitive, and/or medical characteristics of the person being screened (such as those that might be used by the speech-language pathologist, educator, psychologist, social worker, parent, or physician members of Bellis's [2003] multidisciplinary screening team discussed above). This is a noted limitation of this chapter.

Observational Tools (Questionnaires and Checklists)

Auditory Processing Domains Questionnaire (APDQ)

The APDQ (O'Hara, 2006), previously called The Listening Questionnaire (TLQ), is a 52-item questionnaire that uses a 4-point Likert-like scale that "takes a broad, differential approach to CAPD where auditory processing skills are rated along with nonauditory listening factors such as attention control and language." It provides four scale scores (adjusted auditory processing, attention control, language and target auditory processing), the scores for which can be used to classify the child as being "at risk" or "not at risk" of listening difficulties. The APDQ/TLQ was designed for use by parents for children aged 7 to 17 years. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Checklist of Auditory Perceptual Subskills

The Checklist of Auditory Perceptual Subskills (Kelly, 1995) is a 20-item

questionnaire that uses a 3-point Likert-type scale to rate a child's listening behavior that is considered to consist of "foundation tools for auditory perceptual skill area development" (p. 250). Each item is analyzed separately. The Checklist of Auditory Perceptual Subskills was designed for teachers and parents to complete for infants and toddlers. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Children's Auditory Performance Scale (CHAPS)

The CHAPS (Smoski, Brunt, & Tannahill, 1998), originally reported as the Children's Auditory Processing Performance Scale (CHAPPS) (Smoski, 1987), is a 36-item questionnaire that uses a 7-point Likert-type scale to rate a child's listening behavior against that of his or her classmates in the conditions of noise, quiet, ideal, and multiple inputs, as well as the child's auditory memory sequencing and auditory attention span. It provides scores for each condition and for the questionnaire as a whole, which can be classified as being "normal" or "at risk." The CHAPS was designed for teachers and parents to complete for children aged 7 years and older. It is listed in ASHA (2005) and AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Children's Home Inventory of Listening Difficulties (CHILD)

The CHILD (Anderson & Smaldino, 2000) is a "family-centered" pair of 15 item questionnaires that use an 8-point Likert-type scale to rate the child's ability to hear in a range of listening environments in and around the home. One questionnaire in the pair is completed by a family member, while the second is completed by the child with the help of the parent and/or audiologist. Each item is paired between the questionnaires to allow direct comparisons between the family member's rating and the child's own rating. A total score is also provided that can be used for pretest-posttest comparisons. The CHILD was designed for home use with children aged 3 to 12 years for the questionnaire completed by the parent, and 7 to 12 years for the questionnaire completed by the child. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Fisher's Auditory Problems Checklist (FAPC)

The FAPC (Fisher, 1976) is a 25-item questionnaire that uses a simple check or no check system to identify auditory behaviors of concern. It provides a single score, which can be classified as suggesting or not suggesting the "need for further evaluation." The FAPC was designed for teachers and parents to complete for children aged 5 years to 11 years and 11 months. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Listening Inventory For Education-Revised (LIFE-R)

The LIFE-R questionnaires were designed to quantify how well a student may hear, is hearing, or has been hearing in different listening environments in the classroom. The Before–LIFE-R (Anderson, Smaldino, & Spangler, 2011c) contains six questions that require the student to mark items

that best describe his or her classroom listening setting. The LIFE-R Student Appraisal of Listening Difficulty (LIFE-R SALD) (Anderson, Smaldino, & Spangler, 2011a) is a 15-item questionnaire that uses a 5-point Likert-type scale whereby the student rates his or her perceived level of difficulty in different listening environments in the classroom (supporting pictures of these environments are available if required). The After LIFE-R (Anderson, Smaldino, & Spangler, 2011b) contains six questions that require the student to mark items that best describe how he or she responds when faced with six difficult listening environments in the classroom. Each of these questionnaires is analyzed on an item-by-item basis. As each requires self-report by the student, they are together considered appropriate for students approximately 8 years (third grade) or older.

The LIFE-R questionnaires are also available for teachers to rate the listening abilities of students. The LIFE-R Teacher Appraisal of Listening Difficulty (LIFE-R TALD) (Anderson, Smaldino, & Spangler, 2011d) is a 15-item questionnaire that uses a 5-point Likert-type scale whereby the teacher rates the student's level of challenge when listening and learning in different listening environments in the classroom. It also provides a total score that is rated on a 5-point Likert-like scale, and can be used as a pre- and posttest assessment if required. The LIFE-R Teacher Checklist: Self-Advocacy and Instructional Access (Anderson, Smaldino, & Spangler, 2011e) is an 8-item questionnaire that uses a 5-point Likert-type scale whereby the teacher rates when the student uses self-advocacy strategies in the classroom. The items on this questionnaire are analyzed on an item-by-item basis.

The original LIFE questionnaires were available in two versions only: the LIFE-SALD (Anderson & Smaldino, 1998a) and the LIFE-TALD (Anderson & Smaldino, 1998b). The LIFE-R questionnaires are listed in AAA (2010, p. 13) as tools that have been suggested as screeners for CAPD.

Scale of Auditory Behaviors (SAB)

The SAB (Schow, Seikel, Brockett, & Whitaker, 2007) is a 12-item questionnaire that uses a 5-point Likert-like scale to rate the frequency of auditory behaviors, including difficulty hearing or understanding in background noise, inconsistent responses to auditory information, and short attention span. It is used to support findings of the five-test Multiple Auditory Processing Assessment (MAPA) battery (see below). It provides a total score that can be compared against expected scores for age. The SAB was designed for parents and/or teachers to complete for children aged 8 to 11 years. The MAPA is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Screening Instrument for Targeting Educational Risk (SIFTER)

The Preschool SIFTER (Anderson & Matkin, 1996), SIFTER (Anderson, 1989), and Secondary SIFTER (Anderson, 2004) are three versions of a 15-item questionnaire that uses a 5-point Likert-type scale to rate a child's performance against those of his or her other classmates in regard to academics, attention, communication, class participation, and school behavior in the classroom setting. All three versions provide scores for each performance

area. The Preschool SIFTER provides an indication of a pass or at risk performance for each performance area as well as derived scores that indicate expressive communication and socially appropriate behavior. The SIFTER and Secondary SIFTER provide an indication of a pass, fail, or marginal performance for each performance area. The Preschool SIFTER, SIFTER, and Secondary SIFTER were all designed for teachers and parents to complete for children aged 3 years through to kindergarten, primary school age, and secondary school age, respectively. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

The Listening Inventory

The Listening Inventory (Geffner & Ross-Swain, 2010) is a 103-item questionnaire that uses a 5-point Likert-like scale to rate specific behaviors in the areas of linguistic organization, decoding/language mechanics, attention/organization, sensory/motor, social/behavioral, and auditory processes. It provides index scores that can be compared with criterion-based cutoff scores. The listening inventory was designed for parents and teachers to complete for children aged 4 to 17 years. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Evaluation of Children's Listening and Processing Skills (ECLIPS)

The ECLIPS is currently in development in the United Kingdom as a 37-item questionnaire that uses a 5-point Likert-like scale to rate level of agreement with statements on whether a child demonstrates key presenting symptoms for suspected CAPD. It is being designed in line with BSA (2011a,b) and Moore et al. (2012) with statistical modeling being used to show it assesses five factors: speech and auditory processing, environmental and auditory sensitivity, language/literacy/laterality, pragmatic and social skills, and memory and attention. The ECLIPS is to be completed by parents and teachers for children aged 6 to 11 years.

Screening Tests

Hearing in Noise Test (HINT) and Hearing in Noise Test for Children (HINT-C)

The HINT (Nilsson, Soli, & Sullivan, 1994) and HINT-C (Gelnett, Sumida, & Soli, 1994; Nilsson, Soli, & Gelnett, 1996) assess sentence recognition in background noise. These tests are used to present phonetically balanced sentences in spectrally matched, speech-shaped masking noise. A total of 250 sentences are included in the HINT, having been adapted from 336 Bamford-Kowal-Bench (BKB) sentences (Bench & Bamford, 1979) and categorized into 25 lists. A total of 130 sentences are included in the HINT-C, categorized into 13 lists. Both tests use an adaptive technique to determine the subject's reception threshold for sentences in noise. The HINT was designed for adults while the HINT-C was designed for children.

Pediatric Speech Intelligibility (PSI) Test

The PSI test (S. Jerger & Jerger, 1984) assesses sentence recognition in noise. It is used to present words or sentences in the presence of competing sentences presented ipsilaterally or contralaterally.

The testee responds by pointing to the appropriate picture (in a set of four pictures) that best represents the target word or sentence. Message-to-Competition Ratios (MCR) and Performance-intensity (PI) functions can be obtained for the different test conditions. The PSI can be applied to children aged 3 to 6 years.

Test of Everyday Attention for Children (TEA-Ch)

The TEA-Ch (Manly, Robertson, Anderson, & Nimmo-Smith, 1999) assesses a range of attentional abilities in children. The screening version of the test consists of four subtests to measure selective attention, sustained attention, attentional control/switching, and sustained-divided attention across the visual and auditory modalities. The TEA-Ch can be applied to children aged 6;0 to 15;11 years.

University of Queensland Understanding Everyday Speech Test (UQUEST)

The UQUEST (Kei et al., 2003) assesses understanding of everyday speech in noise. It is used to present short speech passages (30 to 40 s) describing real life situations such as a birthday party or a visit to a park, under headphones, in the presence of a background speech noise, and with the simultaneous presentation of an accompanying video animation. The animation serves to attract a child's attention without providing cues such as lipreading. At the end of each video, the child answers four multiple-choice (four-option), content related questions (presented auditorily and visually) about the real life situation he or she had just observed. The UQUEST has been normalized on grade 3 and 4 Australian children, making it suitable for children aged between 6 and 10 years.

Screening Test Batteries

In considering screening test batteries, the question arises: When does a screening test battery become a diagnostic test battery? Wilson and Jungner (1968, p. 30) argue that this may be "a matter of degree rather than of kind; the screening test (which of its nature should be easy and quick to perform) is allowed to possess a higher margin of error and may be less valid than a diagnostic test." It will be seen that the line between screening and diagnostic test battery is blurred for some of the screening test batteries listed below.

Differential Screening Test for Processing (DSTP)

The DSTP (Richard & Ferre, 2006) assesses auditory and language processing skills at three major levels: acoustic, acoustic-linguistic, and linguistic. It contains three level one acoustic subtests (dichotic digits, temporal patterning and auditory discrimination), two level two acoustic-linguistic subtests (phonemic manipulation and phonic manipulation), and three level three linguistic subtests (antonyms, prosodic interpretation and language organization). The DSTP was designed for use by professionals on children aged 6 to 12 years. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Multiple Auditory Processing Assessment (MAPA)

The MAPA (Schow & Seikel, 2007; Schow et al., 2007) is a test battery that assesses

a range of skills by using prerecorded stimuli that the testee has to repeat in whole or in part, and a questionnaire that has to be completed by the testee's parents and/or teachers (the Scale of Auditory Behaviors described above). The five tests involving prerecorded stimuli provide information on three skill areas: monaural low redundancy (monaural separation closure) through the monaural Selective Auditory Attention Test (mSAAT), temporal (auditory pattern temporal ordering) through the Tap Test (not to be confused with the Test of Auditory Processing Skills [TAPS] below) and the Pitch Pattern (PP) Test, and dichotic speech (binaural integration/binaural separation) through Dichotic Digits (DD) Test and Competing Sentences (CS) Test. Three supplemental tests involving prerecorded stimuli are also included in the form of the MAPA Duration Pattern (DT) Test, the MAPA Speech in Noise for Children and Adults (MAPA SINCA), and the MAPA Fusion Test (MAPA AFT-R). Raw scores are reported against mean and standard deviation values obtained from normally performing children. The MAPA was designed for audiologists or speech-language pathologists to assess children aged 8 to 12 years and adults aged 21 to 49 years, and its authors report it can be used as a screener or as a preliminary diagnostic tool for CAPD. It is listed in AAA (2010, p. 13) as a tool that has been suggested as a screener for CAPD.

Test of Auditory Processing Skills-3rd edition (TAPS-3)

The TAPS-3 (Martin & Brownell, 2005) is a test battery that assesses a range of skills by using prerecorded and live voice stimuli that the testee has to repeat or respond to in whole or in part. It provides information for four areas: auditory attention (an auditory figure-ground screener), basic phonological skills (word discrimination, phonological segmentation, and phonological blending), auditory memory (number memory forward, number memory reversed, word memory, and sentence memory), and auditory cohesion (auditory comprehension and auditory reasoning). Scores can be reported as raw, stanines, scaled, T-scores, standard scores, and percentile ranks. The TAPS-3 was designed to assess children aged 4 years to 18 years and 11 months by "speech-language pathologists, audiologists, special education and resource teachers, and other clinicians who are interested in, and have been trained to assess, a child's use of auditory information" (Martin & Brownell, 2005, p. 5). Its immediate predecessor was the Test of Auditory Perceptual Skills-Revised (TAPS-R; Gardner, 1996).

Tests for Auditory Processing Disorders for Children (SCAN-3:C) and Adolescents and Adults (SCAN-3:A)

Both of these test batteries (Keith, 2012a, 2012b) assess a range of central auditory processing skills by using prerecorded stimuli that the testee has to repeat in whole or in part. Both batteries include a tonal gap detection task, an auditory-figure ground task (a monosyllabic word-in-noise [i.e., multitalker babble] task at +8dB SNR, with +12 dB and 0 dB SNR versions also included), a competing words task (dichotic, monosyllabic words) in free recall and directed recall formats, a filtered words task (monosyllabic words low-pass filtered at 750

Hz), a competing sentences task (a dichotic sentence task with directed recall from the target ear only), and a time compressed sentences task (time compressed at 60%). Both batteries offer the gap detection, auditory-figure ground (+8 dB for the SCAN-3:C and 0 dB for the SCAN3-A) and competing words (free recall) tasks as a screening test battery for CAPD, and both were designed for audiologists, speech-language pathologists, and other professionals trained in standardized assessments to assess children aged 5 to 12 years (SCAN-3:C) or 13 to 50 years (SCAN-3:A). Both test batteries are based on their predecessors: the SCAN-C (Keith, 1994c), SCAN-A (Keith, 1994b), and the original SCAN (Keith, 1986). The SCAN, SCAN-C, and SCAN-A are listed in ASHA (2005, p. 5) and the SCAN-3:C and SCAN-3:A are listed in AAA (2010, p. 10) as tools that have been suggested as screeners for CAPD.

J. Jerger and Musiek (2000)

In their report of the Consensus Conference on the Diagnosis of Auditory Processing Disorders in School-Aged Children, J. Jerger and Musiek (2000) recommended that a direct screening test procedure for CAPD should include a dichotic digits test and a gap detection test. The dichotic digit test should consist of two digits in each ear, using a free-recall response mode, to minimize the linguistic load imposed by less well-learned speech tokens. The gap detection test should involve a short silent gap inserted in a burst of broadband noise. These tests were recommended for children aged 6 years or older. The J. Jerger and Musiek (2000) recommendation is listed in ASHA (2005, p. 5) and AAA (2010, p.10) as a tool that has been suggested as a screener for CAPD.

Who Is Using Which Tools to Screen for CAPD?

In the past 10 years there have been three surveys that directly addressed screening for CAPD, the results of which were published in the peer reviewed scientific literature. In an update on professional education and clinical practices in central auditory processing for audiologists in the USA, Chermak, Silva, Nye, Hasbrouck, and Musiek (2007) reported that 31/68 (46%) respondents indicated they screened for CAPD. The most used screening tool was acoustic reflexes (4/31, 13%), followed by questionnaires (3/31, 10%) and the SCAN (2/31, 6%). The respondents screened on average less than one percent of their caseload, with 4.3% of those persons screened going on to be diagnosed with CAPD. It was interesting to note that a small number of respondents also reported using the FAPC, SCAN-A, SCAN-C, CHAPS, Speech in Noise (Etymotic Research, 1993), Auditory Continuous Performance Test (Keith, 1994a), Selective Auditory Attention Test (Cherry, 1980, 1992), or TAPS in their evaluation of CAPD, with their ratings of the efficiency of these tests being generally split between "not efficient" and "efficient."

In a survey of common CAPD diagnostic and management practices in the USA, Emanuel, Ficca, and Korczak (2011) reported 52% of the 195 responding audiologists screened for CAPD. The screening tool most used by the respondents who did screen for CAPD was the SCAN-A or SCAN-C (69%), followed by questionnaires (56%) and classroom observation

(33%). Three (2%) respondents reported using a speech-language evaluation as part of the screening process, with no other reports of multidisciplinary testing. When asked if they distributed questionnaires to teachers and/or parents prior to conducting CAPD testing, 75% of the respondents said yes to parents and 65% said yes to teachers. Among these respondents, the most popular questionnaires were the FAPC (63%), CHAPS (51%), SIFTER (39%), and site generated questionnaires (31%). When asked to indicate other professionals from whom they "preferred" or "required" evaluations prior to diagnosis, the respondents selected speech-language pathologists (90%), psychologists (86%), education specialists (84%), neurologists (31%), and otolaryngologists (26%).

In an investigation into the prevalence of clinical referrals of persons with hearing thresholds within normal limits in the United Kingdom, Hind (2006) reported on 19 audiology and speech-language-therapy services whose staff indicated that they screened for CAPD. A wide range of methods were used, which were categorized broadly into audiological assessments, cognitive/processing tests, questionnaires, and/or profiles (education/medical). Eleven of these services used more than one method of screening, five used "fairly extensive" (p. 16) batteries incorporating audiological, electrophysiological, language, and cognitive assessment, and the most popular single method (used by six respondents) was the SCAN-C.

Overall, the results of the three surveys reported above suggest the number of audiologists who screen for CAPD in the USA and the United Kingdom has increased over the past 10 years, with the SCAN-C and questionnaires (particularly the FAPC, CHAPS, and SIFTER) being the most used tools in this regard.

Reviewing the Use and Effectiveness of Current Screening Tools for CAPD

Assessing the use of screening tools for CAPD is difficult for at least four reasons. First, data of this type are lacking for many of the tools proposed as screeners for CAPD. Second, we lack a universally accepted definition of CAPD, diagnostic criteria for CAPD, and a gold standard test or tests for CAPD (discussed above). This effectively prevents any assessment of a measure's true sensitivity and specificity to CAPD. Third, many measures of central auditory processing have been used both for screening and/or diagnosis, and even as both in the same study (e.g., in examining the screening potential of the four tests within the MAPA test battery, Domitz and Schow [2000] compared the sensitivity of various combinations of these tests to CAPD diagnosed on the basis of failing ≥1 of these tests by ≥2 SD). Finally, it could be argued that any measure or combination of measures of central auditory processing could be used to screen for CAPD. Examining all possible combinations was beyond the scope of this chapter. With these difficulties in mind, this section considered the effectiveness of the screening tools discussed above that had data from persons with and without CAPD (as determined using clearly stated diagnostic criteria) that were published in the peer reviewed, scientific literature in a form that could be used to assess its performance as a potential screening tool for CAPD. This literature is summarized in Tables 10–2, 10–3, and 10–4.

Table 10–2. The Ability of Questionnaires and Checklists to Identify CAPD

Question-naire/ Checklist	Subjects Assessed	Separated Groups
CHAPS	40 children aged 8 to 15 years assessed at an audiology clinic: 20 diagnosed with CAPD for failing (at least monaurally) ≥2 tests of CAP (tests: one test for each of the following processes: auditory closure, binaural interaction, binaural integration, auditory figure ground, temporal processing/patterning, and auditory attention [screening]) (Drake et al., 2006)	No
	23 children aged 7:0 to 10:6 years referred for CAPD assessment: 13 diagnosed with CAPD for failing ≥2 tests of CAP by ≥2 SD (tests: SSW, CS, DD and two non-standardized tests of STM). CHAPS only completed for 17 children (Lam & Sanchez, 2007)	No (p<0.05)
	89 children aged ~10±2 years referred to a specialist CAPD clinic: 32 diagnosed with CAPD for failing the composite SCAN test score >1 SD and failing ≥1 other test of CAP (n = 32) (other tests: RGDT, GIN, PP or DP). (Dawes et al., 2008)	No (p<0.05)
	68 children aged 7-12 years: 49 diagnosed on the basis of failing ≥2 tests of CAP by ≥2 SD or ≥1 tests of CAP by ≥3 SD (tests: DD, FP, RGDT, TCRS and MLD) (Sharma et al., 2009)	Sensitivity to CAPD = 37%
	25 children aged 10:4±2:5 years diagnosed with CAPD on the basis of scoring below the recommended clinical cutoff on the SCAN-C or SCAN-A, and failing ≥1 other test of CAP (n = 32) (other tests: RGDT, PP or DP), and 19 children aged 10:1±1:6 years diagnosed with dyslexia (Dawes & Bishop, 2010)	Yes (p<0.05, effect size = 0.24)
	856 children aged 6 to 11;11 years, the worse performing 5% of whom were classified as having CAPD (tests; backward masking, simultaneous masking, frequency discrimination, temporal resolution and frequency resolution) (Moore et al., 2010)	Yes (p<0.05)
	104 children aged 6:9 to 14:3 years referred for a CAPD assessment: 39 diagnosed with CAPD for failing (binaurally) ≥2 tests of CAP by ≥2 SD, and 74 diagnosed with CAPD for failing ≥1 test by ≥2 SD within a single CAP domain (tests: LPFS, CS, DD and FP) (W. J. Wilson et al., 2011)	No (p<0.05)

continues

Table 10–2. *continued*

Questionnaire/ Checklist	Subjects Assessed	Separated Groups
CHAPS *continued*	88 children aged 6 to 13 years: 19 diagnosed with CAPD on the basis of parental report of difficulties hearing in background noise, expressing or clearly using speech, understanding when listening, remembering complex and multistep instructions, staying focused, 22 diagnosed with SLI and 47 recruited from mainstream schools (Ferguson et al., 2011)	Yes ($p<0.001$)
	97 children aged 11;4 to 12;7 years: 45 had been referred for CAPD assessment of whom 25 were diagnosed with CAPD for failing ≥2 tests (at least monaurally) of CAP by ≥2 SD with ≥1 fail on a test of nonspeech stimuli (tests: GSiB, GDD, FP, DP, RGDT and MLD) and 20 were not diagnosed with CAPD for passing these tests, and 39 controls with no listening, hearing or academic difficulties (Iliadou & Bamiou, 2012)	Yes ($p<0.0001$)
FAPC	As per Dawes et al. (2008) above	No ($p<0.05$)
LIFE	10 children aged 8;2 to 15;7 years diagnosed with CAPD for failing ≥2 tests (at least monaurally) of CAP by ≥2 SD (test groups: auditory figure-ground, dichotic listening, phonemic awareness and auditory sequencing), and 13 children aged 8;2 to 13;2 years who passed the CAP test battery (Johnston et al., 2009)	Yes ($p<0.05$, two items only)
SIFTER	As per Johnston et al. (2009) above	Yes ($p<0.05$, one item only)
	As per W. J. Wilson et al. (2011) above	No ($p>0.05$)

CAP, central auditory processing; *CELF*, Clinical Evaluation of Language Functions; *CS*, competing sentences; *DD*, dichotic digits; *DP*, duration pattern test; *FP*, frequency pattern test; *GDD*, Greek dichotic digits; *GFW*, Goldman-Fristoe-Woodcock; *GIN*, gaps in noise test; *GSiB*, Greek Speech in Babble test; *LPFS*, low-pass filtered speech; *MLD*, tonal masking level difference test; *PP*, pitch pattern sequence test; *RGDT*, random gap detection test; *SLI*, specific language impairment; *SSW*, Staggered Spondaic Words; *TCRS*, time compressed and reverberant speech.

Table 10–3. The Ability of Screening Tests to Identify CAPD

Screening Test	Persons Assessed	Separated Groups
HINT	20 adult workers aged 18 to 55 years: 10 of whom had been exposed to solvents and who as a group performed significantly ($p<0.05$) worse than the non-exposed group on HINT, DD, FS, PP and RGDT, but not MLD or RGDT (Fuente et al., 2006)	Yes ($p<0.05$)
	10 children aged 8;2 to 15;7 years diagnosed with CAPD for failing ≥2 tests (at least monaurally) of CAP by ≥2 SD (test groups: auditory figure-ground, dichotic listening, phonemic awareness and auditory sequencing), and 13 children aged 8;2 to 13;2 years who passed the CAP test battery (Johnston et al., 2009)	Yes ($p<0.05$, HINT-quiet only)
	92 adult workers aged ~36±~7 years: 46 of whom had been exposed to solvents and who as a group performed significantly ($p<0.05$) worse than the non-exposed group on DD, PP, FS, RGDT and HINT SRT, but not HINT (Fuente et al., 2011)	No ($p<0.05$)
PSI	21 children aged 3;2 to 8;4 years: 10 with auditory CNS lesions and 11 with non-auditory or resolved CNS lesions (S. Jerger, 1987)	Yes
	34 children aged 3;4 to 8;6 years: 10 with non-auditory CNS lesions, 17 with auditory CNS lesions (S. Jerger et al., 1988)	Yes
TEA-Ch	23 children aged 7;0 to 10;6 years referred for CAPD assessment: 13 diagnosed with CAPD for failing ≥2 tests of CAP by ≥2 SD (tests: SSW, CS, DD and two non-standardized tests of STM). CHAPS only completed for 17 children (Lam & Sanchez, 2007)	No ($p<0.05$)
UQUEST	25 children aged 7 to 17 years and 19 children aged 7 to 14 years referred for a CAPD assessment: 7 to 20 diagnosed with CAPD for failing ≥2 tests of CAP by ≥2 SD binaurally, ≥2 tests of CAP by ≥2 SD monaurally, or ≥1 test of CAP by ≥2 SD binaurally within one or more domains (tests: LPFS, CS, DD and FP) (W. J. Wilson et al., in press)	Yes ($p<0.01$, 50 dB HL, +5 SNR only)

Abbreviations as per Table 10–2 plus AEPs, auditory evoked potentials; *FS*, filtered speech; *LAC*, Lindamood Auditory Conceptualization Test; *PST*, phonemic synthesis test; *SRT*, speech reception threshold (in quiet); and *STM*, short-term memory.

Table 10–4. The Ability of Screening Test Batteries to Identify CAPD

Screening test battery	Persons assessed	Separated groups
MAPA	81 children aged 8;8 to 9;9 years from 3rd grade public school classrooms: 20 diagnosed with CAPD for failing ≥1 tests of CAP by ≥2 SD (tests: mSAAT, PP, DD, CS) (Domitz & Schow, 2000)	Sensitivity to CAPD mSAAT = 40%; mSAAT + PP or DD = 65%; mSAAT + PP + DD = 90%
SCAN	SCAN: As per Domitz and Schow (2000) above	Sensitivity to CAPD = 45%
	SCAN-C: 40 children aged 5 to 11 years referred for a CAPD assessment: 20 diagnosed with CAPD for failing (degree not stated) ≥2 tests of CAP (tests not stated) within the same process (processes: auditory closure, binaural interaction, binaural integration, auditory figure ground, temporal processing and auditory attention (screening) (Madison et al., 2005)	No
	SCAN-A: 23 children aged 7;0 to 10;6 years referred for CAPD assessment: 13 diagnosed with CAPD for failing ≥2 tests of CAP by ≥2 SD (tests: SSW, CS, DD and two non-standardized tests of STM). CHAPS only completed for 17 children (Lam & Sanchez, 2007)	Yes ($p<0.05$, CS only)
TAPS	TAPS-R: 104 children aged 6;9 to 14;3 years referred for a CAPD assessment: 39 diagnosed with CAPD for failing (binaurally) ≥2 tests of CAP by ≥2 SD, and 74 diagnosed with CAPD for failing ≥1 test by ≥2 SD within a single CAP domain (tests: LPFS, CS, DD and FP) (W. J. Wilson et al., 2011)	No ($p<0.05$)

Abbreviations as per Tables 10–2 and 10–3 plus *CW*, competing words and *mSAAT*, monaural Selective Auditory Attention Test.

Effectiveness of Questionnaires and Checklists

Of all the questionnaires and checklists discussed above, only the Children's Auditory Performance Scale (CHAPS), Fisher's Auditory Problems Checklist (FAPC), Listening Inventory for Education (LIFE), and Screening Instrument for Targeting Educational Risk (SIFTER) were found to have data that met the inclusion criteria. Of the nine studies that

included the CHAPS, five reported that it could not (Dawes, Bishop, Sirimanna, & Bamiou, 2008; Drake et al., 2006; Lam & Sanchez, 2007; Sharma, Purdy, & Kelly, 2009; W. J. Wilson et al., 2011) and four reported that it could (Dawes & Bishop, 2010; Ferguson, Hall, Riley, & Moore, 2011; Iliadou & Bamiou, 2012; Moore, Ferguson, Edmondson-Jones, Ratib, & Riley, 2010) separate children with or without CAPD (for two of the latter studies, some or all of the children without CAPD had specific language impairment [Dawes & Bishop, 2010; Ferguson et al., 2011]).The single studies that included the FAPC and LIFE reported that the FAPC could not (Dawes et al., 2008) and the LIFE could (for two of its items only) (Johnston, John, Kreisman, Hall, & Crandell, 2009) separate children with or without CAPD. Finally, of the two studies that included the SIFTER, one reported it could (Johnston et al., 2009, academics subscore only) and one reported it could not (W. J. Wilson et al., 2011) separate children with or without CAPD.

Effectiveness of Tests

Of the tests discussed above, only the Hearing in Noise Test (HINT), Pediatric Speech Intelligibility Test (PSI), Test of Everyday Attention for Children (TEA-Ch), and the University of Queensland Understanding Everyday Speech Test (UQUEST) were found to have data that met the inclusion criteria. Of the three studies that included the HINT, one reported that it could (Fuente, McPherson, Munoz, & Espina, 2006) and one reported that could not (Fuente, McPherson, & Hickson, 2011) separate adults with or without CAPD, and one reported that it could (Johnston et al., 2009, HINT-quiet score only) separate children with or without CAPD. Of the two studies that included the PSI, both reported that it could (S. Jerger, 1987; S. Jerger, Johnson, & Loiselle, 1988) separate children with or without confirmed auditory CNS lesions. Finally, the single studies that included the TEA-Ch and UQUEST reported that the TEA-Ch could not (Lam & Sanchez, 2007) and the UQUEST could (W. J. Wilson et al., in press, under specific conditions) separate children with or without CAPD.

Effectiveness of Test Batteries

Of the test batteries discussed above, only the Multiple Auditory Processing Assessment (MAPA), Test of Auditory Processing Skills (TAPS), and Tests for Auditory Processing Disorders for Children (SCAN) were found to have data that met the inclusion criteria. The one study that included the MAPA (Domitz & Schow, 2000) showed its ability to separate children with CAPD or without CAPD improved as the number of subtests included in the battery increased. Of the three studies that included the SCAN, one reported that the SCAN (Domitz & Schow, 2000) and one reported that the SCAN-C (Madison, Hallberg, Anfinson, DeMaio, & Drake, 2005) could not separate children with or without CAPD, and one reported that the SCAN-A (Lam & Sanchez, 2007, competing sentences subtest only) could separate children with or without CAPD. Finally, the one study that included the TAPS-R (W. J. Wilson et al., 2011) reported that it could not separate children with or without CAPD.

While no studies were found that specifically reported on the use of the dichotic digits test and a gap detection

test, the direct screening test procedure recommended by J. Jerger and Musiek (2000), it is worth considering an example from the literature (in addition to the MAPA study by Domitz and Schow [2000] discussed above) of the potential sensitivities, specificities, and efficiencies of combinations of central auditory processing tests to CAPD. Musiek, Chermak, Weihing, Zappulla, and Nagle (2011) investigated two groups: an experimental group of 20 persons aged 13 to 59 years with confirmed cortical lesions that involved, but were not limited to, the CANS and a control group of 29 persons aged 20 to 57 years with no history of neurological involvement of the CANS (all participants had normal hearing sensitivity). All participants underwent dichotic digit (DD), frequency pattern (FP), filtered speech (FS), and competing sentence (CS) testing and the results were analyzed using varying combinations of tests and pass/fail criteria. Of the >40 test and criteria combinations examined, the sensitivity values ranged from 20% to 100%, the specificity values ranged from 59% to 100%, and the efficiency values ranged from 63% to 98%. The authors concluded that the two-test battery of DD and FP with a strict criterion (both tests failed in ≥1 ear by ≥2 SD) achieved the best balance of sensitivity (80%), specificity (93%), and efficiency (88%) to cortical lesions involving the CANS, and that such a two-test battery could serve as an efficient diagnostic test battery (let alone a screening battery) for CAPD.

Summary of Effectiveness of Current Screening Tools for CAPD

Overall, the evidence is equivocal on the effectiveness of current screening tools for identifying persons with CAPD. Of the 25 studies reviewed above and in Tables 10–2, 10–3, and 10–4, 12 reported the tool being examined could not and 13 reported it could separate persons with or without CAPD. None of the tools stood out as a best candidate for screening for CAPD, although individual screening tests appeared to slightly outperform questionnaires, checklists, and screening test batteries.

The Future of Screening for CAPD

The future of screening for CAPD is an exciting one indeed, with new tools, improvements to existing tools, and investigations into their reliability and validity already under way in many centers worldwide. Rather than offering a formal prediction of the future of screening for CAPD, however, I offer two postulations to challenge the reader.

Postulation One

The value of questionnaires and checklists will continue to lie more in their ability to "supplement and contextualize the behavioral test findings after a diagnostic battery confirms CAPD" (Schow & Seikel, 2007, p. 143) rather than in their ability to efficiently identify persons at risk for CAPD. For children in particular, it remains difficult to see how parental and/or teacher opinion of a child's behaviors could serve to efficiently identify risk for CAPD in the face of so many confounding variables. These include the overlap of behaviors shown by persons with CAPD versus those with other disorders, the potential subjectivity and biases

of respondents, and the need to distil the complexity of CAPD down to a limited number of questions that can be reliably and validly completed by a respondent in a reasonable period of time. It is noted that this postulation directly contradicts BSA's (2011a, p. 6) statement that not only might a carefully constructed parent/caregiver evaluation (e.g., a questionnaire) be a sensitive screening instrument for CAPD, the development of such an instrument for listening difficulties might also lead to a gold standard for the diagnosis of CAPD (see Moore et al., 2012, for critiques of this suggestion).

Postulation Two

If the efficiency of diagnostic test batteries for CAPD continues to increase, the need for a screening tool for CAPD could decrease. This potential is suggested by the 88% efficiency to CAPD achieved by Musiek et al.'s (2011) recommended diagnostic test battery of DD and FP (where CAPD had been diagnosed on the basis of cortical lesions involving the CANS), and the 90% sensitivity to CAPD achieved by Domitz and Schow's (2000) screening test battery of mSAAT, PP and DD (where CAPD had been diagnosed by failing ≥1 of the following tests by ≥2 SD: mSAAT, PP, DD, and CS), both of which are discussed above. As Musiek et al.'s (2011) proposed diagnostic test battery of DD and FP can be completed in under 10 minutes (including scoring and interpretation), it could be more efficient to proceed directly from a primary screening based solely on teacher or parental concern to such a diagnostic CAPD assessment by an audiologist. Adding a secondary screening (by questionnaire, checklist, test, or test battery) between these steps could be redundant. This postulation depends, of course, on a proposed diagnostic test battery such as DD and FP being applicable to a wide range of CAPD populations.

Conclusion

Screening for CAPD remains as controversial (and as exciting) as the topic of CAPD itself. Many screening tools have been offered in the literature, including questionnaires, checklists, individual tests, and test batteries, but only a limited number have been assessed against clearly defined CAPD diagnostic criteria. The evidence for the use of these tools is equivocal, with no single tool standing out as a best candidate for screening for CAPD. Based on these results, it appears that two statements by AAA (2010, p. 13) on screening measures for CAPD should remain in place:

1. "While a number of questionnaires have been used to screen for CAPD . . . , they generally have poor specificity, tend to over-refer, and have not been validated" and
2. "Further studies are needed to determine the efficiency of currently available screening instruments, including the efficiency of diagnostic tests used for screening purposes, and to develop new screening tools for CAPD."

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CHAPTER 11

TEST BATTERY PRINCIPLES AND CONSIDERATIONS

JANE A. BARAN

Introduction

As one prepares to undertake the assessment of an individual at risk for a central auditory processing disorder (CAPD), there are a number of important variables and factors that should be given serious consideration. Failure to do so may result in an incomplete assessment of the extent of the individual's auditory deficits, or the misdiagnosis of a nonauditory deficit (e.g., a cognitive disorder, such as attention deficit or a working memory disorder, or a language disorder, such as specific language impairment) as an auditory deficit.

The audiologist who is involved in the evaluation and management of CAPD should be well grounded in the anatomy and physiology of the auditory system. The central auditory nervous system (CANS) is a complex system of neural pathways whose function underlies a number of different auditory processes that serve as the foundation for normal auditory function. This system extends from the cochlear nucleus complex in the region of the low brainstem on each side of the brain to the primary auditory reception areas of the temporal lobes in each hemisphere. The auditory pathways then course from the primary auditory cortices of each hemisphere to other regions of the brain in the same hemisphere (e.g., association areas) as well as to both homololateral and heterolateral areas of the brain in the opposite hemisphere. As sensory information travels within this system via both ipsilateral and contralateral pathways, the signal undergoes several levels of processing.

This processing occurs in both a hierarchical or serial fashion, as well as an overlapping or parallel manner. The result of this combined serial and parallel processing of information is a highly efficient, but redundant system (Baran & Musiek, 1999; Chermak & Musiek, 1997; Musiek & Baran, 2007). In addition to the ascending pathways, there are descending pathways, which can moderate the response of the auditory system to an incoming acoustic signal or signals (Musiek & Baran, 2007).

Due to the complexity of the CANS and its intricate neural networks, the processing of auditory information is not a single unitary process that can be readily identified and assessed using a single test. In fact, the processing of auditory information involves a number of subprocesses that are necessary for the normal and efficient processing of an auditory signal to occur. Although several definitions of central auditory processing have been offered, most of these definitions identify a number of specific component skills or subprocesses that underlie normal auditory processing (e.g., AAA, 2010; ASHA, 1996; 2005; Baran & Musiek, 1999, 2003; Bellis, 2003; Chermak & Musiek, 1997). For example, a task force convened by the American Speech-Language-Hearing Association (ASHA) has offered the following definition of central auditory processing:

> (C)AP [(Central) Auditory Processing] includes the auditory mechanisms that underlie the following abilities or skills: sound localization and lateralization; auditory discrimination; auditory pattern recognition; temporal aspects of audition, including temporal integration, temporal discrimination (e.g., temporal gap detection), temporal ordering, and temporal masking; auditory performance in competing acoustic signals (including dichotic listening); and auditory performance with degraded acoustic signals. (ASHA, 2005, p. 2)

As readily seen as one considers this definition, normal auditory processing involves a number of distinct subprocesses or skills. A breakdown or deficit in any one of these skills or subprocesses is possible and it is not necessary that all subprocesses, or even a majority of these processes, be affected for a CAPD to be present. In fact, it is unusual that an individual with CAPD will experience a deficit in each of these areas. Therefore, the selection of a comprehensive and valid test battery is an important clinical skill that the audiologist must master. If a test battery does not include an assessment of a particular subprocess or skill and that subprocess or skill is a deficit area for a given individual, then the presence of a CAPD may go undiagnosed, even if a test battery approach is utilized.

In addition to the considerations mentioned above, there are a number of test principles and features that are important to consider when one is selecting tests for inclusion in a test battery. What specific auditory process (or processes) is (are) being assessed by the test? What other nonauditory influences (e.g., fatigue, lack of motivation) might there be on test performance? If these influences exist, what are the potential effects of these nonauditory influences on test performance? Are the test materials appropriate to the age and linguistic and/or cognitive abilities of the individual being tested? Are there normative data available, and if so, are these appropriate for the individual being tested? How have these norms been derived and are they

valid for the purpose(s) for which they are being used? How efficient is the test (or the battery of tests) in differentiating the presence versus the absence of a CAPD? What are the effects of comorbid conditions on the auditory tests being administered? Are some tests less resistant to the potential confounding effects of a comorbid condition, and therefore are they better choices for inclusion in a test battery? What does the patient's case history and presenting symptomatology tell the audiologist about his or her patient's auditory processing abilities, and how can this information be used to inform the development of a test battery to assess the individual's auditory processing abilities?

It is essential, therefore, that each of these factors and influences (specific auditory deficits, comorbid disorders or disabilities, age, native language and/or general language ability, motivation, fatigue, general malaise, task/test demands, and so forth) be taken into consideration when designing an evaluation protocol for an individual considered to be at risk for CAPD. Failure to do so is likely to result in the development of an assessment battery that will not meet with optimal diagnostic success and may lead to an inaccurate diagnosis.

Case History

A comprehensive and carefully elicited case history is central to the planning, conduct, and interpretation of a central auditory test battery (AAA, 2010; ASHA 2005). The information obtained during this process can prove invaluable in helping the clinician begin to form clinical hypotheses about the nature and extent of the disorder, its potential etiology, and the presence of other comorbid conditions that can seriously impact the test processes and/or confound the test interpretation. It can also provide critical information about the functional implications of the disorder and the impact of the behavioral deficits on the individual's everyday functioning. At a minimum, the history should include information on the patient's personal and familial medical history, including known or potential genetic abnormalities, the individual's prenatal, perinatal, and postnatal history and development, the patient's general health status, including the presence of any significant medical conditions or psychological factors, the individual's communication, listening, and auditory skills and behaviors, the person's social development and linguistic and cultural background, and any prior therapies and/or current treatments (AAA, 2010; ASHA 2005). This information can be obtained by direct interview of the individual, his or her parents or other family members, or from another informant who is responsible for the individual. In addition, important information can be obtained through direct observation techniques; that is, an audiologist who is a good observer of patient behavior is likely to be a good diagnostician, as insights gained from observation of the patient during the interview process or in some other context that can be arranged can help inform the selection of tests for inclusion in the test battery, the need for modifications of test procedures, and so forth (AAA, 2010; Baran, 2007).

Another method of gaining at least some of this information is through the use of self-assessment tools or behavioral checklists (AAA, 2010; ASHA, 2005). A number of such tools are available and

these can provide valuable insights into the individual's behaviors and functional deficits. The behavioral checklists or self-assessment instruments can be completed by the patient's parents, his or her teachers, or by the patient, depending on the nature of the checklist or assessment tool and the ability of the individual to complete the requirements associated with reviewing and completing the self-assessment form or behavioral checklist. (See Chapter 10 for a discussion of the use of behavioral checklists with individuals at risk for CAPD.)

Case history information is more efficiently elicited if the interviewer has some knowledge of the etiological bases of the disorder for which the individual is seeking evaluation and the presenting symptomatology and behavioral manifestations. Equipped with this knowledge, the interviewer can carefully probe relevant medical, educational, and behavioral areas that could reveal important insights into the individual's history and deficits (AAA, 2010; ASHA, 2005).

Research on children and adults with CAPD has revealed a number of etiological bases for the auditory deficits experienced by patients with CAPD. In some patients, the deficits can be linked to frank neurological lesions or disease processes that compromise the integrity and function of the CANS, such as trauma, neoplasms, degenerative disorders, metabolic dysfunction (including neonatal hyperbilirubinemia), neurotoxicity, viral infections, and surgical lesions (Musiek, Baran, & Pinheiro, 1994; Musiek, Baran, Shinn, & Jones, 2012). In other individuals, the deficits are related to benign CANS dysfunction. These etiological bases include delays in the neuromaturational development of the CANS (Musiek, Gollegly, & Baran, 1984), cerebromorphological abnormalities (also referred to as neuromorphological or neuroanatomical abnormalities) involving the CANS (Musiek, Gollegly, & Ross, 1985), and age-related changes within the CANS (Baran & Musiek, 1999). For a more exhaustive review of the etiological bases of CAPD, the reader is referred to Baran and Musiek (1999; 2003), Bamiou, Musiek, and Luxon (2001), Bellis (2004), Chermak and Musiek (1997, 2011), Musiek, Baran, and Pinheiro (1994), and Chapter 4 of this volume of the Handbook and Chapter 16 of Volume 2.

There are also a number of behavioral characteristics that are commonly noted in individuals with CAPD. Some of the more commonly reported symptoms include: (1) difficulty hearing spoken messages in the presence of other competing speech messages or in noisy or reverberant environments, (2) difficulty localizing the source of a signal, (3) difficulty learning a foreign language or other novel speech materials, such as technical language, (4) frequent requests for repetition, (5) difficulty processing rapid speech, (6) inconsistent or inappropriate responding to verbal stimuli, (7) inability to detect humor or sarcasm that is signaled by subtle changes in prosody, (8) being easily distracted by external stimuli, (9) difficulty maintaining attention, (10) difficulty following directions, (11) poor musical ability, and (12) reading, spelling, and/or learning problems (AAA, 2010; ASHA, 2005). The above listing of symptoms also presents a quick overview of many of the items appearing on some of the more commonly used behavioral checklists of listening and related behaviors (see Chapter 10).

As readily seen by reviewing this list of symptoms or behavioral characteristics, many if not most of these symptoms

are not unique to individuals with CAPD. In fact, there is considerable overlap with the symptoms commonly noted in a variety of other disorders, including but not limited to learning disabilities, attention deficit disorder with or without hyperactivity, language disorder, difference, or delay, disorders on the autism spectrum, other cognitive and/or behavioral disorders, and peripheral hearing loss (Baran, 2007; Baran & Musiek, 1999, 2003; Bellis, 2003; Chermak & Musiek, 1997). Therefore, the presence of any of these behavioral characteristics should not be interpreted as evidence that the individual has a CAPD, but rather as an indication that further diagnostic testing is warranted.

Test Principles

There are a number of test principles that should be considered when a central auditory test battery is being developed. Many of these are simply good test principles that should be applied whenever any type of an audiologic assessment is being undertaken, whereas others are test principles that are unique considerations for the development of a central auditory test battery. It is important to note at the outset that central auditory testing should not commence until a comprehensive audiologic evaluation has been completed.

Several test principles that are critical for the development of an efficacious central auditory test battery were enumerated in a 2005 Technical Report on Central Auditory Processing Disorders published by ASHA (2005). These test principles along with some related considerations are discussed below.

(1) It is important that the individual who is administering and interpreting a test battery do so in an ethical and efficient manner. The audiologist who is going to undertake central auditory assessments must have the requisite knowledge, preparation, and skills necessary for the administration and interpretation of the central auditory tests to be administered.

(2) As CAPD represents a heterogeneous group of auditory deficits, it is important that a test battery approach be used so that different underlying processes, as well as different levels of functioning within the CANS, can be assessed. Reliance on a single test or a limited battery of tests may fail to uncover an existing auditory deficit if the deficit is in an area not tapped by the selected test procedure or procedures. Likewise, the use of a single test or a battery of tests that assesses CANS function only at one level of the auditory system or within a limited region of the auditory system may fail to uncover compromise within the CANS.

(3) The test battery used with any given patient should be individualized as the presenting complaints and auditory behaviors that the individual experiences can be, and often are, quite diverse. A single test battery approach, which is test driven, will not result in the most comprehensive and sensitive assessment of every patient who presents in the clinic as being at risk for CAPD.

(4) There are numerous tests of central auditory processing that have been developed over the past 50 to 60 years. However, not all of these tests are equal in their ability to identify auditory processing disorders. It is important that tests chosen for inclusion in a test battery be valid and reliable measures of the auditory processing skill and/or level

of CANS functioning that the test purports to assess. Selection of tests to be included in a test battery should be based on a consideration of the test's sensitivity, specificity, and overall efficiency. (See Clinical Decision Analysis below and Chapter 12 for more extensive discussion of this principle.)

(5) Test batteries should include a variety of test stimuli, as well as test procedures. It is strongly recommended that test batteries include both verbal and nonverbal test stimuli as well as both behavioral and electrophysiologic measures to explore different auditory processing skills and levels of auditory processing within the CANS. Research findings have shown that speech signals provide access to different CANS processing mechanisms than do nonspeech signals (Grossman, Oberecker, Koch, & Friederici, 2010) and that a greater degree of temporal processing is required for the processing of speech as opposed to nonspeech signals (Fitch, Miller, & Tallal, 1997; Griffiths, Rees, & Green, 1999; Shannon, Zeng, Kamath, Wygonski, & Ekelid, 1995; Zatorre & Belin, 2001). It also has been shown that in at least some cases, central auditory processing deficits may be revealed with speech tasks, but not with similar nonspeech tasks (Johnson, Nicol, & Kraus, 2005; Russo, Nicol, Zecker, Hayes, & Kraus, 2005). Therefore, the processing of speech signals may be more vulnerable to disruption by CANS dysfunction than the processing of nonspeech stimuli. As indicated above, it is advisable that a test battery include electrophysiologic test measures as well as behavioral measures, as electrophysiologic measures can play an important role in the objective demonstration of neural deficits within the CANS. They also may help to identify patients who are likely to benefit from auditory training and then they can be used to document treatment efficacy for those individuals for whom auditory training is initiated (King, Warrier, Hayes, & Kraus, 2002; Russo et al., 2005). Finally, it is important to mention that tests should be carefully selected for inclusion in a test battery based upon a consideration of their potential value in differential diagnosis of CAPD from cognitive and/or language disorders, which often can masquerade as auditory-based problems (see Interpreting Test Results below).

(6) It is essential that any tests included in the test battery be appropriate to the individual's age, background, level of intellectual functioning, and peripheral hearing status. It is important, therefore, to take into consideration the patient's developmental age, cognitive abilities, level of language functioning, motivation, level of alertness and potential for fatigability during testing, cultural background, native language, and hearing sensitivity when selecting tests for administration as part of a test battery.

(7) Related to the previous principle, it is important to be sure that if a patient has a cognitive and/or behavioral problem for which he or she is being medicated, the individual be appropriately medicated during testing. If not, the chances of a nonauditory problem significantly influencing the test results are greatly increased.

(8) As fatigue and motivation can affect many, if not most, of the tests to be administered in a central auditory test battery, it is important to employ a test strategy that will provide the greatest amount of information in the most time-efficient manner. Careful monitoring of the patient's alertness, energy level, and motivation throughout the test session

is essential. Tests that require considerable attention and mental effort should be administered early in the test session, whereas tests that are not likely to be affected by these variables, or only minimally affected by these variables, should be positioned toward the end of the battery. Frequent breaks may be needed to maintain attention and motivation in some individuals. In others it may be necessary to bring the patient back for a second testing session to complete the testing (i.e., if this is possible). If this is not possible, then it becomes even more important that the audiologist administer the selected tests in a carefully planned sequence in order to maximize the amount of information available at the conclusion of testing so that he or she can make as comprehensive an assessment as is possible given the testing limitations.

(9) The audiologist must carefully review the recommended testing procedures, normative data, and background information that accompany the tests and use only the test procedures that were recommended by the test developer when administering tests. Any modifications to test procedures or use of a test with populations other than those upon which the test was normed will limit the applicability of the test findings and may lead to an inappropriate diagnosis. Often the audiologist will be faced with the pressure to assess auditory processing in young children. However, when testing children younger than 7 years (developmental age), task difficulty, response demands, and performance variability will severely limit the utility of these test measures for assessment purposes. There are a limited number of tests that have been specifically developed for use with young children (e.g., Pediatric Speech Intelligibility [PSI] Test [Jerger & Jerger, 1984]; Test for Auditory Processing Disorders in Children-Revised [SCAN-C; Keith, 2000]). In addition, there are some other tests that were not specifically developed for use with children that can be used with young children due to the simplicity of the tasks involved (e.g., Gaps-In-Noise [GIN] test; Amaral & Colella-Santos, 2010; Chermak & Lee, 2005; Shinn, Chermak, & Musiek, 2009; and the Listening in Spatial Noise Test [LiSN] Cameron, Dillon, & Newall, 2006). Unfortunately, the limited number of tests appropriate for use with young children restricts the number and variety of auditory processing skills that can be assessed reliably in this population. In the absence of diagnostic tests of certain auditory processes, the audiologist will have to rely more on informal assessments of auditory processing skills in young children. Behavioral checklists that can be completed by parents and/or teachers, behavioral observations by the audiologist or another adult observer, and some screening procedures may be used to identify a child who is "at-risk" for a CAPD. However, a definitive diagnosis of a CAPD should be withheld until such time as appropriate and comprehensive testing can be completed. With this caveat in place, it is strongly recommended that periodic follow-up and monitoring of the young child's auditory performance be maintained so that an appropriate diagnosis can be rendered as early as is clinically feasible.

(10) As there is a high comorbidity of other cognitive and linguistic disorders in individuals with CAPD, it is important to involve other professionals (i.e., multidisciplinary team approach) in the assessment of individuals considered to be at risk for CAPD, especially when case history information, behavioral observations

during the test session, or test results obtained during the CAPD evaluation point to potential speech and/or language delays, deficits, or disorders, intellectual, behavioral, or psychological disorders, or academic or learning deficits or problems. Generally, if these types of assessments can be completed prior to the central auditory assessment, the interpretation of the central test results can be made with appropriate levels of caution, and this is likely to result in the accurate interpretation of the test results. If not, then it will be important that a cautionary note be included in the clinical report indicating the limitations of the diagnostic assessment and the potential that some other cognitive or linguistic deficit may be contributing to the test results noted and the diagnosis rendered. In some cases, the existence of significant cormorbid conditions (e.g., severe to profound hearing loss, severe developmental delay) may preempt central auditory assessment altogether.

(11) Finally, it is important to realize that the assessment of auditory processing abilities in a clinical setting assesses auditory function in only one context, and that context is often ideal; that is, testing occurs in a sound-treated booth with minimal extraneous distractions. Also, the individual who is coming in for testing typically comes in well rested, on appropriate medications, and so forth. Therefore, the test results, which are derived under such favorable test conditions, may not fully reveal the effects of the CAPD on the individual's ability to function in everyday situations. It therefore is strongly recommended that assessment not be limited to formal diagnostic testing, but should include behavioral and systematic observation of the individual's performance in daily activities, whenever feasible (Baran, 2007). This type of information can be used to corroborate test findings and to assist in management planning. (See Chapters 13 and 14 in Volume II of this Handbook for additional information on observation techniques in everyday settings, and see ASHA, 2005 for additional discussion of test principles.)

ypes of Behavioral and ectrophysiologic Tests

Interest in the assessment of central auditory processing deficits associated with compromise of the CANS can be traced back to the mid-1950s when a group of Italian physicians (Bocca, Calearo, & Cassinari, 1954; Bocca, Calearo, Cassinari, & Migliavacca, 1955) used a distorted speech test (i.e., low-pass filtered speech test) to assess auditory performance in patients with confirmed temporal lobe lesions. These physicians noted the failure of routine peripheral hearing tests to uncover the auditory difficulties being experienced by their patients and the ability of a low-pass filtered speech test to document the presence of auditory deficits in many of these individuals. Since the initial efforts of these Italian physicians to develop a test that would be sensitive to the auditory difficulties of patients with CANS compromise, a number of other tests have been developed or utilized for this purpose. These tests have included both behavioral and electrophysiologic tests. (See Baran and Musiek, 1999, for a comprehensive review of this history.)

Since the mid-1950s there have been a number of central auditory tests developed. These tests differ in terms of the auditory processes that they assess, the types of stimuli used in the tests, the test

procedures employed, and the level of CANS that is being evaluated. In an effort to categorize these tests, a number of classification approaches have been used. However, for the purposes of the present discussion (as well as for the organization of this Handbook), a five-category classification system will be used. The categories include binaural interaction tests, dichotic speech tests, monaural low-redundancy speech tests, temporal patterning and processing tests, and electroacoustic and electrophysiologic procedures. Following a brief review of each of these test categories, the reader will be referred to the pertinent chapters in this Handbook for additional coverage of the various tests and test procedures that fall within each of the test categories introduced here.

The following overview provides a description of the properties of the test categories, a brief discussion of a representative test within each of the categories, and information regarding the sensitivity of the tests to CANS dysfunction at various levels within the CANS. This latter information is also summarized for the reader in Table 11–1, and additional information can be found in a number of other sources (Baran, 1997; Baran & Musiek, 1999; 2003; Bellis, 2003; Chermak & Musiek, 1997).

Binaural Interaction Tests

Binaural interaction tests include those tests that require the efficient integration of acoustic information from both ears in order to mediate the fusion or synthesis of acoustic information that differs in time, intensity, or frequency between the two ears. Test stimuli used in binaural interaction tests include both speech stimuli and tonal stimuli.

The tests in this category are designed to assess the ability of the CANS to take disparate information presented to the two ears and to unify this information into a single perceptual event. This unification of the disparate auditory information being presented to two ears is presumed to occur in the brainstem. For this reason, tests that fall into this category are believed to be sensitive to brainstem pathology. They can, however, also be affected by cerebral lesions, although the probability of an abnormal finding with lesions above the level of the low brainstem is quite low (as in the case for masking level differences discussed below) (Lynn, Gilroy, Taylor, & Leiser, 1981). Due to the nature of the tests in this category, a "binaural" deficit is expected—however, since the ears are not tested independently, it is not possible to differentiate the performance of one ear versus the other (see Table 11–1) (Baran & Musiek, 1999).

One of the more sensitive tests in this category is the *Masking Level Difference* (MLD) test. This test involves the presentation of a stimulus, either spondee words or a pulsed pure tone, to both ears at the same time that a broadband masking noise is delivered to the two ears (Licklider, 1948). The patient is tested under two conditions—a homophasic and antiphasic condition. In the homophasic condition the stimulus and the noise are presented in-phase to both ears (SoNo), whereas in the antiphasic condition one of the two signals is presented 180 degrees out-of-phase while the other signal is maintained in-phase between the two ears. For example, in the SπNo antiphasic condition, the noise is maintained in-phase between the two ears and the signal is presented 180 degrees out-of-phase. Subtracting the threshold established in the homophasic condition

Table 11–1. Patterns of Central Test Results That May Be Observed in Patients with Lesions at Various Sites Along the CANS

Test Category	Low Brainstem	High Brainstem	Auditory Cortex	Inter-hemispheric Pathways
Binaural Interaction	Binaural deficit[a] (2)	Little or no deficit (3)	Little or no deficit (3)	Little or no deficit (3)
Phase Tests (e.g., MLD)	Binaural deficit[a] (2)	Little or no deficit (3)	Little or no deficit (3)	Little or no deficit (3)
Dichotic Speech	Ipsilateral ear deficit (2)	Contralateral ear deficit (2) Bilateral deficits (2) Ipsilateral ear deficit (2)	Contralateral ear deficit (3) Bilateral deficits (1)	Contralateral ear deficit (3)
Monaural Low-Redundancy Speech	Ipsilateral ear deficit (2)	Contralateral ear deficit (2) Bilateral deficits (2) Ipsilateral ear deficit (1)	Contralateral ear deficit (3)	No deficit (3)
Temporal Patterning	Ipsilateral ear deficit (1)	Contralateral ear deficit (1) Bilateral deficits (1) Ipsilateral ear deficit (1)	Bilateral deficits[b] (3)	Bilateral deficits[b] (3)
Auditory Brainstem Response[c]	Ipsilateral abnormality (3) Bilateral abnormalities (1) Contralateral abnormality (1)	Bilateral abnormality (2) Ipsilateral abnormality (2) Contralateral abnormality (1)	No deficit (3)	No deficit (3)
Middle Latency Response[c]	Ipsilateral ear effect (1)	Contralateral ear effect (2) Bilateral effects (1) Ipsilateral effect (1)	Abnormality at electrode nearest pathology (2) Contralateral ear effect (2)	Little or no deficit (3)

Table 11–1. *continued*

Test Category	Low Brainstem	High Brainstem	Auditory Cortex	Inter-hemispheric Pathways
Late Response (N1 and P2)[c]	Ipsilateral ear effect (1)	Contralateral ear effect (1) Bilateral ear effects (1) Ipsilateral ear effect (1)	Abnormality at electrode nearest lesion (2) Contralateral ear effect (2)	Little or no deficit (3)
Auditory Cognitive (P3)[c]	Same as late response	Same as late response	Non-localizing abnormality (2)	Little or no deficit (3)

Source: From "Central auditory processing disorders in children and adults" by Baran and Musiek (1995). Adapted with permission from Butterworth-Heinemann Medical Publishers.

Key: (3) high probability of occurrence, (2) moderate probability of occurrence, (1) low probability of occurrence.

[a]Binaural is used in this context since both ears are receiving segments of the stimulus and only one score is derived.

[b]Specified deficits would be predicted if the patient was asked to verbally describe the patterns perceived.

[c]Abnormal results may be noted for one or more of the indexes derived during the electrophysiological procedure (see Chapter 17). The use of the singular form in this context indicates that any abnormalities that exist are limited to one ear.

from that found in either of the antiphasic conditions results in a difference score that is referred to as the masking level difference. In individuals with normal brainstem function, the threshold noted in the antiphasic condition is better (i.e., more sensitive) than the threshold obtained in the homophasic condition. This improvement in hearing sensitivity in the antiphasic condition is considered to represent a *release from masking*. This release from masking effect is considered to originate at the level of the CANS where information from the two ears is first integrated. (See Chapter 16 for discussion of clinical tests of binaural interaction and Chapter 2 for psychoacoustic considerations related to binaural interaction, including sound localization.)

Dichotic Speech Tests

Dichotic speech tests involve those tests in which different speech materials are presented to the two ears in a simultaneous or overlapping manner. Stimuli used in these tests can involve any type of speech stimulus, such as consonant-vowel combinations (CVs), digits, monosyllabic words, and sentences. Some of the tests in this category require patients

to divide their attention (i.e., to repeat all stimuli heard in both ears), whereas others require patients to direct or focus their attention to a target ear (i.e., repeat only the stimuli perceived in the right ear or the left ear, as directed). Research findings suggest that when the CANS is presented with dichotic speech materials, the weaker ipsilateral pathways tend to be suppressed, and the neural impulses travel up the preeminent contralateral pathways to reach the auditory reception areas of the cerebrum (Kimura, 1961a, 1961b).

Dichotic speech tests are particularly sensitive to lesions of the auditory cortex and the interhemispheric fibers, and to a lesser degree to auditory brainstem lesions (see Table 11–1) (Baran & Musiek, 1999). Most typically, contralateral ear effects are noted with lesions of the auditory cortex, although binaural deficits can be noted if there is significant compromise of the left side of the brain. With lesions involving the corpus callosum and/or the interhemispheric pathways, left ear deficits are commonly noted. In cases of brainstem pathology, ipsilateral ear deficits are commonly observed in patients with extra-axial lesions (i.e., lesions originating from the periphery of the brainstem), whereas bilateral, contralateral, or ipsilateral ear effects may be observed with intra-axial lesions (i.e., lesions originating from within the brainstem) (Baran & Musiek, 1999).

One of the more commonly used tests in this category is the Dichotic Digits Test (Musiek, 1983). For this test, two digit pairs (i.e., four digits) typically are presented to the patient at 50 dB SL re: SRT, with one digit from each pair being delivered to each ear in an overlapping manner, and the patient is asked to repeat all digits perceived. Although the Dichotic Digits Test stimuli are commonly presented at 50 dB SL re: SRT, the stimuli can be administered effectively at other presentation levels. The digits, which are carefully aligned in terms of their stimulus onsets, include all of the single syllable numbers from 1 to 10; the number 7 is not included as it is a two-syllable number. Patients are encouraged to guess if they are not sure as to the digits heard and they are informed that it is not necessary to repeat the digits in any particular order. A percent correct score is derived for each ear and compared to age-appropriate norms. (See Chapter 14 for further discussion of dichotic listening tests.)

Monaural Low-Redundancy Speech Tests

Monaural low-redundancy speech tests include tests in which speech stimuli have been degraded by modifying the frequency, temporal, or intensity characteristics of the undistorted signal. A common feature in all these tests is the monaural presentation of a speech stimulus that has undergone some type of signal degradation.

Clinical research has demonstrated that these types of tests tap auditory closure abilities and are moderately sensitive to cortical lesions (see Table 11–1). With cortical lesions, contralateral ear deficits are most commonly noted (Lynn & Gilroy, 1977), although in some cases with extensive left hemisphere compromise, bilateral deficits may be noted (see Baran & Musiek, 1999). In these latter cases, it is likely that the auditory areas subserving speech recognition have been compromised. Monaural low-redundancy tests are less sensitive to brainstem lesions,

and as was the case for the dichotic speech tests, the laterality effects noted are likely to differ with the specific location of the lesion. Finally, test performance on monaural low-redundancy speech tests is typically not affected in patients with interhemispheric pathway compromise (Baran & Musiek, 1999).

An example of a monaural low-redundancy speech test is the *Compressed Speech* test (Beasley, Forman, & Rintelmann, 1972; Beasley, Schwimmer, & Rintelmann, 1972). Some of the more commonly used versions of this test utilize monosyllabic words that have been compressed using time compression ratios of 60% or 65% (see Baran & Musiek, 1999). This time compression is achieved by removing brief segments of the original speech signal until either 60% or 65% of the original signal has been removed. The remaining segments of the original signal are then strung together to achieve a new speech signal that contains only 40% or 35% of the original signal. These compressed stimuli are presented to each ear individually at 40 dB SL to 50 dB SL (re: SRT) and percent correct scores are derived for each ear. These scores are compared with age-appropriate norms, which differ for tests developed using computer waveform editing software in contrast to the older electromechanical compression techniques. (See Chapter 13 for further discussion of monaural low-redundancy speech tests.)

Temporal Patterning and Temporal Processing Tests

Temporal processing involves a number of subprocesses, as can readily be seen in the definition of central auditory processing that was presented earlier in this chapter. Until recently, most of the tests used clinically to assess temporal processing skills involved the use of temporal patterning tests. More recently, however, other temporal processing tests have found their way into the clinical assessment arena. These tests assess other temporal processes, such as temporal resolution (Lister, Roberts, & Lister, 2011; Lister, Roberts, Schackelford, & Rogers, 2006; Musiek, Shinn, Jirsa, Bamiou, Baran, & Zaidan, 2005).

Temporal patterning tests assess feature detection abilities, frequency or duration discrimination, and acoustical pattern contour recognition. In addition, if the patient is asked to label the patterns perceived, then language processing also is required. Temporal patterning tests have been shown to be sensitive to compromise of the auditory cortex in the right hemisphere, which is the hemisphere responsible for the processing of the acoustical contour of the patterns (Musiek & Pinheiro, 1987) (see Table 11–1). In addition, if a verbal response is required, the test is sensitive to lesions in the left hemisphere (i.e., the hemisphere responsible for verbally labelling the patterns perceived) and/or the interhemispheric pathways (Musiek, Kibbe, & Baran, 1984). Deficits are less common in patients with brainstem lesions.

The *Frequency Pattern Sequences* test is one of the more popular tests within this category (Musiek & Pinheiro, 1987). It is composed of test sequences consisting of three tone-bursts. Two of the tone-bursts in each sequence are of the same frequency, whereas the third tone-burst is of a different frequency. The two tone-bursts used on commercially available frequency pattern sequence tests include a low-frequency tone (880 Hz) and a high-frequency tone (1122 Hz).

Each tone-burst has a 10 ms rise/fall time and a total duration of 150 ms; there is a 200 ms interstimulus interval between successive tones in each sequence. Given these parameters, a total of 6 different sequences are possible: *high-high-low, high-low-high, high-low-low, low-low-high, low-high-low, and low-high-high*. Patients are typically asked to describe the sequences perceived using the words *high* and *low*. Thirty sequences typically are presented at 50 dB SL re: SRT to each ear individually and a percent correct score is derived for each ear. Although testing is commonly completed at the sensation level mentioned above, the test can be administered at other presentation levels (Musiek, 1994). Clinical experience has shown, however, that ear differences are uncommon on this test. Therefore, it is possible to derive a single score (i.e., diotically under headphones or in the soundfield). In addition, if a patient is unable to describe the sequences, he or she may be asked to hum the acoustic patterns. If this is done, the test assesses primarily right hemisphere function. In either instance, test scores are compared with age-appropriate norms for the specific test procedure administered. (See Chapter 15 for further discussion of temporal processing tests.)

Electrophysiologic and Electroacoustic Procedures

Electrophysiologic procedures can be used to evaluate function of the auditory pathways beginning with the cochlear nerve and progressing through the cortical levels of the CANS. Electroacoustic procedures (e.g., otoacoustic emissions, acoustic reflex thresholds, acoustic reflex decay) are useful in identifying involvement of the low brainstem and in differentiating CAPD from *auditory neuropathy* (see Musiek, Baran, Shinn, & Jones, 2012). They also can be used to differentiate *central deafness* (i.e., hearing loss caused by significant and usually bihemispheric involvement of the CANS) from deafness or hearing loss of a peripheral origin (Baran & Musiek, 1999, 2003; Musiek, Charette, Morse, & Baran, 2004). The remainder of this section will focus on electrophysiologic procedures. The reader is referred to Chapters 17 and 19 for discussion of the use of electroacoustic procedures in the differential diagnosis of peripheral versus central compromise within the auditory system.

Electrophysiologic procedures provide objective measures of neural functioning and, as such, can serve as valuable adjuncts to the behavioral tests discussed above. The auditory brainstem response (ABR) is an early latency response that is frequently abnormal in patients with lesions of the cochlear nerve and/or caudal brainstem, whereas lesions in the thalamic regions and above are unlikely to result in abnormal ABR findings (see Table 11–1). Ipsilateral and bilateral deficits are common findings when utilizing the ABR; true contralateral findings are rare (Musiek, Gollegly, Kibbe, & Verkest, 1988).

The middle latency response (MLR) is sensitive to lesions located more rostrally within the CANS (thalamus and primary auditory projections), and the late auditory evoked response (LAER) and P300 assess cortical functions (Kileny, Paccioretti, & Wilson, 1987; Musiek, Baran, & Pinheiro, 1992) (see Table 11–1). Abnormal findings for both of these procedures are more commonly noted with CANS compromise affecting the auditory cortex and subcortex, but deficits can be noted with lesions located more caudally within the CANS. With cortical lesions,

deficits are more often noted from an electrode positioned over the compromised side of the brain, but contralateral ear abnormalities also are observed. In cases with compromise of the CANS in the high brainstem region, abnormalities are somewhat less frequently observed and no typical pattern of results is commonly observed. Contralateral, bilateral, and ipsilateral abnormalities can be noted. Finally, with compromise of the low brainstem, abnormalities on these electrophysiologic tests are relatively uncommon. When these are observed, the most common result is an ipsilateral abnormality (Kileny et al., 1987; Musiek, Baran, & Pinheiro, 1992).

Recently, interest has grown in the use of the mismatch negativity (MMN) responses to assess an individual's ability to discriminate or selectively attend to certain stimuli. To date, however, the procedure has been used almost exclusively in research investigations. With continued research and development of the test procedures, this procedure may provide audiologists with another objective test that can be used to assess selected auditory subprocesses that are important for the normal and efficient processing of auditory information. The reader is referred to Hall (1992), Musiek and Lee (1999), Musiek et al. (1994), and Musiek et al. (2012), as well as to Chapter 17 for additional information on the use of electrophysiologic protocols in the assessment of CAPD.

Assessment of CAPD in Special Populations

As alluded to in the information presented above, there are certain populations of patients who present unique challenges for the audiologist who is attempting to assess their central auditory processing abilities. Young children may not have the linguistic and cognitive skills needed to meet the task demands associated with some of the CAPD tests. Since most of the behavioral central tests currently employed in the assessment arena were initially developed for use with adults, these tests may not be appropriate for use with young children because of the nature of the stimuli, the task demands associated with the test, and so forth. This situation may limit the test options that the audiologist has for use with this particular population of patients.

Many individuals who present for CAPD assessments have other comorbid conditions (e.g., peripheral hearing loss, learning disabilities, autism spectrum disorder, speech and language disorders or delays, attention deficit disorder with or without hyperactivity, cognitive decline associated with aging). The presence of one or more these comorbid conditions in a given patient may well impact the individual's performance on many of the behavioral and/or electrophysiologic tests that the audiologist would like to include in the test battery, as these comorbid conditions exist along a continuum of severity from mild to severe. In some cases, the conditions will be so severe as to preclude assessment. In other cases, however, the presence of the comorbid condition will not necessarily preclude assessment, but the administration and interpretation of test results will require particular diligence on the part of the audiologist. The reader is referred to Chapters 18, 19, and 20 for an in-depth discussion of differential diagnosis among these comorbid conditions.

Finally, with the changing demographics in the United States (Day, 1996), nonnative speakers of English (and to some

extent, non-English-speaking patients) are presenting at an ever increasing frequency in our audiology clinics for central auditory assessments. Clearly, these patients can present with unique testing challenges. Many of the behavioral tests used for central auditory assessments employ English words or sentences as test stimuli. This factor can severely limit the applicability of many of the commonly employed CAPD tests for use with this population of patients—thus leaving the audiologist with only a small subset of central auditory tests (e.g., electroacoustic and electrophysiologic procedures, nonverbal behavioral tests such as frequency pattern sequences or auditory duration patterns) that can be used to assess CANS function in English language learners or in patients who do not speak English without running the risk that the patient's limited experience (or lack of experience) with English (i.e., knowledge and use) will negatively affect the test results. As was the situation when testing individuals with comorbid conditions, the testing of individuals for whom English is not the native language requires a deliberate and thoughtful approach to both test selection and the subsequent interpretation of test results.

Patients With Hearing Loss

The presence of a peripheral hearing loss in an individual being assessed for a potential CAPD presents certain challenges for the audiologist. First, it must be recognized that peripheral auditory dysfunction can lead to central auditory dysfunction. Transsynaptic degeneration of central auditory structures can occur subsequent to sensory deprivation (e.g., noise-induced or conductive hearing loss, longstanding peripheral hearing loss, severely impoverished auditory environments) (Hardie & Shepard, 1999; Schwaber, Garraghty, & Kaas, 1993; Webster & Webster, 1977). Moreover, comorbid peripheral hearing loss will affect an individual's performance on most central auditory tests, with few exceptions, as noted below.

If a comorbid peripheral hearing loss is present in an individual being seen for a central auditory assessment, then one must question whether the findings noted during the testing are the result of compromise of the CANS or whether the performance deficits being noted are simply the manifestations of the peripheral hearing impairment. Since cochlear distortion effects are common in individuals with a peripheral hearing loss, the possibility exists that abnormal performance on a central auditory test is not reflecting a CAPD, but rather that the auditory deficits being detected during central auditory testing are the result of distortion effects that are being introduced into the auditory system at the level of the periphery. The presence of distortion effects originating at the auditory periphery may well affect the performance of the individual with such compromise on many, if not all, of the central auditory tests to be administered. Clearly, if depressed scores are noted on routine speech audiometric procedures, then depressed scores would be anticipated for any central auditory test that requires the processing of speech stimuli. In these cases, it may not be possible to determine if a CAPD actually coexists with the peripheral impairment; that is, unless some unique profiles of test results emerge (see discussion below). For many years, it was common practice for the audiologist to not administer any

of the central auditory tests if a peripheral hearing loss was found to be present in an individual considered to be at risk for CAPD. Although such a practice avoided the need to account for the potential contribution of the peripheral hearing loss to the central test results, it often failed to meet the needs of the patient who was denied testing, as a central hearing disorder may very well coexist with a peripheral hearing impairment and its identification may have important implications for management of the individual with hearing loss (Baran & Musiek, 1999, 2003; Musiek & Baran, 1996; Stach, 1990; Stach, Spretnjak, & Jerger, 1990).

There have been a number of investigations that have addressed the effects of peripheral hearing loss on central auditory test results. These investigations have shown that the presence of a peripheral hearing loss can negatively impact the results of a number of central auditory tests (Divenyi & Haupt, 1997a, 1997b; Fifer, Jerger, Berlin, Tobey, & Campbell, 1983; Grimes, Mueller, & Williams, 1984; Kurdziel, Noffsinger, & Olsen, 1976; Miltenberger, Dawson, & Raica, 1978; Musiek, Gollegly, Kibbe, & Verkest-Lenz, 1992; Noffsinger, 1982; Olsen, Noffsinger, & Carhart, 1976; Orchik & Burgess, 1977; Roeser, Johns, & Price 1976; Speaks, Niccum, & Van Tasell, 1985). However, in spite of these findings (i.e., that virtually all central auditory tests can be affected by peripheral hearing loss), some studies have shown that certain central tests may be more resistant to the confounding effects of peripheral hearing loss than others. Among the central tests that use speech recognition measures, both the dichotic digits test (Musiek, Gollegly, Kibbe, & Verkest-Lenz, 1991; Speaks, Niccum, & Van Tasell, 1985) and the dichotic sentence identification test (Fifer et al., 1983) appear to be less affected by the presence of mild to moderate hearing losses than the other speech-based CAPD tests. However, it should be noted that Humes et al. (1996) reported that the dichotic digits test (single digits version) and the dichotic sentence identification tests were among five central tests that were negatively affected by hearing loss in their investigation of the effects of hearing loss and aging on auditory test performance. In addition, the frequency pattern sequences test (Humes et al, 1996; Musiek & Pinheiro, 1987) and the auditory duration patterns test (Humes et al., 1996; Musiek, Baran, & Pinheiro, 1990) have been shown to be relatively resistant to the potentially confounding effect of mild to moderate peripheral hearing loss. Given these findings, these tests should be given serious consideration for administration whenever a central auditory assessment is being conducted on an individual with a peripheral hearing loss.

Despite the potential confounding effects of peripheral hearing loss and the limited number of tests that appear to be somewhat resistant to these effects, the assessment of central auditory function in individuals with mild to moderately severe peripheral hearing impairment should not be withheld. The presence of either normal or abnormal central auditory function can be implicated in a number of clinical situations , and the identification of CAPD or normal CANS function can lead to the more effective management of the individual with hearing loss. The situations in which a determination of the status of the CANS and the individual's auditory processing abilities can potentially be made include the following: (1) If hearing loss is present in one or both ears and the central test results

fall within the normal range for both ears, then the presence of CANS compromise or a CAPD can be ruled out; (2) if a bilaterally symmetrical hearing loss is present and the central test results are more depressed in one ear relative to the other ear, then the presence of a CAPD and CANS dysfunction is implicated; (3) if a hearing loss is present, but it is a unilateral or asymmetrical loss and the *better ear* shows the poorer performance on central auditory testing, then CAPD and CANS dysfunction are implicated; (4) if a symmetrical hearing loss is present and abnormal middle and/or late potentials are noted from electrodes positioned over one hemisphere versus the other (i.e., a significant electrode effect), CANS involvement should be suspected and as such would implicate the presence of a CAPD; (5) if a symmetrical hearing loss is present and an *ear effect* is noted during electrophysiologic testing, then the possibility of CANS involvement and a CAPD should be entertained; and (6) if an asymmetrical hearing deficit is noted on a given test measure (such as word recognition or an MLR amplitude measure) and the binaural presentation of the acoustic stimulus results in a poorer performance or measure than that which is noted for the better ear for a monaural presentation condition (i.e., a binaural interference effect), then CANS compromise and a CAPD are implicated (Jerger, Silman, Lew, & Chmiel, 1993; Musiek & Baran, 1996). In these cases, a determination as to the presence or absence of a CAPD can be made with a certain degree of confidence. However, for those individuals with significant peripheral hearing loss whose test results do not fit neatly into one of these categories or profiles, then the diagnosis of a CAPD is difficult, if not impossible, to make. In spite of this significant limitation in the audiologist's ability to definitively diagnose the presence of CAPD or CANS dysfunction in cases of coexisting peripheral hearing loss, the administration of central auditory testing may lead to the identification of auditory deficits, which if managed appropriately can significantly improve the quality of life for the patient with these auditory difficulties even though the etiological basis for the deficits cannot be determined. Additional assessments outside the field of audiology, such as modern day neuroimaging, can often provide more definitive information about CANS involvement, when clinically necessary. (See Chapter 18 for a discussion of CAPD and comorbid hearing loss in older adults and Chapters 3 and 23 for an overview of use of radiologic techniques in assessment of CAPD.)

Young Children

There are a number of patient variables that are important to take into consideration when one is preparing to administer a central auditory test battery. One of these variables is patient age. As mentioned above, most of the tests that are currently available for clinical use were originally developed for use in the assessment of auditory processing abilities in adults. In fact, many of these tests were initially developed for clinical research purposes to assess auditory processing skills in adults with frank neurological involvement of the CANS (Baran, 2007; Baran & Musiek, 1999). Following the initial clinical investigations of the auditory processing abilities in patients with documented lesions within the CANS, the tests were then used in the assessment of adults considered to be at risk for CAPD, as the earlier research studies had established linkages between

specific impairments of auditory function and various sites of compromise within the CANS. It was only after the application of some of these tests to the adult population of individuals considered to be at risk for CAPD that much attention was directed toward the use of these tests with children. Initially these tests were administered to children with language and learning disabilities (who were considered to be at risk for CAPD), and then testing was expanded to other children who did not necessarily present with these types of comorbid diagnoses (see Baran & Musiek, 1999), as well as to children with circumscribed lesions of the CANS (see Chermak & Musiek, 2011).

Available evidence on the development of the CANS suggests that this system does not reach the adult stage of maturity until adolescence (AAA, 2010; Yakovlev & Lecours, 1967). Therefore, it is important that age-appropriate norms be available for any test that is to be administered to children below this age range. It is also advisable that the audiologist carefully scrutinize the tests to be administered to be sure that the test items are within the child's receptive vocabulary (i.e., if the test involves speech recognition measures), and that the child being tested is capable of providing the type of response required by the test.

Other Populations

There are other populations for which the administration of central auditory testing will require special considerations. Space limitations preclude a detailed discussion of each of these populations; however, brief comments are offered in an effort to raise the audiologist's awareness of these populations and the need to consider the following: (1) whether central auditory testing is appropriate for a given individual from a special population, (2) whether modifications in test procedures are needed, and if they are, what are the implications of these modifications on test interpretation, and (3) whether there are methods and procedures available that will facilitate a differential diagnosis for individuals who may have comorbid conditions.

The audiologist who is involved in central auditory testing is likely to encounter the situation of being asked to assess an individual with a developmental delay. Several of the same considerations noted above when testing young children would apply for the development, execution, and interpretation of a test battery administered to an individual with a developmental delay. It will be important to verify that the tests to be administered are appropriate to the individual's level of cognitive and language functioning, and that the individual is capable of responding in the desired manner. Interpretation of the test results will also require special considerations. If the individual is developmentally delayed, then use of chronologically based age norms is not likely to be appropriate. In these cases, the audiologist may choose to use norms based upon mental age, language age, or some other measure of intellectual functioning. Even with such accommodations, diagnosis of a CAPD must be rendered with due caution, as any deficits noted during the testing may be reflecting nonauditory factors (attention, impaired intellectual functioning, etc.) rather than a true auditory deficit.

Similar considerations should be undertaken when testing individuals who are either at risk for or diagnosed with cognitive or behavioral deficits or disabilities (e.g., individuals with attention deficit disorders, learning disabilities, Alzheimer's

and other forms of dementia, head injuries, the elderly, particularly the very old). In many cases, a diagnosis of these disorders or deficits will be known at the time of testing; however, in some cases, especially those with subtle deficits, these deficits may not be known to the patient and/or his or her family, and they may not be readily apparent even to the trained observer. These subtle deficits, however, may still negatively impact the individual's performance on an auditory test battery, and it will be important for the audiologist to watch for patterns of responses that might suggest a nonauditory basis for abnormal performance on an auditory test. In some instances when the existence of a comorbid condition is known, it may be possible for the audiologist to establish the existence of CAPD in an individual with a comorbid condition. For example, if an individual with attention deficit hyperactivity disorder (ADHD) shows significant deficits in one ear on a dichotic speech test, it is likely that the deficit is auditorily based and not simply a manifestation of the attention or behavioral regulation problem, as it would be expected that the attentional deficits noted in ADHD would affect performance in both ears. For additional information on the assessment of special populations, the reader is referred to Chapter 18 (the elderly) and Chapter 20 (ADHD), and Chapter 17 of Volume 2 (cognitive-communicative and language factors).

[redacted]inical Decision Analysis

An important decision that the audiologist involved in central auditory assessments must make involves which tests to include in a central auditory test battery. As discussed above (see the section on Test Principles), the audiologist will want to include a variety of tests that assess various auditory processes and skills, as well as the efficiency of neural processing at various levels within the CANS. One potential strategy for developing a comprehensive test battery for central auditory testing would be to select one test from each of the test categories mentioned above. Although such a strategy for test selection may appear to be logical, it may in fact not necessarily result in the most efficient and valid test battery, as different tests have different test properties. Therefore, it will be important for the audiologist to carefully consider the individual test properties of the various tests within each test category to determine their individual test efficiencies prior to rendering a decision on the composition of a central auditory test battery.

Clinical decision analysis is a process that can be used by the audiologist to evaluate the performance of individual diagnostic tests, as well as various combinations of diagnostic tests, and to better understand the probabilistic uncertainties associated with the administration of these tests or test batteries (Musiek, Chermak, Weihing, Zappulla, & Nagle, 2011; Turner & Hurley, 2009; Turner, Robinette, & Bauch, 1999). Clinical decision analysis is based on the assumption that only two patient states or conditions are possible; that is, either the patient has the disorder or dysfunction or the patient does not have the disorder or dysfunction (Figure 11–1). Test outcomes are then compared with these two states or conditions to derive a number of measures of test performance. Take for example the information presented in Figure 11–1. On the left-hand side of the figure is informa-

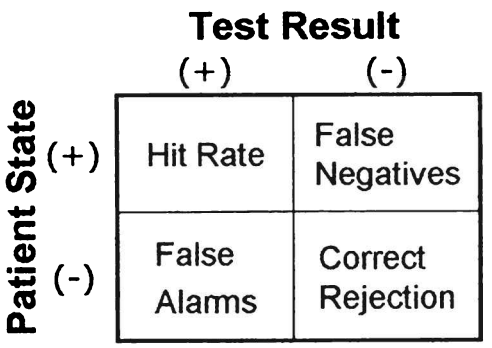

Figure 11–1. A decision matrix for diagnostic tests depicting the potential outcomes when a diagnostic test is administered to patients without and without a specific disease (defined as patient state in this figure; see text for additional discussion of the test outcome results shown in the four cells of this matrix). Musiek, F. E., and Rintelmann, W. F., *Contemporary Perspectives in Hearing Assessment*, 1st ed., © 1999, p. 438. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

tion about the state or condition. There are only two options: A positive indicator (+) indicates that the state or condition is present, while a negative indicator (–) indicates that the state or condition is not present. Test results or outcomes are recorded in the columns of the table shown in Figure 11–1. In this figure, a positive indicator (+) as shown in the left-hand column signifies that the test result confirmed the existence of the state or condition and a negative indictor (–) as shown in the right-hand column indicates that the test results were not consistent with the presence of the state or condition. If one administers a test that is specifically designed to assess a given state or condition, and the test result obtained confirms the expected state or condition, then the test outcome is said to be a true positive (or a hit). If, however, on the other hand, the test does not result in the predicted outcome, then the test outcome is said to be a false negative or a false alarm. In an ideal situation, the test performance should be positive for every patient with the state or condition (often referred to as the hit rate), and negative for all individuals tested that do not have the condition (commonly referred to as the correct rejection rate). Unfortunately, such an ideal situation does not exist in our current testing protocols. What often occurs is that a patient with a given state or condition will perform differently from the expectation, rendering a negative finding in the presence of the state or condition (i.e., a false negative), and some individuals without the state or condition will test positive for the state or condition (i.e., a false positive or false alarm).

Several measures can be derived from the information presented in Figure 11–1, but two measures in particular are central to our discussion of test efficiency (i.e., test sensitivity and test specificity). Sensitivity, or hit rate, refers to the percentage of patients with a given condition or pathology that the test accurately identifies as having the condition or pathology (see Equation 11–1). For example, if a test is specifically designed to identify brainstem pathology, as would be the case for the MLD test, then the sensitivity measure provides an indication of how many patients in an experimental population of subjects with compromise at this level of the CANS actually performed abnormally on this test. A second measure that is important to consider when selecting a test for administration is the test's specificity or correct rejection rate (CR). Specificity refers to the percentage of individuals without the condition or pathology for which the test was

designed to test who perform normally (or at least differently from the population of patients for whom the test was designed) (see Equation 11–2).

$$HR = tp/dp \qquad \text{Equation 11–1}$$

$$CR = tn/np \qquad \text{Equation 11–2}$$

where HR = hit rate, tp = number of true positives, dp = number of diseased patients, CR = correction rejection rate, tn = number of true negatives, and np = number of nondiseased individuals (Turner et al., 1999).

The best tests for administration as part of a CAPD test battery would be those tests that are highly efficient; that is, they are high on both of these measures. However, for most tests there is a trade-off between the two measures. If a cutoff criterion on a given test is selected to increase one of these measures, the other measure generally suffers. For example, it may be possible to select a cutoff criterion that is so lax that any patient with a given condition, pathology, or disorder will fall outside of the range of normal performance; however, as the criterion is relaxed the number of individuals without the condition who are inaccurately identified as having the condition, pathology, or disorder is likely to increase (i.e., the number of false alarms or false positives will increase).

In these cases, the audiologist will be faced with the decision as to what type of error is more acceptable. Would it be better to identify more patients with the disorder, recognizing that this increase in the sensitivity of the test is likely to be accompanied by coincident decrease in the specificity of the test? Or would it be clinically preferable to limit the number of false alarms and accept the fact that some patients who would have been identified as having the disorder if an alternative criterion had been used would be missed? Obviously, these are important decisions that can be made only following careful consideration of a number of factors, including the morbidity rate associated with the condition (if any), the financial and emotional costs of over-referrals, the potential long-term financial costs if intervention is delayed, and so forth.

Another approach that can be used to assess the clinical utility of a test or test battery is posterior probabilities. This approach considers both prevalence data as well as sensitivity and specificity measures to derive two measures: a positive posterior probability (D+) and a negative posterior probability (N–). The D+ measure represents the likelihood that a patient has the disorder when a positive test result is noted (see Equation 11–3), whereas the N– measure provides an indication of the probability that a patient does not have the disorder when a negative test result is found (see Equation 11–4).

Equation 11–3

$$[D+] = \frac{1}{1 + \frac{(FA) \times (1 - PD)}{(HT) \times (PD)}}$$

Equation 11–4

$$[N-] = \frac{1}{1 + \frac{(1 - HT) \times (PD)}{(1 - FA) \times (1 - PD)}}$$

where HR = hit rate, FA = false alarm rate, and PD = prevalence (Turner et al., 1999).

As discussed by Musiek et al. (2011), these calculations, which include preva-

lence data, may lead to different findings and clinical decisions than those that are based solely on a consideration of test sensitivity, specificity, and efficiency. In addition, small changes in any of the variables used in the calculation of posterior probabilities can result in large changes in the posterior probabilities indices.

As hit rates and false alarm rates can vary significantly with the criterion used to differentiate normal versus abnormal performance, many researchers have begun to use the receiver operating characteristic (ROC) curve as a means for analyzing test performance. The ROC curve plots hit rate versus false alarm rate (i.e., 1—specificity, if proportional values are used, or 100—specificity, if percentages are used) for different cutoff criteria. A visual inspection of the data displayed in the ROC curve can lead to a decision regarding the best criterion to be used to maximize both the sensitivity and the specificity of the test. For example, the following function (Figure 11–2) was derived from gap detection threshold data obtained by Musiek et al. (2005). Based upon these data, the researchers concluded that a 5-msec cutoff criterion for a measure of a gap detection threshold resulted in a sensitivity of 73% and a specificity of 84%. In addition, the area under the curve can be calculated and can provide the investigator an indication

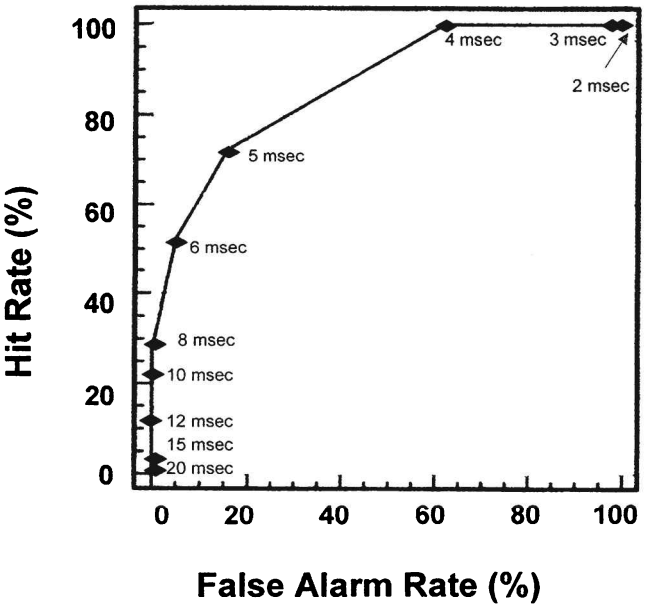

Figure 11–2. Receiver operating characteristic (ROC) curve for the approximate threshold (A.th.) measure derived during the administration of the Gaps-in-Noise (GIN) test. Hit rate (i.e., test sensitivity) is plotted on the *y*-axis and false alarm rate (100 % minus the correct rejection rate or specificity) is plotted on the *x*-axis (adapted from Musiek et al., 2005, with permission).

of the *goodness* of the test. In the case of the ROC derived by Musiek et al. (2005), the area under the curve was 0.87. According to Hanley and McNeil (1984), a ROC curve with an area under the curve falling between 0.7 and 0.9 represents a *good* test.

The use of these types of clinical analysis can help inform clinical decisions about the effectiveness and utility of any test and/or test battery that is subjected to such analysis. Space limitations preclude a detailed discussion of all of these test measures as well as other measures that can be derived to help inform clinical test decisions about which test and/or tests to administer as part of a clinical assessment (e.g., cost-effectiveness). The reader interested in a more in-depth discussion of clinical decision analysis procedures is referred to Turner et al. (1999) and to Chapter 12 in this Handbook.

Recently there has been an increasing number of clinical investigations that have attempted to determine test efficiency. Data are emerging that some tests are quite high on both sensitivity and specificity measures. For example, dichotic digits (Musiek, 1983; Hurley & Musiek, 1997), duration patterns (Hurley & Musiek, 1997; Musiek, Baran, & Pinheiro, 1990), frequency patterns (Musiek & Pinheiro, 1987), and MLR and P300 (Musiek, Baran, & Pinheiro, 1992; Musiek et al., 1999) have all been shown to perform quite well on both sensitivity and specificity measures. More recently, Musiek et al. (2011) examined the protocol performance (sensitivity, specificity, and efficiency) of four central auditory tests (dichotic digits, competing sentences, frequency patterns, and filtered speech) for a population of 20 individuals with confirmed lesions of the auditory cortex (12 right hemisphere, 4 left hemisphere, and 4 bilateral lesions) and 29 control subjects. The four tests were combined to form all possible combinations of two-test, three-test, and four-test batteries, and both lax (i.e., failure on only one test required) and strict (i.e., failure on all tests in the battery required) performance criteria, as well as intermediate criteria (i.e., failure on more than one, but not on all of the tests in the battery required) were included in their analyses. The authors found that a two-test combination (dichotic digits and frequency patterns) using a strict criterion provided the best balance between sensitivity and specificity across the test batteries examined in their study. However, they caution that this test battery is not necessarily the same test battery that should be used for every individual being seen for a central auditory assessment, as patients present with different histories, complaints, and auditory and nonauditory behaviors. It is also important to note that the participant pool in the Musiek et al. study was limited to patients with lesions involving the auditory cortex for the most part (although some individuals did have lesions that extended beyond the auditory cortex) and only four central auditory tests were included in the analyses. It is therefore important that similar analyses be conducted with other patient populations, as lesion sites within the CANS are not limited to the auditory cortex, and also with other central auditory tests, as there are many central auditory tests that have not been subjected to this type of detailed analysis. For more detailed information on the assessment of test battery protocols, see Musiek et al., 2011, Turner & Hurley, 2009, and Chapter 12 in this Handbook. As more behavioral and electrophysiologic tests used in the assessment of CAPD are subjected to

these types of clinical performance analyses (both individual tests and test batteries), audiologists will be better equipped to make informed decisions regarding the selection of a given test or test batteries for each individual patient who is being seen for a CAPD evaluation.

At this point in the discussion, a cautionary note appears to be in order. Inherent in the discussion offered above is the existence of a *gold standard* against which test performance can be gauged to establish measures of test sensitivity, specificity, and efficiency (a combination of both sensitivity and specificity measures). Such a gold standard should be derived from a well-defined population of patients with the disorder, as well as from testing of a population of subjects who can be documented as not having the disorder. Unfortunately, because of the variability in both the causes and the manifestations of CAPD, such a gold standard does not readily exist, at least not in most clinical populations studied. Efforts to attempt to establish test efficiency measures (for the identification of CAPD) based upon the presence or absence of a behavioral symptom (e.g., difficulty hearing in noise) are fraught with problems, as there is considerable overlap in the behavioral symptoms of many comorbid conditions. In the example given above (i.e., difficulty hearing in noise), there is no way to establish *a priori* that the origin of this behavioral symptom is CAPD—the difficulty experienced may be due to a CAPD, but it may also be related to other behavioral, psychological, and/or learning problems, and even peripheral hearing impairment. Therefore, the percentage of individuals in the group who performed abnormally on a test measure designed to assess speech recognition in noise does not reflect the percentage of individuals with CAPD, but rather the percentage of individual who have difficulty hearing in noise, regardless of the etiology of this problem. Likewise, the administration of a central auditory processing test to a population of subjects with a comorbid condition (e.g., learning disabilities or ADHD) cannot be used to definitively establish test performance measures for a central auditory test for similar reasons—that is, there is no way to establish that each individual within the population of individuals with the cormobid condition being tested also exhibits CAPD or has a CANS compromise. The best population for the establishment of these types of measures includes subject groups with confirmed lesions of the CANS as evidenced by radiologic findings (AAA, 2010). In these cases, sensitivity and specificity measures can be derived with a degree of confidence in the accuracy of the test measures, as it is possible to link site of pathology with test performance. Most of the available data on test efficiency has been derived from investigations of the performance of adults with confirmed lesions of the CANS on one or more of the central auditory tests. In many cases, distinctive patterns of results have been observed in patients with confirmed lesions of the CANS, with similar patterns of results noted in other populations (e.g., children or adults) for whom confirmed CANS compromise is not available. By inference, if similar test results or patterns of results are seen in children and/or adults, then it may be reasonable to use sensitivity and specificity data derived from patients with known CANS lesions to guide test selection for individuals who are considered to be at risk for CAPD but for whom evidence of confirmed CANS involvement is

lacking (AAA, 2010; ASHA, 2005; Baran & Musiek, 1999; Chermak & Musiek, 2011; Musiek, Gollegly, & Baran, 1984).

Interpreting Test Results

There are a number of different approaches that can be used to interpret the results of diagnostic tests used in the assessment of CAPD. These include both norm-based (intersubject) approaches as well as a number of potential intrasubject (patient-referenced) comparisons. For norm-based approaches, the performance of the individual being tested is compared with the performance of a group of normal subjects who have served as the subjects for a normative study. Criterion for normal performance can be established by deriving a mean performance score or measure for the group and then adding and/or subtracting one or more standard deviations to or from the mean to establish a cutoff criterion or criteria for normal performance. For example, if a threshold measure or a latency measure is being derived, then typically the standard deviation measure (most often two standard deviations) would be added to the value. If, on the other hand, a percent correct score is being derived, then the standard deviation measure would be subtracted from the mean. In some cases, there may be a range of normal performance in which the standard deviation measure is both added to and subtracted from the mean. This procedure is generally used if the normal performance on the measure is typically at or near 50% (as is the case of the dichotic rhyme test; Musiek, Kurdziel-Schwan, Kibbe, Gollegly, Baran, & Rintelmann, 1989). Another approach would be to establish a cutoff criterion based upon percentile ranks; that is, the test developer may establish a cutoff criterion value that is set to some percentile value (e.g., the 90th percentile or the 75th percentile).

Intrasubject analysis involves the comparison of an individual's performance relative to his or her own baseline performance. One such approach includes the comparison of an individual's performance on a given test under different conditions (e.g., interaural [ear] differences, interhemispheric differences, divided versus directed dichotic listening, multimodality differences). For example, on the MLR or LAER tests, the amplitudes of the responses derived from electrodes over one hemisphere can be compared with those derived from electrodes positioned over the other hemisphere. The assumption is that within an individual the size of the responses derived from the two sides of the brain should be approximately equal. Significant differences in the size of the response from one side of the brain compared to the other can signal CANS compromise. Other intrasubject assessment approaches can include an *intertest* comparison, which involves the comparison of trends across the test results obtained during the administration of a test battery, with the requirement that the pattern of test results be consistent with an anatomical site of lesion/dysfunction and neuroscience principles, or a *cross-discipline* analysis, which involves the comparison of test results obtained from the central auditory assessment and from related disciplinary assessments, such as speech and language, psychological, educational, and academic assessments. These sorts of intrasubject comparisons can also provide insights regarding differential diag-

nosis of comorbid conditions. For example, the finding of a greater deficit on a dichotic test administered in a divided attention mode relative to a focused or directed mode might indicate a nonauditory source of the problem, perhaps a cognitive deficit, rather than a CAPD. (See Chapters 17 and 20 for additional discussion of intrasubject strategies.)

As noted above, CAPD is a heterogeneous disorder and there is no one pattern of results that will be seen in all patients with this disorder. Therefore, the diagnosis of CAPD requires due diligence. Many professionals advocate that the diagnosis of a CAPD be reserved for individuals who fail at least two central auditory tests (i.e., with criterion of two standard deviations below the mean) (AAA, 2010; ASHA, 2005; Chermak & Musiek, 1997). Obviously the greater the number of tests failed, especially if the tests assess diverse auditory processing abilities and neural functioning at different levels of the CANS, the more confident the audiologist can be in the diagnosis of a CAPD (Musiek et al., 2011). With limited evidence, the interpretation of the test results should always be made with caution. A below normal performance on a single test may indicate the presence of some nonauditory deficit or subject-variable effect (fatigue, boredom, malaise), but it may well be signaling that CAPD is in fact present and in need of remediation. Although there may be some hesitancy on the part of the audiologist to diagnose a CAPD on the basis of a single abnormal test result, additional support for the presence of the deficit may be found in a careful analysis of the case history information. If the deficit noted is consistent with the behavioral complaint or complaints, then the audiologist can be more confident in rendering a diagnosis. Another strategy that the audiologist can use to increase his or her confidence in the test findings is to either readminister the failed test a second time or to administer a second test to the patient that assesses the same (or a similar) underlying process(es). As noted above, there are several tests in each of the various test categories discussed in this chapter; therefore, the audiologist is not limited to the use of only one test to assess a given auditory process or a level of functioning within the CANS. The administration of an alternative test from within the same category of CAPD tests is a reasonable approach to confirming central auditory deficits. Another strategy that has been advocated by the ASHA (2005) task force for diagnosing CAPD under these conditions (i.e., failure on only one test or test measure) is to use a more stringent criterion for abnormal performance; for example, to use a cutoff criterion that is three standard deviations above or below the mean. In these instances, additional confidence in appropriateness of a CAPD diagnosis is afforded when there is sufficient evidence in the patient's case history of functional difficulties in auditory areas subserved by the deficient process (AAA, 2010).

As there is considerable overlap in the behavioral manifestations of several other cognitive and learning disorders, it is important that the audiologist carefully observe the individual's performance throughout the test battery. Inconsistent behaviors or responding patterns are likely to signal a nonauditory problem or deficit rather than (or in addition to) a CAPD. Likewise, pervasive deficits noted on all tests within a central test battery may well signal a cognitive or supramodal deficit, such as an attention or memory

deficit, or some other nonauditory influence on test performance, such as motivation, fatigue, boredom with the testing process, and so forth, rather than a CAPD.

Finally, it should be noted that although the identification of a CAPD is one of the primary goals inherent in the evaluation of the individual at risk for CAPD, a second and equally important goal is the identification of the auditory processes that are in need of remediation. Careful selection of the tests administered during a CAPD test battery coupled with meticulous scrutiny of the test results can, and should, lead to the development of a comprehensive intervention plan. There are a number of resources that identify specific auditory processes, the diagnostic tests that assess these processes, and the intervention strategies that can be used if deficits are identified during the testing process (Musiek, 1999; Musiek, Baran, & Schochat, 1999; Musiek & Schochat, 1999). These resources can provide the audiologist with some guidance in terms of test selection and interpretation, and the subsequent development of a management plan, if a CAPD is identified. In addition, a number of other authors have proposed classification systems that can be used to classify individuals diagnosed with CAPD (Bellis, 2003; Bellis & Ferre, 1999; Katz, 1992). These classification systems were developed in an attempt to relate diagnostic test performance to both specific behavioral symptoms and performance difficulties, both auditory and nonauditory (e.g., communication difficulties, academic difficulties, difficulties in the workplace). Although these classification systems are not universally accepted at this time (ASHA, 2005), they may serve as guides to facilitate interpretation of test results and to develop deficit-specific interventions programs. (See Chapter 8 in this volume and Chapters 13 and 20 in Volume II of this Handbook for further discussion of CAPD subprofiling.)

Concluding Comments

CAPD represents a complex and heterogeneous group of auditory deficits that can result from a variety of etiological bases and from dysfunction at multiple sites along the CANS. For this reason, it will be important that assessment of the individual considered to be at risk for CAPD include a number of different behavioral, electroacoustic, and electrophysiologic tests specifically chosen to assess different auditory processes and skills, as well as CANS function at various levels within this system. One classification system for categorizing the available central auditory tests includes a five category system. The five categories include binaural interaction tests, monaural low-redundancy speech tests, dichotic speech tests, temporal processing tests, and electrophysiologic and electroacoustic measures. Within each of these categories, there are a number of tests that can be selected for administration as part of a central auditory test battery, although not all tests within each of these categories are equal in terms of their test performance characteristics. A consideration of test performance measures, such as test efficiency, sensitivity, and specificity, can help inform the selection of tests, which when included in an individualized test battery should lead to the efficacious and comprehensive assessment of the patient's auditory processing abilities. Finally, a number of test principles that can impact the selection and

the administration of the tests, as well as the interpretation of the test results, were discussed. Careful consideration of these principles will help ensure that an accurate and comprehensive assessment of an individual's central auditory processing skills and CANS functioning can be achieved. This, in turn, will help to inform the subsequent development of an efficacious management plan that will target the specific auditory deficits that were uncovered during the assessment process.

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CHAPTER 12

PRIMER ON CLINICAL DECISION ANALYSIS

JEFFREY WEIHING and SAMUEL R. ATCHERSON

Introduction

Clinical decision analysis (CDA) is an objective, mathematical approach to determining the relative merits of different clinical tests. The most effective clinical test would be one that always detects pathology when patients present with a disorder, while also correctly identifying patients without pathology as having normal function. Most, if not all, clinical tests will never achieve this goal, as there will always undoubtedly be some margin of error in diagnostic accuracy. As a result, some patients with pathology will go undetected, while other patients with normal functioning will be erroneously identified as disordered. Although perfect diagnostic accuracy may not be realistic, it is possible to characterize the degree to which a clinical test approaches this goal, and our methods for accomplishing this are rooted in CDA. Importantly, a well-rounded understanding of CDA and how it applies to common clinical tests can assist the audiologist in making appropriate clinical decisions.

The primary aim of this chapter is to provide an overview of the calculations (i.e., indexes) used when performing CDA. There are several different ways in which the diagnostic ability of a test can be characterized, and this chapter will consider each in turn. In an attempt to reinforce the relevance of CDA to audiology practice, most CDA indices are described in the context of a real audiology issue. Thus, readers will find themselves considering not only how

to calculate an index, but also how this information has actually been used in the past to improve the practice of audiology.

A secondary, equally important aim of this chapter is to discuss the utility of CDA in establishing diagnostic tests of central auditory processing disorder (CAPD). Current best practice recommendations characterize CAPD as a dysfunction of the central auditory nervous system (CANS). Thus, CDA performed in neurological populations with known CANS involvement can provide information on the relative merits of a test in detecting CAPD. The value of this application of CDA, along with several alternatives, are considered in the present chapter.

tory and Overview: elevance of CDA

Early writings about CDA in audiology began as early as 1972 when Schultz published a book titled *An Analysis of Clinical Behavior in Speech and Hearing*. Work by Jerger (1983), Jerger and Jerger (1983), and Turner and his colleagues (Turner & Nielsen, 1984; Turner et al, 1984) helped to usher in the widespread use of CDA in audiology. CDA has been applied most commonly to tests that are used to identify acoustic tumors (Musiek, McCormick, & Hurley, 1996; Turner & Nielsen, 1984) and more recently with CAPD tests (Hurley & Musiek, 1997; Musiek, Chermak, Weihing, Zappulla, & Nagle, 2011; Singer, Hurley, & Preece, 1998).

CDA was born out of the principles and concepts of signal detection theory (SDT). In SDT, the aim of a test is to discriminate between two mutually exclusive states, such as the presence or absence of a signal (McFall & Treat, 1999). The signal of interest usually is embedded in noise events, and the noise events may mimic or erroneously be mistaken for the signal. McFall and Treat (1999) state that SDT has two important components: the perceptual index and the decisional index. The perceptual index provides a measure of diagnostic accuracy that indicates quantitatively how well a test can discriminate between the two possible states. The decisional index, on the other hand, quantitatively indicates how a criterion or cutoff score influences the test result.

In audiology, the concepts of SDT are at work during pure-tone audiometry at a single frequency. As tones are presented to the patient, the patient will likely be influenced by a variety of different potential noise events (physically external, physiological, and cognitive) that may come to alter the patient's internal criterion from which to decide if a tone (signal) is present or absent. Testing in a sound booth that does not exceed maximum permissible ambient noise levels (MPANL; ANSI S3.1-1999 [r2008]) and giving clear instructions with age- and/or cognitively appropriate response mode are well-known ways to keep the test quality and perceptual index high. Specifically, patients should be able to discriminate whether they hear a tone or not as the audiologist performs a psychophysical bracketing procedure. If the patient breathes loudly during testing or has no interest in being tested, the signal may be difficult to detect near true threshold. Alternatively, a patient may be aggressive and decide to perform his or her best at all costs and end up with results closer to true threshold. These two scenarios paint a picture of how

one's internal criterion (the decisional index) may vary from person to person, and one's internal criterion may also vary from the beginning of the test to the end of the test. At the conclusion of the audiometric test, the audiologist will make an overall judgment about the quality of the test results (e.g., good, fair, poor). On what does the audiologist make this judgment? This decision is typically made by observing the patient's behaviors and the pattern of responses to the presentation of tones. Consistent and repeatable responses are likely to be judged reliable (good), whereas inconsistent and unpredictable responses are likely to be judged unreliable (poor). From here, the audiologist will make judgments about the next clinical or diagnostic course of action, or whether to terminate testing altogether.

A broader application of SDT to clinical tests, and the most frequently encountered CDA application, is to examine the ability of a test to accurately detect a disorder in groups of patients. In this situation, the "signal" is the disorder and we are interested in how often the test can detect the disorder when it is present while also correctly identifying when the disorder is not present. This application is necessary due to within-group performance variability, as illustrated in Figure 12–1. Examined across the group, performance on a test has a measure of variability in both normal

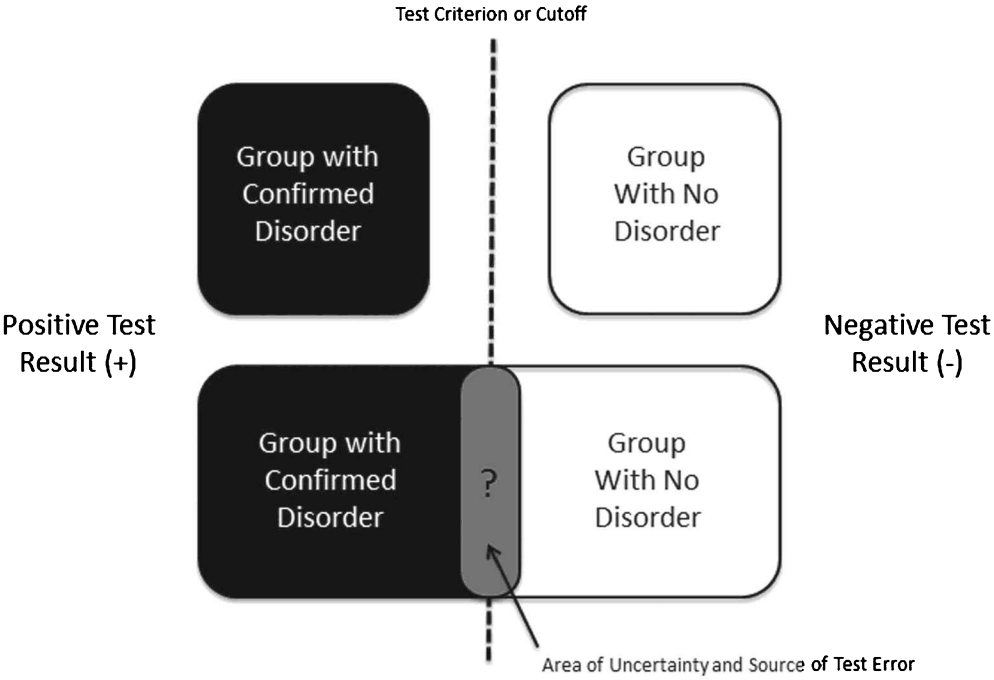


Figure 12–1. Test performance uncertainty (*gray area*) in differentiating between a group with confirmed disorder (*black box*) and a group with no disorder (*white box*). Group variability is indicated by the width of the individual boxes, and the vertical dashed line indicates the test criterion or cutoff score.

and disordered groups. The range of scores demonstrated by each group is represented by the width of the group's squares, where wider squares indicate greater group variability. The top scenario in Figure 12–1 represents an ideal situation, where group variability is low and the normal and disordered distributions do not overlap. This situation yields perfect group discrimination, and the performance of this test as measured by CDA would be high. The bottom scenario in Figure 12–1 more realistically depicts an actual clinical test, where variability is somewhat larger and the performance of both groups shows some degree of overlap. This variability and overlap contributes "noise" to our signal detection process, making it harder to detect abnormal or normal function. Thus, it is within the overlap that clinical decision errors can occur: disordered patients can be identified as having normal function, and normally functioning patients can be identified as having the disorder. It is reasonable to expect that all tests approach the bottom situation to some degree, and CDA can assist the clinician in determining how detrimental is the overlap.

In addition to within-group variability, several other factors influence the ability of clinical tests to discriminate groups. First, as discussed below, the prevalence of a disorder, known or unknown, introduces a measure of uncertainty about how tests will perform. If the disorder is more prevalent, this tends to enhance the ability of a test to detect the disorder. On the other hand, if the disorder is infrequent, this tends to enhance the ability of the test to detect normal function. Second, clinicians are reliant upon a given test's reliability and validity. If a test is unreliable and provides inconsistent scores on retest, or if it fails to measure what the clinician believes it is measuring, then its clinical utility is questionable.

Indexes

Sensitivity, Specificity, and Efficiency

The goal of CDA is to determine how efficacious a test is in identifying disordered from normal patients or to discriminate between two different disorders (e.g., retrocochlear vs. cochlear). All tests will have a single best criterion or cutoff score that attempts to discriminate between the two possibilities. We begin with the assumption that a given test is valid, that is, it has the ability to detect a disorder for which it was designed. Secondly, we assume that test is not only valid, but reliable, that is, it will yield a similar result no matter how many times it has been administered to the same person or if different people administer the test. Third, we assume that no test is 100% accurate (Swets, 1988). In other words, patients who do not have the disorder may fail to be identified with a test, and patients who do not have the disorder may incorrectly be identified as having a disorder. Finally, we assume that we have *a priori* knowledge of whether the disorder is present or absent in the individual. Given these assumptions, administration of the test to a patient can yield one of four possible outcomes for a single criterion or cutoff:

- True positive (TP): A true positive indicates that a test has correctly identified that the patient is positive

for a disorder. A true positive is also known as a hit. From a clinical standpoint, this is a desirable outcome.

- False positive (FP): A false positive indicates that a test has incorrectly identified a patient without the disorder as positive. A false positive is also known as a miss. From a clinical standpoint, this is an error.
- False negative (FN): A false negative indicates that a test has incorrectly identified a patient with a disorder; that is, the patient has no disorder but tested positive. A false negative is also known as a false alarm. As with a false positive, this is also an error.
- True negative (TN): A true negative indicates that a test has correctly indicated that the patient does not have a disorder; that is, the test yielded a negative result. A true negative is also known as a correct rejection. As with a true positive, this is also a desired outcome.

These four possible test outcomes can be represented in a 2 × 2 grid, or decision matrix, and the results of a sample population can be inserted into each box based on the test results by computing frequency counts. Each box, row, column, and total sample population can be calculated and used with various mathematical formulas associated with CDA. Figure 12–2a illustrates how the 2 × 2 decision matrix is constructed using a Venn diagram. First, we will generally know *a priori* who has the disorder and who does not. Second, we will know the outcome of the test for each patient, which will be either a positive (disorder) or negative (no disorder) result. It can be seen in Figure 12–2a that desirable results will be a test that accurately identifies patients with the disorder (TP or *hit*) or accurately identifies patients who do not have the disorder (TN or *correct rejection*). The two undesirable results will be a test that misdiagnoses patients who do (FN or *miss*) or do not (FP or *false alarm*) have the disorder. Figures 12–2b and 12–2c show examples of tests that would be considered good and poor, respectively. Specifically, Figure 12–2b has high frequency counts in boxes indicating desirable results (TP and TN), whereas Figure 12–2c has high frequency counts in boxes indicating undesirable results (FP and FN).

The two most familiar CDA terms to clinicians are the sensitivity and specificity of a test. These terms, along with the efficiency of a test, are defined as follows, with examples provided in Figures 12–3a and 12–3b using the frequency counts indicated in Figures 12–2b and 12–2c.

- Sensitivity: The sensitivity of a test is indicated by the number of true positives divided by the total number of patients with the disorder. This is the percentage of patients identified correctly (positive result) with the disorder from the group of patients who actually have the disorder. Ideally, sensitivity should be high.
- Specificity: The specificity of a test is indicated by the number of true negatives divided by the total number of patients without the disorder. This is the percentage of patients identified correctly (negative result) without the disorder from the group of patients who do not have the disorder. Ideally, specificity should be high.

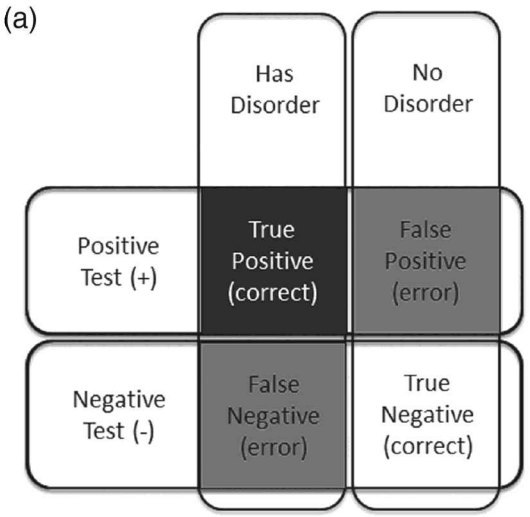
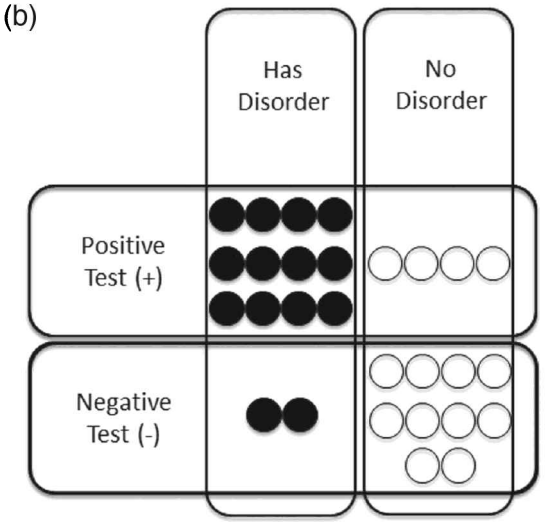


Figure 12–2. A decision matrix for diagnostic tests (a) showing correct and error relationships among true positive (TP), false positive (FP), false negative (FN), and true negative (TN) with respect to patients who do and do not have a disorder and whether the test outcome is positive or negative. Frequency counts of patients with (*black circles*) and without (*white circles*) disorder are placed according to their test results. Good (b) and poor (c) tests are based on frequency counts.

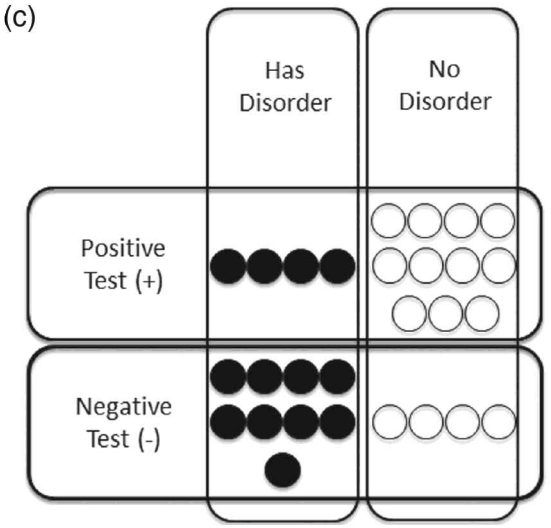


True Positive (TP) = 12

False Positive (FP) = 4

False Negative (FN) = 2

True Negative (TN) = 10



True Positive (TP) = 4

False Positive (FP) = 11

False Negative (FN) = 9

True Negative (TN) = 4

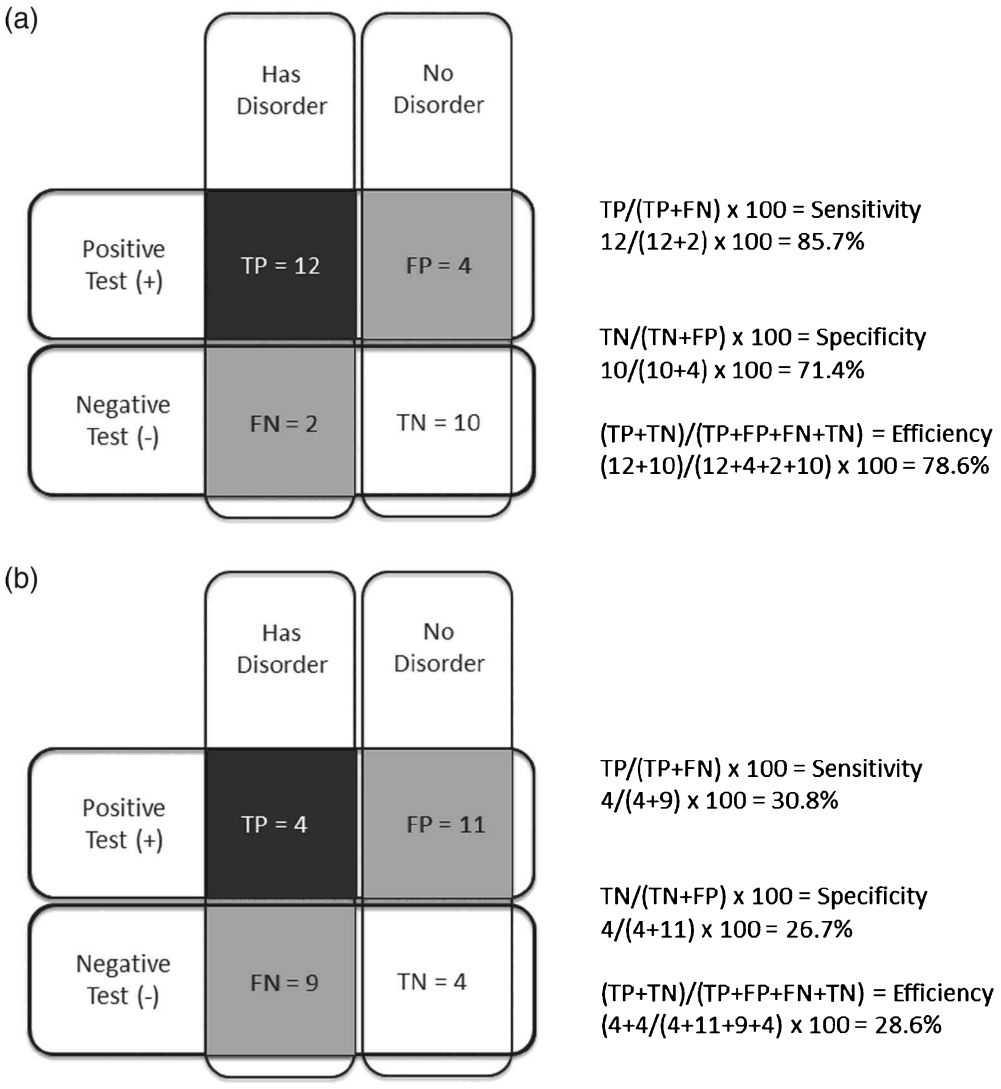


Figure 12–3. Good (a) and poor (b) tests based on the calculation of sensitivity, specificity, and efficiency. Greater frequency counts within the true positive (TP) and true negative (TN) boxes with lesser frequency counts within the false positive (FP) and false negative (FN) boxes should yield a better test.

- Efficiency: The efficiency of a test is indicated by the number of true positives and true negatives divided by the total patients from both groups with and without the disorder. As discussed further below, the prevalence of the disorder being diagnosed will impact the sensitivity, specificity, and efficiency of a test when applied to actual clinical populations. For this reason, an alternative computation for efficiency has been provided by Turner and Nielsen (1984) that takes into

account the prevalence of the disorder. They report efficiency equal to (TP * prevalence) + (1 – FP) * (1 – prevalence). In this chapter, we refer to the efficiency calculation that does not take prevalence into account as "efficiency," and the calculation that incorporates prevalence as "efficiency$_{prev.}$."

There are numerous examples in the audiology research literature in which sensitivity, specificity, and/or efficiency have been calculated to answer a clinical question. We discuss just two here as illustrations of the utility of these indexes. Musiek et al. (1989) examined the ability of the interaural latency difference in wave V of the auditory brainstem response (ABR) to accurately discriminate patients with cochlear hearing loss from those with acoustic neuromas. Both groups had a total of 15 patients each. The authors noted that using an interaural latency difference (ILD) cutoff of >0.4 msec, 14 of 15 patients with confirmed cochlear hearing loss were correctly identified as not having a neuroma, and all 15 patients with acoustic neuromas were correctly identified as disordered. This yielded a sensitivity of 15/15, or 100%, as all disordered patients were identified as disordered. It yielded a specificity of 14/15, or 93%, as almost all patients without an acoustic neuroma were identified as normally functioning. Finally, the measure had an efficiency of 29/30, or 97%, since all but 1 patient were discriminated correctly. This is an extremely favorable result, indicating strong diagnostic utility of the wave V ILD measurement. As a comparison, wave V absolute latency measures for the ABR have sensitivity and specificity that approach only 60% sensitivity and 80% specificity (Musiek & Lee, 1995), much poorer than the diagnostic accuracy of the interaural wave V ILD measurement.

One of the more compelling clinical questions that has been addressed with one or more of these diagnostic indices is how the diagnostic value of the ABR procedure compares with magnetic resonance imaging (MRI) in the detection of acoustic neuromas. Prior to the introduction of hemodynamic methods, ABRs showed considerable favorable sensitivity and specificity to this pathology when compared with other available methods at the time. However, with the introduction of MRI and the level of precision it provides, it is not uncommon for medical professionals to bypass ABR testing in favor of MRI in cases of suspected acoustic neuroma.

To obtain a better understanding of how these two measures compare, Mangham (1991) administered ABRs and MRIs to a large sample of patients with surgically confirmed unilateral acoustic neuromas. In this sample, the sensitivity of the ABR was approximately 84%, indicating that roughly 16% of the tumors were FNs and went undetected. The sensitivity for MRI was much higher at approximately 94% (i.e., ~6% of tumors went undetected). Although initially these findings argued in favor of the MRI, the authors observed that if a combination of both indices were utilized, a similar degree of sensitivity could be obtained at the benefit of improved cost effectiveness. For instance, if an ABR was ordered when the interaural asymmetry in hearing sensitivity was 5 to 20 dB, and an MRI was ordered when the asymmetry was greater than that value, then a 92% sensitivity could be obtained (i.e., ~8% FNs). The cost savings of using ABRs in cases of smaller interaural hearing asymmetries

was almost $25,000 per every tumor diagnosed. This was the case because MRIs were the most costly procedure and by not performing them when interaural asymmetries were less than 20 dB, the total number of these procedures performed was reduced. Thus, one can see how sensitivity calculations can provide extremely practical information in the making of clinical decisions.

Receiver Operating Characteristic Curves, Area Under the Curve, and A′

Since the 2 × 2 grid of test outcomes is computed for only a single test criterion or cutoff, it fails to show how sensitivity and specificity would change as a function of different test criteria. For instance, the sensitivity and specificity of a test using an 80% performance cutoff as normal would be entirely different from the sensitivity and specificity of the test using a 90% cutoff. This being the case, how is it that one can identify the best cutoff to use for a particular test so that sensitivity and specificity are maximized? Although one could in theory devise a 2 × 2 grid with TP, FP, FN, and TN recomputed for every possible criterion, the receiver operating characteristic (ROC) curve provides a more streamlined way to approach this issue.

ROC curves are a common diagnostic index used in CDA calculations. The ROC curve comprises a series of points, each of which represents a different test criterion. The cutoff is plotted relative to 100-specificity on the *x*-axis (i.e., FP rate) and its sensitivity on the *y*-axis (i.e., TP rate). Thus, better specificity values occur toward the left of the *x*-axis and higher sensitivity values occur toward the top of the *y*-axis. In this way, the best criteria are those that yield points in the upper left quadrant of the graph, as it is in this area where sensitivity and specificity are both high.

Figure 12–4 provides an example ROC curve that is adapted from Musiek and Lee (1995). The figure shows two separate ROC curves that reflect the ability of a measure to detect acoustic neuromas. The triangles reflect the sensitivity and specificity of the I–III ABR interpeak latency (IPL) interval, while squares show the same diagnostic index values for the I–V IPL interval. For the I–V ROC curve, each point is labeled indicating the cutoff for that measure that yielded that particular sensitivity and specificity combination. Thus, using a >4.3 msec I–V interval as a cutoff for pathology, sensitivity and specificity of 60% and 90%,

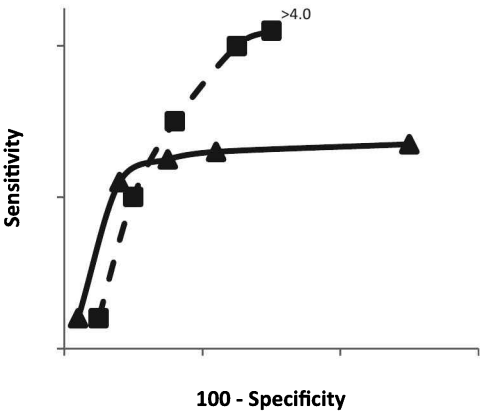


Figure 12–4. ROC curves for the I–III latency difference (triangles) and the I–V latency difference (squares) in the detection of acoustic neuromas (adapted from Musiek & Lee [1995]). Due to the overlap between the two ROC curves, cutoff criteria for a given point along the ROC curve are provided for the I–V measure only in order to maintain figure clarity. The *x*- and *y*-axes are also truncated for this reason.

respectively, are obtained. Similar values are not plotted for the I–III interval in order to maintain figure clarity. The *x*- and *y*-axes are also truncated for this reason. It is evident from the comparison of these two ROC curves, however, that the I–V interval provides the better diagnostic advantage. Both indices appear to achieve maximum efficiency at the points just below 80% specificity, or a 100-specificity value of 20% as plotted on the graph. At this point, the I–V interval shows better sensitivity than the I–III interval, approximately 70% versus 65 % sensitivity, respectively. Thus, using this curve we would conclude that the I–V interval shows a diagnostic advantage over the I–III interval and that a >4.2 msec cutoff for the I–V interval is the criterion that appears to yield the best test efficiency.

An index very closely related to ROC curves is the "area under the curve" measurement. Generally speaking, tests that are more sensitive and specific will have a greater portion of the ROC curve fall into the upper left quadrant of the graph. If one calculates the area under the ROC curve, tests that fall into the upper left quadrant will also tend to have larger values on this area measure. Figure 12–5 provides two hypothetical ROC curves: The solid line represents a measure with very good test efficiency that has most of its points in the upper left quadrant, while the dashed line represents a measure with poor efficiency. It is clear that the area under the ROC curve will be much larger for the test with high efficiency represented by the solid line. Actual computation of the area is mathematically complex and typically performed by statistics software. The interested reader is referred to Metz (1978) for more information on these calculations. Generally speaking, values of this index that are closer to 1.0 denote better area under the curve values and, subsequently, better diagnostic accuracy.

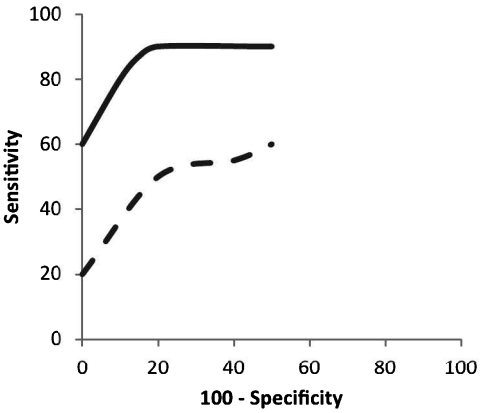


Figure 12–5. Hypothetical ROC curves, where the solid and dotted lines represent tests with good and bad efficiency, respectively. The area under the curve will be much larger for the better test represented by the solid line.

A final index that is associated with ROC curves is A′ (A-prime). Practically speaking, it is an average of all potential maximum and minimum area-under-the-curve values that pass through a given point on the curve (Turner & Nielsen, 1984). Since A′ values are specified relative to individual points along the ROC curve, it does not reflect on information from multiple criteria or cutoffs. The equation for A′ is provided in Table 12–1. The index can range from 0.5 to 1.0, with values closer to 1.0 indicating better diagnostic accuracy of the test. The A′ index does not incorporate the prevalence of the disorder in the general population. Where sensitivity and specificity are high for a given criteria, A′ tends to be high as well.

Table 12–1. A′ Calculation

$$A' = .5 + \frac{(TP - FP) * (1 + TP - FP)}{4 * TP * (1 - FP)}$$

Results reported in Turner & Nielsen (1984).

d′ (d-prime)

As Figure 12–1 depicts, the variability in the distribution of scores for disordered and normally functioning patients can range from small to large values. The greater this variation, the greater the likelihood that the performance distribution for the two groups will overlap. A test that cannot differentiate between a disorder being present or absent probably has performance distribution curves that overlap considerably for the two groups. One way in which this overlap can be quantified is with the d′ (d-prime) index. If the disordered and normal functioning distributions are Gaussian distributed (i.e., bell-shaped) and have equal standard deviations, then d′ is calculated as the difference in means between the distributions divided by the standard deviation of the two groups (Turner & Nielsen, 1984). Higher d′ values are associated with distributions that have a larger mean difference and smaller standard deviation (i.e., the within-group variability is small and the distributions overlap less). Smaller d′ values occur when the two distributions show a small mean difference and have large standard deviations (i.e., the within group variability is large and distributions overlap more). Although it is idealized that human variability tends to conform to a Gaussian performance distribution curve, clinical data often diverge from this idealized distribution (Turner & Nielsen, 1984). In cases where distributions are not Gaussian distributed and have unequal standard deviations, d′ can be calculated from the TP and FP values if they are known. Although it is beyond the scope of this chapter to provide an exhaustive table of all possible d′ values for given TP and FP values, Table 12–2 provides some example d′ values. A more exhaustive table can be found in Appendix A of Swets (1964). When comparing tests, the one that shows the greater d′ value may be considered the better diagnostic test (Turner & Nielsen, 1984). Note that d′ does not incorporate the prevalence of the disorder in the general population, and as such is driven by TP and FP values.

Posterior Probabilities

Both sensitivity and specificity calculate the diagnostic accuracy of a test without taking into consideration the relative prevalence of the disorder. In reality, the probability of obtaining a TP or FP is also dictated by the prevalence of the disorder in the general population. If prevalence values are high, there is a greater probability that any individual selected from the population has the disorder, and a positive test result tends to be correct more often. Conversely, if prevalence is low, there is a greater probability that selected individuals have normal function, and a negative test result tends to be correct more often. Put another way, sensitivity is enhanced when the prevalence of the disorder in the population is high, while specificity is enhanced when the prevalence of the disorder is low.

Calculations of posterior probabilities combine prevalence information with TP

and FP values. Two indices are used in this capacity, D+ and N–, and their equations are included in Table 12–3. Both reflect the probability of an outcome given the known prevalence of the disorder in the population. D+ reflects the probability that the patient has a disorder when a positive result is obtained, whereas, N– indicates the probability that a patient does not have a disorder when a negative result is obtained.

The relationship between both D+ and N– and prevalence is detailed in Figure 12–6. In this example, for a test with 90% sensitivity and specificity, the probability of identifying a TP is greatest for 80% prevalence. However, since so few people actually have normal func-

Table 12–2. Example d' Values for Given True and False Positive Rates

	TP	.1	.2	.3	.4	.5	.6	.7	.8	.9	.99
FP											
.1		0	.44	.76	1.02	1.28	1.54	1.80	2.12	2.56	3.60
.2		–.44	0	.32	.58	.84	1.10	1.36	1.68	2.12	3.16
.3		–.76	–.32	0	.27	.52	.78	1.05	1.36	1.80	2.84
.4		–1.02	–.58	–.27	0	.26	.51	.78	1.10	1.54	2.58
.5		–1.28	–.84	–.52	–.26	0	.26	.52	.84	1.28	2.32
.6		–1.54	–1.10	–.78	–.51	–.26	0	.27	.58	1.02	2.06
.7		–1.80	–1.36	–1.05	–.78	–.52	–.27	0	.32	.76	1.80
.8		–2.12	–1.68	–1.36	–1.10	–.84	–.58	–.32	0	.44	1.48
.9		–2.56	–2.12	–1.80	–1.54	–1.28	–1.02	–.76	–.44	0	1.04
.99		–3.60	–3.16	–2.84	–2.58	–2.32	–2.06	–1.08	–1.48	–1.04	0

Results reported in Swets (1964).

Table 12–3. Posterior Probabilities

$$D+ = \frac{1}{1+ ((FP) * [1 - \text{prevalence}]) / (TP * \text{prevalence})}$$

$$N- = \frac{1}{1 + ([1 - TP] * [\text{prevalence}]) / ([1 - FP] * [1 - \text{prevalence}])}$$

Multiply by 100 to convert to a percentage.

Results reported in Turner & Nielsen (1984).

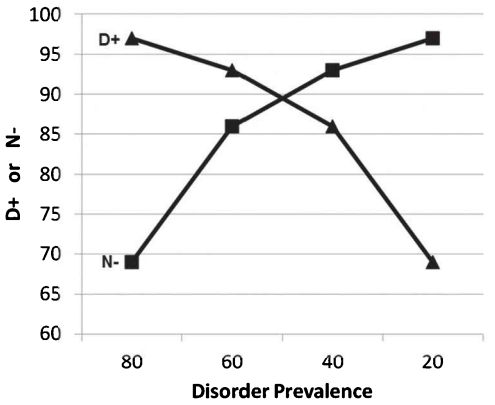


Figure 12–6. D+ (*triangles*) and N– (*squares*) values for a given prevalence. As the prevalence of a disorder decreases, N– increases, while D+ becomes poorer.

tioning in this population, the probability of identifying a TN at this prevalence is much lower. The relationship is reversed at 20% prevalence, as now more people in the population have normal than abnormal function.

[redacted]actical Application

Performing CDA

In this section we describe the minimal steps that need to be taken to conduct a CDA for clinical purposes. Prior to performing the analysis, it needs to be decided which two groups you are trying to discriminate using the clinical test. Generally, the two groups are individuals with an auditory disorder and individuals with normal auditory function. For the purposes of our example, we will decide to perform CDA on a new screening test that has been developed to detect acoustic neuromas. Our two groups will be individuals with known acoustic neuromas and individuals with normal auditory function who do not have acoustic neuromas. When the purpose of the test has been decided, one next needs to obtain access to both disordered and normally functioning individuals. As previously mentioned, the assignment of individuals to the disordered group requires *a priori* knowledge of whether the individual has the disorder or not. In our example, as we are interested in screening for acoustic neuromas, we would need to confirm that individuals in our disordered group actually have the disorder. Typically, this would be accomplished by administering a gold standard test for detection of the disorder, such as MRI.

Once we have recruited and assigned individuals to their respective groups, the screening test that we have developed would be administered. We would administer the test to all subjects and then collect the data for analysis. We include our illustrative data from this fictional test in Table 12–4. Our first step in analysis would be to construct an ROC curve as indicated earlier, with our criteria or cutoffs being percent correct performance. The construction of an ROC curve is typically done via computer software, but can also be calculated by hand. For our example, we are interested in only five criteria: the ability of the test to discriminate groups when a cutoff of >50% is considered normal, and when the cutoff (criterion) is >60%, >70%, >80%, or >90%. Calculating sensitivity for each individual point in our dataset gives us the ROC curve in Figure 12–7. Clearly, using the 50% or 90% criteria is not satisfactory, as sensitivity is near zero in the former case and specificity is equally poor in the latter.

Determining which of the remaining three points provides the best balance

Table 12–4. Scores for the Disordered and Normal Functioning Group From a Fictional Diagnostic Test of Acoustic Neuromas

Disorder Group Scores	Normal Group Scores
40%	45%
51%	55%
51%	55%
51%	65%
51%	71%
51%	71%
61%	85%
61%	85%
71%	85%
95%	95%

Each line represents a different patient in the group, and the scores are ranked from highest to lowest within the group.

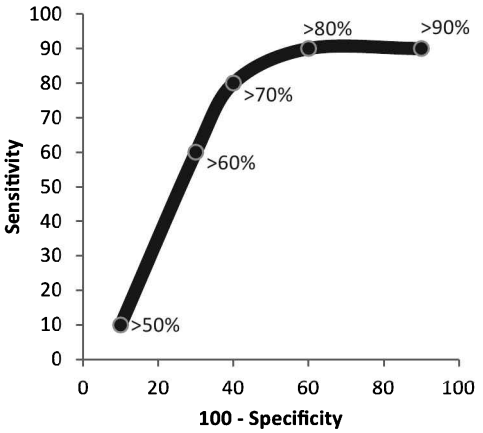


Figure 12–7. Hypothetical ROC curve, where best group discrimination is obtained at the >70% criterion.

of sensitivity and specificity can be accomplished by calculating efficiency at each of these values. In our hypothetical example, using our known sensitivity and specificity values at each of these points, efficiency for >60%, 70%, and 80% are 65%, 70%, and 65%, respectively. Thus, calculating efficiency for the remaining points reveals that the >70% cutoff actually gives the best combination of sensitivity and specificity with 70% efficiency. If we so desired, we could then go on to examine the effect of prevalence on the diagnostic accuracy of the test. Assuming that the prevalence of acoustic neuromas in individuals with asymmetric hearing has the very low prevalence of 1 in 1000 (0.001%), then the D+ and N– values for this test at the 70% efficiency level (i.e., 8/10, or .8 TP; 4/10, or .4 FP) are approximately .2% and 99%, respectively. Thus, given the very low prevalence, there is a *much* greater probability that a negative result reflects normal function than a positive result reflects abnormal function.

Collective Consideration of Multiple Indices

Examination of the combined results of test sensitivity, specificity, efficiency, A′, d′, and posterior probabilities can assist in identifying each test's diagnostic value. In a literature review of 175 papers that addressed diagnosis of acoustic neuromas, Turner et al. (1984) calculated these indices for a wide range of audiologic tests. Some of their results are presented in Table 12–5, where tests are ranked by their efficiency$_{prev}$. Note that efficiency was calculated by the authors using the second efficiency equation described in this chapter. This equation incorporates prevalence of the disorder into the efficiency estimate and, therefore, its value is not solely dependent on sensitivity and specificity.

Table 12–5. The Relationship Between Posterior Probabilities, d′, A′, and Test Efficiency

Test	Sensitivity	Specificity	$Efficiency_{prev}$	D+	d′	A′
Rollover	74%	96%	95%	48%	2.4	.92
Acoustic reflex decay	63%	96%	95%	47%	2.1	.89
ABR	95%	89%	89%	31%	2.9	.96
Acoustic reflex threshold	73%	90%	89%	28%	1.9	.89
ABLB	59%	90%	88%	23%	1.5	.84
Tone decay	70%	87%	86%	22%	1.6	.87
SISI	65%	84%	83%	17%	1.4	.83

ABR, auditory brainstem response; *ABLB*, alternate binaural loudness balance; *SISI*, short increment sensitivity index

Results reported in Turner et al. (1984).

Depending on which index is being considered and the desired goal of test administration, several different trends are observed. If the clinician is attempting to achieve the highest TP rate in detecting acoustic neuromas, the ABR clearly yields the best sensitivity. Further, measures that incorporate sensitivity but not prevalence, such as d′ and A′, also indicate that the ABR provides the best diagnostic accuracy in this regard. Conversely, if the clinician is attempting to achieve a good balance of sensitivity and specificity while also considering the prevalence of a disorder, then rollover on word discrimination tasks and acoustic reflex measures provide a very slight advantage over the ABR when $efficiency_{prev}$ is considered. This slight advantage occurs because the relative prevalence of acoustic neuromas in the population is low and both rollover and acoustic reflex show slightly better specificity than ABR. Recall that specificity tends to be enhanced when the prevalence of the disorder is low, as is the case here.

For this particular analysis, results for test $efficiency_{prev}$ and D+ were almost perfectly correlated (98% shared variance). This is attributed to the fact that both indices utilize sensitivity, specificity, and prevalence values. If efficiency were calculated without taking prevalence into account, then the relationship between efficiency and D+ would be smaller. In the current example, no new information is gained from D+, as its trends are nearly wholly represented by $efficiency_{prev}$. Thus, clinical decisions based primarily on $efficiency_{prev}$ would be identical to those made on D+.

To summarize, a collective consideration of these indices indicates that the ABR is better at detecting neuromas than the other measures presented in this

table when prevalence is not taken into account. If one aims to optimize sensitivity, then clearly the ABR is the test to be selected from this battery. However, as the occurrence of acoustic neuromas in the general population is infrequent, the probability that a positive ABR finding indicates pathology in any individual patient is relatively low. When the low prevalence of acoustic neuromas is considered, rollover and acoustic reflex decay show a small advantage in efficiency$_{prev}$ rates, which is a consequence of these measures having a slightly better specificity than the ABR. In this case, this information can assist audiologists in both reaching their clinical decision and counseling patients. For example, in clinical practice, use of the ABR is desirable because of its enhanced sensitivity, but it is equally helpful to let patients know during counseling that the probability that they actually have the disorder when an ABR is positive is extremely low (<1%). It is combined information of sensitivity and posterior probabilities that allows the clinician to achieve both of these aims.

Cost Effectiveness

It is often practical to consider not only the diagnostic accuracy of a test but its cost effectiveness as well. The most cost effective test would be one that is less costly to administer, shows high sensitivity and specificity to a disorder, and correctly detects a disorder that has a high prevalence. In addition to indices discussed thus far, this provides another metric on which the value of two tests can be compared. That is, it is possible for two tests to have drastically different cost effectiveness values yet be similar in all the other regards (e.g., similar sensitivity, specificity).

Cost effectiveness computations have been approached in several different ways. Hurley and Musiek (1997) reported one in which the cost per each TP diagnosis is equal to (cost per test * number of tests) / (sensitivity * prevalence) x 100. Hurley and Musiek (1997) used this equation to determine the cost effectiveness of a behavioral CAPD test (i.e., duration patterns) and an electrophysiological test (i.e., auditory P300). The behavioral test was noted to have better sensitivity than the electrophysiological measure. In computing the cost effectiveness for each test, the cost was always estimated to be $40 for a single behavioral test and $200 for a single electrophysiological measure. The prevalence of CAPD was considered to be 5% regardless of the test being considered. The cost effectiveness analysis revealed that the cost per TP was much lower for the behavioral measure of CAPD than for the electrophysiological measure, $9.64 versus $70.18. This outcome was observed in large part because of the better sensitivity and lower cost of the behavioral measure.

Gorga and Neely (2003) reported an alternative cost effectiveness calculation that takes into account the different costs that accurate or inaccurate diagnoses place on the health care system. Additionally, it utilizes specificity information and examines the question as cost per patient instead of cost per TP. Their equation, outlined in Table 12–6, was used by Gorga and Neely to determine the cost effectiveness of universal newborn screening protocols. They estimate the cost per TN and FN to be $25, which is the clinical cost of administering the test, and the cost per FP to be $500, which includes the cost of the initial test along with the cost of all unnecessary follow-up diagnostic tests. They estimated the cost per TP at −$1,000,000, since this

Table 12–6. Cost Effectiveness

Cost per patient = (CTN * PTN) * (CTP * PTP) * (CFN * PFN) * (CFP * PFP)	
CTN	Cost per true negative
PTN	Probability of a true negative = (1 – prevalence) * specificity
CTP	Cost per true positive
PTP	Probability of a true positive = prevalence * sensitivity
CFN	Cost per false negative
PFN	Probability of a false negative = (1 – prevalence) * (1 – specificity)
CFP	Cost per false positive
PFP	Probability of a false positive = prevalence * (1 – sensitivity)

Results reported in Gorga & Neely (2003).

is the savings accrued by successfully identifying the hearing loss; untreated hearing loss would be expected to have a significant financial impact on the health care system, and diagnosing it early prevents these costs from occurring. Using these cost values and known sensitivity, specificity, and prevalence values, the cost effectiveness of the protocol was estimated to be equal to ($25 * .94715) + (–$1,000,000 * .00180) + ($525 * .04985) + ($25 * .00120), or roughly = –$1,750. The authors noted that the negative value indicates that application of the test yields long-term benefits that exceed immediate costs.

[redacted]e of CDA in Central Auditory Testing

Relevance of CDA in Central Auditory Testing

CDA is used in CAPD research as a means to validate CAPD tests. The logic behind this approach is that if central auditory tests are a measure of central auditory nervous system (CANS) function, then establishing the sensitivity and specificity of a central auditory test in subjects with known neurological lesions of the CANS gives us some information about whether the test is measuring this system. Note that this approach does not imply that all individuals with CANS dysfunction have neurological involvement. It merely shows that a central auditory test is sensitive to CANS dysfunction. This method for validation on patients with CANS dysfunction is considered the best currently available to the field, and is also considered the gold standard for establishing a diagnostic central auditory test (AAA, 2010). It should be noted, however, that this is not the only form of validation. There are additional ways in which CANS involvement could be associated with central auditory tests. Hemodynamic and structural imaging provides a noninvasive means by which CANS function can be correlated with central auditory test performance. Additionally, transcranial magnetic stimulation could temporarily disrupt CANS processing and, if

decrements are seen on central auditory tests during stimulation, this may also be construed as validation. Thus, the neurological model, while useful, may be surpassed by alternative methods in the future. Regardless of which method is considered the gold standard, it is important to utilize rigorous validation in the establishment of central auditory tests.

One criticism of the neurological approach to validation of central auditory tests is that CANS lesions are heterogeneous in their etiology, size, and location. There is natural variance to CANS lesions across subjects; even on an individual basis, the lesion may affect the CANS differently over time. This means that two studies using similar central auditory tests could reach different conclusions because of the variability in lesion site from sample to sample. For example, Musiek et al. (2011) observed sensitivity and specificity of 90% and 83%, respectively, for the dichotic digits test in detection of CANS dysfunction. Other studies have noted a much lower sensitivity (~75% to 80%) and slightly improved specificity (90%) for the identical test and criterion (Musiek, 1983a; Musiek et al., 1991; Hurley & Musiek, 1997). For this reason, the neurological model is perhaps at its best when examining the sensitivity and specificity of central auditory tests administered within the same sample. This relative comparison of tests tends to yield more consistent results across studies. For instance, Musiek et al. (2011) showed that dichotic digits and frequency patterns had the best test sensitivity, that competing sentences showed slightly less sensitivity than either of these measures, and that filtered speech showed the worst test efficiency, performing at chance levels. This is consistent with other studies that have examined multiple central auditory tests in their samples. For instance, Musiek (1983b) noted that the Dichotic Digits Test was more sensitive than competing sentences. Therefore, the actual sensitivity and specificity values for any given test may not be as important as is consideration of which central auditory measures rank most highly on CDA indices when the tests are administered in a test battery to a sample of patients with confirmed CANS involvement.

Clinical Decision Analysis in Central Auditory Test Batteries

When testing for CAPD, a battery of clinical measures is typically administered during the clinical evaluation. The logic behind the battery approach is that CAPD can arise from deficits in more than one auditory process or insult to more than one region of the CANS. For this reason, it becomes more likely that pathology will be detected by the inclusion of several tests in the clinical evaluation. The CDA indexes described above (e.g., sensitivity, specificity, efficiency) can also be calculated when considered as part of a larger test battery. The question changes in this situation from: What is the diagnostic accuracy of a single test? to: What is the diagnostic accuracy of the battery as a whole? Regardless of the shift in focus away from individual tests to test batteries, the goals of CDA are the same: to determine under what conditions maximum sensitivity and specificity are achieved. We consider here some of the choices that must be made when calculating diagnostic accuracy for test batteries, as well as highlight the role that test batteries play in the diagnosis of CAPD.

Diagnostic accuracy of a test battery can be approached in one of two ways. The first approach asks the question: What is the sensitivity and specificity of the test battery when the patient must fail all of the tests for a positive diagnosis? This has been termed the "strict" approach because it utilizes the most stringent test battery criterion (Bellis, 2002). Alternatively, the second approach asks the question: What is the sensitivity and specificity of the test battery when the patient must fail only some of the tests in a battery for a positive result? This has been termed the "lax" criterion, since the requirements for failure are less stringent. Thus, if we administer a three test battery, employing the strict criterion would require that a patient fail all three of the tests for a positive result, while employing the lax criterion would require that the patient fail only one or two tests, depending on the specific lax criterion adopted.

Employing a strict or lax criterion will have different effects on the sensitivity and specificity of a test battery. The use of a strict criterion will improve the specificity of a test battery, as it is less likely that an individual without the disorder will fail all of the tests in the battery. However, this criterion will simultaneously decrease sensitivity, as it is also less likely that an individual with the disorder will fail all tests in the battery. Conversely, the lax criterion yields the opposite trends. In regard to sensitivity, administering multiple tests and then requiring that patients need fail only a subset of those tests (e.g., as few as one) will increase the sensitivity, as every additional test represents an additional chance to diagnose the disorder in those who have it. Specificity, however, will become poorer with the lax criterion, as individuals without the disorder will also have an additional opportunity to be incorrectly diagnosed as having a deficit. An additional factor that will influence the sensitivity and specificity of a test battery is the number of tests included. For a strict criterion, increasing the number of tests in the battery will improve the specificity and decrease the sensitivity further. For the lax criterion, the situation is reversed, with the addition of tests to the battery increasing the sensitivity while decreasing the specificity further.

A recent example of CDA applied to central auditory test batteries is found in Musiek et al. (2011). The authors examined the efficiency of various central auditory test batteries as a function of: (1) the number of tests patients were required to fail for a positive diagnosis, (2) the specific tests included in the battery, (3) which criterion, strict or lax, was adopted, and (4) whether normative values for single tests used less than two standard deviations or three standard deviations as the criterion differentiating normal from abnormal function. The tests administered included the dichotic digits, competing sentences, frequency patterns, and low-pass filtered speech tests. Pathological subjects were those with lesions located in the CANS, and normal controls were those with no known neurological involvement. In regard to the number of tests patients were required to fail for a positive diagnosis, using failure of two and three tests yielded much better battery efficiency (~90%) then one or four test failures (~75%). This suggests that in administering central auditory test batteries, subjects should be required to fail at least two tests for a positive diagnosis. In regard to which tests should be included in a battery, it was found that incorporating the frequency patterns test with

either the dichotic digits test or the competing sentences test most often yielded the best efficiency. Adding the low-pass filtered speech test in any battery greatly decreased the efficiency of the battery. In regard to strict versus lax criterion, the greatest efficiency values tended to be encountered when the lax criterion was used, although there was considerable overlap between the two criteria depending on which tests were used and how many were incorporated into the battery. Finally, it was determined that using three standard deviations as the cutoff criterion instead of two standard deviations slightly improved efficiency, as was seen for all measures, except competing sentences. The average improvement in efficiency for the other tests was approximately 3%.

Based on these findings, Musiek et al. (2011) concluded that a two- or three-test battery that utilized a lax criterion and included both the frequency patterns along with the dichotic digits or competing sentence test yielded the best diagnostic efficiency. If administering the frequency patterns and competing sentence tests using the lax criterion, the efficiency of the battery surpassed the efficiency obtained by any one test alone. This indicates there is value to using the test battery approach in CAPD diagnosis. Finally, if the patient performed poorly on only a single test in the battery, it was better to use a three standard deviation criterion instead of the two standard deviation cutoff criterion because the former cutoff yielded slightly improved test efficiency.

Summary

This chapter has described calculations related to CDA and how they have been used in the field of audiology to answer important clinical questions. The role of CDA in CAPD diagnosis also was considered, with particular emphasis on how CDA can be applied to test batteries. Although CDA utilizing neurological patients may be an imperfect gold standard for CAPD diagnosis, it currently remains the best option available for validation of these tests (AAA, 2010). The data-driven approach to making clinical decisions presented in this chapter provides an important tool for evidence-based practice regardless of the type of audiology test or pathology under consideration.

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SECTION 3

Evaluation of Central Auditory Processes

CHAPTER 13

MONAURAL LOW-REDUNDANCY SPEECH TESTS

SRIDHAR KRISHNAMURTI

Introduction

Monaural low-redundancy speech tests are among the oldest tests used to assess the central auditory nervous system (CANS) (Bocca, Calearo, & Cassinari, 1954; Bocca, Calearo, Cassinari, & Migliavacca, 1955). These tests are administered monaurally using stimuli that have been degraded electroacoustically or digitally in the frequency/spectral, temporal, or intensity domain. Because the stimuli are degraded, the inherent redundancy of the signal is reduced and the tests are considered "sensitized" to detection of CANS pathology.

Redundancy facilitates auditory processing. Extrinsic redundancy is a characteristic of the speech signal itself. Extrinsic redundancy arises from multiple and overlapping acoustic (i.e., frequency, intensity, temporal) and linguistic cues inherent to speech and language (i.e., phonemic cues, prosodic cues, morphological cues, syntactic cues, and semantic cues) (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Sanders & Goodrich, 1971). Intrinsic redundancy is due to the structure and physiology of the auditory pathways whereby multiple and parallel pathways concurrently and sequentially transmit information across the CANS (Hall & Mueller, 1997).

The degree of redundancy associated with speech stimuli can exert significant effects on the performance of listeners in intelligibility tasks. Low-redundancy speech materials (e.g., nonsense syllables) are far less intelligible than high-redundancy speech materials (e.g., sentences). Miller, Heise, and Lichten (1951)

studied speech intelligibility in normally hearing listeners for words in sentences, digits 0 to 9, and nonsense syllables. Digits (a small, closed stimulus set) reached 100% intelligibility at signal-to-noise ratios (SNRs) of −10 dB, while listeners required an SNR of 18 dB to achieve the same performance level for words in sentences. In contrast, listeners attained a maximum of only 70% intelligibility for low-redundancy nonsense syllables at the highest SNR of 18 dB.

Four behavioral domains have emerged from recent research employing the statistical approach of factor analysis to tests of central auditory processing: auditory pattern/temporal ordering (APTO); monaural separation/closure (MSC); binaural integration/binaural separation (BIBS); and binaural interaction (BI) (Domitz & Schow, 2000, Schow & Chermak, 1999). Monaural low-redundancy speech tests factor within the MSC category. Several factor analyses have demonstrated that tests involving auditory closure on the part of the listener and presumed to evaluate monaural auditory performance decrements due to degradation or competition load on the MSC factor (Domitz & Schow, 2000; Schow & Chermak, 1999). Tests of monaural competition and degradation included in these analyses were the Selective Auditory Attention Test (SAAT; Cherry, 1980) and the Auditory Figure-Ground and Filtered Word subtests of the Screening Test for Auditory Processing Disorders or SCAN (Keith, 1986). Findings from another factor analysis of the SCAN-C based on data compiled from 99 children, ages 6 to 10 years, showed that two of the SCAN-C subtests (Filtered Words and Auditory Figure-Ground) loaded on a "monaural low-redundancy/degradation" factor, while two other subtests (Competing Words and Competing Sentences) loaded on a "binaural separation/competition" factor (Dawes & Bishop, 2007).

Interaction Between Extrinsic Redundancy and Intrinsic Redundancy

Extrinsic redundancy has been most often degraded or reduced by: (a) altering the frequency or spectral aspects of speech (e.g., low-pass filtered speech tests), (b) adding background noise or speech to introduce competition (e.g., speech-in-noise tests), and (c) altering the temporal aspects of speech (e.g., time-compressed speech tests). Less commonly, intensity alterations (e.g., low sensation level speech tests) or temporal alterations other than compression (e.g., interrupted speech) have been examined. Reduced intrinsic redundancy typically reflects a dysfunction in the CANS.

As seen in the first panel of Figure 13–1, when a listener with normal intrinsic redundancy (normal CANS) listens to unaltered speech with normal extrinsic redundancy, normal speech recognition performance is expected. Even when speech is degraded (i.e., extrinsic redundancy is reduced by filtering, adding competition, or altering temporal aspects), listeners with normal intrinsic redundancy can fill in the missing information by means of their auditory closure skills and achieve normal speech recognition performance (see panel 2 of Figure 13–1). When a listener with reduced intrinsic redundancy (CANS dysfunction) listens to unaltered speech with normal extrinsic redundancy, normal speech recognition performance is still expected. However, as shown in

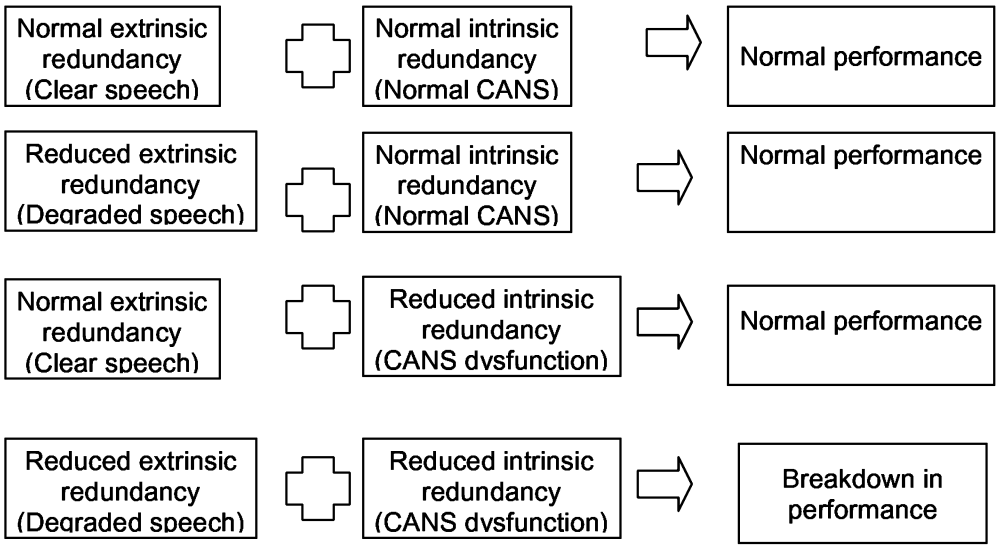

Figure 13–1. Interaction between extrinsic and intrinsic redundancy in outcome of monaural low-redundancy tests.

panel 3 of Figure 13–1, when speech is degraded (i.e., extrinsic redundancy is reduced), listeners with reduced intrinsic redundancy (due to CANS dysfunction) show a significant deficit in speech recognition performance (see panel 4 of Figure 13–1).

Not depicted in Figure 13–1 are the effects of peripheral hearing loss, which, like CANS dysfunction, also reduce the intrinsic redundancy of the auditory system. The presence of peripheral hearing loss reduces the ability of the auditory system to resolve spectral detail. Hence, central auditory tests employing stimuli placing significant demands on cochlear processing to resolve frequency and intensity transitions of the acoustic signal (e.g., consonant-vowel syllables, words) are subject to the potential confound of peripheral hearing loss (Chermak & Musiek, 1997). Since all monaural low-redundancy tests are heavily dependent on resolution of rapid frequency-intensity interactions, the clinician should balance the value of these tests in the central auditory diagnostic battery against their potential to confound results. Results should be interpreted with caution when used as part of a central auditory test battery with individuals with known hearing loss (Baran & Musiek, 1999). Peripheral hearing loss can significantly influence performance on monaural low-redundancy speech tests because of the dependence of these tasks on basic auditory skills, such as frequency and intensity discrimination, speech recognition, and auditory closure. Individuals with peripheral hearing loss experience auditory discrimination deficits caused by the degradation of the signal by the internal filtering caused by the hearing loss and/or internal noise or distortion, which can reduce phonological contrasts (Elliott & Hammer, 1988; Elliott, Hammer, & Scholl, 1989). Poor performance on filtered speech tests due to auditory closure

deficits have been reported in individuals with mild sensorineural hearing loss (Neijenhuis, Tschur, & Snik, 2004).

Conceptual Framework for Use of Monaural Low-Redundancy Speech Tests

The contribution of factors related to speech intelligibility skills are shown in a pyramidal format in Figure 13–2. Peripheral hearing acuity is at the base of the pyramid, indicating its basic and key role prior to undertaking any evaluation of central auditory processing. Most individuals with central auditory processing disorder (CAPD) show normal hearing sensitivity on pure-tone audiometric testing, and even excellent speech intelligibility scores on word recognition testing in quiet. This confirms that the listener's basic auditory ability to handle acoustic cues (frequency, intensity, and timing) is intact. In those individuals with peripheral hearing loss, spectral filtering and temporal resolution deficits imposed by the sensorineural hearing loss can significantly influence speech intelligibility on monaural low-redundancy speech tests. Nonetheless, as explained above, central auditory dysfunction is expected to reveal itself during audiological testing only when the CANS of limited intrinsic redundancy interacts with the (acoustically degraded) speech stimuli (e.g., filtered speech, speech-in-noise, time-compressed speech). Tests of peripheral auditory function typically do not test higher level central auditory processing; therefore, we must ascend the pyramid (i.e., assessment of central auditory function). It is valuable to recognize that inclusion of degraded speech tests (e.g., filtered speech and speech-in-noise) that assess auditory closure can result in improvements in the sensitivity of test batteries used to identify CAPD in children, albeit at the expense of the test battery's specificity (Dawes & Bishop, 2009).

The degradation of information in the CANS requires the approach shown in Figure 13– 2. At the apex of the pyramid, we find cognitive factors. Although CAPD

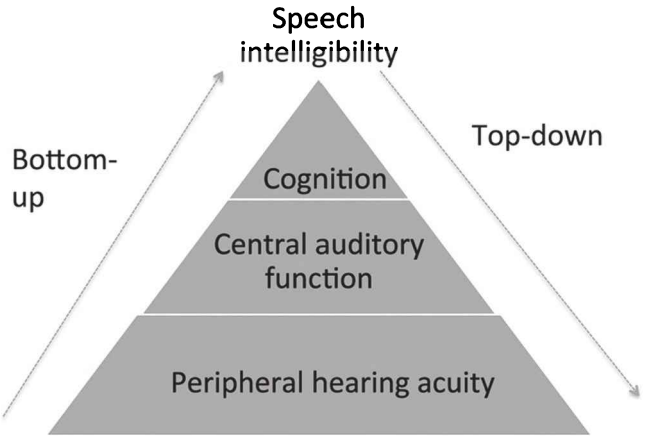

Figure 13–2. Interaction of peripheral and central auditory function variables with cognition in determination of speech intelligibility outcomes.

is not a cognitive disorder, if there is cognitive decline, as seen in elderly listeners (e.g., with Alzheimer's disease), performance on monaural low-redundancy speech tests can significantly decline, relative to controls who show no cognitive decline (Krishnamurti, Snell, King, & Drake, 2013). In this study, performance on measures of auditory closure (filtered words subtest of SCAN-A) and speech-in-noise (auditory figure-ground subtest of SCAN-A) declined significantly in the left ear only for older individuals with cognitive decline relative to a control group with no cognitive decline. The ear effect reflects the more direct connection of the right ear with the presumed language-dominant left hemisphere. This performance outcome also indicates limitations in information processing resources in the central nervous system for listeners with cognitive decline, thereby making it difficult for them to compensate for the spectral and temporal degradation imposed by monaural low-redundancy speech tests. See Chapter 18 in this volume and Chapter 15 in Volume 2 of the Handbook for discussion of CAPD in elderly populations.

History of Monaural Low-Redundancy Speech Tests

Monaural low-redundancy speech tests (and central auditory tests in general) can be traced back to the pioneering work of Italian investigators (Bocca et al., 1955) in the 1950s. They were the first to recognize that patients with temporal lobe lesions had normal pure-tone audiograms and speech recognition in quiet, but still complained of qualitative reduction of speech quality. Upon investigation with filtered speech tests, Bocca et al. (1955; Bocca, 1958) found depressed scores in patients' ears contralateral to the affected hemisphere. Subsequently, speech-in-noise tests were introduced to detection of lesions in the CANS. Abnormal scores on speech-in-noise tests were reported for patients with temporal lobe lesions (Sinha, 1959) and for patients with eighth nerve tumors and multiple sclerosis (Olsen, Noffsinger, & Kurdziel, 1975). Time-compressed speech tests also have received attention beginning with the normative studies by Beasley and his colleagues (Beasley, Schwimmer, & Rintelmann 1972). Normal listeners showed essentially normal performance for recognition of monosyllabic NU-6 words when lower time compression (30%–60%) was applied; however, their performance broke down when a greater proportion of temporal segments were removed (i.e., 70% time compression). Kurdzeil, Noffsinger, and Olsen (1976) showed that patients with diffuse temporal lobe lesions exhibited poorer performance on time-compressed speech tests in the ear contralateral to the lesion at 40% and 60% time compression. Auditory discrimination deficits have been reported for speech stimuli in noisy backgrounds in children with language learning problems and learning disabilities (Ferre & Wilber, 1986).

Classification of Monaural Low-Redundancy Speech Tests

Monaural low-redundancy speech tests continue to be among the most widely used tests to evaluate central auditory function (Musiek, 1999). They con-

tinue to be used, despite only moderate sensitivity to CANS lesions (Musiek & Baran, 2002), primarily because they have been incorporated into several popular clinical test batteries (Keith, 1986, 1994, 2000, 2009a, b; Willeford, 1977). These tests are easy to administer, score, and interpret. They also provide insight as to functional deficits (i.e., listening in noise and auditory closure problems) and therefore offer practical information for intervention (Bellis, 2003; Bellis & Ferre, 1999).

Commercially available monaural low-redundancy speech tests currently used can be classified as: (1) low-pass filtered speech tests, (b) speech-in-noise tests, (c) speech-in-message competition tests, and (d) time-compressed speech tests. A classification tree for monaural low-redundancy speech tests is shown in Figure 13–3. As shown in this classification, speech is degraded in the spectral domain by low-pass filtering (i.e., low-pass filtered speech), or by adding background noise (i.e., speech-in-noise), while temporal degradation is achieved by artificial time-compression applied electronically (i.e., time-compressed speech). These tests have moderate sensitivity for brainstem and cortical dysfunction (as outlined in following sections).

Low-Pass Filtered Speech Tests

The use of low-pass filtered speech (LPFS) can be traced historically to research conducted in neurological patients by

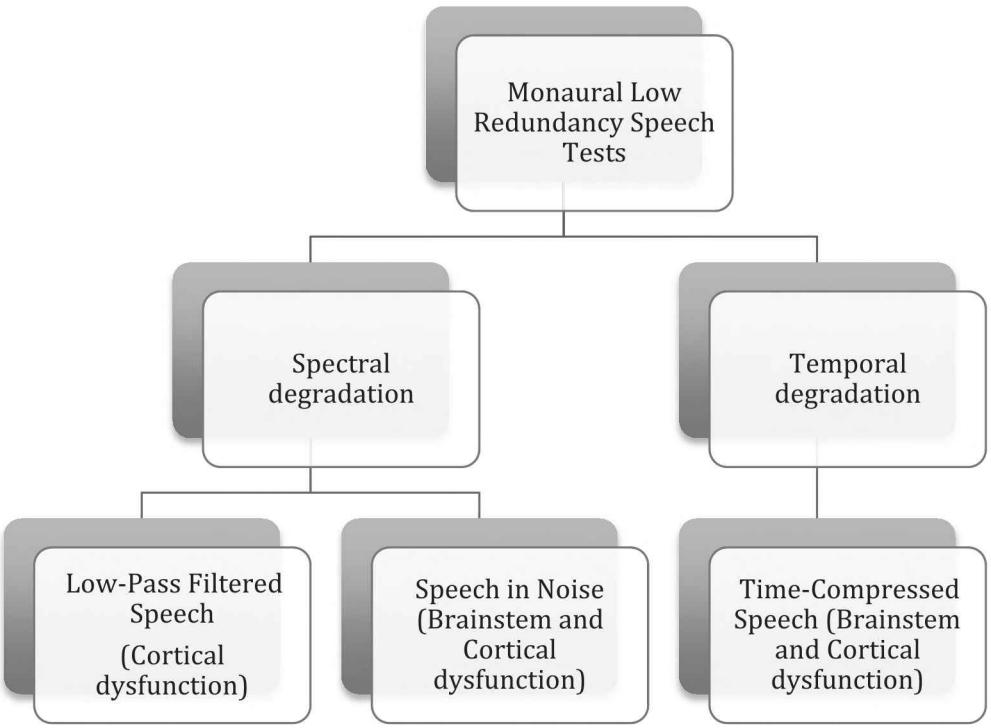

Figure 13–3. Types of monaural low-redundancy speech tests.

Bocca et al. (1954). They found that patients who were referred from neurological wards presented normal hearing sensitivity (as revealed by pure tone air conduction threshold testing); however, they exhibited temporal lobe damage. To probe their auditory function in more detail, Bocca et al. (1954) devised tests for higher order auditory processes and reported reduced performance in the ear contralateral to the temporal lobe lesion for most patients. See Chapter 11 for discussion of expected central auditory findings in CANS lesions.

Willeford (1977) proposed one of the earliest clinical test batteries to probe CAPD in children, aged 5 to 10 years. Included in his battery was a subtest comprising two 50-item lists of filtered consonant-nucleus-consonant (CNC) words. The words were presented at 50 dB HL and filtered at a 500 Hz cutoff frequency at a rate of 18 dB per octave. A slight maturational effect, reflected by larger standard deviations and ranges for younger children, was reported; however, no major ear asymmetry was seen (in contrast to ear asymmetry seen on dichotic speech tasks) (Willeford, 1977). Despite sounding muffled due to the filtering, the CNC words are rather intelligible to listeners with normal peripheral and central auditory function (Bellis, 2003). In contrast, individuals with CANS dysfunction evidence considerable difficulty, reflecting deficits in auditory closure (Bellis, 2003).

Keith (1986, 1994, 2000, 2009a, 2009b) developed several of the most widely used tools (SCAN, SCAN-A, SCAN-C, SCAN-3) to probe central auditory processing in children and adults (Emmanuel, 2002). One of the major advantages of these tests is that they look at multiple auditory processes such as monaural auditory closure (i.e., filtered words), monaural separation (i.e., auditory figure-ground), binaural integration (i.e., competing words), and binaural separation (i.e., competing sentences). A second strength of these tests is that subtest raw scores are converted into standard scores, which facilitates objective interpretation of performance. In this chapter, comments are limited to the monaural low-redundancy subtests.

The Screening Test for Auditory Processing Disorders (SCAN) was the first test developed specifically for children (Keith, 1986). This test included three subtests (Filtered Words [FW], Auditory Figure-Ground [AFG], and Competing Words [CW]). Instructions and scoring were straightforward and both raw scores and standard scores (compared with age norms) were utilized. Clinicians should be aware, however, of questions raised regarding the test-retest reliability of the SCAN. Amos and Humes (1998) reported that children (first- and third-grade) obtained significantly higher scores on the AFG and CW subtests of the SCAN upon retest 6 to 7 weeks following the first administration, indicating concerns about test reliability in younger school-age children.

The adult equivalent of the SCAN was the SCAN-A, a test of auditory processing disorders for adolescents and adults (Keith, 1994). This test included four subtests (i.e., FW, AFG, CW, and Competing Sentences [CS]). The filter cutoff (750 Hz) for the FW subtest in the SCAN-A rendered it more challenging than the cutoff of 500 Hz used in the child (SCAN) version. Likewise, the AFG subtest used a more challenging signal-to-noise ratio (SNR) (+4) for the SCAN-A than the +8 SNR applied in the SCAN. The SCAN-C, a revised version of the SCAN test for

children (Keith, 2000), was revised again in 2009 as the SCAN-3C; the SCAN-3A is the revised version of the SCAN-A (Keith, 2009a, b). Among the differences between the SCAN-3 and earlier versions of the SCAN is the inclusion of both screening and diagnostic subtests, including both a screening and diagnostic subtest of AFG, both using a +8 dB SNR, as well as an FW (750 low-pass filter) diagnostic subtest.

A different version of a low-pass filtered speech (LPFS) test is available on a Veterans Administration compact disc (VA, 1992). The standardized version consists of two lists of low-pass filtered NU-6 words (lists 3C and 4C) with a filter cutoff of 1500 Hz. Standardization of this LPFS test has been achieved by playing recordings of the female talker version of monosyllabic NU-6 words at different filter cutoffs (800 Hz, 1200 Hz, 1500 Hz, and 1700 Hz) to eight young normal hearing adults (Bornstein, Wilson, & Cambron, 1994). The target cutoff frequency that produced 70% to 80% correct word recognition performance at a comfortable listening level (50 dB HL) was found to be 1500 Hz, with normal young adults achieving on average 76.5% at this cutoff frequency (Bornstein et al., 1994).

Another version of an LPFS test is the Auditec recording of the filtered NU-6 words. According to Wilson and Mueller (1984), four low-pass filtered versions of this LPFS task (500 Hz, 700 Hz, 1000 Hz, and 1500 Hz) are available. Filtering using a 500 Hz cutoff frequency was found to pose difficulties even for listeners with normal peripheral and central auditory function and, therefore, is not recommended for clinical assessment of central auditory function (Wilson & Mueller, 1984). The 1000 Hz cutoff, male talker version has been recommended for clinical use (Wilson & Mueller, 1984), with norms available for NU-6 lists #1 to 4 (75%, 80%, 83%, and 78%, respectively) for normal hearing young adults. Bellis (2003) reported pediatric norms for the Auditec recording (1000 Hz cutoff frequency) at a presentation level of 50 dB HL: 62% (7 year old), 70% (8 year old), 68% (9 year old), 72% (10 year old), 75% (11 year old), and 78% or higher (12 year old to adult).

Limited sensitivity and specificity data for LPFS tests have been published; however, there is some evidence that these tests are more sensitive to temporal lobe lesions than interhemispheric and brainstem lesions. Lynn and Gilroy (1972, 1977) studied 34 patients with temporal lobe tumors and 27 patients with parietal lobe tumors. They found that 74% of patients with temporal lobe lesions showed expected contralateral deficits on LPFS, while 74% of patients with parietal lobe tumors presented normal performance on LPFS. Other studies reported similar outcomes: lower scores in the ear contralateral to the temporal lobe lesion (Bocca, 1958; Jerger, 1960). Karlsson and Rosenhall (1995) reported that filtered speech tests showed modest sensitivity (62%–64%) to brainstem lesions and slightly higher sensitivity (65%–67%) to temporal lobe lesions.

The diagnostic accuracy of established central auditory test batteries has been examined for patients with known lesions of the central nervous system (Musiek, Chermak, Weihing, Zappulla, & Nagle, 2011). Musiek and colleagues reported that the sensitivity of LPFS when used alone was the lowest (50%) of three other tests when they were used as a one-test "battery." The authors showed

that the use of multiple central auditory tests can help to: (a) potentially reduce diagnostic error and improve efficiency, (b) increase the face validity of the battery, and (c) help in establishment of the most appropriate intervention goals.

In contrast to the findings in temporal lobe lesions, LPFS performance has been reported to be essentially normal in listeners with interhemispheric lesions (Baran, Musiek, & Reaves, 1986; Musiek, Wilson, & Pinheiro, 1979). In patients with brainstem lesions, LPFS findings vary considerably (Calearo & Antonelli, 1973; Musiek & Guerkink, 1982). Some of this variability may be due to size and location of the lesion. Large, diffuse lesions are likely to show bilateral deficits, lower brainstem lesions are likely to reveal ipsilateral deficits, and higher brainstem lesions may present with contralateral deficits (Musiek & Guerkink, 1982). O'Beirne, McGaffin, and Rickard (2012) evaluated the performance of 33 normally hearing adults and 30 normally hearing children (aged 8–11 years) using a four-alternative, forced choice adaptive LPFS test (i.e., University of Canterbury Adaptive Speech Test [UCAST]). The UCAST varies the cutoff frequency of the LPFS to track the corner frequency at which participants correctly identify a certain percentage of the word stimuli. Results showed that adult participants performed significantly better than children on the UCAST-FW, indicating the measure's sensitivity to maturational changes in auditory processing ability.

Age and cognitive decline can influence performance on LPFS tests. Previous studies (Gates et al., 2008, 2011) have shown not only that central auditory dysfunction is prevalent among elderly individuals with Alzheimer's dementia, but that presence of central auditory processing deficits can actually be a harbinger of the onset of dementia. In a study that compared performance of elderly listeners with and without cognitive decline, LPFS scores were significantly poorer in individuals with Alzheimer's disease than LPFS scores for elderly listeners without cognitive issues (Krishnamurti, Drake, & King, 2011). These findings demonstrate that along with peripheral hearing loss, age, and cognition must be taken into account when evaluating central auditory function.

In summary, LPFS tests are moderately sensitive to CANS lesions, especially those located in the temporal lobe. In addition, some of these tests are useful to assess central auditory processing skills of children suspected of CAPD because they simulate listening in poor (degraded) listening conditions and therefore reveal functional deficit, even in the absence of true CANS lesion.

Speech-in-Noise Tests

In this category of tests, speech typically is embedded in a background of noise or speech competition. Early applications of these tests can be traced back to research by Sinha (1959), who reported deficits in the ear contralateral to cortical lesion in patients with CANS pathology. Subsequent studies also have shown speech-in-noise deficits contralateral to the hemisphere with auditory cortex involvement (Heilman, Hammer, & Wilder, 1973, Morales-Garcia & Poole, 1972). Other studies also have reported poor scores in patients with eighth nerve and extra-axial lesions (Dayal, Tarantino, & Swisher, 1966) and in patients with intra-

axial brainstem lesions (Morales-Garcia & Poole, 1972). Olsen, Noffsinger, and Kurdziel (1975) computed difference scores by subtracting NU-6 word recognition scores in noise from NU-6 word correct scores in quiet. Significant differences (greater than 40%) between quiet and noise were seen for patients with eighth nerve lesions, Ménière's disease, and temporal lobe lesions.

Recently, strong recommendations have been made to ensure that speech-in-noise tests be used and interpreted with extreme caution when assessing central auditory processing in children with coexisting peripheral hearing loss (Bantwall & Hall, 2011). Peripheral hearing loss imposes filtering and distortion on speech inputs, and poor spatial separation between speech and noise can make speech intelligibility difficult in noise. However, the majority of individuals with CAPD have normal peripheral hearing and hence their speech-in-noise problems are qualitatively different from those with peripheral hearing loss. Many of the speech-in-noise tests discussed in the following paragraph have been standardized for use with children and adults suspected of CAPD.

There are at least three standardized versions of speech-in-noise tests currently in clinical use: (1) the Auditory Figure Ground (AFG) subtest of SCAN/SCAN-C/SCAN-A/SCAN-3; (2) Synthetic Sentence Identification (Jerger & Jerger, 1974) with Ipsilateral Competing Message (SSI-ICM); and (3) the Pediatric Speech Intelligibility Test (Jerger & Jerger, 1984) with Ipsilateral Competing Message (PSI-ICM). A key factor in using speech-in-noise tests is the SNR. Perhaps the most commonly used SNR is 0 dB, which indicates that the sound pressure level of the speech is equal to the overall level of the noise (Rintelmann, 1985). The type of noise used in speech-in-noise tests can also vary considerably, making comparisons among tests difficult. White noise, multitalker babble, and even competing discourse have been used in various speech-in-noise tests.

The screening and diagnostic AFG subtests in the SCAN-3C version for children (Keith, 2009a) use an SNR of +8 dB. Both the screening and diagnostic tests in the SCAN-3A version for adolescents and adults use a 0 dB SNR in the AFG subtest.

In the SSI-ICM task, synthetic sentences are presented at 30 dB HL in competition with connected discourse (i.e., a story about Davy Crockett). The synthetic sentences are 10 third-order approximations of English sentences. The seven word sentences are semantically meaningless. They were designed to reduce a listener's reliance on linguistic skills while preserving the syntax and temporal feature of the English language (Jerger & Jerger, 1974). The SSI also can be presented with contralateral competition (SSI-CCM) (see Chapter 14). The SSI should only be administered to individuals with normal hearing sensitivity through 1000 Hz because the important audiometric frequency region underlying correct identification of sentence materials centers around 750 Hz (Hall & Mueller, 1997).

In the SSI-ICM task, the synthetic sentences are presented at a fixed level (i.e., 30 dB HL) and the level of the competition is varied to achieve varying MCR ratios (i.e., +10, 0, −10, −20). In contrast to the other tests reviewed above, the SSI requires that the listener be able to read the list of printed synthetic sentences. Also differentiating the SSI-ICM from the other tests reviewed above, the

SSI employs a closed message set (i.e., 10 sentences) and a closed response set (i.e., selection from among those same 10 sentences). Also, rather than repeating what is heard, the subject is asked to report the number of the sentence heard, thereby reducing the potential confounds of memory and language. Percentage correct scores are reported for each ear. Normative cutoffs for the SSI-ICM vary with MCR, for example, +10 dB MCR: 100%, 0dB MCR: 85%, −10 dB MCR: 70%, −20 dB MCR: 45% (Jerger & Jerger, 1974).

The SSI-ICM test has been shown to be sensitive to lesions of the brainstem (Jerger & Jerger, 1974, 1975). Jerger and Jerger (1974) showed that all (11/11) patients with intra-axial lesions in their study showed SSI-ICM deficits. Jerger and Jerger (1975) also showed that the average SSI-ICM loss approximated about 40% in the ears contralateral to brain stem lesions in 10 patients with intra-axial brainstem lesions, while for patients with eighth nerve lesions, the average SSI-ICM loss approximated 50% in the ear ipsilateral to the lesion. In the same study (Jerger & Jerger, 1975), patients with eighth nerve lesions showed a loss for monosyllabic, phonetically balanced words for the ipsilateral ear, while patients with brainstem lesions showed a loss for monosyllabic, phonetically balanced words bilaterally, with greater loss in the contralateral ear.

The Pediatric Speech Intelligibility (PSI) test is an adaptation of the SSI test for the pediatric population (Jerger & Jerger, 1984). This test is appropriate for children aged 3 to 6 years. Like the SSI, the PSI allows for the construction of performance versus intensity functions (PI Fn) and message to competition functions. Moreover, the PSI constructs these functions based on the child's responses to both monosyllabic words (i.e., 20 simple nouns) and sentence stimuli (i.e., 10 simple sentences with animal agents). Twenty competing sentences also involve animal agents in simple contexts. Like the SSI, the PSI can be administered with competition in the contralateral ear (PSI-CCM; see Chapter 9). The child is asked to point to the picture that is heard. In the competing conditions, the child is told to point to the picture said by "your man" and to ignore the "trick man." Like the SSI, the PSI employs a closed message set (i.e., 20 words and 10 sentences) and a closed response set (i.e., selection from among five pictures). The PSI closed response set requires the subject to point to the picture corresponding to the sentence heard, thereby reducing the potential confound of memory. The presentation level recommended for PSI-ICM is 30 dB HL with a 0 dB MCR for sentences and +4 dB MCR for words.

Based on norms reported in the test manual, SSI-ICM scores less than 80% fall outside the 95% confidence interval for normal hearing children (Jerger & Jerger, 1984). Sensitivity and specificity data were not reported by Jerger and Jerger (1984); however, Jerger, Johnson, and Loiselle (1988) reported that children suspected of CAPD presented deficits on the PSI similar to those demonstrated by children with confirmed temporal lobe lesions.

In summary, available data indicate that speech-in-noise tests may be marginally to moderately sensitive to a variety of CANS disorders (Jerger & Jerger, 1975; Morales-Garcia & Poole, 1972). Patients with low to mid-brainstem involvement, as well as cortical lesions, typically perform poorly on speech-in-noise tests. Given the variability related to stimuli, norms, and interpretation, it is extremely important for clinicians to avoid diagnosis

of CAPD based solely on a speech-in-noise test result (Bantwall & Hall, 2011). Several other tests, such as the Hearing In Noise Test (HINT; Nilson, Soli, & Sullivan, 1994), the Words In Noise Test (WIN; Wilson & Burks, 2005), and the QuickSin (Killon, Niquette, Gudmundsen, Revit, & Banerjee, 2004), have shown promise for quantifying hearing in noise difficulties.

Time-Compressed Speech Tests

Time-compressed speech tests employ speech that is compressed electronically by systematically sampling and discarding segments of the signal without distorting the frequency aspects of the signal (Beasley & Maki, 1976; Beasley, Schwimmer, & Rintelmann, 1972). Time compression (TC) currently is accomplished using waveform editing software that alters the rate of speech without altering the power spectrum or pitch. Artificial time compression of speech normally is performed in a linear fashion by reducing all speech segments (i.e., consonants, vowels, and pauses). By speeding up speech digitally, speech information and voice are presented without a high-pitched "Mickey Mouse"-like quality.

Naturally produced, speech spoken quickly (i.e., fast speech) has been found to be much less intelligible than mechanically time-compressed speech. Janse, Noteboom, and Quene (2003) assumed that the enhanced prosodic pattern found in naturally produced fast speech might improve the intelligibility of artificially time-compressed speech, and if speakers naturally time-compress their speech in a selective way that preserves prosodic patterns, for the sake of the listener, then listeners should benefit from such nonlinear, electronic time compression. Their study demonstrated that when speakers speed up their rate of speech, unstressed syllables in disyllabic words are affected more in duration, relatively, than stressed syllables. The problem is that for speech produced naturally but at faster rates, the consonant durations in syllables are influenced differentially than vowel duration, making the intelligibility of naturally time-compressed speech poorer than for electronically time-compressed speech. Several versions of electronically time-compressed speech tests are available (Beasley, Schwimmer, & Rintelmann, 1972; Bornstein, Wilson, & Cambron, 1994; Kurdziel, Noffsinger, & Olsen, 1976; Wilson, Preece, Salamon, Sperry, & Bornstein, 1994). Commercial availability of time-compressed speech materials allows clinicians to include this type of testing to evaluate auditory closure in children and adults.

Time-compressed speech tests assess the auditory system's capacity to process rapidly changing acoustic spectra. The amount of compression is expressed as a percentage of the original signal that is eliminated. TC of 45%, for example, indicates that the signal now occupies 55% of its original time frame, with 45% of signal having been removed. Again, this is accomplished without altering the acoustical aspects (e.g., frequency, intensity) of the signal.

Time-compressed speech tests are sensitive to cortical lesions (Karlsson & Rosenhall, 1995; Kurdzeil, Noffsinger, & Olsen, 1976), with performance typically reduced in the ear contralateral to the temporal lobe lesion (Calearo & Antonelli, 1973). Karlsson and Rosenhall (1995) studied the sensitivity of time-compressed speech in patients with brainstem lesions and temporal lobe

lesions. While time-compressed speech was only moderately sensitive to brainstem lesions (62%–64%), its sensitivity was much higher (80%) for patients with temporal lobe lesions (Karlsson & Rosenhall, 1995). Kurdzeil et al. (1976) studied the effects of several degrees of time compression (0%, 40%, and 60%) on speech recognition scores in 15 patients with diffuse cortical lesions and 16 patients with anterior temporal lobe surgical lesions. Patients with diffuse lesions showed a significant deterioration in speech recognition at 60% time compression in the ear contralateral to the lesion. In contrast, patients with discrete (i.e., anterior temporal lobe) lesions demonstrated essentially normal performance bilaterally.

Normative studies have explored the percentage of time compression that separates listeners with normal central auditory function from those with CAPD. Wilson, Preece, Salamon, Sperry, and Bornstein (1994) studied time-compressed speech for NU-6 monosyllables at 45% and 65% time compression in young adults with normal hearing. They found that normal listeners showed difficulty at 65% time compression; however, they exhibited normal performance at 45% TC. In contrast, listeners with central auditory dysfunction demonstrate reduced performance at 45% TC (Wilson et al., 1994). Based on these studies, 45% TC is the recommended standard compression value used clinically (Bellis, 2003). To make time compression even more challenging, a 0.3 msec reverberation has been electronically added to the time-compressed speech on the VA (1992) CD.

These are at least two standardized recordings of time-compressed speech currently in clinical use. One recording is included on the VA (1992) CD. This recording consists of two lists of monosyllabic NU-6 words (lists 7A and 8A) administered at a level of 55dB HL. Wilson et al. (1994) reported normative data (at 55 dB HL presentation level) with criterion values (i.e., two standard deviations below the mean) for young normal hearing adults (i.e., 45% TC = 86.5%; 45% TC plus reverberation = 34.9%). Bellis (2003) reported pediatric norms calculated at two standard deviations below the mean using the VA (1992) CD recording of 45% TC presented at 55 dB HL (i.e., 65% [9 year olds]; 68% [10 year olds]; 78% [11 year olds]; and 85% [12 year olds to adults]).

The second standardized recording of TC speech is the *Time Compressed Sentences Test* (TCST) for children aged 6 to 11 years developed by Keith (2002a). In this test, sentences are presented at 55 dB HL and compression rates are varied for subtests (0%, 40%, and 60% TC). All lists are composed of 10 sentences. In subtest 1 of the TCST, one list is presented at 0% (no) time compression; this easy baseline condition is used for practice and preliminary screening. Subtest 2 of the TCST is composed of two lists, each at 40% time compression, with one list presented to each of the two ears monotically. Subtest 3 is administered as is subtest 2 using 60% TC. Although Keith argued that sentences are more realistic and meaningful stimuli for children, sentence stimuli can increase the potential for language and memory confounds. The latest SCAN-3 versions for children and adults (Keith, 2009a, 2009b) provide additional (supplemental) time-compressed speech materials for optional testing (i.e., 60% time-compressed sentences).

No sensitivity or specificity data have been published for the TCST; however, Keith (2002a) published normative data

for children aged 6 to 11 years. Based on published norms (Keith, 2002a), pediatric norms calculated for right/left ears at two standard deviations below the mean for 40% TC presented at 55dB HL—that is, 75.6%/70.8% (6 year olds), 88.3%/89.3% (7 year olds), 87.5%/86.2% (8 year olds), 86.8%/91.4% (9 year olds), and 96.9%/91.4% (10 and 11 year olds). For detailed norms on the TCST, the reader is referred to the manual (Keith, 2002b).

Comparison of Bellis' (2003) and Keith's (2002a) norms for children (ages 9–11 years) indicate that the children of the same age did relatively better on the TCST than on the TC NU-6 VA (1992) CD recording. These performance differences may be attributed to the considerable difference in the intrinsic redundancy of the stimuli (sentences versus NU-6 monosyllables), resulting in the TCST being an easier task for children to perform.

In summary, time-compressed speech tests are only moderately sensitive to brainstem lesions; however, they present much greater sensitivity to diffuse lesions of the auditory cortex (Kurdzeil et al., 1976) and temporal lobe lesions (Karlsson & Rosehall, 1995). Sensitivity and specificity data are not available for these tests for children with CAPD, and this is a need that must be addressed in future research.

For many monaural low-redundancy speech tests, norms are available to allow comparisons by age, gender, ethnicity, and caregiver education level, for example, SCAN-3 test batteries (Pearson technical report, 2009a, b). However, in these test batteries, increased sensitivity must be weighed against specificity concerns associated with test instruments for CAPD screening and diagnosis. According to the test developers of the SCAN-3 (Pearson Technical Report, 2009a, 2009b), the sensitivity (ability to identify CAPD) and specificity (ability to correctly classify children without CAPD as not having CAPD) varied depending on the cutoff scores on the tests. For the SCAN-3A screening and diagnostic subtests, a score falling below 8 on the screening/diagnostic subtests led to sensitivity and specificity values of 93% and 49 %, respectively (Pearson Technical Report, 2009a). For the SCAN-3C, a score falling below 8 on the screening/diagnostic tests led to sensitivity and specificity values of 90% and 20%, respectively.

Implications for Intervention

As noted earlier, monaural low-redundancy test data provide information regarding real-life functional deficits and can be used to plan deficit-specific intervention. Poor performance on monaural low-redundancy speech tests implicates deficits in auditory closure and suggests that the listener could benefit from environmental noise reduction, signal enhancement, auditory training, and metalinguistic approaches discussed in Volume 2 of this Handbook.

Summary and cluding Comments

Monaural low-redundancy speech tests continue to be one of the most widely used types of tests for central auditory dysfunction (Chermak et al., 2007), despite the rather limited number of published reports regarding these tests' sensitivity and specificity, especially with

pediatric populations. Since performance on monaural low-redundancy speech tests is not affected by interhemispheric involvement (i.e., corpus callosum), they are especially useful when used alongside dichotic tests or pattern tests that are sensitive to hemispheric as well as interhemispheric involvement (Musiek, Kibbe, & Baran, 1984). That is, a left ear deficit on a dichotic listening test coupled with normal left ear monaural low-redundancy performance suggests interhemispheric involvement. In contrast, a left ear deficit on a dichotic listening test coupled with abnormal left ear monaural low-redundancy performance suggests either right hemisphere involvement or possibly involvement of both the right hemisphere and interhemispheric transfer (Musiek et al., 1984). In addition to assisting the audiologist in identifying the site/level of dysfunction, monaural low-redundancy tests are often included in the central auditory test battery because the test stimulates listening in poor (degraded) acoustic conditions, and, therefore reveals functional (i.e., auditory closure, listening in noise) deficits. Monaural low-redundancy speech tests can be useful, therefore, in assessing central auditory processing skills of children suspected of CAPD and in suggesting directions for deficit-focused intervention. (See Chapters 7, 9, and 10 for discussion of test battery interpretation.)

Available data suggest that low-pass filter speech tests are moderately sensitive to temporal lobe lesions (Karlsson & Rosenhall, 1995; Lynn & Gilroy, 1977). Speech-in-noise tests may be marginally to moderately sensitive to a variety of CANS disorders (Jerger & Jerger, 1975; Morales-Garcia & Poole, 1972), including patients with low to mid-brainstem involvement, as well as cortical lesions. The PSI-ICM is a well-developed test that employs speech in competition to assess young children's ability to extract speech presented in background competition. At least one study suggests the PSI's sensitivity to CANS lesions or dysfunction (Jerger et al., 1988). Time-compressed speech tests appear to be sensitive to diffuse lesions of the auditory cortex (Kurdzeil et al., 1976) and are also sensitive to temporal lobe lesions (Karlsson & Rosenhall, 1995).

Additional research is needed to determine the sensitivity of monaural low-redundancy speech tests to CANS lesions and CAPD in children who do not present with evidence of lesions, but rather present more diffuse neurobiological dysfunction. See Chapter 4 for discussion of the causes of CAPD in children. Additional normative data are needed as well for many of the tests reviewed in this chapter.

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CHAPTER 14

DICHOTIC LISTENING TESTS

JEFFREY WEIHING and SAMUEL R. ATCHERSON

Introduction

Overview

Assessment of dichotic processing is an important component of central auditory processing test batteries. Indeed, in a recent survey of practicing audiologists, Emanuel, Ficca, and Korczak (2011) noted that well over 50% of respondents reported using some type of dichotic test as part of these evaluations. The utility of these tests arises in part from the sensitivity of these measures to central auditory nervous system (CANS) dysfunction (Musiek, Chermak, Weihing, Zappulla, & Nagle, 2011) and the relationship between dichotic listening and selective attention (Hugdahl, 2000). By definition, a dichotic listening task is one in which a different auditory stimulus is presented simultaneously to each ear. It is distinguished from a diotic task in which the same auditory stimulus is presented to each ear, and a monotic task in which an auditory stimulus is presented to only one ear. During a dichotic task, patients are generally asked to direct their attention to one ear and repeat what is heard, or to repeat back what was heard in both ears.

This chapter provides a comprehensive view on what is currently known about dichotic listening using speech stimuli. Topics to be covered include the history of dichotic testing, mechanisms thought to underlie dichotic difficulties, and methods by which dichotic listening can be assessed. An emphasis is placed on the clinically important measurement of dichotic listening and how to use test

dichotic test results to differentiate central auditory processing disorder (CAPD) from more global processing difficulties.

History of Dichotic Testing

The definition of "dichotic listening" is credited to Stevens and Davis (1938), who described it as "the simultaneous stimulation of both ears, but with a different stimulus in each ear" (p. 451). Early research investigated the effect of stimulus parameters on dichotic performance, including word length, familiarity, and intelligibility (Ptacek, 1954). Concurrently, Broadbent (1954) published the now commonly used dichotic digit procedure as a means to test the ability of air-traffic controllers to monitor different auditory streams. Another line of inquiry in early research on dichotic listening investigated the nature of ear dominance effects, or the tendency of one ear to have an advantage and outperform the other when engaged in dichotic listening. In participants of various ages, Kimura noted that a right ear advantage on dichotic listening was observed in individuals with confirmed left hemisphere language dominance, while a left ear advantage was noted in patients with right hemisphere dominance (Kimura, 1961a, 1963a, 1963b). This ear advantage was thought to be attributed to stronger connections between the language dominant cortex and the contralateral ear (Kimura, 1961a; Satz, 1989). It also was initially thought that handedness predicted the dominant ear, such that right-handed individuals showed right ear advantages on dichotic listening, and left-handed individuals showed left ear advantages. Although this relationship is most frequently observed (Demarest & Demarest, 1981; Hugdahl & Andersson, 1984; Lishman & McMeekan, 1977), a percentage of the population does not conform to this predicted handedness-ear dominance relationship (Briggs & Nebes, 1976; Carr, 1969; Lishman & McMeekan, 1977). Further complicating the issue has been the finding that ear dominance shows a great deal of within-subject variability from trial to trial (Blumstein, Goodglass, & Tartter, 1975; Speaks & Niccum, 1977).

Although mechanisms are discussed in greater detail below, it is worth mentioning briefly some of the proposed mechanisms for dichotic listening that have emerged both historically and through more recent research. In a now classic paper, Kimura (1961b) examined the effect of neurological lesions (i.e., temporal or frontal lobectomies) on dichotic performance. For patients undergoing temporal lobe procedures, it was observed that there was a significant decrease in dichotic performance at the ear contralateral to the removed lobe. Similar effects were not witnessed for patients undergoing frontal lobectomies. Furthermore, the effect of temporal lobectomies was greater in cases that involved Heschl's gyrus. Based on these findings and the research of Tunturi (1946) and Rosenzweig (1951), Kimura proposed an anatomic and physiologic explanation of these clinical observations. In Kimura's model of dichotic listening, the ipsilateral ascending auditory pathways are generally weaker than the contralateral pathways. Following temporal lobectomy, only the ipsilateral and contralateral pathways ascending to the unaffected hemisphere remain active. Since the ipsilateral connections are weaker, their activity is generally suppressed by the stronger contralateral pathways. Thus, on dichotic stimulation, only activity from the contralateral ear reaches the unaffected hemisphere because the ipsilat-

eral signal is suppressed (Figure 14–1). This yields a distinct ear advantage at the ear contralateral to the unaffected hemisphere. Based on these findings, it can be said that the temporal lobe contralateral to the test ear contributes importantly to normal dichotic listening.

Other neurological investigations performed shortly thereafter highlighted the important role of the corpus callosum in dichotic listening (Geschwind, 1962; Geschwind & Kaplan, 1962). Patients undergoing split-brain procedures would show complete left ear extinction on dichotic listening following the surgical procedure, despite having normal dichotic function preoperatively (Musiek et al., 1989; Musiek & Wilson, 1979; Sparks & Geschwind, 1968). As discussed below, the explanation for this finding was that the left ear pathway depends on the "disconnected" corpus callosum during dichotic processing while the right ear does not (as it maintains access to the left hemisphere "speech processor" through direct contralateral pathways). Interestingly, clinical groups, such as younger children with suspected maturational delays of the corpus callosum (Musiek & Gollegly, 1988; Musiek, Gollegly, & Baran, 1984) and older adults with aging-related changes to this same region (Gootjes et al., 2006; Jerger & Lew, 2004) also presented similar left ear deficits on these dichotic measure. In part because of this line of research and others (Bellis, Billiet, & Ross, 2011; Bellis & Wilber, 2001), emphasis has been placed on the interaural asymmetry in dichotic performance when testing for CAPD clinically. That is, individuals with greater dichotic difficulties tend to be those who show the greatest difference in performance between ears, and it is believed

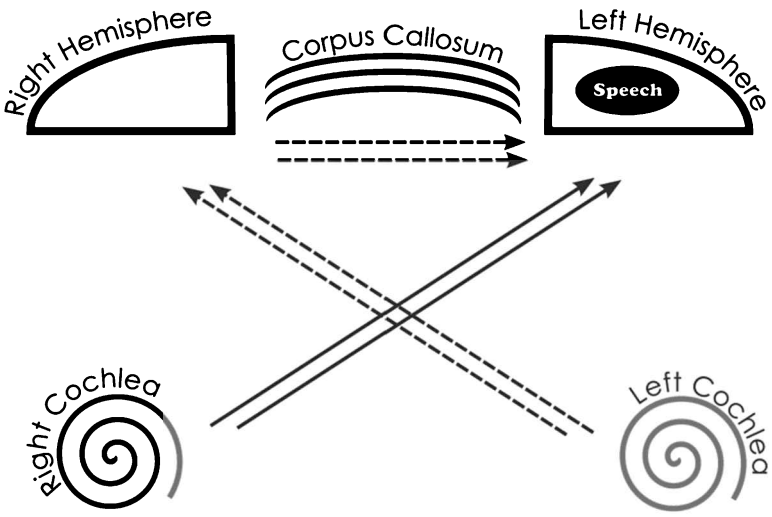

Figure 14–1. Dominate pathways during dichotic listening using verbal stimuli for a case of left hemisphere language dominance. Dashed line represents the left ear pathway, while solid lines represent the right ear pathway. Note that the left ear pathway utilizes the corpus callosum, whereas the right ear does not. Not pictured here are weaker ipsilateral pathways thought to be suppressed during dichotic listening.

that the corpus callosum plays an important role in mediating the magnitude of these differences.

Studies investigating ear dominance and dichotic listening also have considered the role played by modulating attention. These types of investigations generally utilize very short consonant vowel (CV) syllables that are differentiated only on their initial, brief consonant information. Such paradigms typically yield two scores: the number of stimuli correctly identified in the attended ear, and the number of times the stimulus in the nonattended ear intruded and was reported by the subject. As would be expected, when attention is directed to one ear, the number of CVs reported from that ear increases and the number of intrusions from the nonattended ear decreases (Asbjornsen & Hugdahl, 1995). For patients who show a right ear advantage on dichotic testing, attending to the left ear can switch their right ear advantage to a left ear advantage, and attending to the right ear can increase the magnitude of the right ear advantage when it is present (Asbjornsen & Hugdahl, 1995; Keith, Tawfik, & Katbamna, 1985). Some studies have shown a less consistent effect of modulating attention. For instance, Hugdahl and Hammar (1997) and Gadea, Gomez, and Espert (2000) showed that right-handed participants less consistently showed a left ear advantage when directing attention to the left ear. Additionally, studies using consonant-vowel-consonant (CVC) stimuli, where only the initial consonant differs between ears, have shown that performance is not as affected by directing attention (Shinn, Baran, Moncrieff, & Musiek, 2005).

This observed effect of attention on dichotic listening performance raised interesting questions about cognitive control abilities and executive function largely mediated by structures associated with the frontal cortex (Hugdahl, 2000; Hugdahl et al., 2009). The finding that the right ear advantage increases with right ear directed attention, while left ear directed attention reduces the right ear advantage (or increases the probability of a left ear advantage) does not hold true for certain clinical groups, particularly where top-down processes may be suspect. As one example, otherwise healthy aging adults maintained a right ear advantage in a left ear directed-attention task, indicating a breakdown, conflict, or failure of attentional processes to override the stimulus-driven right ear advantage (Thomsen et al., 2004a). Similar deviations or progressive changes to the patterns of dichotic listening performance also have been observed in participants with nonauditory specific issues, such as patients diagnosed with schizophrenia, loneliness, and/or stuttering (Cacioppo et al., 2000; Foundas, Corey, Hurley, & Heilman, 2004; Hugdahl et al., 2009).

Why Test Dichotic Listening?

Current interest in dichotic listening stems from several important audiological applications of dichotic measures. Important among these is applying dichotic tests to help explain listening difficulties encountered by children and diagnose CAPD in this population. In some of the earliest studies in this population, Musiek and colleagues (Musiek & Geurkink, 1980; Musiek, Geurkink, & Kietel, 1982) observed dichotic processing deficits in children referred for audiologic evaluation due to listening difficulties. More recently, Moncrieff (2006) noted in a larger sample that approximately 20% to 50% of children

referred for listening difficulties showed deficits on dichotic tests. Dichotic listening also appears to co-occur with some developmental disorders, although the relationship is imperfect. For example, research has shown significant correlations between dichotic processing and learning disabilities (Ayres, 1977; Johnson, Enfield, & Sherman, 1981; Koomar & Cermak, 1981; Obrzut, Hynd, & Obrzut, 1983), and between dichotic processing and reading disabilities (Arciuli, Rankine, & Monaghan, 2010; Asbjornsen & Bryden, 1998; Asbjornsen & Helland, 2006; Helland, Asbjornsen, Hushovd, & Hugdahl, 2008; Hugdahl et al. 1998; Iliadou, Kaprinis, Kandylis, & Kaprinis, 2010; Martinez & Sanchez, 1999; Moncrieff & Musiek, 2002; Morton & Siegel, 1991; Richardson & Firlej, 1979; Welsh, Welsh, & Healy, 1980).

Although currently there is considerable interest in understanding dichotic listening in children, studies also have shown that assessment of dichotic skills provides useful information in explaining the listening difficulties of older adults. In this context, dichotic testing is used to assess "central presbycusis," or aging-related changes to the CANS. It is estimated that approximately 30% of older adults referred for audiometric evaluation show a dichotic processing issue consistent with central presbycusis (Weihing et al., 2006). Chmiel and Jerger (1993, 1996) have reported that dichotic ability is very closely related to "hearing handicap" in older adults. When hearing handicap is rated by their significant others, older adults with dichotic deficits show more difficulties than older adults with cognitive deficits or those with normal functioning (Chmiel & Jerger, 1993). Further, older adults with dichotic deficits do not show significant reduction in hear-ing handicap following hearing aid fittings. This is in stark contrast to similar patients who do not have dichotic deficits and show hearing handicap benefits post-hearing aid fittings. These observations also have been reported by Givens, Arnold, and Hume (1998) who noted that poorer dichotic performance was correlated with reduced hearing aid satisfaction.

Additional clinical applications of dichotic tests in the older adult population include diagnosis of binaural interference. Binaural interference refers to a phenomenon in which, despite the indication for binaural amplification based on audiometric performance, a patient performs better with one ear amplified than with two (Jerger, Silman, Lew, & Chmiel, 1993). It is estimated that approximately 6% of older adults experience binaural interference (Allen, Schwab, Cranford, & Carpenter, 2000). Importantly, dichotic listening can be useful in identifying who may be at risk for experiencing this phenomenon (Carter, Noe, & Wilson, 2001; Chmiel, Jerger, Murphy, Pirozzolo, & Tooley-Young, 1997; Cox, Schwartz, Noe, & Alexander, 2011).

Clarification of Terminology

A variety of terms have been used to describe patient response modes on dichotic testing. The two most commonly used in CAPD consensus statements are *binaural integration* and *binaural separation* (AAA, 2010; ASHA, 2005). Binaural integration refers to attending and repeating back both left and right ear dichotic stimuli, whereas binaural separation refers to attending to only a single ear and repeating back what is heard while ignoring the other ear. Analogous terms sometimes used for binaural integration are free recall, free report,

divided attention, and nonforced (NF) attention. For binaural separation, terms such as directed attention and directed report are used, where instructions must be qualified as forced left (FL) or forced right (FR) to indicate the attended ear. In an effort to remain consistent with what has been recommended by consensus statements, this chapter generally uses the binaural integration and separation terminology, though not exclusively. In particular, we use the NF, FL, and FR terms when discussing current psychological literature, as these are the terms that are typically used in this research.

Mechanisms of Dichotic Listening

There are at least two lines of research that attempt to explain performance on dichotic listening tasks. Examination of the performance of patients with neurological lesions of the central nervous system has provided information on what auditory regions contribute importantly to dichotic listening. Additionally, functional magnetic resonance imaging (fMRI) studies have yielded information on which central regions are most active during the processing of dichotic information. These methods of inquiry have led to separate models that attempt to explain what underlies reduced dichotic performance. The first "transmission line" or "structural" theory highlights the importance of the temporal lobe in dichotic processing, as well the interaction of the two hemispheres via the corpus callosum. The second theory highlights the importance of the frontal lobe and attention in dichotic listening, and emphasizes the role of the corpus callosum in exerting beneficial inhibition. How the available evidence relates to each of these two theories is discussed in greater detail below.

Neurologic Studies: Temporal Lobe and Corpus Callosum

Studies investigating dichotic listening in patients with cortical lesions have commonly noted a contralateral ear effect. This ear effect manifests as poorer performance at the ear contralateral to the central lesion, particularly when there is involvement of the temporal lobe (Harris, 1994; Hugdahl & Wester, 1992; Mazzucchi & Parma, 1978; Musiek, 1983a; Musiek, 1983b; Sparks, Goodglass, & Nickel, 1970; Speaks, Gray, & Miller, 1975; Tanabe, Nishikawa, Okuda, & Shiraishi, 1986). As the contralateral ascending auditory pathway is stronger than the ipsilateral pathway, it is thought that damage to the cortex in one hemisphere prevents the ear contralateral to the lesion from effectively stimulating the cortex. Right hemisphere lesions tend to yield larger interaural asymmetries on dichotic testing (Mazzucchi & Parma, 1978; Sparks et al., 1970). When there is complete removal of an affected hemisphere, extinction of performance at the contralateral ear is noted (de Bode, Sininger, Healy, Mathern, & Zaidel, 2007). These ear effects have been less commonly observed in patients without temporal lobe involvement (Bergman, Najenson, Hirsch, & Solzi, 1985). The anterior pole of the temporal lobe does not appear to contribute to dichotic listening, as removal of this region does not produce a predictable decrease in performance (Olson, 1983). However, in some cases where pathology localized to this

region affects other regions (e.g., as with epilepsy), removal of the temporal pole can sometimes yield an improvement in dichotic performance (Musiek, Bromley, Roberts, & Lamb, 1990). It is thought that this can occur because there is a spread of epileptic activity from the temporal pole to the more posterior areas of the temporal lobe, and even across the corpus callosum.

Unlike cortical lesions, brainstem lesions have been associated with primary deficits for the ear ipsilateral to the lesion (Musiek, 1983a). This relationship is less consistent, however, and depends more precisely on where in the brainstem the lesion is located. Some studies have reported a contralateral ear effect for some types of brainstem lesions, such as those that are intra-axial or in the region above the superior olive (Cho et al., 2005; Jerger & Jerger, 1975). It also has been noted that electrical stimulation of the left thalamus will increase dichotic performance in the contralateral ear (Ojemann, 1985; Wester & Hugdahl, 1997), and removal of this same structure greatly reduces performance in the contralateral ear (Wester & Hugdahl, 1997). Interestingly, stimulation of the right thalamus does not produce similar trends (Ojemann, 1985; Wester & Hugdahl, 1997).

Although much of the research on the impact of neurologic dysfunction on dichotic performance has focused on adults, there have been some studies that have examined this relationship in developing children. The contralateral ear effect seen in adults is typically seen in children with neurological dysfunction as well (Cranford, Kennalley, Svoboda, & Hipp, 1996; Netley, 1972; Plaza, Rigoard, Chevrie-Muller, Cohen, & Picard, 2001; Wester, Hugdahl, & Asbjornsen, 1991; Woods, 1984). Additionally, children who acquired their lesions from a very young age tended to show contralateral ear effects less consistently, possibly as a result of beneficial plasticity (Woods, 1984). It is also suspected that plasticity accounts for greater post-surgery dichotic recovery in children (Cranford et al., 1996). Another cortical neurologic finding in children that correlates with dichotic performance are abnormal perisylvian polymicrogyri (Boscariol et al., 2010, 2011). These polymicrogyria are characterized by small and/or ectopic neural groups, and tend to occur bilaterally. Boscariol and colleagues have shown that children with language-learning disabilities who demonstrate these polymicrogyri tend to show poorer dichotic performance in both ears relative to children with similar disabilities who do not have polymicrogyri and/or to normal hearing controls.

Neurologic studies also have demonstrated that the corpus callosum makes essential contributions to successful dichotic listening. Typically, callosal lesions yield large interaural asymmetries in dichotic performance, with left ear performance typically below the right ear. The initial observation of left ear deficits following corpus callosum lesions is attributed to Geschwind and colleagues, where the observed lesions were caused by strokes that damaged the callosal fibers (Geschwind, 1962; Geschwind & Kaplan, 1962). Similar dichotic effects also were observed at the time in patients undergoing "split-brain" procedures for intractable epilepsy (Bogen & Vogel, 1962). Later studies would begin to correlate abnormally large interaural asymmetries on dichotic measures with other neurological disorders known to affect the corpus callosum, including multiple sclerosis (Jacobson, Deppe, & Murray,

1983; Rubens, Froehling, Slater, & Anderson, 1985).

When the role of the corpus callosum in dichotic listening was initially identified, there was some question as to which region of this structure made the most important auditory contributions. It is generally well accepted that the trunk of the corpus callosum (e.g., sulcus) contains auditory fibers and contributes to left ear deficits on dichotic tasks (Alexander & Warren, 1988; Kaga, Shindo, Gotoh, & Tamura, 1990; Springer & Gazzaniga, 1975). Research investigating the role of the region posterior to the sulcus, or the splenium, is more equivocal. While some studies have indicated that the splenium lesions induce left ear deficits on dichotic tasks (Gadea et al., 2002, 2009; Pollmann, Maertens, von Cramon, Lepsien, & Hugdahl, 2002; Sugishita et al., 1995), others have noted significant left ear deficits in patients without splenium lesions (Risse et al., 1988; Springer & Gazzaniga, 1975). Lesions to the anterior region of the corpus callosum have indicated that this region does not contribute to dichotic performance (Baran et al., 1986; Sugishita et al., 1995), Although isolated cases of left ear deficits due to lesions localized to anterior regions of the corpus callosum have been reported (Pollman et al., 2002; Risse et al., 1988).

Compelling evidence for the role of the corpus callosum in dichotic listening comes from several studies using within-subject designs. These studies demonstrate that dichotic ability fluctuates within-subject concurrently with changes to the corpus callosum. For example, in patients undergoing commisurotomy, normal dichotic performance was seen before surgery, but extinction of the left ear response was observed following surgery (Damasio, Damasio, Castro-Caldas, & Ferro, 1976; Milner, Taylor, & Sperry, 1968; Musiek et al., 1989; Musiek & Wilson, 1979; Sparks & Geschwind, 1968; Springer, Sidtis, Wilson, & Gazzaniga, 1978). In patients with intermittent callosal pathology due to exacerbations and remissions seen in multiple sclerosis (MS), progressively greater involvement of the posterior corpus callosum also tends to correspond with progressively greater asymmetries on dichotic tests (Gadea et al., 2009). Fujimoto, Ito, Iwasaki, Nakao, and Sugasawa (2006) reported a case in which 5-FU (fluorouracil)-induced leukoencephalopathy, which caused temporary injury to the posterior corpus callosum as confirmed by diffusion weighted imaging (DWI), led to dichotic deficits confirmed nine days post-incident, with almost a 100% performance difference between the ears. Similar differences were not noted on a haptic recognition test. Approximately nine weeks later following treatment, DWI abnormalities were mostly absent and the patient performed within normal limits on the dichotic measure. Research investigating the effect of traumatic brain injury (TBI) also provides similar evidence. The corpus callosum is affected in cases of TBI because the impact exerts shearing and tearing forces to the callosal fibers (Levin et al., 1989). Peru, Beltramello, Moro, Sattibaldi, and Berlucchi (2003) reported a case in which a severe head trauma contributed to left ear extinction on dichotic tasks. Among other abnormalities, the patient showed a reduction in signal intensity in the posterior third of the corpus callosum. This region of the corpus callosum and left ear dichotic performance returned to normal levels approximately three years later.

As described briefly above, a mechanism that explains callosal involvement

in these neurological cases is based on theories formulated by Kimura (1961a, 1961b). In her "transmission line" or "structural" model, the contralateral ascending auditory pathways are stronger than the ipsilateral pathways. During dichotic listening, the contralateral pathways are most stimulated and this suppresses activity in the ipsilateral pathways. Thus, the only route by which auditory signals can reach the cortex are via the contralateral route. When asking patients to make a verbal response in a dichotic task, the primary auditory cortex and related areas are recruited. In a majority of patients this neural center is located in the left hemisphere. As such, both left and right ear signals must reach the left hemisphere in order to be repeated back verbally. The right ear signal has a direct route to this region, as it ascends contralaterally to the left hemisphere. The left ear signal, however, ascends to the right hemisphere and then must be transmitted across the corpus callosum in order to reach the left hemisphere. When the corpus callosum has neurologic involvement, the left ear signal is unable to be transmitted to the left hemisphere, and the patient performs much better on right ear than left with dichotic stimuli. This model offers a cogent explanation for interaural asymmetries typically observed in dichotic performance. A final detail to note regarding neurologic studies of the temporal lobe and corpus callosum is the phenomenon known as "paradoxical ipsilateral extinction" (Sparks et al., 1970). This unusual phenomenon occurs when dichotic performance is worse in the ear *ipsilateral* to the cortical lesion (Bergman et al., 1985; Harris, 1994; Jerger & Zeller, 1989; Sparks et al., 1970). Studies of reduced left ear performance in cases of left hemisphere involvement have noted that there is typically damage in the left hemisphere white matter that is lateral and posterior to the posterior lateral ventricles, very near where the corpus callosum enters the left hemisphere (Damasio & Damasio, 1979). Thus, although this effect does not involve the corpus callosum directly, it does appear to influence the output of the callosal signal into the left hemisphere, thereby yielding a finding similar to a callosal lesion (e.g., an ipsilateral ear effect).

Neurologic Studies: Frontal Lobe

Although not as common as the dichotic effects caused by callosal and temporal lobe lesions, frontal lobe lesions and other lesions not involving the CANS also have been reported to contribute to interaural asymmetries in dichotic performance in some cases (Hugdahl & Wester, 1994; Hugdahl, Wester, & Asbjornsen, 1991). Effects have sometimes been shown to be stronger when lesions are in the left frontal lobe (Hugdahl, Bodner, Weiss, & Benke, 2003a). Hugdahl and Wester (1994) emphasized the importance of considering potential cognitive contributions to dichotic listening because of these observed effects. As noted by Kinsbourne (1970), it may be that the left and right hemispheres orient auditory attention to the contralateral hemifield and that this attention is modulated by an inhibitory feedback system that incorporates the corpus callosum. Under this model, inhibitory processes mediated by the corpus callosum allow for relatively equal degrees of attention in the two hemispheres. When corpus callosum dysfunction occurs, it causes

attention to be distributed more asymmetrically due to loss of this beneficial inhibition (Tweedy, Rinn, & Springer, 1980). Thus, according to Kinsbourne's theory, the right hemisphere would have difficulty allocating attention to the left hemifield because, in the absence of inhibition, these attentional resources are being taxed by the left hemisphere.

Functional Magnetic Resonance Imaging Studies

The use of fMRI has permitted study of the neural and structural underpinnings for hemispheric specialization and dichotic listening (Hugdahl, 2000; Jäncke, Buchanan, Lutz, & Shah, 2001) to both nonverbal and verbal stimuli. Jäncke, Specht, Shah, and Hugdahl (2003) used dichotic tones presented in NF, FL, and FR conditions. While presented with pairs of dichotic tones (800, 1000, 1200, or 1400 Hz), healthy participants were instructed to press a button whenever they heard either the 800 or 1400 Hz tone. Blood oxygenation changes (reflecting activation regions) during dichotic listening (compared with resting state) were found in multiple areas of the brain, including the superior temporal gyrus and planum temporale (perisylvian regions of the temporal lobes), the inferior and middle frontal gyrus (frontal areas), the anterior supplementary area (presupplementary motor area), and right superior and inferior parietal areas. That activation within the temporal perisylvian regions of the temporal lobes was seen is not surprising given the auditory stimuli used. Activation of the frontal areas also is not surprising given the task-related cognitive demands (attention and working memory) and button-press motor planning. Finally, greater activation in the right hemisphere compared with the left hemisphere is likely related to the processing of tones, rather than speech. An important across-task finding was that the NF task revealed stronger activation in the presupplementary motor area compared with FL and FR tasks. The authors argued that the presupplemental motor area may be involved, not only for motor planning, but also for processes requiring mediation of higher cognition, particularly in the NF task.

More commonly, fMRI studies of dichotic listening have used speech stimuli. Thomsen et al. (2004b) examined diotic (binaural) and dichotic CV stimuli (e.g., /ba/, /da/, /ga/, /pa/, /ta/, /ka/) under all three task conditions: NF, FR, and FL. For dichotic stimuli, typical behavioral performance patterns were exhibited with right ear advantage during NF and FR conditions, whereas a left ear advantage was evident during FL. As expected, when compared with dichotic performance, diotic performance was better in all conditions. To reveal brain activations for the various listening and attention conditions, several subtractive permutations can be made with the fMRI scans. Most salient are the comparisons between diotic and dichotic listening during a NF task, as well as the analogous diotic and dichotic conditions for FL and FR. For the NF task, dichotic minus diotic scans revealed activation in the left cingulate gyrus, inferior frontal gyrus (bilaterally), and the left middle frontal gyrus. For the FR tasks, dichotic minus diotic scans revealed activation in the left superior temporal gyrus in the vicinity of the planum temporale, along with the cingulate gyrus (bilateral), inferior frontal gyrus (bilateral), and the left middle frontal gyrus. Finally, for the FL

task, dichotic minus diotic scan revealed similar activations as the FR task; however, there was an additional activation in the anterior cingulate. Taken together, Thomsen et al. (2004b) implicated the prefrontal cortex as playing a role in dichotic speech tasks in addition to activation of the left hemisphere for speech and language processing.

The effect of aging on dichotic listening has also been investigated using fMRI. Thomsen et al. (2004a) reported the expected right ear advantage in the NF and FR tasks; however, older adults maintained a significant right ear advantage even in the FL. The fMRI scan comparisons between younger and older adults revealed decreased left middle frontal gyrus activity, which is supported by structural MRI data showing reduced gray matter density in older adults. Functionally, these data point to the notion that there is a breakdown, conflict, or failure of attentional processes in stimulus-driven right ear advantage in older adults, even in the absence of peripheral hearing loss.

This persistent right ear advantage even in FL conditions in some clinical populations is an interesting phenomenon that has spawned further studies investigating contributions made by attention to dichotic listening. Although it is well accepted that the auditory system has a stimulus-driven, bottom-up processing bias to the right ear/left hemisphere for dichotic speech stimuli, the lingering question is, what happens during the left ear directed-attention task? One approach is to consider a model in which cognitive control is different between FR and FL conditions. Due to the right ear advantage bottom-up processing bias, a situation is set up where there is top-down cognitive conflict during left ear directed attention tasks (e.g., the right ear pathways dominate, but attention is being directed away from them to the left ear). During this cognitive conflict, the listener must maintain attentional focus on the left ear and depend on inhibitory control to counteract the stronger right ear advantage (Hugdahl et al., 2009). This is supported by Thomsen et al. (2004a) who highlighted differences in fMRI brain activations in the prefrontal cortical areas between the FR and FL conditions. Moreover, there is evidence of reduced left middle frontal gyrus activity in older adults (Thomsen et al., 2004a) and complete inability to activate the anterior cingulate cortex areas in patients in schizophrenia during FL tasks (Hugdahl et al., 2003b, 2009). Both of these latter findings point to changes in cognitive control and failure to modulate the stimulus-driven right ear advantage.

Although the actual processing network underlying cognitive conflict during FL tasks remains to be determined, paradigms that present dichotic CV stimuli at various interaural intensity differences may yield additional insight (Falkenberg, Specht, & Westerhausen, 2011; Westerhausen et al., 2010). The rationale for using interaural intensity differences in this context is that greater attention would be required at the attended ear if the intensity of the stimulus in that ear were less than the unattended ear, while less attention would be required if intensity favored the attended ear. Results have shown, first, that top-down processes are clearly at play in order to suppress correct behavioral responses in the nonattended ear when the salience (i.e., intensity level) is reduced in the attended ear. Moreover, the number of correct responses for all three conditions (NF, FR, and FL) increased somewhat

linearly as the interaural intensity difference shifted from one ear to the other, but was generally steepest (most correct responses) during the FR and FL conditions. Furthermore, when the salience progressively favored the attended ear, the right ear advantage was increased during FR while the left ear advantage was increased in FL.

According to fMRI scans, there seems to be at least two brain networks associated with cognitive conflict during dichotic listening with interaural intensity differences (Falkenberg et al., 2011; Westerhausen et al., 2010). The first network is viewed as the top-down cognitive control network involving multiple fronto-medial-parietal areas including the pre-supplemental motor area, precentral gryus, anterior cingulate cortex, inferior frontal junction, and inferior parietal lobe (Falkenberg et al., 2011; Westerhausen et al., 2010). The second network appears to involve top-down processes that influence activation in the superior temporal gyrus and the postcentral gyrus (Falkenberg et al., 2011). One interesting finding in the fMRI scans is that attending to and reporting stimuli from the ear with less salience produces greater activation with the frontal and parietal regions than when the salience favors the attended ear (Westerhausen et al., 2010). A second interesting fMRI finding is that of an inverse interaction of higher activation in the superior temporal gyrus and postcentral gyrus during the least demanding interaural intensity difference conditions (e.g., FR with high right ear salience and vice versa). These collective behavioral and fMRI results with the interaural intensity difference paradigm seem to reflect roles and inherent biases offered by the top-down cognitive control system and bottom-up (sensory) processes, and that in healthy participants can exhibit appropriate attentional and cognitive control is exhibited while suppressing information in the unattended ear.

In summary, fMRI scanning in conjunction with the study of binaural integration and binaural separation dichotic listening tasks reveal the close interplay between the classical auditory, bottom-up (sensory) processing network and the nonclassical, top-down (cognitive) network of broadly distributed brain areas. Clearly, the influence of top-down processes during dichotic listening cannot be ignored, and should be addressed during differential diagnosis. See Chapter 6 for elaboration of some of the scientific foundations of dichotic listening.

Application of Mechanisms to Clinical Observations

Interaural asymmetries on dichotic listening tests are often noted in certain clinical populations. We consider here some of these populations and, based on the neurologic and fMRI studies described, offer some explanation for why these effects are observed. Although we do not intend to imply that these clinical populations show neurologic lesions or processing exactly akin to the fMRI research, we do speculate on how these two lines of research may offer us a better understanding of these clinical phenomena.

Dichotic Listening in Children

There is a normal maturational time course toward adultlike levels of dichotic listening in children. Adultlike performance usually is not seen until 10 years of age. Because right ear performance typically matures before left ear performance, most children will show normal interaural asymmetries on dich-

otic tests throughout the development process. When this asymmetry exceeds what would be expected for a child of a given age, or when the asymmetry fails to resolve to adultlike levels, concern should be raised that the child has not followed the normal maturational time course for dichotic listening. Given performance sufficiently below normative values and use of the appropriate dichotic tests, a child who falls into this category would be expected to be diagnosed with CAPD in the area of dichotic processing. Considered below is the nature of dichotic processing maturation and what may contribute to performance deficits in children who trail behind the expected maturational time course.

Several studies have noted an increase in left ear performance with age, contributing to a progressively smaller right ear advantage. In a cross-sectional study, Pohl et al. (1983) examined binaural integration maturation of left and right ear performance on a dichotic digit test. They noted that the right ear advantage decreased significantly after age 5 years, and then again after age 7 years. This change in right ear advantage was driven primarily by improvements in the left ear, yielding smaller right ear advantages. Lamm and Epstein (1997) demonstrated on a binaural integration dichotic digits task that kindergarteners showed a smaller interaural asymmetry throughout a 1-year period primarily because the left ear performance improved. Persinger, Moulden, and Richards (1999) noted a significant decrease in left ear errors between 8 and 10 years of age using a dichotic word binaural separation task. A similar reduction in errors was not noted for the right ear. This improvement in left ear performance was replicated by Moulden and Persinger (2000) using this same test. Stollman, Neijenhuis, Jansen, Simkens, Snik, and van den Broek (2004) administered a dichotic CVC word test in a binaural integration task. Results revealed that 4 year olds performed significantly more poorly in the left ear when compared with children aged 5 or 6 years, while 5 and 6 year olds did not differ significantly. For the right ear, children aged 4 and 5 years both performed more poorly than 6 year olds. In contrast to studies showing asymmetric maturation of dichotic processing, several studies have noted that each ear tends to mature at the same rate (Bissell & Clark, 1984; Nagafuchi, 1970; Schulman-Galambos, 1977), Porter and Berlin (1975) suggested that conflicting findings of maturation effects on dichotic performance might be reconciled in part by consideration of the stimuli and tasks being used in each study.

In an attempt to provide the reader with some idea as to what the "normal" maturational course would be, we recreate in Table 14–1 normative values for various clinical dichotic processing tests administered to children with normal peripheral and central auditory function. These norms were established by Musiek and colleagues at the Dartmouth Hitchock Medical Center. Cutoff values reported in this table represent two standard deviations below the mean for the SSW and competing sentences tests, and three standard deviations below the mean for the dichotic digits. These norms reflect the asymmetric maturation of dichotic performance that is described above, as in most cases the right ear performance exceeds the left ear and the right ear appears to reach adult levels (~90%) more quickly. Partly as a result of this maturational trend, there are few dichotic test norms for children younger than 8 years of age. To better illustrate this maturational trend for interaural

Table 14–1. Dartmouth Hitchcock Normative Values for Dichotic Tests, Established by Musiek and Colleagues

Test	Age Range (Years, Months)	Left Ear	Right Ear
Dichotic Digits	7–7;11	55%	70%
	8–8;11	65%	75%
	9–9;11	75%	80%
	10–10;11	75%	85%
	11+	90%	90%
SSW	7–7;11	55%	75%
	8–8;11	75%	80%
	9–9;11	75%	80%
	10–10;11	80%	85%
	11+	90%	88%
Competing Sentences	7–7;11	–	–
	8–8,11	40%	82%
	9–9;11	75%	90%
	10–10;11	90%	90%
	11+	90%	90%

Cutoff values reported in this table represent 2 standard deviations below the mean for the SSW and Competing Sentences tests, and 3 standard deviations below the mean for the Dichotic Digits.

SSW, Staggered Spondaic Words.

asymmetry, the right minus left ear normative cutoffs from Table 14–1 also are plotted in Figure 14–2. All three tests reach 0% or near 0% interaural asymmetry by age 11 years or earlier. The competing sentence test shows the largest interaural asymmetry at age 8 years, but matures to adultlike levels one year earlier than the other two tests.

The corpus callosum likely plays some role in dichotic processing issues in children (Musiek et al., 1984; Musiek & Gollegly, 1988). This conclusion is reinforced by the observation that interaural asymmetry in dichotic performance is the hallmark of a CAPD-related dichotic deficit in children (Bellis et al., 2011). The corpus callosum is a heavily myelinated structure and rapid transmission of interhemispheric signals is dependent on the integrity of this myelin (Bear, Connors, & Paradiso, 2007; Musiek et al., 1984). Neuroanatomic studies have shown that children do not have adultlike levels of myelin at birth, and the normal time course of myelin develop-

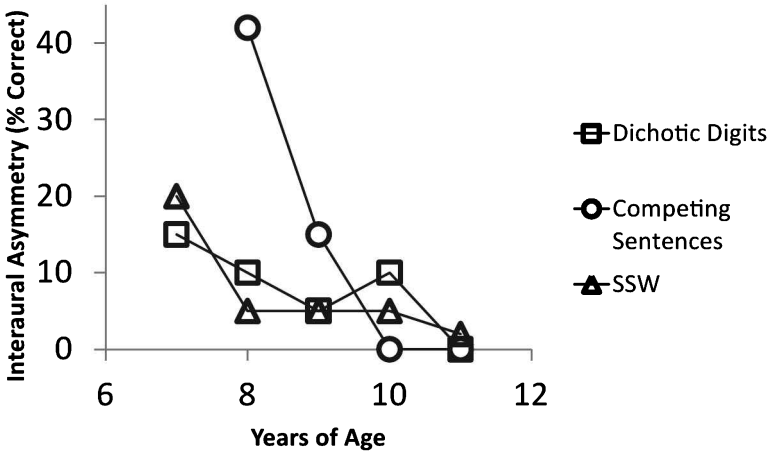

Figure 14–2. Interaural asymmetry values for three commonly used clinical dichotic processing tests, based on normative values for left and right ear dichotic test performance (Musiek, Dartmouth Hitchcock Medical Center Norms). Interaural asymmetry is calculated as right minus left ear percent correct, and 0 values indicate no interaural difference.

ment is approximately 10 to 12 years of age (Yakovlev & LeCours, 1967), which, not surprisingly, corresponds with the developmental time course of dichotic listening. Imaging data also has shown age-related changes to the corpus callosum past the first decade of life. Giedd et al. (1996) showed a linear increase in callosal area from 4 through 18 years of age, where increases in the posterior regions exceeded those seen in the anterior regions. In a longitudinal study, Westerhausen et al. (2011) measured callosal thickness and dichotic listening at 6 and 8 years of age. They noted that left ear dichotic performance was correlated with the thickness of the isthmus at age 6 years but not at age 8 years. Surprisingly, smaller callosal thickness was associated with better left ear dichotic performance. The authors suggested that this may indicate a refinement process of the interhemispheric fibers that occurs during maturation, during which axons may undergo beneficial pruning. This finding contrasts somewhat with other studies that have shown that larger callosal regions are associated with smaller interaural asymmetry (i.e., better dichotic performance), though not necessarily in pediatric samples (Clarke & Zaidel, 1994; Morton & Rafto, 2006; Yazgan, Wexler, Kinsbourne, Peterson, & Leckman, 1995).

In a classic study, Jerger, Johnson, and Loiselle (1988) compared performance on dichotic measures in children with neurologic involvement of the temporal lobe, those with neurological involvement not encompassing auditory regions of the central nervous system, and in children suspected of CAPD, but with no known neurological issues. Results showed that children with temporal lobe involvement generally had bilateral dichotic deficits, although one subject presented with a contralateral ear effect. Children suspected of CAPD tended to show left ear deficits in most cases, and

bilateral deficits in approximately 30% of the cases. Notably, children with lesions to areas of the central nervous system not involving auditory regions did not show any dichotic deficits. That children with lesions to the temporal lobe and those with suspected CAPD both showed dichotic difficulties reinforces the inference of CANS involvement in children shown to have dichotic deficits in the clinic.

There is also some evidence to suggest that auditory deprivation secondary to otitis media may contribute to dichotic listening deficits in children. Lewis (1976) reported data from two groups of two children of similar age, IQ, and hearing levels at the time testing. One group had a significant history of documented otitis media, while the other had a normal hearing history. Results showed that the interaural difference on dichotic digit testing was approximately 10% greater for the children with a history of otitis media. Welsh, Welsh, and Healy (1983) examined competing sentence performance of children with a history of otitis media who presented with normal hearing sensitivity at the time of testing. Twenty-three percent of the children showed an abnormality on one or more competing sentence measures. Of this 23%, 71% showed left ear, but not right ear, performance that was greater than two standard deviations below norms. Asbjornsen and colleagues (Asbjornsen et al., 2000, 2005; Klausen, Moller, Holmefjord, Reisaeter, & Asbjornsen, 2000) examined the impact of otitis media history on dichotic CV performance. They noted that children with a history of otitis media did not show as much improvement in left ear performance as normal hearing controls when attending to the left ear on binaural separation tasks. This suggests that a persistence of deprivation induced dichotic deficits.

Central Presbycusis

Another clinical population in which dichotic deficits frequently are noted are older adults with central presbycusis, or aging-related changes to the CANS. Reductions in dichotic performance have been shown to emerge between 60 and 70 years of age, and to become progressively poorer in each subsequent decade for binaural integration and/or binaural separations tasks (Alden, Harrison, Snyder, & Everhart, 1997; Andersson, Reinvang, Wehling, Hugdahl, Lundervold, 2008; Barr & Giambra, 1990; Bouma & Gootjes, 2011; Clark & Knowles, 1973; Goncales & Cury, 2011; Gootjes, Van Strien, & Bouma, 2004; Hallgren, Larsby, Lyxell, & Arlinger, 2001; Hommet et al., 2010; Jerger, Alford, Lew, Rivera, & Chmiel, 1995; Jerger, Chmiel, Allen, & Wilson, 1994; Martin & Cranford, 1991; Mukari, Umat, & Othman, 2010; Panek & Rush, 1981; Rodriguez, DiSarno, & Hardiman, 1990; Roup, 2011; Roup, Wiley, & Wilson, 2006; Takio et al., 2009). There is some evidence to indicate that males are affected by central presbycusis earlier than females (Golding, Taylor, Cupples, & Mitchell, 2006; Jerger et al., 1994).

The typical dichotic trend seen in central presbycusis is a decline in left ear performance with age (i.e., an increase in the right ear advantage). For example, Jerger et al. (1994) reported that the interaural asymmetry on the Dichotic Sentence Identification (DSI) test increased from 50 through 70 years, starting at roughly 5% interaural asymmetry with the right ear outperforming the left at 50 years, and increasing to about 10% asym-

metry in adults approaching 70 years. At 80 years, there was sharp increase in interaural asymmetry, with the right ear again performing better than the left. This was observed for both binaural integration and separation response modes. Bellis and Wilber (2001) also examined interaural asymmetry using a dichotic digits test, a common clinical measure. They administered the test in four different age groups: 25 to 25 years, 35 to 40 years, 55 to 60 years, and 70 to 75 years. Bellis and Wilber (2001) reported that the difference between left and right ears was significantly smaller for the younger two groups than for the older two, by approximately 3%. The two oldest groups also differed significantly from each other, but by less than 1%. Both findings have been replicated in several more recent studies (Andersson et al., 2008; Roup, 2011; Takio et al., 2009). That these interaural differences in dichotic performance emerge despite symmetrical peripheral hearing suggests a central locus for these effects (Neijenhuis, Tschur, & Snik, 2004; Roup, 2011; Roup et al., 2006).

Dichotic effects in central presbycusis are modulated by additional variables. First, response mode can significantly impact the size of the aging-related decrements observed on a dichotic task. Martin and Cranford (1991) administered dichotic digits to older adults twice, once requiring binaural integration and a second time requiring binaural separation. A significant effect of age was seen for both tasks; however, whereas overall score for the binaural integration task yielded a 13% difference between the youngest and oldest groups, the binaural separation task yielded only a 3% difference between groups. Rodriguez et al. (1990) also noting greater interaural symmetries on a binaural integration task relative to the binaural separation task. Type of test stimuli y also may affect dichotic performance in older adults, with digits discriminating younger and older subjects more so than sentence material on binaural separation tasks (Hallgren et al., 2001). Finally, stimulus uncertainty also appears to influence performance, where greater uncertainty is defined as larger variability in talker-voices encountered in each trial. Humes, Lee, and Coughlin (2006) reported that while younger and older adults show statistically equal performance on binaural separation tasks in which stimuli presented less uncertainty (e.g., fewer number of talkers across trials), younger adults performed significantly better than older adults for stimuli with greater uncertainty (e.g., a greater number of talkers across trials). For binaural integration tasks, younger adults always performed better than older adults regardless of the level of stimulus certainty. These findings suggest that older adults may benefit from high levels of stimulus certainty, but only on binaural separation tasks.

As with children, the corpus callosum has been invoked to explain to explain-age-related changes in dichotic listening. As mentioned above, the corpus callosum plays an important role in dichotic processing, and changes to this structure can yield changes to performance on dichotic tasks. In a now classic study, Jerger et al. (1995) examined dichotic performance in several groups that included older adults with aging-related hearing loss and dichotic deficits, and neurological subjects with lesions of the corpus callosum. They observed that older adults showed a left-ear deficit on dichotic tasks that

was nearly identical to that found for the neurologic group. The similarity in these findings provides support for the suggestion of callosal involvement in older adults with dichotic deficits. Gootjes et al. (2006) found that corpus callosum size was correlated with dichotic performance in healthy older subjects. Specifically, decreased size of the isthmus and splenium was associated with increased interaural asymmetry on binaural separation tasks, approximately 18% regardless of which ear was attended.

Other studies have emphasized the role of regions other than the corpus callosum in this age group. Gootjes, Scheltens, Van Strien, and Bouma (2007) used imaging in older adults to assess contributions to dichotic listening. For binaural integration tasks, they noted that right ear performance was correlated with abnormal hyperintensities in frontal and parietal regions of both hemispheres, while left ear performance was correlated only with hyperintensities in frontal, temporal, and occipital regions of the left hemisphere only. The authors interpreted these findings to mean a different processing route for left and right ear signals on dichotic tasks.The recruitment of the frontal and parietal regions likely emphasizes the contributions of attention on this task. Interestingly, controlling for corpus callosum variables did not change the significance of these correlations, suggesting that the present findings and callosal mechanisms may reflect separate contributions to dichotic processing.

Studies of cognition also have shown a relationship between this variable and dichotic listening in aging. Hommet et al. (2010) reported that both left and right ear performance on binaural integration tasks were significantly correlated with a measure of attention and cognition, with approximately 30% shared variance. Humes et al. (2006) noted that binaural separation performance was significantly correlated with a digit-span memory task, with shared variance ranging from 30% to 40%. Dichotic tests also have been shown to be related to reaction time on a variety of different auditory tasks in the aging population (Bellis & Wilber, 2001; Hallgren et al., 2001). Jerger, Jerger, and Pirozzolo (1991) found that speed of processing, as indicated by a digit symbol task, was significantly correlated with performance on the DSI. Notably, many of these studies have not examined the relationship between interaural asymmetry and cognition. Jerger, Mahurin, and Pirozzolo (1990) observed that interaural asymmetries on dichotic tests may more likely be associated with central auditory declines than with cognitive deficit, as cognitive deficits would typically not be expected to affect the ears asymmetrically. Thus, it is unclear if these cognition relationships would persist for interaural asymmetry dichotic calculations.

Clinical Dichotic Tests

There are a wide range of clinical tests that can be used in the assessment of dichotic function. We limit our discussion to the more common diagnostic behavioral tests used for dichotic testing, and do not cover screening tests of this ability. There are also additional diagnostic tests of equal utility that are not mentioned here because they are uncommonly used. Diagnostic dichotic tests vary widely in the stimuli employed, response modes used, and overall clinical feasibility. Wherever possible, we comment on the relative utility of some dichotic test over

others; however, a reasonably good argument could be made for any of the tests discussed below. For tests that have pediatric normative values, these values are included in Table 14–1. See Chapter 10 for discussion of screening.

Dichotic Digits

The Dichotic Digits Test (Musiek, 1983a) is a measure of binaural integration that typically employs a verbal report mode. Two separate digit pairs are presented simultaneously to the ears, with the onsets of each digit aligned between the ears. Patients are asked to repeat all four digits, and are encouraged to guess even if they are not completely sure of all numbers heard. There are three practice trials and 20 trials in the test proper. It is generally recommended that the test be presented at 50 dB sensation level (SL) re: PTA or SRT. If this level is uncomfortable for the patient, then it can be presented at most comfortable loudness (MCL) as long as interaural presentation levels are within 5 to 10 dB. Each ear is scored separately, and a result is considered abnormal if one or both ears are below normal limits. Normative values for adults with normal peripheral hearing are 90% for each ear. Norms for adults with mild to moderate hearing loss are decreased to 80% correct for each ear. This reduces the number of patients misdiagnosed with dichotic deficits because of confounding peripheral issues (Musiek, Gollegly, Kibbe, & Verkest-Lenzl, 1991). The sensitivity of the Dichotic Digits Test to CANS dysfunction has been shown to range from 70% to 90%, with specificity ranging from 80% to 90% (Hurley & Musiek, 1997; Musiek et al., 2011). Advantages of this test include its good sensitivity and specificity, its resistance to hearing loss effects due to its small closed set, and its quick administration time (<5 minutes). It should be noted that the digits test may be more dependent on working memory than some other dichotic tests (Lawfield, McFarland, & Cacace, 2011). See Chapter 18 in Volume 2 of the Handbook for discussion of dichotic testing and working memory.

Staggered Spondaic Word Test

The Staggered Spondaic Word (SSW) test (Katz, 1968) is a measure of binaural integration that uses a verbal response mode. Two different spondees are presented to each ear. These stimuli are temporally offset relative to each other, such that one spondee occurs first and "leads," and the other spondee occurs second and "lags." The second syllable of the leading spondee overlaps in time with the first syllable of the lagging spondee. A total of 40 spondee pairs are presented, which the patient is asked to repeat. The test is administered using a presentation level of 50 dB SL re: PTA or SRT in each ear. Although Katz describes a detailed scoring paradigm that is thought to extract additional information from patient responses, the test also can be scored as a straightforward measure of binaural integration. This allows the audiologist to score each ear separately based on the correct repetition of the spondee. Normative values for adults are 90% in the left ear and 88% in the right. Separate norms have been established for patients with peripheral hearing loss (Amerman & Parnell, 1980). The SSW is advantageous in that it is a measure of binaural integration that includes stimuli more complex than digits.

Dichotic Words Listening Test

The Dichotic Words Listening Test (DWLT) (Roberts et al., 1994) is a measure of binaural integration that uses a verbal response mode. The patient is presented 20 monaural practice items, during which they adjust the volume of the test to comfortable levels. The actual dichotic test is then administered at these levels in a short form (i.e., 30 dichotic pairs) or long form (i.e., 60 dichotic pairs). The test is scored as left ear, right ear, and "both ear" performance (e.g., left + right ear score), reflecting performance at one or both ears in the binaural integration task. Research has indicated that the short and long forms are roughly equivalent (Roberts et al., 1993). Although we do not reprint them here, normative values for adults for each decade of life from 16 years through approximately 80 years of age are provided by Meyers, Roberts, Bayless, Volkert, and Evitts (2002), while normative values for children have been reported by Roberts et al. (1993). For older adults, Meyers et al. (2002) noted a general decrease in performance with aging that does *not* appear to affect the ears asymmetrically. In children, there is gradual increase in left ear performance between 5 and 7 years of age. The test also appears to be sensitive to neurological dysfunction in pediatric and adult patients (Meyers et al., 2002; Roberts et al., 1993).

Competing Sentences

The Competing Sentences Test (Willeford, 1977) is a measure of binaural separation that typically employs a verbal response mode. Two separate sentences that are semantically related are presented to each ear (e.g., "This watch keeps good time" in the left ear and "I was late to work today" in the right ear). Patients are asked to repeat what they heard in either the left or right ear. They are told to indicate which words they did hear if they do not understand the entire sentence. A total of 20 trials are presented, 10 in which the patients attend to the left ear and another 10 in which they attend to the right. It is our experience that some patients benefit from an additional training trial in each ear that familiarizes them with the task. The target practice sentence is presented monaurally so they know what to listen for, and then again with dichotic competition. (See Chapter 18 for discussion of the benefits of presenting central tests in a nonmanipulated trial condition to assist in sorting cognitive and language from central auditory issues.) The test is typically administered at 35 dB SL re: PTA or SRT in the attended ear and 50 dB SL in the unattended ear. There are a variety of scoring schemes that have been described for this measure. A common method is to allot 10 points if the sentence is said 100% correctly, 7.5 points if one keyword is missed, 5 points if half of the sentence is repeated correctly, 2.5 points if one keyword is identified, and 0 if no words are repeated correctly. Normative values for adults are 90% in each ear. There are no normative values for this measure established for peripheral hearing loss. Advantages for this test are that it has greater ecological validity than some of the other dichotic tests. The test has better specificity but worse sensitivity than the dichotic digits in the assessment of CANS dysfunction (Musiek, 1984; Musiek et al., 2011). Disadvantages of the measure are its high linguistic load, which could confound interpretation of

bilateral deficits, and variations in scoring schemes used by different clinics.

Dichotic Sentence Identification

The Dichotic Sentence Identification (DSI) test (Fifer, Jerger, Berlin, Tobey, & Campbell, 1983) uses speech stimuli, but a nonverbal response mode. Two separate sentences are presented to each ear that are several orders of approximation away from conversational English (e.g., "Go change your car color is red"). The sentences are printed on a response board next to a number, and the patient's task is to indicate which numbers correspond to the two sentences they heard (e.g., binaural integration). It has also been used in binaural separation mode, where patients indicate the number of the sentence heard in only one ear (Jerger et al., 1994). It is recommended that the test be administered at 50 dB hearing level (HL) or 50 SL re: PTA. Fifer et al. (1983) noted that since the test is scored as an interaural performance difference, using HL as opposed to SL, or vice versa, has no significant impact on performance. Patients are provided with 10 monaural practice trials, a total of 5 in each ear, followed by 20 dichotically presented trials. For the test proper, a total of 90 dichotic items are presented. The effects of hearing loss on this test have been well established. For patients with better ear pure tone averages (PTAs) less than 40 dB HL and interaural PTA differences less than 20 dB, a >16% interaural difference on the DSI is considered below norms. For patients with better ear PTAs between 40 and 59 dB HL and interaural PTA differences less than ~26 dB, a >38% interaural difference on the DSI is considered abnormal. The test is not recommended for more severe PTA hearing losses or larger peripheral interaural asymmetries, as it is difficult to separate central from peripheral effects.

Dichotic CVs

The use of dichotic CVs is common in psychological studies of sensory laterality and attention, where they are typically administered as either binaural integration or separation tasks (i.e., free or directed recall). Administered in this context, it is thought that binaural integration tasks assess primarily bottom-up processes, since attention is not being directed toward a specific ear, while binaural separation tasks assess primarily top-down processes since attention is focused toward either the left or right side. In the clinic, however, these stimuli are uncommonly used. This may be because dichotic CV test performance appears to be more influenced by peripheral hearing loss than other dichotic tests (Roeser, Johns, & Price, 1976; Speaks, Niccum, & Van Tasell, 1985).

Dichotic Rhyme

Another test sometimes used in the assessment of dichotic function is the Dichotic Rhyme Test (Musiek et al., 1989; Wexler & Halwes, 1983). This test differs slightly from other dichotic measures in that it makes use of a "fusion" phenomenon using the initial consonants of the dichotically presented words. Each stimulus is a CVC, where the VC component of every dichotically presented CVC pair is identical. The two initial consonants differ between the ears, but because of

their production elements (e.g., voicing), only one of the consonants is perceived. Lists are arranged in such a way so that in normal hearing individuals, approximately 50% of the right ear stimuli will be perceived, while the remaining ~50% of perceived stimuli will be those that originate from the left ear. The patient is asked to simply repeat the word heard on each trial. Patients are not made aware that the stimuli differ slightly between the ears, nor is there generally a perception of two auditory stimuli. Normative values for adults are 30% to 60% in the left ear and 32% to 78% in the right ear, a much larger range than typically is encountered for adult normative values on other dichotic tests. A main advantage of this design is it reduces the influence of attention on dichotic performance (Shinn et al., 2005). As with many other dichotic tests, it has been shown to be sensitive to pathologies of the corpus callosum (Musiek et al., 1989). Therefore, it is assumed that this measure utilizes traditional dichotic pathways, at least in part. As is the case with most dichotic tests, a 50 dB SL re: PTA or SRT presentation level is used.

Development of New Dichotic Tests

Using current digital technology, clinicians and researchers can create their own dichotic tests using a variety of waveform editors, such as Audacity. Good-quality monaural speech recordings can be made using these programs with an appropriate microphone. Assignment of one monaural recording to the left channel of a stereo sound file and the other monaural recording to the right channel of this file allows for the creation of dichotic stimuli. During stimulus creation, the recordings in the left and right channels can be carefully aligned using the waveform editor. Subtle interaural level differences between channels that were inherent to the original recordings can be controlled for to some degree by adjusting the individual channel amplitudes or normalizing the left and right channels. Indeed, these software programs provide robust tools that, with some practice, allow one to generate new dichotic tests. A more extensive undertaking would be the establishment of normative values, as well as investigating the reliability and validity of any new stimuli created.

Interpreting Dichotic Tests

Scoring Tests

Over the course of several decades of research on dichotic listening, there have been a variety of methods recommended for calculating dichotic performance. For most tests described above, normative values are generally provided for the percentage performance at each ear. Although a useful calculation, this type of comparison does not identify what degree of interaural asymmetry would exceed normal performance on a dichotic test. As dichotic deficits are frequently asymmetric, normative values for interaural difference can provide another useful way to determine if an abnormality is present. Indeed, Moncrieff (2006) suggested that the occurrence of dichotic issues goes under reported if one only examines absolute ear score. Using the competing words subtest of the SCAN-C,

she noted that the occurrence of binaural integration deficits using standard scores that combine ear performance was 23%. When also including patients with abnormal interaural asymmetry in this total, the occurrence of dichotic issues increased by more than half to 51%.

In addition to possible improvements in detection of dysfunction, it is also thought that computation of interaural asymmetry controls for some within-subject variables that presumably are comparable at the left and right ears and have linear effects. For example, if someone had an issue with a cognitive function, such as working memory, we might expect that negative influence of that issue to have a relatively equal effect on the left ear and right during dichotic testing. By computing the dichotic performance difference between the ears, we may subtract out the negative impact of this variable. These "intratest" measures have been shown to discriminate children with CAPD from those with supramodal issues, such as attention deficit hyperactivity disorder (ADHD) (Bellis et al., 2011). In these cases, children with CAPD show a much larger interaural asymmetry than children with more global deficits. It should be noted, however, that computing interaural asymmetry does not control for any interactive effects between cognition and central auditory dysfunction. See Chapters 11 and 18 for discussions of strategies for distinguishing effects of peripheral hearing loss, cognitive issues, and CAPD for differential diagnosis.

The easiest way to calculate interaural asymmetry would be simply in terms of the percentage difference between the ears. If one subtracts left ear performance from right ear performance, positive values indicate that right ear performance exceeds the left ear, and negative values indicate the opposite. An alternative interaural difference calculation was reported by Halwes (1969), who suggested dividing the difference by the total left ear and right ear percent correct score and multiplying the result by 100. This yields a normalized interaural asymmetry metric that is relative to the patient's total percent correct across the two ears.

Unfortunately, with the exception of the DSI, none of the diagnostic tests described above have normative values established for any interaural asymmetry calculation. Bellis et al. (2011) reported values for interaural asymmetry on dichotic digits in children with CAPD who were approximately 10 years of age. Using mean and standard error values reported in her figures, we can estimate the lower limit of the confidence interval for this group to be approximately 16%. Thus, patients 10 years of age who exceed 15% asymmetry on the dichotic digits do not have performance that is significantly different from similarly aged patients with CAPD. Weihing et al. (2007) also noted in adults with neurologic lesions that using a >14% dichotic digits interaural asymmetry to detect pathology yields sensitivity and specificity values that are near identical to what can be achieved based on performance at either ear alone.

A novel way in which to quantify dichotic performance was proposed by Bergman and colleagues (Bergman, Hirsch, & Solzi,1987a; Bergman, Hirsch, Solzi, & Mankowitz, 1987b) and is referred to as the "threshold of interference." Bergman described this metric using sentence-level dichotic materials. Sentences were presented in the attended ear at 20 dB SL re: sentence threshold, and the competing

sentence in the contralateral ear was initially presented at a low level. Over the course of the test, the presentation level of the competing message is increased gradually until the patient can no longer obtain at least 66% correct in the attended ear. When this level is obtained the audiologist has identified the threshold of interference. Bergman and colleagues (1987a, 1987b) reported that the threshold of interference is much smaller in individuals with lesions of the central nervous system. That is, these patients are unable to tolerate the same levels of competition as individuals with normal auditory function.

Structural and Cognitive Contributions to Dichotic Test Interpretation

Response Modes

It has been suggested that comparing binaural integration and binaural separation response modes can yield information regarding which mechanisms are contributing to an interaural asymmetry on dichotic testing. Jerger et al. (1994) proposed that dichotic performance can be defined as the linear combination of four factors: a general speech understanding ability (SU), a deficit introduced to left ear performance because the central auditory system generally shows left hemisphere dominance and contralateral pathways dominate (LED), a deficit introduced because of the increased cognitive task demands (i.e., greater attention) that primarily are present in the binaural integration response mode and affects both ears equally (TD), and an additional cognitive deficit reflecting an interaction effect of the LED and TD that affects the left ear (IE). The goal in using this model is to infer values for LED (i.e., the bottom-up structural contributions to dichotic processing), TD (i.e., the top-down cognitive contributions to dichotic listening), and IE (i.e., an additional cognitive deficit that affects the left ear). Binaural integration and binaural separation tasks are defined for the left and right ears using the Jerger et al. (1994) model in Table 14–2. To compute LED, we would subtract right ear and left ear binaural separation performance, or SU – (SU – LED). To compute TD, we subtract right ear binaural separation from right inte-

Table 14–2. Contributions to Binaural Integration and Separation Performance for the Left and Right Ears, as Proposed by Jerger et al., 1994

Task	Ear	Equation
Binaural Integration	Right	= SU – TD
	Left	= SU – TD – LED – IE
Binaural Separation	Right	= SU
	Left	= SU – LED

SU, Speech understanding; *LED*, Left ear deficit; *TD*, Task demands; *IE*, Interaction effect.

Detailed definitions for the units specified above are included in the text.

gration performance, or SU – (SU – TD). Computing IE is the most complex, and requires subtracting right and left ear binaural integration performance from right and left ear binaural separation performance, or ([SU – TD] – [SU – TD – LED – ID]) – ([SU] – [SU – LED]). These three computations yield the three variables of interest: LED (structural component), TD (cognitive component), and IE (an additional cognitive component).

Using these computations, Jerger et al. (1994) attempted to describe aging-related effects on dichotic listening interaural asymmetries. They examined the structural and cognitive contributions separately, where the structural contributions were LED and cognitive contributions were TD + IE as defined above. Interestingly, the cognitive contributions to interaural asymmetry were relatively constant from age 50 through 90 years, and reflected about 10% of the asymmetry effect. The structural contributions, however, varied as a function of age, ranging from 5% of the asymmetry at 50 years to 40% in the 80+ age range. They interpreted these findings to mean that most of the aging effects seen in dichotic interaural asymmetries can be attributed to structural mechanisms. This approach, of mathematically separating out the unique structural and cognitive contributions to dichotic listening, may provide a useful means to explain dichotic deficits in patients seen by audiologists, although more research is needed before meaningful use of this approach can be realized.

Utilization of different response modes also has been advocated by Martin, Jerger, and Mehta (2007), who examined this paradigm in children ranging in age from 8 to 13 years (mean age approximately 11 years) with and without dichotic deficits. Participants performed a simple dichotic task, designed to reduce the demands placed on psychological variables. On binaural integration tasks, a probe word was presented diotically, followed by dichotic presentation of two words. If the probe word was either of the dichotically presented words, participants pushed a button indicating "yes" the word was present, otherwise they indicated "no" the word was not in the dichotic pair. The next probe word would then be presented. Binaural separation tasks were similar, except that the probe word was presented to only one ear and the participant had to attend to that ear to determine if the probe word occurred on that side in the dichotic pair that followed. Children with normal central auditory function performed both tasks well, whereas the children with known dichotic issues had difficulty reporting back left ear stimuli on the binaural integration task. The authors suggested that if the behavioral deficit in dichotic listening was occurring because of CANS dysfunction, it would be present in both binaural integration and separation response modes as both utilize similar auditory structures. That the behavioral data showed difficulties only in the binaural integration response mode suggests that the deficit may be localized to the cognitive demands inherent to that task (e.g., attention, memory). Thus, it is seen how differential comparison of the two response modes may yield information regarding the locus of the dichotic deficit.

Interaural Presentation Level and Timing Effects

Subtle interaural differences in stimulus level and onset can influence dichotic test performance. For this reason, it is generally recommended that dichotic tests be

presented at 50 dB SL re: PTA or SRT, and that stimuli be carefully aligned when created. However, certain rehabilitative procedures (see Chapter 9 of Volume 2 of the Handbook) make use of these differences in order to train dichotic ability. For this reason, we consider here some of the effects of introducing interaural level and timing difference on patients' dichotic scores. In regard to interaural intensity differences, reducing the intensity of one of the dichotically presented stimuli will yield a decrease in performance in that ear and yield an increase in the performance in the contralateral ear (Hugdahl, Westerhausen, Alho, Medvedev, & Hamalainen, 2008; Speaks & Bissonette, 1975; Tallus, Hugdahl, Alho, Medvedev, & Hamalainen, 2007; Westerhausen et al., 2008). This effect occurs even though the reduced-intensity stimulus remains audible. Interestingly, forcing an interaural intensity difference to favor the weaker ear (on a dichotic measure) allows this ear to achieve normal performance. Thus, Musiek and Wilson (1979) observed that left ear deficits in split-brain patients could be significantly improved by decreasing the presentation level in the right ear.

A similar effect can be obtained by manipulating interaural timing differences, such that the onsets of the dichotic stimuli at each ear are misaligned relative to each other. In this context, one dichotic stimulus is called the "lead" stimulus, and its onset occurs first. The other dichotic stimulus is called the "lag" stimulus, and its onset follows the lead stimulus in time. The timing difference between the two ears typically is reflected in milliseconds (msec). Under such conditions, the lagging stimulus is more easily comprehended by the patient than the leading stimulus. Benefits start to emerge in the lagging ear at approximately 30 msec interaural difference, and reach maximum difference around 60 msec (Berlin, Lowe-Bell, Cullen, & Thompson, 1973; Studdert-Kennedy, Liberman, Harris, & Cooper, 1970). There are several explanations for why the lag effect occurs. Studdert-Kennedy et al. (1970) speculated that the leading stimulus cannot be properly processed because attention is being switched to the lagging stimulus while important acoustic information is being presented to the leading ear. The "recency effect" also may provide an explanation for why this lag effect occurs. The recency effect refers to the well-known psychological finding that more recent stimuli are better recalled than earlier presented stimuli (Duncan & Murdock, 2000; Greene, 1986).

Summary and Future Trends

Administration of dichotic tests can reveal robust ear advantages that are abnormally large in certain populations. These larger than expected ear differences often are referred to as "left ear deficits," since the right ear typically outperforms the left on dichotic listening. It is suspected that these left ear deficits contribute negatively to listening difficulties in thesepatients, such as children with maturational delays of the corpus callosum and older adults with aging-related degradations to this same region. Contributions to dichotic deficits arise from important structural regions of the CANS, including the ascending auditory pathways, the corpus callosum, and the auditory cortex. Successful dichotic listening also is dependent on contribu-

tions from the frontal lobe, including attention mechanisms.

Current clinical dichotic tests are able to successfully diagnose dichotic issues in patients with CAPD. However, contemporary research suggests there may be certain advantageous applications of dichotic tests that are being underutilized. For example, with the growing concern that cognitive factors may confound dichotic test interpretation (Moore et al., 2008), use of intratest measures, such as interaural asymmetry calculations, can assist in differential diagnosis (Bellis et al., 2011). If CANS dysfunction affects dichotic performance asymmetrically across the two ears, but cognitive dysfunction affects it symmetrically, then a computation of the ear difference can yield information on the nature of auditory-specific contributions. Although some studies have shown that the frontal lobe and cognitive processes can have asymmetric influences on dichotic listening (Hugdahl et al., 2009), the more typical finding that CAPD affects the ears asymmetrically whereas supramodal deficits affect the ears symmetrically allows one to effectively differentiate patients with CANS involvement from those with other central/cognitive issues (Bellis et al., 2011). Notably, a significant limitation in the application of the interaural asymmetry index is that many existing dichotic tests do not have norms for this calculation. Establishing these norms could greatly improve the diagnosis of auditory-based dichotic issues. Opportunities to improve application of dichotic testing also may be achieved by comparing performance on different response modes as described above. Generation of additional measures and stimuli that make use of multiple response modes within the same test could be beneficial in this regard.

Future dichotic tests also may attempt to reduce the ceiling effects commonly seen with current clinical dichotic measures. Although dichotic tests can discriminate patients with CANS dysfunction (Musiek et al., 2011), there is little variance in performance among participants who have normal CANS function. That is to say that most normally functioning respondents show no interaural asymmetry on common dichotic tests. Martin, Gibson, and Huston (2012) have suggested that ceiling effects can be reduced by using dichotic filtered words. These stimuli aim to increase the reliance on auditory processing without increasing the cognitive demands. This is contrasted with other ways of increasing dichotic test difficulty that may make the test more intensive cognitively, such as increasing the linguistic load or the semantic complexity of the sentence. Administered to normal participants, this paradigm has revealed that these stimuli yield larger ear advantages than unfiltered dichotic tests, thereby reducing the ceiling effect commonly noted on dichotic measures.

For many years, dichotic tests have provided useful diagnostic information in the assessment of central auditory dysfunction. Ongoing research across many disciplines aims to enhance the diagnostic accuracy of these measures while simultaneously identifying what dichotic tests tell us about structural integrity and cognitive ability. Importantly, dichotic listening skills reflect meaningfully on the communication challenges encountered by patients who report hearing complaints not predicted by their peripheral hearing levels. With emerging treatments for patients with dichotic deficits (see Chapter 9 of Volume 2 of the Handbook for a review), it is crucial that audiologists evaluate dichotic listening to allow

them to offer recommendations for specific treatmentof dichotic deficits commonly seen in CAPD.

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CHAPTER 15

TEMPORAL PROCESSING TESTS

JENNIFER BROOKE SHINN

Introduction

Auditory temporal processing can be defined as the perception of sound or the alteration of sound within a restricted or defined time domain (Musiek, Shinn, Jirsa, Bamiou, Baran, & Zaiden, 2005). One could argue that temporal processing is the fundamental component of most auditory processing capabilities. This is strongly supported by the fact that many, if not all, characteristics encompassing auditory information are in some way influenced by time (Pinheiro & Musiek, 1985). Temporal processing can be observed at many levels, ranging from the most basic level of neural timing in the auditory nerve to cortical processing for binaural hearing and speech perception (e. g., voice onset time to prosodic detail).

In diagnosing central auditory processing disorder (CAPD), specifically with respect to temporal processing, there are two key knowledge domains with which clinicians should be conversant. First, one should possess a thorough understanding of temporal processing and its four subcomponents: (1) temporal ordering or sequencing, (2) temporal resolution or discrimination, (3) temporal integration or summation, and (4) temporal masking. In addition, one must be keenly aware of the diagnostic tools available, as well as the limitations of current diagnostic techniques.

The purpose of this chapter is to review: (1) clinical behavioral tests that assess the ability to analyze acoustic events over time, (2) the contribution of temporal tests to differential diagnosis, (3) the functional impact of temporal

processing deficits, and (4) primary treatment strategies for temporal processing deficits. The reader is referred to Chapter 2 for elaboration of some of the psychoacoustic principles underlying aspects of temporal processing, to Chapter 16 for a review of tests of binaural interaction, and to Chapter 17 for a review of electrophysiological measures of central auditory nervous system (CANS) timing.

Categories of Temporal Processing and Their Clinical Assessment

The underlying neural mechanisms of temporal processing are not well understood. We do know, however, that although brainstem and subcortical mechanisms support efficient processing, temporal processing appears dependent primarily on cerebral and interhemispheric processing (Pinherio & Musiek, 1985). Before discussing how best to evaluate temporal processing, it is necessary to review the types of temporal processes and their components. (See Chapters 3 and 5 for discussions of neurological timing in the CANS.)

It is well accepted that there are four categories of temporal processing of auditory signals that are critical to central auditory processing abilities. These include: (1) temporal ordering or sequencing, (2) temporal resolution or discrimination, (3) temporal integration or summation, and (4) temporal masking. Although auditory temporal processing has been studied extensively in the research arena, there is a paucity of information on clinical applications. Clinical measures of temporal ordering and temporal resolution are available; however, there are no clinically feasible measures of temporal integration (i.e., summation), or of temporal masking. As discussed below, thorough evaluation of multiple temporal processes is important not only for proper diagnosis, but for targeted intervention designed to improve specific temporal processing deficit(s).

Temporal Ordering or Sequencing

Temporal ordering, or sequencing, refers to the processing of two or more auditory stimuli in their order of occurrence in time (Pinheiro & Musiek, 1985). This has been a highly investigated phenomenon, particularly because of its importance in speech perception (Fu, 2002; Hirsh, 1967; Neff, 1961; Pichora-Fuller & Souza, 2003). Accurate temporal ordering requires that both the left and right hemispheres be anatomically and physiologically intact. Temporal ordering is a complicated process, and while it requires more than sequencing of acoustic events, this is the primary component. In the animal model, it has been demonstrated that following bilateral ablations of the auditory cortex, auditory pattern recognition is severely impaired (Colavita, Szelgio, & Zimmer, 1974; Colavita & Weisberg, 1977; Diamond & Neff, 1957). It also has been demonstrated by Musiek and colleagues (1980, 1987, 1990) that both split-brain patients (i.e., patients with a surgically sectioned corpus callosum) and patients with cerebral lesions demonstrate significant deficits on frequency and duration pattern tests. The effect of cochlear hearing loss also has been investigated for both types of patterning tests (Musiek et al., 1987, 1990). Interestingly, the Duration Pattern Test seems relatively

resistant to the effects of cochlear lesions because it is not highly dependent on good frequency discrimination (Musiek, Baran, & Pinheiro, 1990).

The ability to properly recognize, identify, and sequence auditory patterns involves several perceptual and cognitive processes (Pinheiro & Musiek, 1985). These processes are not restricted to one hemisphere alone, but rather require integration of information from both hemispheres across the corpus callosum (Musiek, Pinheiro, & Wilson, 1980). Until the split-brain study by Musiek and colleagues (1980), it was assumed that the temporal lobe of the left hemisphere was primarily responsible for temporal sequencing (Efron, 1963; Halperin, Nachshon & Carmon, 1973). We now know that neither hemisphere alone can adequately process temporal patterns. Hence, pattern tests are sensitive to hemispheric lesions, as well as interhemispheric dysfunction (Musiek & Pinheiro, 1987; Musiek et al., 1990).

Figure 15–1 illustrates the responsibility of each ear, each hemisphere, and the corpus callosum with respect to processing of tonal stimuli. Information regarding contour recognition must be processed in the right hemisphere and then passed via the corpus callosum to the left hemisphere, where the linguistic label is applied to the signal. In cases where poor verbal responses are produced in the presence of normal

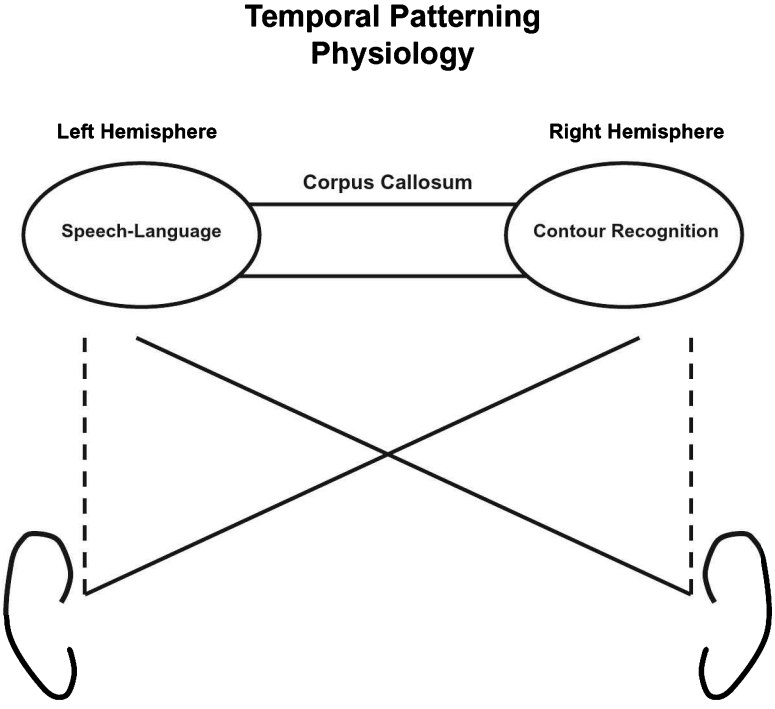

Figure 15–1. Illustration of the physiological mechanisms required for temporal patterning. Contour recognition occurs in the right hemisphere and information is transferred across the corpus callosum to the left hemisphere where linguistic process occurs.

hummed responses, a perceptual auditory deficit cannot be assumed. Rather, an individual who can hum but not verbally label tonal patterns, most likely suffers from dysfunction in interhemispheric transfer to the left hemisphere, or dysfunction in the left hemisphere, the presumed site of the speech processor (Pinherio & Musiek, 1985). Swisher and Hirsh (1972) reported evidence that the auditory cortex is responsible for not only the organization of neural events, but also for maintaining the proper sequence of the acoustic stimuli. In their study, individuals with left temporal lobe lesions needed a much greater onset time difference to identify the order of two rapidly presented acoustic stimuli.

The ability to properly sequence auditory information also is affected by subject and stimulus variables including: (1) subject training, (2) type of stimuli, (3) number of stimuli, 4) duration of stimuli, and (4) rate and manner of stimulus presentation (Pinheiro & Musiek, 1985). All are critical variables that should be taken into account when developing and administering pattern tests. For example, anyone who has ever administered temporal pattern tests is aware of the important effect of subject training. Efron (1963) demonstrated a significant difference in test performance between naïve and trained listeners. (This is true for almost all temporal processing tasks, as these are highly trainable processes.) Additionally, the type of stimuli presented is of importance. Stimuli used to date have included noise, tones, clicks and speech. Also, as the number of components within the stimulus increases, so does the complexity of the task (i.e., three component patterns are more difficult to process accurately than three component patterns). Perhaps one of the most critical considerations is the effect of temporal integration, as reflected in the duration of the components. If components of less than 200 msec duration are used, then subjects will perform more poorly than for those components greater than 200 msec (Warren & Obusek, 1972). This also holds true for the rate of presentation, where shorter interstimulus intervals result in poorer performance (Leshowitz & Hanzi, 1972).

Evidence dating back to the early 1960s (Milner, 1962) suggests that individuals with temporal lobe lesions demonstrate poor performance when asked to identify differences in sequences of tones. Swisher and Hirsh (1972) identified a deficit in the ability of subjects with damage to the cerebrum to order tones with respect to their occurrence in time. A number of additional studies also demonstrated temporal ordering deficits in patients with lesions of the cerebrum (Belmont & Handler, 1971; DeRanzi, Faglioni, & Villa, 1977; Karaseva, 1972).

Undoubtedly the most widely used clinical tests of temporal ordering are the Frequency Pattern Test and Duration Pattern Test (Emanuel, 2002, 2011). The Frequency Pattern Test was first introduced by Pinheiro and Ptacek in 1971. Patients are asked to verbalize the order of a series of three tones (Musiek, 1994). Due to their ease of administration and their efficiency (i.e., sensitivity and specificity), the pattern tests have gained rather widespread clinical acceptance (Emanuel, 2002). They are both sensitive (86%) and specific (92%) with respect to cerebral lesions of the CANS; however, their sensitivity to brainstem lesions is weaker (Musiek et al., 1987, 1990). Additionally, the Frequency Pattern Test bas

been established as an excellent tool to use with young children, ages 8 and older (Musiek, 1994).

The Frequency Pattern Test is composed of three tones that are either "high" (1122 Hz) or "low" (880 Hz) in frequency. As seen in Figure 15–2, there are six possible randomized patterns (HHL, LLH, HLH, LHL, LHH, HLL), with none of the patterns ever consisting of the same frequency appearing in triplicate (i.e., HHH or LLL). Each tone is 150 msec in duration with a 10 msec rise-fall time and an intertone interval of 200 msec. The Duration Pattern Test also is composed of three pure tones per pattern (Figure 15–3). Each tone is 1000 Hz and is either 250 msec (short) or 500 msec (long) in duration, with a 300 msec intertone interval. Again, there are six possible randomizations that occur (LLS, LSL, LSS, SLS, SLL, SSL). Both pattern tests utilize a 6-second interpattern interval (Musiek, 1990).

It is recommended that each test be administered at approximately 50 dB SL in reference to either the speech recognition threshold or the pure-tone average. Since there is little effect of intensity on performance on these tests, they can be presented at levels as low as 20 dB SL and yield essentially the same results (Musiek, 1994). Although tests of temporal processing are not completely resistant to the effects of hearing loss, pattern tests are particularly useful in the assessment of central auditory processing in individuals with peripheral hearing loss.

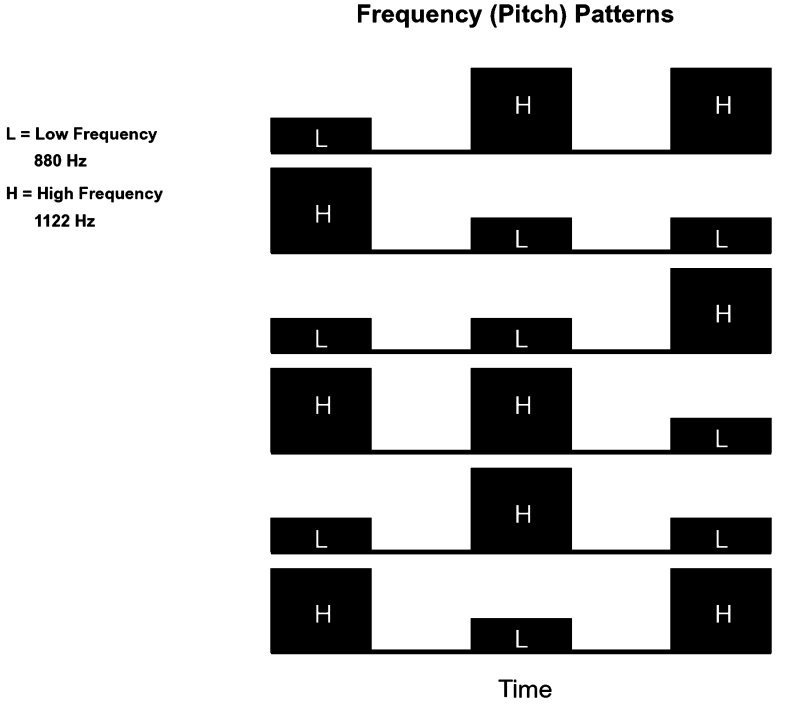

Figure 15–2. The six frequency patterns with time represented on the *x*-axis and amplitude on the *y*-axis.

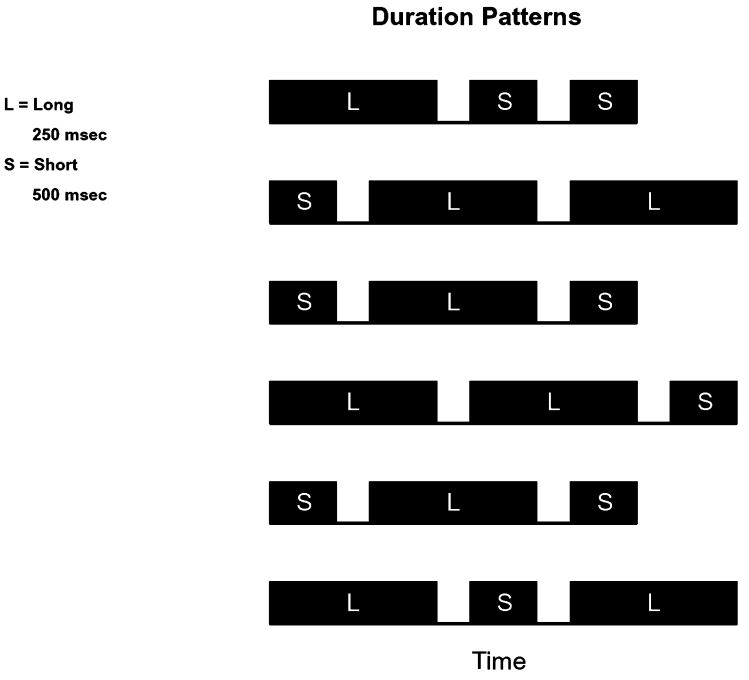

Figure 15–3. The six duration patterns with time represented on the *x*-axis and amplitude on the *y*-axis.

Subjects are instructed that they will hear three tones that vary in either pitch (for the Frequency Pattern Test) or duration (for the Duration Pattern Test). Subjects are asked to repeat the pattern that they hear by verbally indicating either "high or low" or "long or short." It is recommended that the examiner train the patient by first using visual cues (such as finger pointing) in conjunction with the auditory stimuli. Once this has been mastered, then the visual cues should be removed and the examiner should ensure that the patient is able to complete the task in the auditory only condition. Individuals should be encouraged to guess if they are unsure. Both pattern tests are scored in percent correct and have published normative data for both pediatric and adult populations (Tables 15–1A and 15–1B).

Temporal Resolution or Discrimination

Temporal resolution or discrimination refers to the shortest duration of time in which an individual can discriminate between two auditory signals (Gelfand, 1998). For brief sounds, this is generally about 2 to 3 msec (Phillips, 1999). The threshold for temporal resolution is known as *temporal auditory acuity* or *minimum integration time* (Greene, 1971).

Temporal resolution can be assessed utilizing a variety of methodologies. Historically, this has been achieved by determining the temporal modulation transfer function (TMTF) or gap detection threshold (GDT). TMTF assesses one's ability to detect amplitude modulation (Figure 15–4). According to Phillips (1997), one of the most common measures of

Table 15–1A. Normative Values for the Frequency Pattern Test Based On Two Standard Deviations Below the Mean.

Age	Normative Value
7	35%
8	42%
9	63%
10	78%
11	78%
12	80%

Source: Bellis, 2003.

Table 15–1B. Normative Values for the Duration Pattern Test Based On Two Standard Deviations Below the Mean.

Age	Normative Value
7	25%
8	35%
9	54%
10	70%
11	71%
12	73%

Source: Bellis, 2003.

Temporal Resolution

TMTF

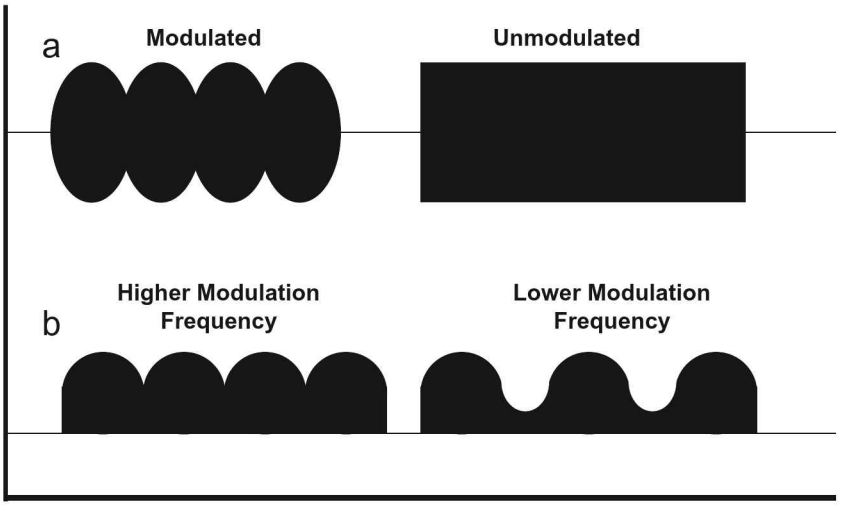

Figure 15–4. Lower and higher modulation frequency sounds used to determine temporal modulation transfer function.

temporal resolution using behavioral methods is gap detection (GD). Temporal resolution can be evaluated "within-channel," in which the stimuli use the same markers, or "between-channel," in which the markers are different (Phillips, 1997). These are mediated by two different operations. It is believed that within-channel processing relies on the detection only of the onset of the second

marker, whereas between-channel processing relies on the detection of the offset of the first marker as well as the onset of the second marker (Phillips, 2004). Our current clinical measures of temporal resolution have been designed to evaluate only within-channel processing. See Chapter 2 for discussion of temporal resolution, including a proposed clinical measure of TMTF.

GD tasks require subjects to indicate whenever they hear a "silent" interval embedded in an ongoing sound or noise burst (Figure 15–5). The interstimulus interval is varied and the GDT is defined as the shortest silent interval in a stimulus a listener is able to detect (Lister, Besing, & Koehnke, 2002). As there are no clinically available tests using TMTFs, the following discussion focuses on clinical measures of gap detection.

One of the issues surrounding the incorporation of temporal resolution measures into clinical practice is that the assessment of temporal resolution abilities has traditionally been accomplished with classic psychoacoustic GD procedures. Such measures often are not feasible in a clinical setting for a variety of reasons, which include the fact that

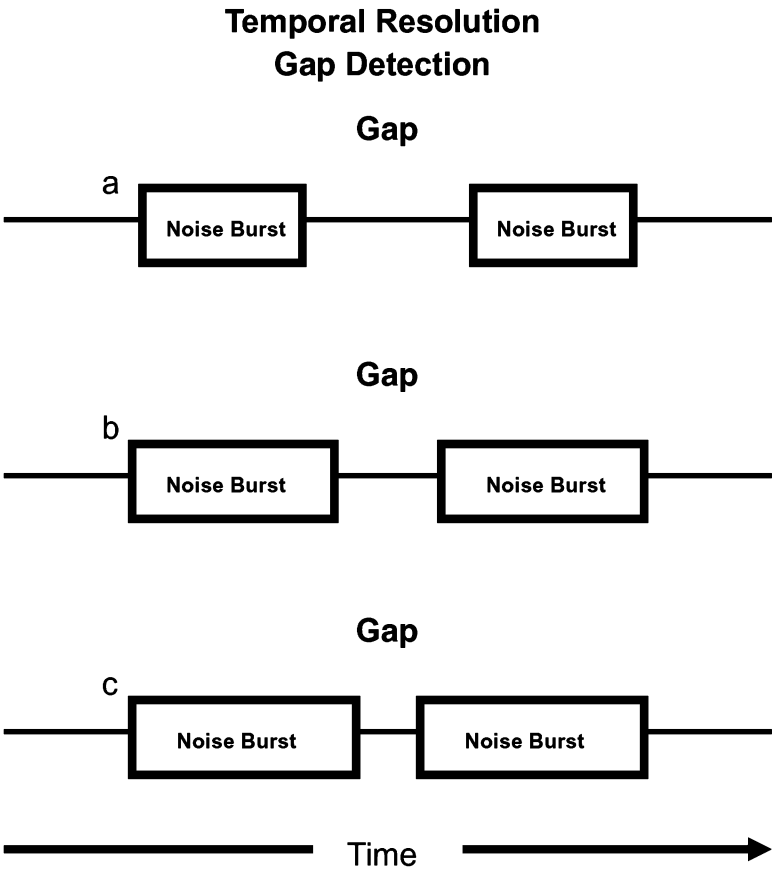

Figure 15–5. An example of gap detection. The progression from **a** to **c** demonstrates larger to smaller gap representations.

they are often time consuming, making them difficult to use within a test battery for patients or children who cannot tolerate long test sessions. Additionally, clinicians may find they do not have the instrumentation necessary to run these classic GD paradigms in the standard audiology clinic.

The investigation of temporal resolution through the use of GD paradigms is not a novel approach, but can actually be traced back to Garner in 1947. Numerous researchers since have investigated the phenomenon of temporal resolution using GD with many different stimuli. Many investigators have explored the effects of a variety of variables (e.g., age, hearing impairment) on temporal resolution abilities. However, as there are some controversies regarding the effects of certain variables, further research is still required. For example, it has been demonstrated that older subjects may present with increased GDTs in comparison with younger control subjects (Bertoli, Smurzynski, & Probst, 2002; He, Horwitz, Dubno, & Mills, 1999; Snell, 1997; Strouse, Ashmead, Ohde, & Grantham, 1998). Other studies, however, have placed in question whether GDT measures are affected by age (Moore, Peters, & Glasberg, 1992). Differences in stimulus and procedural variables may underlie differences in findings across studies. A broadband stimulus may be the best stimulus for use in GDT paradigms, as it is less likely to lead to variability across different age groups or as a function of peripheral hearing status (Musiek et al., 2005).

Until recently, the application of GD paradigms with pediatric populations had received little attention. Although GD research with pediatric participants is somewhat limited, one study reported a maturational effect for GD tasks in normal children (Trehub, Schneider, & Henderson, 1995). It also appears that children with learning disabilities demonstrate abnormal temporal resolution abilities based on GD procedures (Hautus, Setchell, Waldie, & Kirk, 2003). Taken together, these findings suggest that GD procedures should be a useful component of central auditory processing test batteries with pediatric populations.

One commercially available and frequently used test of temporal resolution is the Random Gap Detection Test (RGDT), a revision of the Auditory Fusion Test-Revised, developed by Keith (2000). Subjects are asked to indicate whether or not they have heard one or two tones or clicks either verbally or by raising one or two fingers (i.e., nonverbally). The tones are presented binaurally and are 15 msec in duration with a 1.5-msec rise-fall time. The clicks are 1 msec of white noise. One advantage of using tonal stimuli is that it allows the clinician to obtain frequency specific information regarding temporal resolution skills. There are four subtests, each of which uses nine interpulse intervals ranging from 2 to 40 msec. The RGDT is presented binaurally at 55 dB HL. Normative values for the RGDT tonal stimuli range from 6.0 to 7.8 msec. Currently, there are no published normative values for the click stimuli. Test administration requires approximately 10 minutes. Unfortunately there is only one list available, which limits the ability to retest patients.

The Gaps-In-Noise (GIN©) test is yet another clinical test of temporal resolution (Musiek et al., 2005). It is composed of a series of 6 sec segments of broadband noise. Each segment contains zero to three silent intervals or "gaps" per noise segment. The interstimulus interval between successive noise tokens

(segments) is five seconds and the gap durations presented are 2, 3, 4, 5, 6, 8, 10, 12, 15, and 20 msec. Ten practice items precede the administration of the test items. There are six tokens of each gap duration within each list. Additionally, there are four lists available for testing, which allows for test-retest comparisons. Although the test is administered through one channel, the second channel is used by the examiner to monitor and score the responses. Two measures are derived from the GIN. The approximate threshold (A.th.) for the GIN is defined as the shortest gap duration for which there are at least four of six correct identifications. This is an "approximate" threshold because the exact GDT is not determined in this procedure, in contrast to traditional psychoacoustic procedures. In addition to the A.th., the percentage of correct responses out of the total number of gaps (e.g., 36 out of 60 would be equal to 60% correct) can be computed for each ear.

The GIN is one of the few measures of temporal processing that has published data regarding sensitivity and specificity to CANS lesions. Specifically, the sensitivity of the GIN is 67% and the specificity is 94% (Musiek et al., 2005). The GIN appears to be more sensitive to cortical compromise as opposed to brainstem involvement (Musiek et al., 2005). This sensitivity is similar to the patterning tests, which suggests that temporal processing is primarily mediated in the cerebrum (Pinheiro & Musiek, 1985). In addition, test-retest and interlist consistency are high for the GIN test (Musiek et al., 2005). Finally, there are four available lists that demonstrate interlist equivalence (Musiek et al., 2005). This is important because equivalency across lists allows for comparisons for follow-up testing, various types of monitoring, and assessment of treatment effectiveness. It has been demonstrated that peak performance on the GIN is achieved between a 35 and 50 dB sensation level (Weihing, Musiek, & Shinn, 2007).

Mean thresholds for the GIN are slightly longer than traditional psychoacoustic gap detection thresholds. Most GD in noise thresholds with humans have been reported on the order of 2 to 3 msec (Moore, 2003; Phillips, 1999). The mean A.th's for the GIN are 4.8 and 4.9 msec for the left and right ears, respectively (Musiek et al., 2005). Differences in GDTs likely result from a number of procedural differences, including less training for subjects participating in the normative data collection for the GIN, and the use of somewhat larger gap intervals in the GIN than those used in many traditional psychoacoustic experiments.

The GIN also has well-established normative data for the pediatric population (Shinn, Chermak, and Musiek, 2009). Data indicate that that there is in fact no developmental effect seen in children between ages 7 and 18. Children age 7 demonstrate the same performance as adults. Similar to adults, no ear asymmetry is observed. These two findings would suggest that temporal resolution is likely an auditory process that develops early and symmetrically.

Although the RGDT and the GIN are both measures of temporal resolution, clinicians should be aware of the differences between these temporal tests. These differences include stimulus variables (tones versus noise), presentation mode (binaural versus monaural), response mode (verbal versus motoric), range of interpulse intervals, gap durations, normative values, and test time (Chermak & Lee, 2005). Given the importance of temporal discrimination to auditory processing, clinicians should

include a GD measure in their behavioral test batteries.

Several new clinically viable GD procedures have been introduced demonstrating increased interest in these kinds of temporal processing procedures and their clinical application (see for example Griffiths, Dean, Woods, Rees, & Green, 2001; Lister, Roberts, & Lister, 2006). One such procedure, the Adaptive Test of Temporal Resolution (ATTR), utilizes a computerized, adaptive pyschophysical methodology to evaluate temporal resolution. Thresholds obtained on the ATTR are similar (2.2 msec) to those obtained using traditional psychophysical laboratory procedures (Lister et al., 2006).

Temporal Integration

Temporal integration results from the summation or aggregation of neuronal activity as a function of the additional duration of sound energy (Gelfand, 1998). This summation results in threshold improvements as duration increases up to about 200 msec in normal hearing populations (Durrant & Lovrinic, 1995). As duration is decreased by a factor of 10 (i.e., one-tenth of its original duration), an increase of approximately 10 dB in threshold is observed. This relationship is referred to as a *time-intensity trade.* The time-intensity rule explains why a signal is perceived as being louder as the duration of a brief signal (less than 200 msec) is increased at suprathreshold levels. As depicted in Figure 15–6, temporal summation asymptotes at about 200 msec such that further increases in duration have no affect on threshold.

The primary pathological population on which temporal integration has been studied consists of individuals with cochlear involvement (Buss, Florentine, & Poulsen, 1999; Carlyon, Buss, & Florentine, 1990; Garnier, Micheyl, Berger-Vachon, & Collett, 1999; Moore, 1996; Papsin & Abel, 1988), although several investigations have studied populations with CANS involvement (Baru & Karaseva,

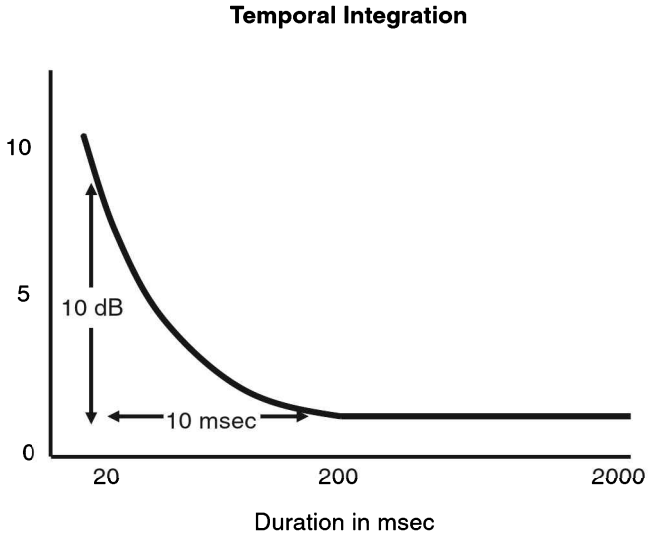

Figure 15–6. Temporal integration function demonstrating the time–intensity tradeoff.

1972; Cranford, 1984; Cranford, Stream, Rye, & Slade, 1982). Temporal integration has been found to be impaired in individuals with central auditory disorders, including temporal lobe lesions. Specifically, subjects with temporal lesions demonstrate elevated frequency difference limens for short duration tones in the ear contralateral to the lesion (Cranford et al., 1982). The finding of a contralateral deficit in temporal integration in cases of temporal lobe lesions has been demonstrated by several investigators (Baru, Gershuni, & Tonkonogii, 1964; Baru & Karaseva, 1972; Gershuni, 1971).

Acoustic stimulus duration is an important factor influencing temporal integration in individuals with normal hearing, as well as those with both peripheral (cochlear) and central disorders. If the duration of an acoustic stimulus (such as a tone) is decreased in duration from 200 to 20 msec, there is about a 10 dB increase in threshold in normal hearing individuals. If durations are increased beyond 200 msec, little change in threshold is noted in normal hearing subjects; however, if the duration is decreased to less than 20 msec, the threshold levels will increase in these same individuals (see Durrant and Lovrinic, 1977, p. 161). For individuals with central nervous system disorders, the short duration stimuli show a much larger differential (i.e., steeper integration functions) between the long and short stimuli, especially when the short tone is less than 10 msec (Baru & Keraseva, 1972; Olsen & Matkin, 1978, p. 395; Wright, 1978). As would be expected, these increased thresholds were found to manifest mostly in the ear contralateral to the side of the brain lesion with the ipsilateral ear performing essentially normally. It is of interest that individuals with cochlear disorders show about the same threshold differences for short and long tones (and possibly less difference) as those with normal hearing (Olsen & Matkin, 1978; Wright, 1978). Also of interest is that stimulus durations markedly affect frequency discrimination—especially for those with central auditory disorders (see Cranford, 1984). Given the different integration functions seen between individuals with normal hearing and those with central disorders, it is surprising that there has not been more recent research on temporal integration (also termed brief tone audiometry) in clinical populations.

It is important to note that absolute thresholds often are undisturbed in cases of CANS involvement (Bocca & Calearo, 1963). This is because stimuli such as pure tones are simple and static. However, dynamic stimuli (i.e., patterns, numbers and speech), such as those used in traditional psychoacoustic assessments of temporal integration are more sensitive to cerebral lesions (Evans, 1974). Clinicians should keep in mind that patients often exhibit CANS lesions despite normal pure tone findings, underscoring the need for inclusion of central auditory tests in audiological batteries. Unfortunately, brief tone audiometry, which was once used clinically to examine temporal integration, is not used at this time, leaving the audiologist with no clinically feasible, behavioral means to measure temporal integration in the clinic.

Temporal Masking

Temporal masking refers to masking that occurs when a signal and a masker do not overlap in time (i.e., the signal and masker are not presented simultaneously). Backward masking occurs when the masker follows the signal, and forward masking takes place when the

masker precedes the signal (Figure 15–7). Interest in temporal masking dates back to the early 1950s, and although the literature clearly demonstrates the effects of temporal masking, the exact mechanisms underlying this phenomenon are unclear (Durrant & Lovrinic, 1995). Temporal masking may reflect a difference in latencies of neural timing within the CANS; however, this has never been fully confirmed.

There are several major parameters surrounding temporal masking that deserve attention. These include the time interval between the masker and signal, masker level, masker duration, and the acoustic similarity between the masker and the signal. With respect to the time interval between the masker and the signal, assuming that all else is equal, temporal masking drastically decreases as the time interval between the masker and the signal increases. In the case of forward masking, when the time interval between the masker and the signal reaches or exceeds 200 msec, essentially no masking occurs (Durrant & Lovrinic, 1995). Interestingly, as noted above, this is the same duration at which temporal integration plateaus and reaches its minimum threshold. In backward masking, we see a reduction in masking effects at about 25 msec separation (Durrant & Lovrinic, 1995).

In a classic study in 1962, Elliott demonstrated several key principles of temporal masking. She found that backward masking is more effective than forward masking: Given the same time interval between maskers, more masking occurs when the masker follows the signal. Masking is more effective when the stimuli are delivered monotically as opposed to dichotically (with masker and

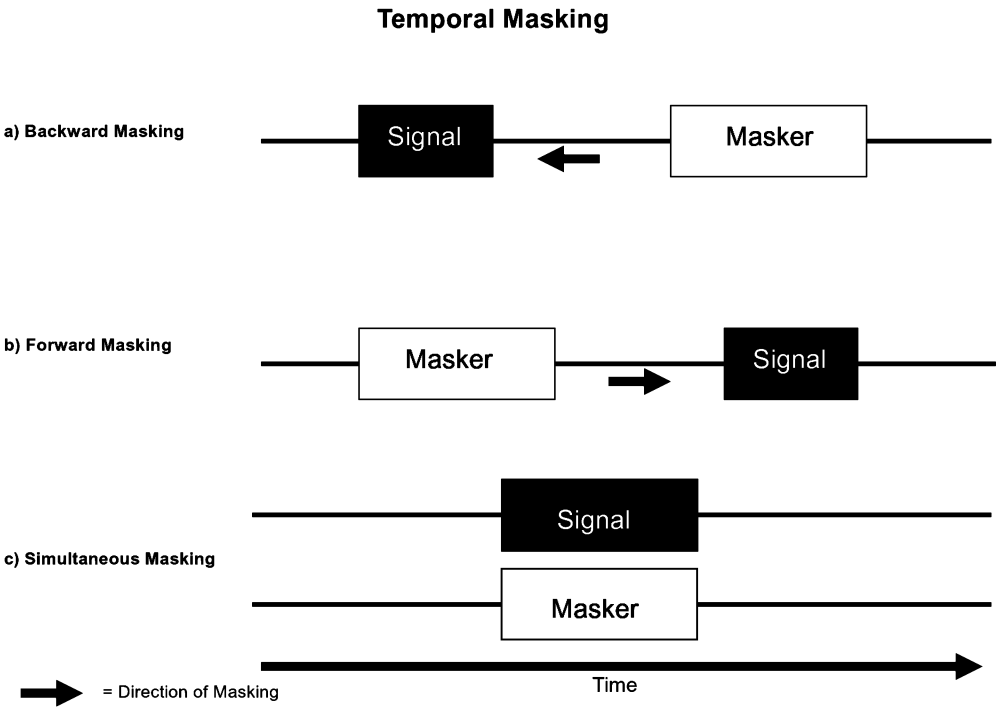

Figure 15–7. Illustration of backward (**a**), forward (**b**), and simultaneous (**c**) masking.

signal presented to opposite ears). Elliott (1967) later demonstrated that the duration of the masker influences forward but not backward masking. Another important variable is the frequency relationship between masker and the signal. The greatest degree of masking occurs when the masker and signal are identical, with the amount of masking decreasing as spectral differences increase between the masker and signal (Elliott, 1967; Wright, 1964). The amount of masking increases as the level of the masker increases, although the relationship between the intensity of the masker and the amount of masking observed is nonlinear. Generally, a 10 dB increase in masking level may result in a threshold shift of only approximately 3 dB (Gelfand, 1998). The simultaneous masking condition always produces greater masking than either backward or forward masking, if all parameters other than temporal distance between signal and masker are held constant (Wilson & Carhart, 1971).

Children appear more susceptible to the effects of backward masking than adults (Buss, Hall, Grose, & Dev, 1999; Hartley et al., 2000). Some researchers attribute this finding to children's poor temporal resolution abilities (Irwin et al., 1985; Wightman et al., 1989); however, given the adultlike norms obtained for children on the GIN, this may not be the contributing factor. Others attribute the findings to processing *inefficiency* in children (i.e., a deficiency in their ability to extract information) (Hill et al., 2002).

Unlike temporal ordering and temporal resolution, few studies have examined the effects of CANS lesions on temporal masking in children; however, children with language impairments have been investigated (Wright, Lombardino, King, Puranik, Leonard, & Merzenich, 1997). Wright and colleagues reported that children diagnosed with specific language impairment demonstrate a severe temporal processing deficit with respect to their ability to separate one sound that is rapidly followed by another sound (backward making). They concluded that this deficit may indeed degrade the "perception of brief acoustic elements of speech" (Wright et al., 1997). Moore and colleagues (2011) recently examined the development of auditory processing in children ages 6 to 11. They found that while there was relatively good within-subject reliability, there is large between-subject variability, particularly on backward masking paradigms.

The evidence that children with language and speech perception deficits demonstrate abnormal temporal masking skills is not surprising when one considers the potential influence of coarticulation for speech perception. Coarticulation, an aspect of speech production resulting from the concurrent movement of multiple articulators, leads to parallel transmission of acoustic information across neighboring phonemes and, therefore, to the potential for temporal masking. This linkage suggests that it is reasonable to hypothesize that speech and language impairments may co-occur with temporal masking deficits. This linkage also underscores the importance of studying temporal masking, as it is a process that may affect our basic communication function. Unfortunately, like temporal integration, there are no clinically available tests of temporal masking. One hopes that, in the near future, clinical scientists will recognize the need for clinical measures of temporal masking and begin to develop clinically feasible tests to assist in the differential diagnosis of temporal processing disorders. See Chapter 2 for proposed new psychoacoustic paradigms to assess temporal masking.

Clinical Considerations in Test Administration and Interpretation

In the following section, we pose and respond to several key questions that continue to arise with respect to administering and interpreting temporal processing tests as part of a central auditory processing test battery.

Which Temporal Tests Do Clinicians Administer as Part of Their Central Auditory Processing Battery?

There is a clear divide between what is occurring in the research arena relative to clinical practices. In 2002, Emanuel surveyed a large group of audiologists to determine auditory processing practices using an open-ended questionnaire. With regard to temporal processing tests, she found the most commonly reported test administered is the Frequency (Pitch) Pattern Test (76% of the respondents), followed by the Duration Pattern Test (44% of respondents). The Duration Pattern Test may be used less frequently because no normative values have been published for the pediatric population. It was reported that 28% of respondents performed the Auditory Fusion Test-Revised, whereas less than 20% of respondents reported using a gap detection measure. A follow-up to this investigation was recently published (Emanuel, Ficca, & Korczak, 2011) and revealed little change in practice patterns among clinicians since the initial survey nearly seven years prior.

Audiologists are beginning to have choices among tests of temporal processing. In comparing several older and more recent temporal resolution tests, Chermak and Lee (2005) noted that despite statistically significant differences among test means, there did not appear to be any clinically significant difference in performance across the temporal gap detection and temporal fusion tests examined for the 10 children with normal hearing studied and that, therefore, all four tests demonstrated high specificity. The GIN, although initially a bit more challenging to administer and score, yields strong face validity and reliability, good sensitivity, and excellent specificity (Musiek et al., 2005).

Is a Reversal Considered Correct on the Pattern Tests?

Reversals are not considered correct on either the Frequency Pattern Test or the Duration Pattern Test. Reversals should be considered errors and marked as such. It is recommended, however, that clinicians keep track of the number of reversals and consider them in their interpretation of test results. A significant number of reversals may be indicative of a larger, global perceptual deficit or gross neurological insult.

What Should the Clinician Try Next If a Patient Cannot Linguistically Label the Frequency or Duration Patterns?

If a child or adult is experiencing significant difficulty labeling the tones as high or low or short or long, the clinician should ask the patient to perform the test in the "hummed" condition. This simply requires having the patient hum rather than label the response. Informa-

tion derived from split-brain patients indicates that if an individual is unable to label the patterns but performs within normal limits in the hummed condition, this localizes the deficit to the interhemispheric pathways (i.e., corpus callosum), or possibly the left hemisphere (Baran & Musiek, 1999).The inclusion of a monaural low-redundancy test in the test battery should help the audiologist localize the deficit as either interhemispheric or left hemisphere (i.e., poor performance for low-redundancy stimuli in the right ear would suggest the deficit is in the left hemisphere). As indicated above, the left hemisphere is responsible for one's ability to linguistically label the stimulus, whereas the right hemisphere supports resolution of acoustic contour and melody. Information must travel across the corpus callosum through the interhemispheric pathway to the left hemisphere in order for the listener to be able to accomplish the linguistic labeling process. By having the patient hum the response, the necessity for interhemispheric transfer and left hemisphere processing is eliminated.

Here is a useful clinical tip: Consider the use of kazoos to help children hum. Occasionally children will become shy when asked to hum the response, because it is a bit like singing and most do not like to sing. Using a kazoo offers children a fun and nonthreatening way to participate in the test and for you, the clinician, an opportunity to obtain meaningful test results.

Can I Administer Any of These Tests in the Sound Field?

Research has demonstrated that generally there are no significant ear differences on temporal patterning tests administered under headphones in either normal or pathological populations. This finding suggests that both the Frequency Pattern Test and the Duration Pattern Test may be administered in the sound field (Musiek & Pinheiro, 1987). There are occasions where subjects demonstrate an ear asymmetry; however, this is rare. Nonetheless, the audiologist should recognize the tradeoff in saving time, at the small risk of reduced accuracy of the test administered sound field. If the patterning tests are administered in the sound field, then a total of only 45 items need be presented to the subject. This allows for a shorter test time without compromising sensitivity and specificity (Baran & Musiek, 1999). Although the patterning tests may be administered in the sound field, this is not true for tests of temporal resolution. Ear differences have been observed on the GIN in neurological populations; therefore, it is recommended that the GIN be administered under headphones.

I Am Concerned About Attention and Fatigue. Must I Administer All 60 Items (30 to each ear) on the Pattern Tests?

As indicated above, if the pattern tests are administered in the sound field, then only 45 items total need be presented to the subject. Additionally, if a subject presents with 18/20 either correct or incorrect responses under headphones or sound field, the test can be terminated. This allows for a shorter test time without compromising sensitivity and specificity. It should be noted that if a patient presents with 18/20 incorrect responses in the labeling condition, presenting the test in the hummed condition is *highly* recommended.

Why Are the Frequencies 880 Hz and 1122 Hz Employed for the Frequency Pattern Test?

This question makes a great graduate exam question. There is a simple and scientific reason why the frequencies of 880 Hz and 1122 Hz were chosen for the Frequency Pattern Test. If one examines the equal loudness contours (i.e., phon curves), it can be observed that these two frequencies are of equal loudness. This is important so as not to bias the test by introducing a loudness cue in addition to the pitch cue.

What Do I Do in Cases Where a Patient Is Referred for a Central Auditory Processing Evaluation But Presents With Hearing Loss?

There are several central auditory tests, including several temporal processing tests, which are relatively resistant to the effects of cochlear lesions. Of all of the temporal processing tests, the Duration Pattern Test appears to be the most resistant to hearing loss (Musiek et al., 1990). In their 1990 study, Musiek and colleagues demonstrated that the performance of patients with cochlear hearing loss on the Duration Pattern Test was almost indistinguishable from those without hearing loss. In fact, over 90% of those individuals with hearing loss, including those with moderate degrees of hearing impairment, performed within normal limits, thus making the Duration Pattern Test an excellent test to use in the presence of cochlear pathology. Other tests of temporal processing, such as the Frequency Pattern Test, may be used with mild, relatively flat hearing losses due to the fact that it uses relatively low frequency tones that might not be impaired in the typical patient with hearing loss. However, because there is a frequency or spectral element involved in this test, it may not be completely resistant to hearing loss (Musiek & Pinherio, 1987). Because the GIN incorporates a broadband stimulus, it also appears promising for use with individuals with cochlear lesions.

There Are Several Tools Available That Are Marketed As Temporal Processing Tests. Which Should I Use?

The answer to this question hinges on test sensitivity (i.e., the ability of a test to correctly detect a disease or disorder when it is truly present) and test specificity (i.e., the ability of a test to correctly identify an individual as normal when they do not present the disease of disorder). In a perfect world, all tests would yield sensitivities and specificities of 100%; however, this is nearly impossible to obtain because there is generally a trade-off between test sensitivity and specificity. That is to say that as sensitivity increases, specificity decreases, and vice versa. (See Chapter 12 for discussion of sensitivity and specificity.)

As discussed above, both the Duration and Frequency Pattern Tests demonstrate good sensitivity and specificity and should be considered a component of the test battery (Musiek, Baran, & Pinheiro, 1990). The clinician should be aware, however, that if language is of concern, the only truly nonlinguistic test of temporal resolution available to date is the GIN. While both the GIN and the RGDT employ nonverbal stimuli, only the GIN employs a nonverbal response mode. The GIN demonstrates sensitivity of a 67% for various central auditory

lesions and high specificity of 94% (Musiek et al., 2005)); however, sensitivity is reported to be 100% in the case of insular lesions (Bamiou et al., 2006). Unfortunately, limited data have been published for the RGDT with respect to validity, reliability, and efficiency data (Chermak & Lee, 2005). Ultimately, the clinician must use sound science and judgment based on the individual patient profile to determine which temporal processing test is most appropriate. See Chapter 12 of this volume for discussion of clinical decision analysis and Chapter 2 of Volume 2 of this Handbook for discussion of evidence-based practice.

Should I Use More Than One Test to Assess Temporal Processing?

It may be beneficial to assess both temporal resolution and temporal patterning abilities during a central auditory processing evaluation. If a deficit is identified in one area of temporal processing, and time allows, it would be beneficial to determine if a deficit also lies within other temporal processing areas as well. The neurophysiology underlying pattern perception and temporal resolution is different. It seems patterns require contour recognition and Gestalt processing in the right hemisphere, appropriate transfer, and then sequencing for linguistic labeling in the left hemisphere (Musiek et al., 1980). GD requires a synchronous offset and onset of auditory fibers consistent with the beginning and ending of the gap (Musiek et al., 2005). In addition, the use of multiple tests may yield information regarding different neurophysiological processes, and information from several different types of temporal processing tests would be highly beneficial with respect to focusing intervention to specific deficits.

How Can Clinicians Test Temporal Integration and Temporal Masking?

Unfortunately, clinically feasible measures of temporal processing are limited to temporal ordering and resolution. Although there are a number of paradigms reported in the literature for assessment of temporal integration and temporal masking, they have not been historically clinically feasible due to the necessary equipment interfacing, as well as subject training and time requirements. Transfer of these paradigms from the lab to the clinic will require communication and cooperation between researchers and clinicians. (See Chapters 1 and 23.)

Does Cognition Play a Role in Temporal Processing?

Recently, there has been considerable attention give to the role of cognition (i.e., top-down processing) with respect to audition, particularly in the area of rehabilitation. Notwithstanding some variability across studies, some data have been reported that support a correlation between temporal processing skills and cognitive abilities across various populations (Foster et al., 2012; Mukari, Umat, & Lee, 2010). It has been hypothesized that the variability across studies may be due to methodological differences. However, there have been very few studies to date that have systematically studied and controlled for cognitive variables among study and patient populations. Given the

organization of the brain and information processing, it would not be surprising to see interactions among perceptual and cognitive processing. Additional research is needed to examine the role of cognition with respect to auditory processing and interactions between top-down information processing and bottom-up sensory processing. (See Chapters 1, 6, 7, 8, 22, and 23 for additional discussion of this potential interaction.)

Age-related effects are perhaps the most highly investigated with respect to cognition and its effect on auditory function or dysfunction. For example, Harris and colleagues (2010) demonstrated that processing speed and cognitive load correlate with temporal resolution abilities in younger and older adults. Performance on the Frequency Pattern Test has been found to positively correlate with working memory capacity (Mukari, Umat, & Lee, 2010). Likewise, a relationship between verbal memory and executive functions (which is predictable by the variance expressed in temporal processing abilities independent of age, gender, and hearing loss) has been seen in healthy young adults (Foster et al., 2012). Some have proposed the controversial *temporal processing hypothesis,* suggesting that measures of auditory temporal processing can predict language outcomes (see for example, Benasich & Tallal, 2002; Tallal & Gaab, 2006). In an investigation that sought to control several variables that have led to disparate results across studies, Smith, Trainor, Gray, Plantinga, and Shore (2008) concluded that GD can be influenced by auditory and nonauditory factors and that some aspects of temporal processing may be related to language outcomes and others may not. Clearly, additional research is needed to fully understand the links between language outcomes and temporal processes. See Chapter 18 in this volume and Chapters 1, 15, and 18 in Volume 2 of this Handbook for additional discussion of the interactions among sensory and nonsensory processes.)

[redacted]ferential Diagnosis

Differential diagnosis cannot be accomplished using one test alone. It important that a battery of tests be employed in order to best determine an individual's overall auditory processing profile (Jerger & Musiek, 2000). Tests of temporal ordering and temporal resolution appear to be most sensitive to cerebral lesions and interhemispheric transfer via the corpus callosum (Musiek, 1990; Musiek & Pinheiro, 1987; Musiek, Pinheiro, & Wilson, 1980). Typically, reduced scores on the patterning tests are seen bilaterally. As explained above, comparison of hummed versus labeled performance scores on the pattern tests provide considerable insight for differential diagnosis; however, other tests in the central auditory battery are necessary to contextualize temporal processing test results to accurately infer site or level of dysfunction, as well as to identify the types of deficient central auditory processes and implications for function in everyday settings. While the central auditory tests described in this chapter (i.e., temporal processing tests) are more sensitive to dysfunction at higher levels of the CANS, other central auditory tests provide more specific insight regarding the integrity of lower levels of the CANS (e.g., masking level differences [MLDs], auditory brainstem response [ABR]). In fact, both the MLD and the ABR can provide insight

into timing and temporal processing of the CANS. See Chapters 2, 16, and 17 in this volume.

Case Studies

The following case studies are intended to demonstrate how measures of temporal processing assist in the differential diagnosis of CAPD.

Case 1. Traumatic Brain Injury

A 16-year-old male was involved in a motor vehicle accident. The radiological report following the injury indicated a severe aneurysm affecting the pericallosal artery, resulting in a bleed into the corpus callosum. Audiological findings indicated pure-tone thresholds within the normal range bilaterally. The central auditory processing evaluation (Figure 15–8) revealed a significant left ear deficit on dichotic listening. Duration Pattern Test performance in the sound field was reduced in the verbal condition but was within the normal range in the hummed condition.

This is an interesting case study relative to the location of the lesion. The use of the dichotic listening data in conjunction with temporal pattern performance leads to the identification of the anatomical site of dysfunction. We see that there is a severe left ear deficit and reduced pattern scores for the labeling condition only. In order to process dichotic stimuli,

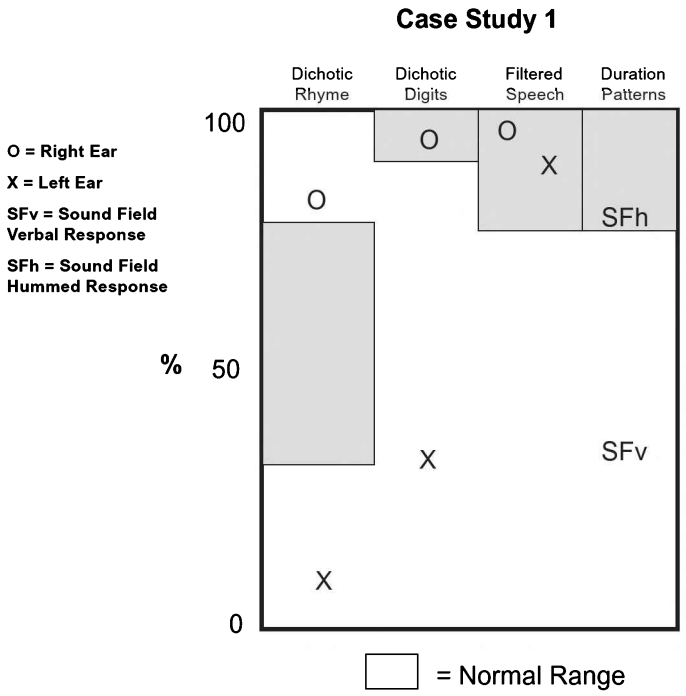

Figure 15–8. Behavioral central auditory processing profile for case study 1.

as well as temporal patterns, information must be transferred across the corpus callosum to the left hemisphere. Figure 15–9 depicts the pathophysiology of this particular case. In this patient, the right ear demonstrates normal scores for the dichotic measures because the right ear has direct access to the left hemisphere, where digits and words are ultimately processed in the so-called *speech processor*. The left ear demonstrates a deficit because information is sent via the contralateral pathway (because it is stronger and contains greater neural substrate) to the right hemisphere (Pinheiro & Musiek, 1985). Although the stimuli may be processed in the right hemisphere, it cannot be transferred across the corpus callosum due to the lesion. The patterning results indicate that the right hemisphere and processing of contour recognition are intact because the patient was able to perform within normal limits in the hummed condition. The dichotic test results coupled with a significant deficit observed in the verbal patterns condition point to disruption of information transfer across the corpus callosum, and hence difficulty in linguistic processing (as reflected in the depressed labeling condition).

Case 2. Auditory Cortical Involvement

Case study 2 involves a 56-year-old female who presented with grand mal seizures and neurological symptoms. Radiological findings indicated a large right-sided glioma affecting the right temporal region.

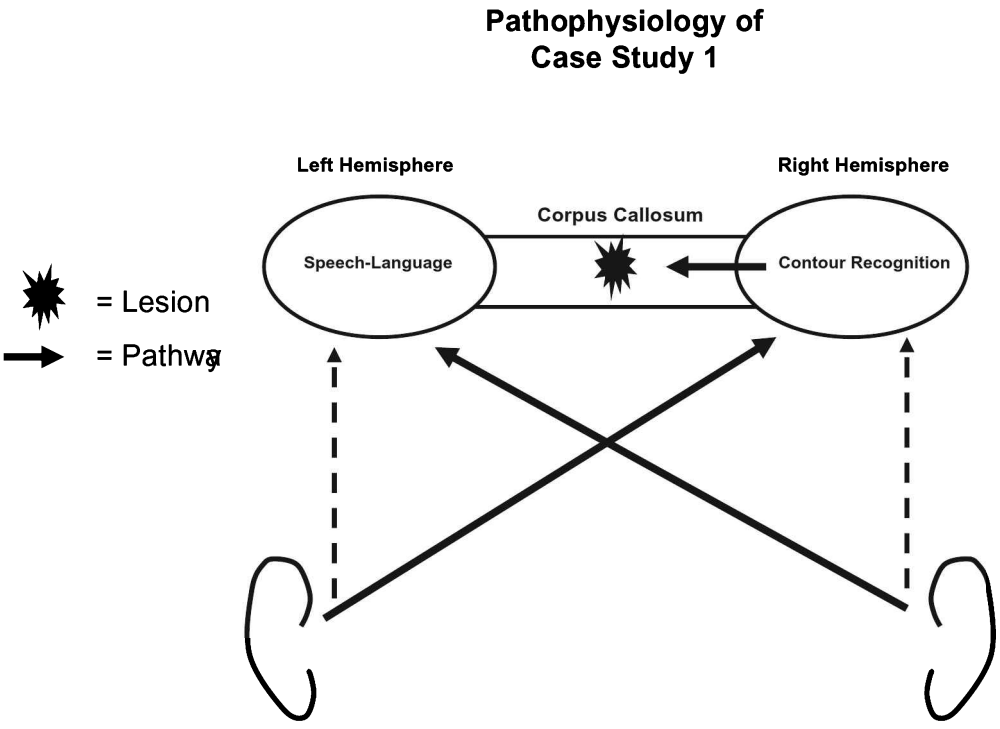

Figure 15–9. Proposed pathophysiology for case study 1.

Audiological findings indicated normal peripheral hearing sensitivity bilaterally. Central auditory processing evaluation revealed a severe left ear deficit on dichotic tests, as well as a severe temporal processing deficit, as reflected on the both the Frequency Pattern Test and the Duration Pattern Test, for both humming and labeling. A right ear deficit was also observed on the GIN (Figure 15–10).

This case provides an excellent example of what may happen in the presence of a right-sided lesion (Figure 15–11). Similar to the previous case, there is a left ear deficit on the dichotic measures. Again, this is a result of the left's ear indirect access to the left hemisphere (where the digits and words are ultimately processed) via the corpus callosum. The left ear, demonstrates a deficit because information is sent via the contralateral pathway (because it is stronger and contains greater neural substrate) to the right ear, where the lesion is located. Unlike the previous case, however, this patient is unable to either linguistically label or hum the pattern tests. This is likely a result of the fact that contour recognition necessary for processing of patterning elements is compromised due to the right-sided neurological insult. Inability to recognize the melodic contour undermines the ability to hum the response. Labeling function remains intact in the left hemisphere; however, in the absence of the necessary auditory contour information processed in the right hemisphere, the left hemisphere is unable to label the pattern.

In this example, we also observe severely increased GDTs for the ear (right) ipsilateral to the lesion. There is strong

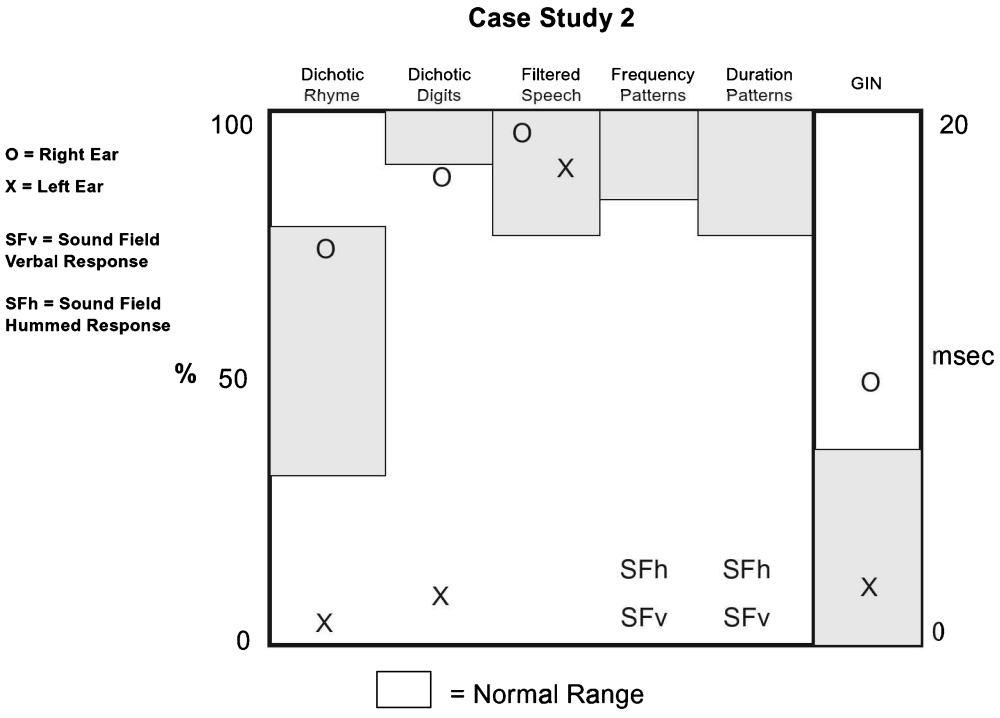

Figure 15–10. Behavioral central auditory processing profile for case study 2.

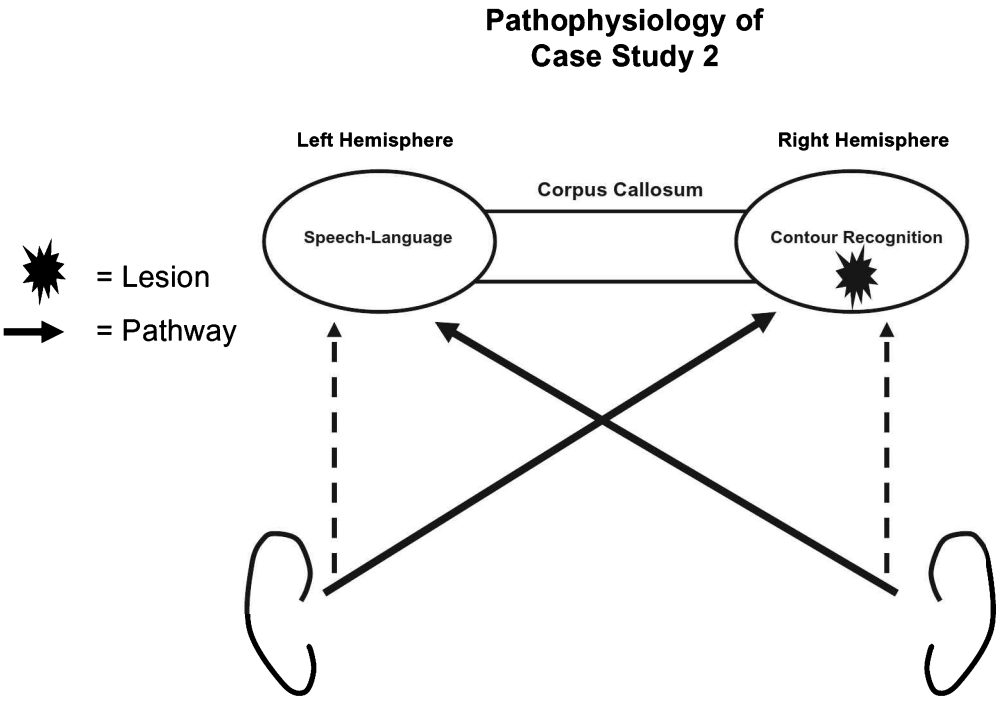

Figure 15–11. Proposed pathophysiology for case study 2.

evidence to support the view that gap detection deficits in cases of neurological insult can be reflected in any number of ways, including the ear ipsilateral or contralateral to the insult, or bilaterally (Bamiou et al., 2006; Musiek et al., 2005).

Case 3. CAPD in Children

A 7-year-old male was seen for a severe articulation deficit and a suspected CAPD. This child presented a history of recurrent otitis media. His parents reported that in addition to the speech deficit, he has difficulty with reading and spelling and that there was a strong family history of learning disabilities. Although there were no concerns regarding hearing loss, the school's staff expressed concerns regarding his ability to hear in the presence of background noise and his poor musical skills. Audiological evaluation confirmed normal peripheral hearing sensitivity bilaterally. The central auditory processing evaluation (Figure 15–12) revealed a right ear deficit on measures of binaural integration (i.e., Dichotic Digits Test; Musiek, 1983) and the Staggered Spondaic Word (SSW) Test (Katz, 1962), bilaterally reduced scores on low-pass filtered speech (LPFS), and severely depressed scores on two-tone frequency pattern tests in both the verbal and hummed conditions.

This child clearly presented with significant auditory processing deficits across several areas. These results may suggest a lesion; however, they are likely consistent with a neuromatural lag or diffuse neuromorphological involvement. Several recommendations were made, the first

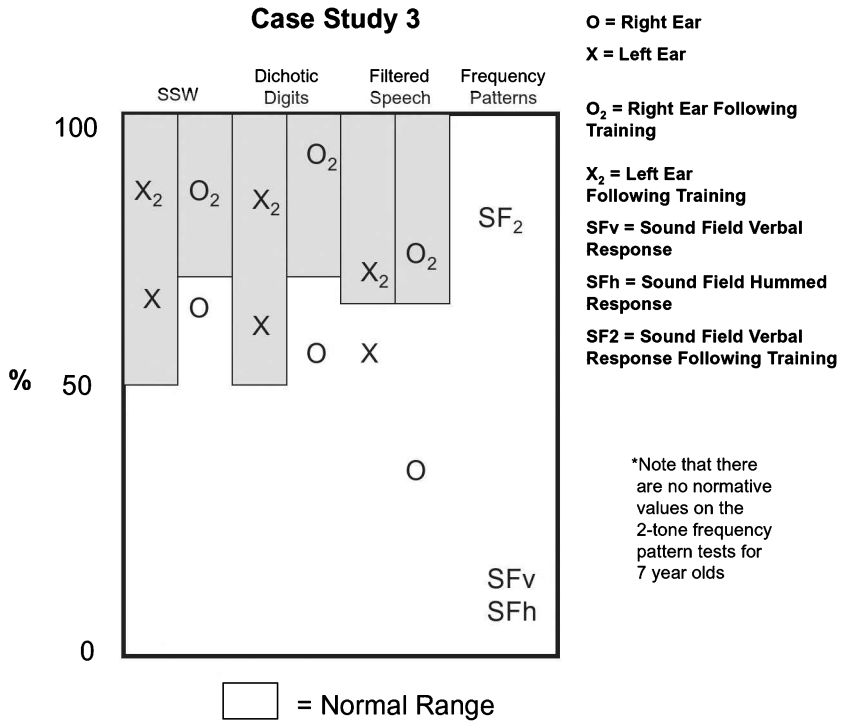

Figure 15–12. Behavioral central auditory processing profile for case study 3.

of which was to implement the use of a FM system to improve the signal-to-noise ratio in the classroom. Improving access to the acoustic signal should lessen his auditory closure and discrimination difficulties (as revealed by his reduced LPFS scores), as well as his depressed auditory performance in competition (as reflected by depressed performance on the Dichotic Digits and SSW tests). In addition, vowel identification and discrimination training were recommended to address his reading and spelling deficits. Finally, the use of the SIMON game (see discussion below under Auditory Training) was recommended to improve his temporal processing skills.

Two months following a six-week, three-hour per week intervention program, the child returned for reassessment. His parents reported significant improvement in his reading and spelling abilities, and even resolution of his articulation deficits! As reflected in Figure 15–12, this child demonstrated significant improvements across all areas of auditory processing, with perhaps the greatest gains made in temporal processing.

ctional Implications Temporal Deficits

Temporal processing deficits are associated with a range of functional deficits. Parents of children with temporal processing deficits often report that their

children present poor musical skills, lack of prosody during speech and reading aloud, poor emotional tone, difficulty understanding poetry, and difficulty understanding jokes. It is highly unlikely that you will find a concert pianist with poor temporal processing abilities, since processing the subtle timing elements of sound is essential to a musician. Take, for example, temporal cueing as it refers to our ability to segment words in the sentence "They saw the *snowdrift* by the window." There are actually two ways in which this sentence could be interpreted. It could be that, "They saw the *snowdrift* by the window" or "They saw the *snow drift* by the window." Although these sentences are exactly the same with respect to their linguistic elements, their interpretation is entirely different depending upon where the individual places the temporal emphasis (Cole & Jakimik, 1980). Children and adults with temporal processing deficits often experience difficulty with such distinctions. Also of clinical significance is the overwhelming evidence of the frequently observed (as in Case 3) comorbid presentation of CAPD and reading and spelling deficits (Farmer & Klein, 1995; Hood & Conlon, 2004; Meng et al., 2005; Meyler & Breznitz, 2005; Putter-Katz et al., 2005; Tallal, 1980; Walker et al., 2002).

Primary Intervention Strategies

Many informal and formal training techniques, as well as a growing number of computer-assisted tools (e.g., Fast ForWord, Earobics) are available for auditory training. It has been our clinical experience that treatment programs are most successful when they are designed to address the specific deficits exhibited by the patient, as identified from the central auditory test battery. For example, many children in our clinic present with deficits in temporal ordering. For these children, we recommend auditory training focused on temporal skills. It is critical that training be directed to the specific area of weakness in order to maximize the ability of the CANS to undergo the plastic changes necessary to elicit improved auditory function. (See Chapters 1, 3, and 5 of this volume and Chapter 1 of Volume 2 of this Handbook for discussions of neuroplasticity.) A brief overview of training techniques that parents, clinicians, and related professionals can implement with children and adults with temporal processing deficits is provided in the next section.

Auditory Training

Auditory training (AT) can be conducted as part of a home or school (re)habilitation program (i.e., informal AT) or within a clinic using highly structured stimuli and tasks (i.e., formal AT). Based on our clinical experience, as well as reports in the scientific literature (Tremblay, Kraus & McGee, 1998), training programs conducted for at least four to six weeks, for an average of two or three hours per week, can be highly effective in (re)training the brain with respect to auditory processing skills. Informal and formal training techniques to enhance temporal processing include reading poetry with proper intonation (thereby exercising prosody), GD training, duration discrimination, sequencing tasks, heteronym differentiation, identifying word boundaries through temporal cues, reading poetry,

and following auditory directives (Chermak & Musiek, 2002).

Perhaps one of the most inexpensive and engaging ways to train children's temporal processing skills is with the game SIMON, a hand-held computer game that has been available for over 25 years. The SIMON game uses sequences of sounds to build temporal ordering and concentration abilities. Visual cues should be removed by having the patient either turned away from the game or blindfolded. SIMON provides a variety of tasks of incremental difficulty, beginning with labeling simple pitch patterns, to counting tones, ultimately to identifying specific tone patterns. A maximum of three tones is used to maintain the focus on auditory skills rather than memory. Tone discrimination can also be exercised. (See Chapters 3, 7, 8, and 9 of Volume 2 of this Handbook for in-depth discussions of auditory training.)

Conclusion

Temporal processing pervades all auditory processing abilities and is linked to many skills ranging from musical perception to speech perception to reading (Musiek et al., 2005). Clinicians must include tests of temporal processing in central auditory processing test batteries to fully examine the integrity of the CANS and to identify or substantiate associated functional deficits. Additional temporal processing tests with documented efficiency (i.e., sensitivity and specificity) that can be administered in the typical clinical setting are needed, especially in the areas of temporal integration and temporal masking.

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CHAPTER 16

MEASURES OF BINAURAL INTERACTION

JENNIFER MCCULLAGH and DORIS-EVA BAMIOU

Introduction

Hearing with two ears is better than with one and leads to improved performance in several auditory tasks, such as sound localization and hearing in background noise, both in real life and in laboratory conditions. This happens because of binaural interaction (BI), which first occurs at the brainstem, and subsequently through processing at higher levels of the central auditory pathways. Binaural interaction mechanisms depend on balanced input from both ears and the mechanisms are more sensitive to the effects of peripheral hearing loss over a longer developmental period than are mechanisms underpinning monaural hearing (Irvine, 1992a, 1992b). Following a brief review of the brainstem and thalamic physiology that underpin binaural interaction, tests of binaural interaction and results in clinical populations are described. The reader is referred to Chapter 2 for psychoacoustic considerations related to binaural interaction underlying localization and to Chapter 5 for additional discussion of the neurobiology of binaural interaction.

Brainstem and Thalamic Pathways of Binaural Interaction

BI first occurs at the level of the superior olivary complex (SOC) in the lower brainstem. It occurs next in two more rostral nuclei of the auditory brainstem, the nuclei of the lateral lemniscus (NLL) and

the inferior colliculus (IC), both in parallel as well as in hierarchical (i.e., serial) fashion relative to the SOC (Moore, 1991). The fibers of the auditory nerve bifurcate upon entering the cochlear nucleus (CN) and synapse with morphologically different types of neurons in the different divisions of the CN. Some CN neurons project directly to the IC, whereas others project to the SOC via the medial nucleus of the trapezoid body (MNTB), as well as to the NLL, and from the NLL on to the IC (Figure 16–1). However, none of the cell populations in the CN project to all brainstem targets, and none of the targets receive inputs from all cell types (Cant & Benson, 2003). Projection of neurons to the IC are both highly convergent and divergent, and the central nucleus of the IC receives projections from 20 identi-

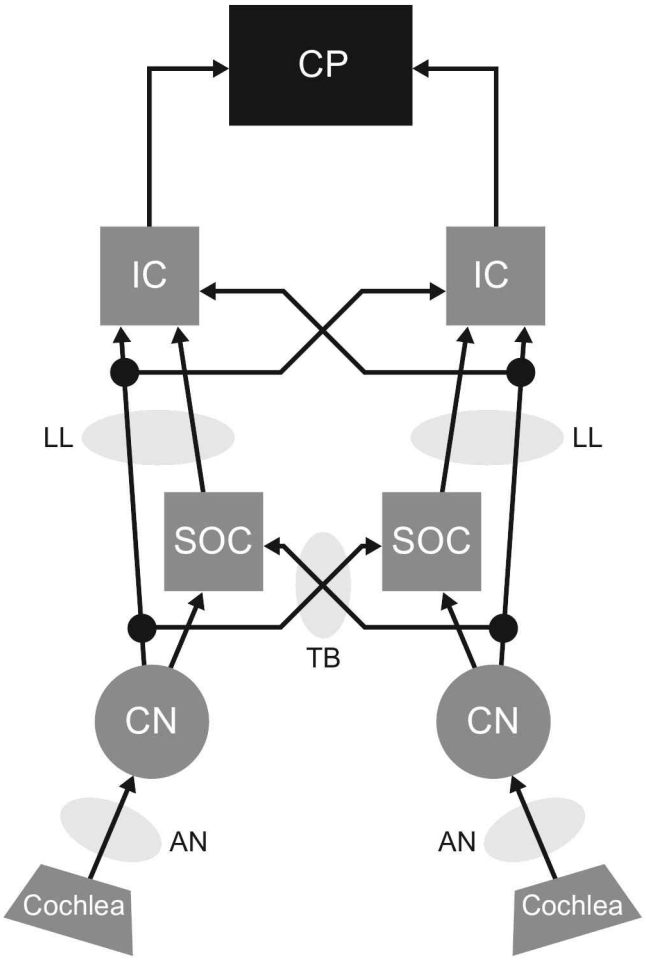

Figure 16–1. Schematic representation of the main brainstem nuclei of the auditory pathway and their main connections. AN = auditory nerve, CN = cochlear nucleus, SOC = superior olivary complex, LL = lateral lemniscus, IC = inferior colliculus, CP = central processor.

fied neuron types in approximately 10 major brainstem nuclei (Irvine, 1992a, 1992b). These neuronal types have different functional properties, due to the differences in received synaptic input and intrinsic membrane properties, thus giving rise to distinct parallel pathways from the CN to the IC, which are responsible for the various aspects of binaural processing (Irvine, 1992a). In addition to these parallel ascending pathways from the CN to the IC, intrinsic connections within certain nuclei of the SOC and IC are important for binaural interaction (Moore, 1991).

At the thalamic level, the medial geniculate body (MGB) receives inputs from the central nucleus of the IC, as well as from the dorsal-medial IC. Most of the inputs to the ventral portion of the MGB from the central nucleus of the IC are ipsilateral (Cant & Benson, 2007; de Ribaupierre, 1997; Morest, 1964). These pathways demonstrate the complex nature of the brainstem and the auditory thalamic anatomy associated with binaural interaction.

Tonotopic Organization of the Brainstem and Auditory Thalamus

All the major brainstem nuclei are tonotopically organized, beginning with the CN and its projections. The medial superior olivary nucleus (MSO) has an expanded representation of low frequencies, due to a major input from the large spherical bushy cell region of the anteroventral CN (Bourk, Mielcarz, & Norris, 1981). In contrast, the lateral superior olivary nucleus (LSO) and the medial nucleus of the trapezoid body (MNTB) have a greater representation of high frequencies. A restricted region of the basilar membrane with a characteristic frequency (CF) is represented in the IC (and in other brainstem nuclei) as a sheet of neurons (Irvine, 1992a, 1992b). The medial portion of central nucleus of the IC is represented by high frequencies with lower frequencies represented in the dorsal cortex (Stiebler & Ehret, 1985). Lastly, in the ventral portion of the MGB the low frequencies are represented laterally and the high frequencies are represented medially (Aitkin & Webster, 1972; Imig & Morel, 1985).

Neurons Receiving Binaural Input

Eighty percent of central auditory neurons are influenced by stimulation of either ear. The binaural neurons at the SOC are classified on the basis of whether they exhibit a predominantly excitatory response (E), inhibitory response (I), or no response to either ipsilateral and/or contralateral stimulation (O), thus leading to nine major categories of response patterns (Goldberg & Brown, 1969) (Table 16–1). However, these effects may not always be apparent in response to monaural stimulation.

The overwhelming majority of neurons at the MNTB are monaural. By contrast, in the SOC, almost all LSO neurons receive inhibitory input in response to contralateral input and excititory input in response to ipsilateral input (i.e., IE input), except for a small proportion receiving no input in response to contralateral input and excitatory input in response to ipsilateral input (i.e., OE input), whereas about 60% of MSO neu-

Table 16–1. Classification of Binaural Neurons at SOC Level on the Basis of Predominant Effect of Monaural Stimulation of Either Ear

		Ipsilateral Stimulation		
		Excitation	**Inhibition**	**No effect**
Contralateral Stimulation	**Excitation**	EE	EI	EO
	Inhibition	IE	II	IO
	No effect	OE	OI	OO

E, excitatory; *I*, inhibitory; *O*, no effect to ipsilateral and contralateral stimulation.

rons receive excitatory input in response to both contralateral and ipsilateral input (i.e., EE input), with a smaller proportion receiving EO, EI or IE input (Golberg & Brown, 1968; Irvine, 1992b). The difference in the response characteristics of the MSO/LSO, in combination with their biased frequency responses (low frequencies in the MSO versus high frequencies in the LSO) (Bourk et al., 1981; Irvine, 1992a, 1992b), result in a correlation between characteristic frequency and binaural response properties that is maintained throughout the central auditory pathway. Thus, in the central nucleus of the inferior colliculus (ICC), EE binaural input occurs more frequently in neurons with CF below 3 to 4 KHz, whereas EI and monaural (excitatory or inhibitory) input patterns occur more frequently in neurons with CF above this frequency range (Irvine, 1992b). At the level of the MGB, the ventral portion is characterized by approximately 53% EE-type, 27% EO-type, and 20% EI-type (Cetas et al., 2002). Binaural inputs have been demonstrated within the ventral nucleus of the MGB (Aitkin & Webster, 1972; Calford, 1983; Ivarsson, de Ribaupierre & de Ribaupierre, 1988; Samson et al., 2000). There is also a remarkable segregation of neurons with respect to their binaural input properties within a frequency band (Irvine, 1992a, 1992b). It has thus been suggested that there is both functional and spatial segregation for different components of binaural function within the human auditory pathway, and this has been confirmed by several human lesion studies (see Tests of Binaural Interaction below for review).

Encoding Interaural Time Differences

If a sound is located at any plane other than the median plane of a listener, the sound must travel a different path length to reach the two ears (as one ear is nearer and one is farther from the sound source). This gives rise to interaural time differences (ITDs), which helps the listener localize sound. Two components of ITDs exist: (1) onset time difference (i.e., difference in the time of arrival of the first waveform at the two ears), and (2) ongoing time difference for sustained sounds (i.e., phase differences for pure tones, and differences in the envelope of the signal for ongoing complex stimuli). ITDs are influenced by stimulus frequency such that the ITD for a given azimuthal displacement decreases with

increasing stimulus frequency (i.e., ITDs are greater for low frequency sounds) (Irvine, 1992a, 1992b). Although neurons in the IC (Carney & Yin, 1989; Yin, Chan, & Carney, 1987) and MGB have been shown to be sensitive to ITDs, the majority of ITD sensitivity is generated in the SOC (Yin & Kuwada, 2010). Whereas the predominance of IE neurons in the LSO suggests that this nucleus is responsible for the representation of interaural level (or intensity) differences (ILDs or IIDs), the MSO has a large proportion of neurons sensitive to interaural time differences (ITDs). This sensitivity has been attributed to simultaneous arrival of neural impulses from the two ears at the MSO, with the variable path length from each anteroventral CN (AVCN) introducing an interaural delay that compensates for the ITD in the real world (Moore, 1991). The MSO neurons are provided with precise phase-locked input by projections of the spherical bushy cells in the anterior part of the AVCN, which preserve auditory nerve discharge characteristics as a consequence of receiving input via the large axosomatic end-bulbs of Held. In the ascending auditory pathway, however, there is a progressive dramatic decrease in the number of neurons exhibiting phase locking, as well as in the upper frequency limit for such phase locking to occur. See Chapter 2 for additional discussion of ITD and ILD.

At the level of the auditory nerve, periodicity of sound is encoded by means of synchronized neural activity, termed as periodicity code. This is replaced by a rate code (i.e., distribution of discharge rate across the neural fibers) at the level of the ICC, with a large proportion of ICC neurons acting as temporal filters (Langner & Schreiner, 1988). In order for information encoded in terms of place/periodicity at the periphery to be used by the central auditory nervous system, this information must be translated to a different code. This observation has led to the postulation of the existence of a "coincidence detector" in the brainstem (Jeffress, 1948), whose output is determined by the temporal correlation between the phase locked input and a delayed replica of this input. Thus, brainstem neurons that process ITDs conduct a cross-correlation process (Yin et al., 1987). In the SOC and ICC, neurons showing sensitivity to click ITDs receive EI or IE input, with maximal response when the stimulus from the ear from which they receive the excitatory input is leading and suppressed response when the stimulus to the inhibitory ear is leading (Irvine, 1992a). Animal data indicate that for both the SOC and the ICC, there is topographic organization of the neuronal sensitivity to interaural time difference and thus for coincidence detection (Irvine, 1992a). Goldberg and Brown (1969) noted that for phase-sensitive neurons, the discharge was maximum when the excitatory inputs originating at the two ears reach the cell in-phase and that the discharge was minimum when the inputs arrived 180 degrees out-of-phase (i.e., binaural facilitation takes place when the inputs arrive in-phase and inhibition occurs when the inputs arrive out-of-phase). Most delay sensitive neurons have a characteristic delay (Rose, Geisler, & Hind, 1966).

Encoding Interaural Intensity Differences

Differences in the sound pressure level of an acoustic signal at the two ears (i.e., interaural intensity differences [IID]), produced by the head "shadow" effect

and the directional amplifying effects of the pinna, provide the major cue for the azimuthal location of high-frequency sounds (Feddersen, Sandel, Teas, & Jeffress, 1957; Rayleigh, 1907). The major class of neurons that are sensitive to IIDs at the level of the SOC are concentrated at the LSO and receive IE binaural input, with excitatory input from the spherical bushy cells at the ipsilateral AVCN and inhibitory input from the principal neurons at the ipsilateral MNTB, which in turn receive input from bushy globular cells in the contralateral AVCN (Irvine, 1992b). Most of these neurons are affected by the base intensity in which IIDs are introduced (Park, Klug, Holinstat, & Grothe, 2004; Tsai, Koka, & Tollin, 2010).

At higher brainstem levels, including the NLL and ICC, neurons sensitive to IIDs receive EI input (Davis, Ramachandran, & May, 1999; Irvine, 1986). IID processing has been shown to be quantitatively different between the LSO and ICC (Greene, Lomakin, & Davis, 2010; Park et al., 2004). Although coding of an auditory space map is uncertain at the level of the MGB, neurons within the MGB have been shown to be sensitive to IIDs (Ivarsson et al., 1988).

Binaural Hearing and Development

The neural connections subserving binaural processing are present at birth, but they are immature (Moore, 1985). The neural circuitry that underpins binaural hearing undergoes a structural reorganization after hearing onset (Kapfer, Seidl, Schweizer, & Grothe, 2002; Magnusson, Kapfer, Grothe, & Koch, 2005). Postnatal maturation is experience dependent (Magnusson et al., 2005) and reduced or altered cochlear output during the early postnatal period may change the normal development of the central auditory system (Kapfer et al., 2002; Moore, 1985). See Chapter 3 for discussion of development of the central auditory nervous system.

Tests of Binaural Interaction

The 2005 American Speech-Language-Hearing Association (ASHA, 2005) technical report on central auditory processing disorder (CAPD) identified binaural interaction as one of the main central processes that may need to be assessed as part of the evaluation for a CAPD, particularly if the referring complaint or clinical information indicates that there may be a deficit in this domain (e.g., difficulty with sound localization or hearing in background noise). There are several tests that have been designed to assess binaural interaction. In general, the neural mechanisms subserving binaural processing are probably best assessed by means of tests that employ dichotic stimulation (i.e., the concurrent delivery of separate and different acoustic stimuli to the two ears). On the other hand, functional performance can be assessed by means of tests that employ presentation of the stimulus in the free field, thus allowing for use of cues provided by the sound transformations produced by the head and outer ear. Binaural interaction tests are thought to be sensitive to brainstem pathology; however, the majority of these tests may also be affected by cortical pathology as well as top-down processes (see each test description for

review). In addition, the presence of peripheral hearing loss may affect test results, which may warrant the need for guarded interpretation.

Masking Level Difference

Theoretical Background

In the real world, when a tone and a noise arriving at the two ears of a listener are produced by sources separated in space, and thus the tone and noise arrive at the ears from different directions, the tone is more detectable than it is when the sound sources are in the same direction relative to the listener (Robinson & Jeffress, 1963). In general, the ability to detect a target signal in a background of masking noise when listening with two ears depends on: (1) the spectral and temporal characteristics of the target signal and the masker, as is true for the monaural listening condition, and (2) the interaural time differences between the target and masker, which enables binaural facilitation by activation of the coincidence detector in the brainstem (Jiang, McAlpine, & Palmer, 1997).

The original description of the binaural masking level difference phenomenon was provided independently by Hirsch (1948) for pure tones and by Licklider (1948) for speech signals. The masking level difference (MLD) refers to the difference in intensity (dB) between signal detection thresholds in two binaural masking paradigms, which differ in phase attributes. The masking paradigms are termed as:

- "homophasic" when the signals in the two channels are in phase with one another (S_0) and the noise maskers in the two channels are also in phase with one another (N_0) (the 0 notation signifies that there is no phase difference). This condition is used as a reference condition, since masking is at its greatest, and the threshold for the signal is the poorest.
- "antiphasic" when either the signal ($S_\pi N_0$) or the noise masker ($S_0 N_\pi$) are 180 degrees (π radians) out of phase. This is the condition that shows the greatest release of masking with a best threshold for the signal (Figure 16–2).

The MLD is defined as the signal detection threshold in the S_0N_0 masking paradigm minus the signal detection threshold in the $S_\pi N_0$ masking paradigm or MLD = $S_0N_0 - S_\pi N_0$.

When the N_0 broadband masker is presented alone, the coincidence detector fires neural discharges of constant amplitude over a broad range of frequencies; therefore, introduction of the S_0 has no effect on the pattern of coincidence counting activity, since the interaural time difference of the target and masker are unchanged. However, introduction of the tone or signal (e.g., 500 Hz) out of phase (S_π) cancels masker components at that frequency (Stern & Trahiotis, 1995), thus improving signal detection threshold. At intermediate values of phase shift from 0° to 180°, the MLD shows a gradual decrease in size (see Chapter 2). Animal electrode recordings have shown that responses at the ICC are indeed consistent both with psychophysical data and with the current coincidence detection theory of binaural interaction models (Jiang et al., 1997). In addition, a more recent study that investigated the neural correlates of the MLD to nonspeech stimuli found activation in the right pulvinar

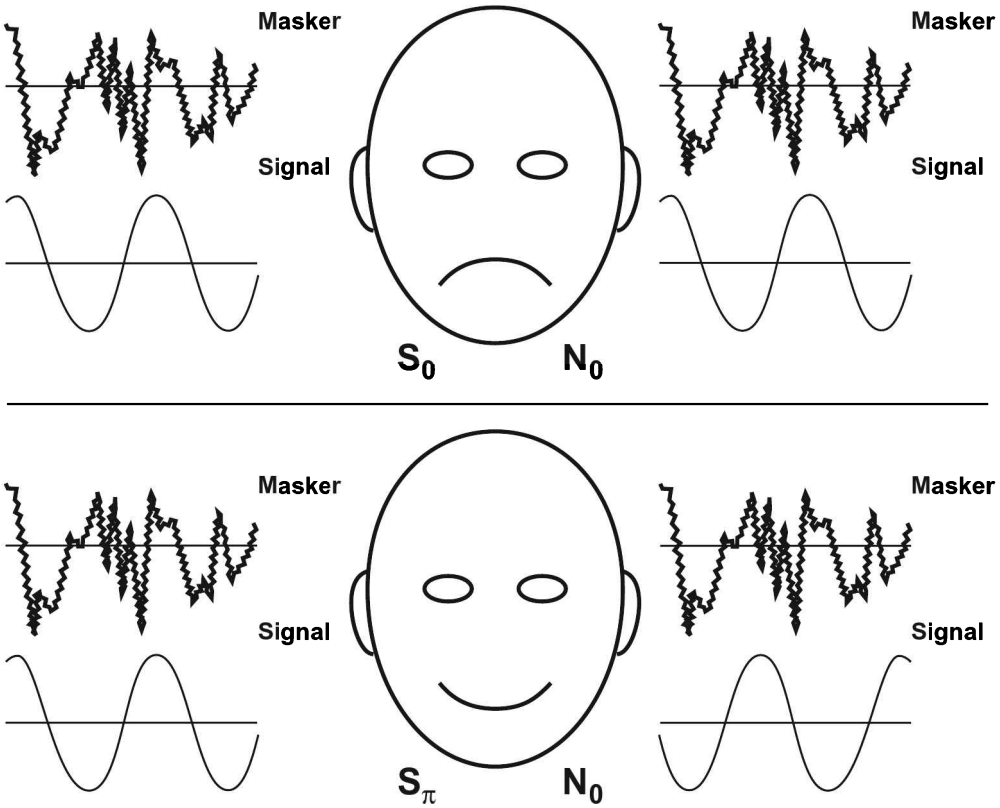

Figure 16–2. The masking level difference test. *Top panel*, homophasic condition; *bottom panel*, antiphasic condition leading to better threshold for signal detection.

thalamus as well as in the corpus callosum and insula, while the audibility of the MLD correlated with activation of the inferior frontal gyrus (Wack et al., 2012). The behavioral MLD may provide a measure of our ability to segregate sounds on the basis of their location in space and is underpinned by early processing of spatial cues in the brainstem and subsequent gating of this information in the thalamus, although contributions by later processing stages also are involved.

Test Description

Masking level differences can be obtained for either pure tones or speech. Several clinical audiometers are programmed for speech or tone MLDs. The MLD is usually obtained for a 500 Hz tone (see below for discussion of stimulus parameters) and a narrow band masker that centers around that test frequency. A Békésy-type tracking method can be used to obtain the thresholds (see Noffsinger, Schaefer, & Martinez, 1985); however, test procedures may differ. The MLD used in the test battery currently developed at the Institute of Hearing Research for the evaluation of CAPD in children employs a three-interval "oddball" paradigm (Moore, 2006). Each trial consists of 3 stimuli delivered sequentially (e.g., masker—masker + $S_{0\ or\ \pi}$— masker) and the child is asked

to pick the odd one out. Test retest reliability is reportedly less than 0.5 dB for both the S_0N_0 and $S_\pi N_0$ conditions for the MLD to a 500-Hz signal (Wilson, Moncrieff, Townsend, & Pilion, 2003).

In addition to MLD paradigms utilizing tonal stimuli, researchers and clinicians have also utilized speech stimuli to elicit MLDs (Johansson & Arlinger, 2002; Wilson, Zizz, & Sperry, 1994). Wilson and colleagues (1994) developed and evaluated an MLD paradigm for 10 spondaic words embedded in 2000-msec bursts of noise at 500 ms after the noise onset (recorded on Tonal and Speech Materials for Auditory Perceptual Assessment, track 2, left and right channels). Based on 60 normal hearing subjects, they reported a 90th percentile MLD value of 5.5 dB (S0N0 – SπN0) for both a 65 dB SPL and 85 dB SPL noise level, and suggested that an MLD < 5.5 dB should be considered as abnormal. MLD paradigms for speech stimuli other than English also have been developed (e.g., Johansson & Arlinger, 2002), with reported MLD normative values broadly similar to those reported in the English literature.

Effects of Stimulus Parameters and Test Procedures

Stimulus parameters may affect the magnitude of the MLD. The MLD depends to a great extent on the frequency and type of target signal. For tonal signals, the MLD decreases as the frequency of the tone increases from 500 to 4000 Hz (Koehnke, Colburn, & Durlach, 1986). The largest MLD is seen for the lower frequencies, with a maximum MLD of 15 dB at 500 Hz (Robinson & Jeffress, 1963). The smallest differences are seen for frequencies above 1000 to 2000 Hz, with an MLD of 3 dB for frequencies greater than 1500 Hz (Jiang et al., 1997). This frequency dependence may reflect reduced neural phase coding with increasing stimulus frequency (Durlach, 1964) and has been postulated as the source of the decrease in the S_0N_0 threshold (Koehnke et al., 1986).

The MLD for speech target signals is smaller than for pure tones, and is in general larger for low-frequency than for high-frequency dominated words, similar to the frequency effect seen for pure tones (Wilson et al., 1994). In addition, the MLD for speech signals is larger for speech detection than for speech recognition tasks (Wilson, Hopkins, Mance, & Novak, 1982). Wilson et al. (1982) found a mean MLD ($S_0N_0 - S_\pi N_0$) of 9.4 dB for a speech detection task versus 7.2 dB for a speech recognition task. The recognition MLDs for the individual spondaic words ranged from 4.4 dB (stairway) to 10.0 dB (oatmeal). In contrast to frequency effects, the duration of the stimulus does not appear to affect the MLD, as the masked threshold decreases with increasing stimulus duration in parallel for both masking conditions (Zwicker & Zwicker, 1984).

The masker level, masker bandwidth, envelope, and fine structure (Eddins & Barber, 1998) affect the MLD. The MLD increases in magnitude with increasing masker level and reaches a shallow maximum at bandwidths of the masker near 30 Hz (Henning & Zwicker, 1984). The interaural correlation of noise has a major effect on the MLD, with the larger MLD found under antiphasic conditions for a 500 Hz tone showing a rapid decrease as the interaural correlation for the noise is decreased slightly from a perfect value of 1. Further decreases in the correlation reduce the MLD further, but at a slower rate (Robinson & Jeffress, 1963) (Figure 16–3).

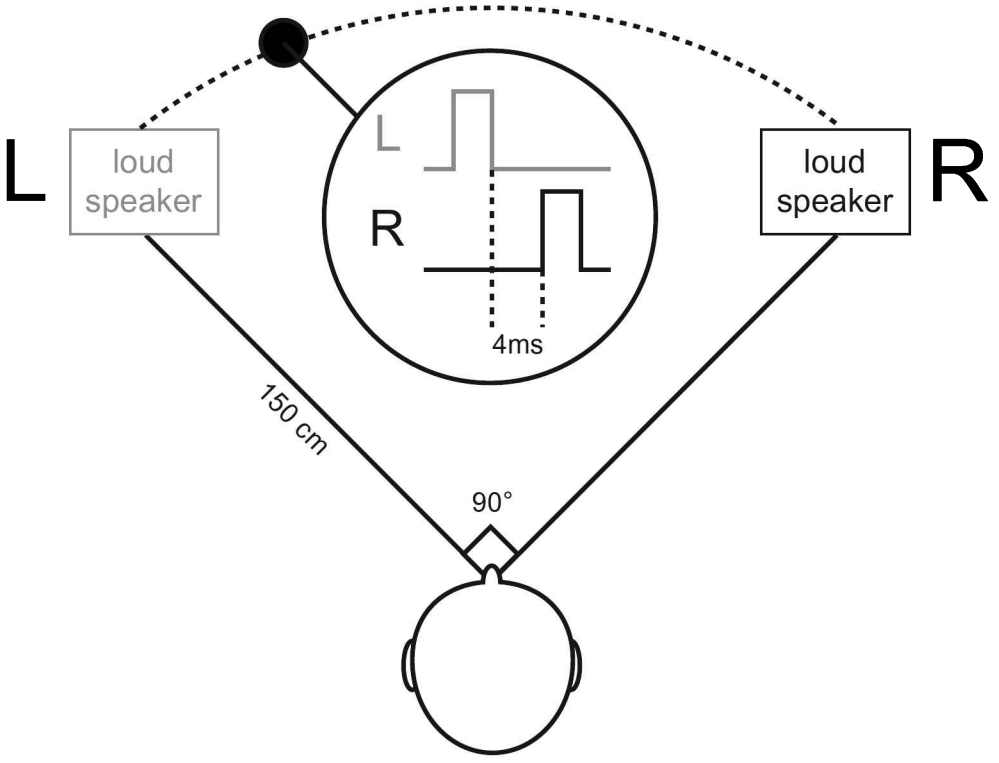

Figure 16–3. Illustration of the precedence effect. A subjects presented with a pairs of clicks, with each click originating from one of two loudspeakers placed at 45° to the left and the right of the subject's midline, will perceive the sound as originating in the left of the midline if the left click precedes the right click by a certain time interval.

Effects of Age

MLD values show age-related changes in the first years of life. Nozza, Wagner, and Crandell (1988) compared MLDs in infants, preschoolers, and adults. After controlling for hearing threshold differences, they found a statistically significant difference between infants and adults, with lower MLD values in the infants. There were no significant differences between preschoolers and adults. In addition, Hall and Grose (1990) demonstrated an increase in MLDs of children up until the age of 5 or 6 for 300-Hz-wide maskers; however, MLDs were still below adult-values for 40-Hz-wide maskers. They proposed two explanations for their findings: Either the MLD is small in young listeners because the interaural time and amplitude cues underlying the MLD are coded with relatively poor precision, or despite accurate peripheral encoding, more central auditory processes are relatively inefficient in extracting the interaural information. In a subsequent study, Hall, Buss, Grose, and Dev (2004) compared MLDs between children aged 5 to 10 and adults. They found that MLDs were greater when the stimuli were placed such that they corresponded with the masker envelope minimum than when they were placed such that they corresponded with the

masker envelope maximum, and that the binaural advantage associated with the masker envelope minima increased with the age of the child. Adults may thus be able to take advantage of the brief, albeit robust interaural decorrelations occurring when the S_π is presented in the masker envelope minima, whereas for young children the temporal window is relatively longer, indicating a developmental improvement over the age range tested. These results indicate a possible developmental change in binaural hearing early in life, which may be underpinned by myelination related synaptic efficiency and changes in neural firing synchrony.

Although researchers have demonstrated differences in MLDs between young children and adults, conflicting evidence exists regarding the effects of aging on MLDs in the adult population. Some researchers have demonstrated no significant differences in MLDs between young adults and older adults (Dubno, Ahlstrom, & Horwitz, 2008; Kelly-Ballweber & Dobie, 1984; Wilson & Weakley, 2005), while other researchers have demonstrated significant differences (Grose, Poth, & Peters, 1994; Pichora-Fuller & Schneider, 1991). Those researchers not finding significant differences in MLD controlled for hearing loss in order to demonstrate the effects of aging, rather than hearing loss, on the MLDs. Methodological differences likely explain the differences across studies, but there does, at least in some conditions, seem to be evidence of poorer binaural processing in older adults compared with younger adults.

Effects of Peripheral Hearing Loss

The presence of hearing loss affects the MLD and the effects may persist even after the hearing loss has been corrected or resolved (e.g., Hall & Grose, 1993a). This effect may be due to poorer encoding of temporal cues inherent to the signal at the periphery (Schoeny & Carhart, 1971). In addition, long-term deprivation or degradation of the acoustic signal at the periphery may lead to abnormal tuning at the brainstem level (Miller & Knudsen, 2001), which may be responsible for the finding of abnormal MLDs even after the resolution/correction of the hearing loss.

The effects of the hearing loss on MLDs depend not only on the frequencies affected by the hearing loss, but also on the age of onset and the duration of the hearing loss. In adults, the presence of high frequency hearing loss does not affect the MLD for 500 Hz stimuli, although the MLD to speech stimuli may be diminished (Olsen & Noffsinger, 1976). However, low frequency hearing loss (e.g., the hearing loss observed in Ménière's disease) may lead to decreased MLDs for both 500 Hz and spondees (Olsen & Noffsinger, 1976).

Researchers demonstrated differences in MLDs related to the type and/or degree of hearing loss. Jerger, Brown, and Smith (1984) studied the MLD at 500 Hz in 651 subjects with conductive or sensorineural hearing loss (both symmetrical and asymmetrical) and 270 normal controls. Consistent with Olsen and Noffsinger (1976), Jerger et al. (1984) also reported that hearing loss confined to 8000 Hz did not affect the magnitude of the MLD; however, if the hearing loss began at 2000 or 4000 Hz, the MLD was reduced by 1 dB, and if the hearing loss started at 1000 Hz, the MLD was reduced by 3 dB. The reduced MLD was presumably due to the peripheral hearing loss resulting from deterioration in threshold

in the antiphasic condition. The effect of symmetrical conductive hearing loss at 500 Hz on the tonal MLD was remarkably similar to the effect of sensorineural hearing loss, with the MLD declining rapidly for a loss up to 30 dB HL and an abrupt reduction in MLD when the loss exceeded 30 dB HL (Jerger et al., 1984). However, the presence of asymmetrical thresholds at 500 Hz between the two ears had a more pronounced effect on the MLD for a sensorineural than for a conductive hearing loss.

Chronic unilateral conductive impairment in adults, due to otosclerosis, tympanic membrane perforation, chronic middle ear infection, or cholesteatoma, may lead to reduced MLDs independent of hearing thresholds, due to central-type changes, as reflected by changes in the ABR (Ferguson, Cook, Hall, Grose, & Pillsbury, 1998). Subsequent restoration of hearing thresholds may lead to gradual recovery of the MLD over time, although not in all subjects. Hall and Grose (1993a) measured the MLD in adult listeners with unilateral otosclerosis before stapedectomy surgery, one month following surgery, and one year following surgery. Their results indicated that the MLD in this group improved significantly over each of the sequential tests; however, two of their eight subjects did not show recovery to a normal MLD value over this time period. The authors postulated that a period of exposure to abnormal binaural auditory input can impair sensitivity to binaural cues. However, there appears to be a "plasticity" type readjustment or adaptation of the central auditory system after restoration of normal hearing, such that binaural cues facilitation may return to a normal or near-normal level one year after the restoration of hearing. The efficiency and rate of readjustment of this process may differ among individuals.

In the pediatric population, the presence of conductive hearing loss, such as in the presence of otitis media with effusion (OME), may have longer term effects on the MLD than in the case of adults with conductive-type hearing loss. MLDs are often abnormally small in children with otitis media with effusion, and may sometimes remain abnormal after surgery for the placement of pressure equalization tubes and after normal hearing sensitivity has returned (Pillsbury, Grose, & Hall, 1991). The MLD is more likely to be abnormally reduced if OME-related hearing loss is asymmetrical between the two ears (Pillsbury et al., 1991). In general, children with a past history of OME have significantly lower MLDs than children without such history, and no correlation exists with the degree of the hearing loss (Moore, Hutchings, & Meyer, 1991). This reduction in MLDs found in children with a history of OME has been attributed to abnormal brainstem processing, as reflected by the finding of concurrent abnormal ABR (Hall & Grose, 1993b).

Changes in MLDs following the resolution of the conductive hearing losses have been demonstrated. Researchers have demonstrated the improvement of MLDs in a group of children with a past history of OME tested 3 years after middle ear surgery. However, a small proportion of subjects with a history of OME continued to have MLDs smaller than normal limits (Hall et al., 2004). Likewise, Hogan, Meyer, and Moore (1996) reported restoration of normal MLD values in a group of teenagers (ages 12–18 years) with a past history of OME before their 5th birthday. Researchers have also demonstrated the normal MLDs in 12-year-old

children with histories of predominantly unilateral OME between the ages of 2 and 4 years (Stollman, Snik, Schilder, & van den Broek, 1996). Finally, the MLD of adults with a past history of persistent OME in childhood has not been found to be significantly different to the MLDs of normal controls (Stephenson, Higson, & Haggard, 1995).

Taken together these results suggest a slow recovery of binaural interaction, as reflected by normalization of MLD values after the resolution of OME and the restoration of normal hearing thresholds occurred. However, this may not necessarily imply normal binaural hearing. Despite normal MLD values, adults with a past history of OME in childhood still complain of auditory disabilities in adulthood (Stephenson et al., 1995), and while the presence of OME in the first year of life does not appear to influence the MLD values in this age group (Hutchings, Meyer, & Moore, 1992), these children may demonstrate deficits during the first decade of life in some aspects of higher order auditory processing (Gravel, Wallace, & Ruben, 1996), possibly due to the mild hearing loss experienced during an important period of early development.

MLD in Special Populations

In the presence of normal and symmetrical hearing thresholds, MLDs are reported to be decreased to both 500 Hz tones and spondees in the presence of a variety of neurological lesions affecting the lower brainstem (Olsen & Noffsinger, 1976) or the auditory nerve, but they remain unaffected by cortical lesions (Olsen, Noffsinger, & Carhart, 1976). Lynn, Gilroy, Taylor, and Leiser (1981) measured speech detection MLDs in 26 patients with cortical, subcortical, and brainstem lesions and in 10 control subjects with normal hearing. No significant differences existed between MLDs of normal subjects and of patients with cortical, subcortical or rostral brainstem lesions. However the MLDs were significantly smaller in the group with pontomedullary-level lesions compared with the other groups.

In addition to lesion studies, other researchers have demonstrated changes in MLDs in individuals with neurodegenerative diseases (Hannley, Jerger, & Rivera, 1983; Hendler, Squires, & Emmerich, 1990; Noffsinger, Olsen, Carhart, Hart, & Sahgal, 1972; Pillon, Moser, & Raymond, 2008). Hannley et al. (1983) demonstrated poorer MLDs in individuals with multiple sclerosis (MS) who lacked wave III of the auditory brainstem response (ABR) that is generated primarily by the cochlear nuclei. Furthermore, Noffsinger and colleagues (1972) reported that almost half of 47 patients with MS had abnormal MLDs at 500 Hz and 71% had abnormal MLDs to spondees, while almost all of these patients had normal thresholds. Similarly, Hendler and colleagues (1990) found reduced MLDs in six patients with MS that were accompanied by abnormal ABRs and middle latency responses (MLRs). They postulated that disrupted neural conduction as a result of demyelinated axons led to deficient processing of phase differences between the two ears. Finally, the sensitivity of the MLD to detection of retrocochlear disease due to MS is comparable to that of ABR and better than that provided by acoustic reflexes (Hannley et al., 1983). For a thorough review of early research in MLDs in special populations, see Durlach, Thompson, and Colburn (1981).

Abnormalities in MLDs have not only been shown in individuals with confirmed

neurological lesions but also in children with learning differences. Researchers have demonstrated significantly poorer MLDs in children with *suspected* central auditory processing disorders (Sweetow & Redell, 1978), as well as children with dyslexia (Putter-Katz, Feldman, & Hildesheimer, 2011) compared with typical children. Sweetow and Redell (1978) reported significantly lower MLDs to pure tones but not to speech stimuli in a group of children with suspected auditory processing deficits (N = 24, age 4 to 12 years) versus the control group of normal children. Other researchers found reduced MLDs in children with dyslexia compared with children with skilled reading abilities (Putter-Katz et al., 2011). These results, although limited, indicate that differences in MLDs exist in atypical children.

Interaural Timing and Lateralization/ Localization Tests

Theoretical Background

A sound produced in an enclosed space reaches the listener's ears after numerous alterations to the wave's travel caused in large part by reflections of the wave from the hard surfaces of the enclosure. In a normal-sized room, the original sound signal and its reflections are perceived as a single "auditory image," and the apparent direction of this image is determined by the interaural cues associated with the earlier-arriving direct sound, with suppression of the later-arriving reflections. This psychoacoustic phenomenon is known as "the precedence effect" (PE) (Wallach, Newman, & Rosenzweig, 1949; Zurek, 1980). The precedence effect is greatest when there is a short delay between the earlier- and late-arriving sounds. In the case of long delays, as occurs when the space is large and the reflecting surfaces are at a greater distance from each other, the listener may perceive an "echo."

The precedence phenomenon has been reproduced in laboratory conditions, with the subject seated in an anechoic room in between two loudspeakers, with one loudspeaker producing the leading sound and the other producing the lagging sound. For short delays, the listener perceives a single sound, or "fused sound image," which originates on the side of the leading speaker, the exact position dependent on the time delay and level differences (Freyman, Clifton, & Litovsky, 1991). However, different mechanisms may underpin localization/lateralization abilities for single-source sounds versus PE paired stimuli. This is illustrated by the fact that single-source localization ability has already reached adult-level maturity, whereas PE localization has not in 5-year-old children (Litovsky, 1997). These findings may indicate that while single-source sound localization requires more basic auditory abilities such as single-source discrimination, PE paired stimuli localization may require more sophisticated skills, such as accommodation of echoes (i.e., sound sources that are not localized as independent auditory events) and possibly other cognitive skills (Litovsky, 1997). See Chapter 4 of Volume 2 of this Handbook for discussion of the precedence effect and room acoustics.

Test Description

Despite the inclusion of localization/lateralization tests in the test categories that

the clinician needs to consider applying in the evaluation of CAPD (ASHA, 2005), no such standard clinical tests are currently available, although more recently developed tests may provide a surrogate measure for localization (Cameron & Dillon, 2007; see section below on Listening in Spatialized Noise, LisN). The reader is referred to Chapter 23 for further discussion of developments in localization testing procedures and their application in the clinical setting. A summary of the main (experimental) test procedures that have been applied to clinical populations follows.

Sound localization tests based on the precedence phenomenon have been developed and researched (Besing & Koehnke, 1995; Cranford, Boose, & Moore, 1990a, b; Moore, Cranford, & Rahn, 1990). Cranford et al. (1990a, 1990b) described a procedure in which subjects were presented with pairs of 100-μsec click stimuli at a presentation level of 50 dB SL, with each click originating from one of two loudspeakers placed at 45° to the left and the right of the subject's midline (see Figure 16–3). The time delay between the two clicks was varied, and the subjects were presented with 3 consecutive identical click pairs and asked whether the sound was perceived as originating in the midline, left or right of the midline.

Although Cranford and colleagues (1990a, 1990b) investigated a "static" precedence effect localization task, other investigators researched a "dynamic" localization task (Besing & Koehnke, 1995; Moore et al., 1990). Moore and colleagues (1990) developed a "dynamic" computerized auditory test that required pursuit auditory tracking of a moving fused auditory image (FAI) based on stimulus conditions that elicit the precedence effect. A pair of two clicks was presented, one each from two loudspeakers placed on opposite sides of the listener, as done in the "static" precedence effect localization task by Cranford et al. (1990a, 1992b). Movement of the FAI was simulated by incrementally varying the delay between the two clicks. Listeners were asked to direct a laser pointer to the perceived location of the FAI. These researchers found that normal listeners were able to track the movement of the FAI accurately, and the perceived location of the FAI varied linearly with the interspeaker delay. Besing and Koehnke (1995) evaluated a similar test of virtual auditory localization with speech signals presented via headphones and with presentation in 9 simulated locations in the horizontal plane from −90° to +90°, in both (simulated) anechoic and reverberant listening conditions. A nine-alternative, forced-choice identification procedure was used to measure localization ability, and the outcome measures were the root-mean-square localization error in degrees and percent correct responses. The test, according to the authors, was both easy to administer and easy to perform, even for the children who performed more poorly on the tests than their peers. In addition, and of considerable interest for audiologists who assess children, subjects found the test more interesting than the MLD.

Other researchers have investigated localization abilities using lateralization tasks (Aharonson, Furst, Levine, Chaigrecht, & Korczyn, 1998; Griffiths, Dean, Woods, Rees, & Green, 2001). Aharonson and colleagues (1998) developed a localization task presented to subjects under headphones (i.e., lateralization). The test procedure consisted of the presentation of two successive short bursts for each trial, separated by an interval of 500 sec. In the first trial, stimuli were presented

diotically; in the second dichotic trial, either the arrival time of the stimulus to one of the earphones was delayed (ITD trials), or the intensity was increased at one ear and decreased at the other ear by the same amount (IID trials). The listener was required to indicate the position of the auditory image of the second burst by pushing one of nine equally spaced buttons on a keyboard (results described below).

Other researchers have demonstrated the use of lateralization tasks as part of an auditory test battery (Newcastle Auditory Test Battery; Griffiths et al., 2001). The test battery includes tests of static and dynamic lateralization for the psychoacoustic evaluation of neurologically impaired and other naive subjects. The battery contains two subtests for the measurement of binaural phase (subtest S1) and amplitude difference (subtest S2) limens for the detection of the "static" lateralization of a 500-Hz tone toward the right or left. "Dynamic" lateralization is measured in another two tests (S3 and S4) in which the phase or amplitude of a 500-Hz tone is sinusoidally advanced at one ear and delayed at the other, thus resulting in perception by the listener of sinusoidal movement of a single sound between the two ears. In subtests S5 and S6, the phase (S5) or amplitude (S6) of a 500-Hz tone is linearly advanced at one ear and delayed at the other, and the listener perceives linear movement of a single sound from the midline toward either side. These tests measure the thresholds for the detection of a 600-ms phase or amplitude ramp to the right or left. A full psychometric function can be plotted for each subtest for each subject. Normative data for 30 naive subjects in two age groups, 20 to 39 and 40 to 55 years, were reported (Griffiths et al., 2001). More research needs to be completed to develop clinically feasible measures of localization/lateralization.

Effects of Age and Peripheral Hearing Loss

The ability to localize has been shown to change from infancy to childhood and again from younger to older adulthood (Clifton, Morrongiello, & Dowd, 1984; Cranford et al., 1990a; Cranford, Andres, Piatz, & Reissig, 1993a; Griffiths et al., 2001; Litovsky, 1997). Infants at the age of 2 months do not appear to localize PE sounds (i.e., paired stimuli), although their ability to localize single source sounds is good (Clifton et al., 1984). By the age of 6 months, infants are reportedly able to localize PE stimuli as well as single-source stimuli (Clifton et al., 1984). Performance in more complex localization tasks shows a longer developmental course than for simpler tasks. Although the ability of 5 year olds to localize a single-source sound has already reached adult-level maturity, their ability to localize PE paired stimuli is still significantly lower than that of adults (Litovsky, 1997). Furthermore, Cranford, Morgan, Scudder, and Moore (1993b) demonstrated that 6- to 12-year-old children performed at normal adult levels for a "static" FAI localization test, yet performed more poorly than adults when the localization task was "dynamic." Additionally, the younger children performed more poorly than the older children on the moving FAI task, indicating a maturational change that occurs between 6 and 12 years of age. At the other end of the age spectrum, researchers have reported poorer performance in elderly compared with younger listeners on PE localization tasks with delay intervals below 0.7 ms (Cranford et al., 1990a, 1993a). Similarly, Griffiths et al. (2001) showed an age effect for

their "dynamic" lateralization task, in that the thresholds for the detection of interaural phase modulation were lower than the threshold for the control binaural frequency modulation detection task by a factor of 6.6 for the age group 20 to 39 versus a factor of 4.8 for the age group 40 to 59, whereas thresholds for the frequency modulation task did not differ in the two age groups. Moreover, other investigators have demonstrated age-related declines in localization abilities using free-field localization tasks to band-pass targets (Abel, Giguere, Consoli, & Papsin, 2000; Dobreva, O'Neill, & Page, 2011). Regardless of procedure, performance on localization tasks does appear to be influenced by the age of the listener.

Localization tasks are adversely affected by the presence of hearing loss and it has been proposed that hearing loss may have a greater impact on the localization performance of elderly subjects than younger subjects (Cranford et al., 1993). Degree, type, and configuration of hearing impairment have also been shown to influence localization abilities (Noble, Byrne, & Lepage, 1994). Furthermore, a past history of OME may have long term impact upon localization performance. Besing and Koehnke (1995) reported that children with a past history of OME showed poorer and more variable localization abilities than children without OME history. These researchers all demonstrated adverse effects of hearing loss, either conductive or sensorineural, on localization abilities.

Localization Tests in Special Populations

The precedence effect, which is underpinned by binaural interaction of disparate sound cues, has been shown to be severely affected by brainstem pathology. Sound lateralization/localization tasks using stimulus conditions known to elicit the precedence effect place greater demands on neural timing and integration than conventional (i.e., single-source) tests of localization, and may provide a more sensitive index of neural function (Moore et al., 1990). Early research demonstrated that individuals with unilateral temporal lobe lesions performed significantly poorer on localization tasks than nonneurologically impaired individuals and errors were greater on the side contralateral to the lesion (Sanchez-Longo, Forster, & Auth, 1957). In a later study, Sanchez-Longo and Forster (1958) found 19 of 21 patients with temporal lobe lesions and 4 of an additional 29 neurologically impaired patients performed poorer than nonneurologically impaired individuals on localization tasks. These studies were limited, however, by the small number of trials and response variability.

Results of a precedence effect localization task were shown to correlate with results of the Synthetic Sentence Identification with Ipsilateral Competing Message test (SSI-ICM; Cranford & Romereim, 1992), a test that is highly sensitive to brainstem pathology (Jerger & Jerger, 1974). Cranford et al. (1990b) found that subjects with MS exhibited problems with delays less than 1 ms and that the sound localization test (see preceding Test Description section) had the greatest sensitivity in separating patients with MS from controls for delays from 0.3 to 0.7 ms. Moreover, Aharonson et al. (1998) reported that lateralization tasks with high-frequency stimuli were more sensitive detectors of abnormality in individuals with MS or stroke than just noticeable differences for any kind of stimulus, or for lateralization tasks using

low-frequency stimuli or clicks. Individuals with MS who have abnormal auditory brainstem responses typically have abnormal ITDs, but not necessarily IIDs (Hausler & Levine, 1980; Levine et al., 1993). ITDs obtained using high-pass noise bursts were more sensitive to neural dsynchrony than low-pass noise bursts. Furthermore, when the MS lesions affected the pontine area of the brainstem (confirmed with magnetic resonance imaging), the auditory brainstem responses and ITDs obtained using high-pass noise bursts were always abnormal (Levine et al., 1993). These results suggest precedence effect localization test sensitivity is related to the type of stimuli utilized and that those with neurological impairment may have poor localization abilities.

Additionally, lesion studies employing localization tasks that are dependent upon ITD and IID interaction indicate poor performance for ITDs or IIDs, depending on the location of the lesion in the brainstem. These studies support the existence of two separate pathways for ITD and IID detection and sound lateralization. Griffiths et al. (1998) conducted detailed tests on a subject with MS affecting the brainstem, with a lesion in the region of the right superior olive and another lesion more dorsally located in the pons. The patient experienced a total deficit in the detection of phase differences between the ears, although detection of IIDs was preserved. Griffiths and colleagues interpreted these findings to indicate that interaural phase detection is subserved by a distinct mechanism to that for IIDs. Also supporting dual pathways, Furst and colleagues (2000) reported that patients with MS with lesions restricted to the caudal pons showed lateralization of the sound to the side of the head for IIDs, but lateralization of the sound to the midline (i.e., more severe impairment) for ITDs. Patients with MS lesions rostral to the trapezoid body, however, showed side-oriented lateralization for both types of interaural differences. In addition they reported that lesions at the trapezoid body and/or at the SOC were correlated to center-oriented lateralization performance, whereas lesions at the level of the LL and/or at the IC were correlated to a side-oriented performance. A further study of patients with MS or stroke lesions (Aharonson & Furst, 2001) showed that most patients who had lesions at or below the SOC perceived all interaural differences in binaural stimuli as small, whereas most patients who had lesions above the SOC perceived all interaural differences as large. Aharonson and Furst (2001) proposed that there is a two-level structure for the estimation of interaural differences in the brainstem, with an initial level at the SOC, and a second level at or above the IC. The first level is thought to detect differences between the left and right input, and if no difference is detected, the default decision is that the two inputs are similar, and consequently, small interaural differences are estimated. The second level at the IC is thought to assess for similarity between the left and right inputs, and if no similarity can be determined, the default decision is that the two inputs are different, and therefore large interaural differences are estimated. Thus, the location of the lesion within the brainstem can influence the degree to which ITDs, IIDs, and subsequently localization abilities are affected.

In addition to lesions of the brainstem, lesions of the auditory cortex also appear to play an important role in discriminating both ITD and IID cues. Patients with bilateral auditory cortical lesions are unable to detect ITDs (Yamada, Kaga,

Uno, & Shindo, 1996). Localization tests based on the precedence effect are affected by cortical lesions, with unilateral lesions resulting in greater difficulty localizing the paired sounds when the speaker contralateral to the lesion is leading (Cornelisse & Kelly, 1987; Cranford et al., 1990b). Moore et al. (1990) has similarly reported that two subjects with unilateral cortical lesions showed failure to track the FAI past the midline to the side contralateral to the lesion. Perception associated with the precedence effect may thus be underpinned by further processing in the ascending auditory pathway up to the cortex, which may contribute to the perception of the size of the acoustic space (Fitzpatrick, Kuwada, Kim, Parham, & Batra, 1999). Additionally, re-searchers have demonstrated poorer IIDs in individuals with temporal lobe epilepsy that did not worsen following temporal lobectomy (Tezer, Ilhan, Erbil, Saygi, Akalan, & Ungan, in press). Similar to brainstem lesions, localization abilities may be negatively impacted by cortical lesions depending on their size and location.

Rapidly Alternating Speech Perception

Test Description

The Rapidly Alternating Speech Perception (RASP) Test (Willeford, 1976) consists of segmented, continuous speech information, which is presented alternately and sequentially between the two ears. Synthesis of this information, which is necessary in order for the listener to perceive and report the complete speech signal, requires binaural interaction, which initially take places in the brainstem (Figure 16–4).

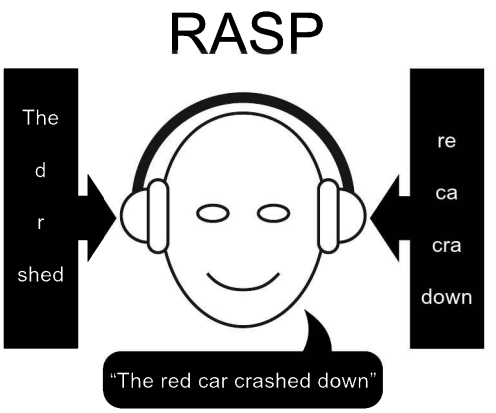

Figure 16–4. Illustration of the Rapidly Alternating Speech Perception (RASP) test.

Bocca and Calearo (1963) tested normal listeners with a test in which speech segments of 20 to 500 ms duration were presented alternately between the two ears, and found normal speech intelligibility. One of the best known tests in this category was developed by Willeford (1977) (see Figure 16–4). In this test, segments of a sentence with approximately 7 words are presented alternately between the two ears, at a 300 ms alternation rate and at 40 dB sensation level (SL) referenced to the pure tone average. Twenty sentences are presented in total, 10 with the right ear receiving the initial sentence segment first, and 10 with the left ear receiving the sentence segment first. It has been argued that since this paradigm requires binaural interaction, a single score rather than separate left and right ear scores is sufficient to identify deficits (Baran & Musiek, 1999).

Rapidly Alternating Speech Perception in Special Populations

Results of the RASP in different populations must be interpreted with caution as this test may also be affected by peripheral hearing loss (Miltenberg, Dawson,

& Raica, 1978). Moreover, although the RASP requires binaural interaction and is thus, in theory, dependent on brainstem integrity, it shows poor sensitivity to brainstem lesions. Lynn and Gilroy (1977) found the test to be abnormal only in patients with lower, but not upper brainstem lesions, while the presence of cortical lesions also affected the test results to some extent. Musiek (1983) reported the RASP had 50% sensitivity to brainstem lesions. Musiek and Geurkink (1980) used this test as part of a battery to assess five children with suspected CAPD and found normal results in all their subjects, despite the fact that two of these children presented abnormal results in a binaural fusion task that used low-pass and high-pass filtered spondees. Similarly, Willeford and Bilger (1978) reported that few children with CAPD had difficulties with the RASP, even at a 30 dB SL. Welsh, Welsh, and Healy (1980) employed a RASP procedure described by Lynn and Gilroy (1972) to assess central auditory function in 77 students with dyslexia (age range 7–18 years) and found abnormal RASP results in only 13% of their population, although 75% of these students performed outside normal limits on a binaural fusion task by Willeford (1976). They noted that their sample had a wide range of scores, and that performance on the RASP improved in the 11- to 12-year-old age group. Given these results, it appears that sentence-based RASP has limited sensitivity and may not provide useful information in a central auditory test battery.

Binaural Fusion Test

Test Description

The binaural fusion test (BFT) involves presentation of different segments of band-pass filtered speech to the two ears with a low-band-pass filtered speech stimulus presented to one ear and a high-band-pass filtered presentation of the same speech stimulus to the other ear (i.e., dichotic presentation). The patient must fuse the information from each channel in order to report the word. Katz and Ivey (1994) proposed that these tests should be more aptly termed "binaural resynthesis," since the listener must combine simultaneous and complementary segments from the two ears into one composite item, rather than fuse or join equal signals into one midline image.

Probably the most well-known test of binaural fusion was included in Willeford's (1977) test battery. In this BFT, 20 spondees filtered through a low-band-pass (500–700 Hz) filter and a high-band-pass (1900–2100 Hz) filter are presented at 30 dB SL referenced to the listener's 500 Hz threshold (low-band) and 2000 Hz threshold (high-band) (Willeford, 1977) (Figure 16–5).

Other researchers have also used filtered words in a BFT paradigm (Neijenhuis, Stollman, Snik, & van den Broek, 2001). Neijenhuis et al. (2001) investigated test-retest reliability in normal hearing adults for their version of the BFT, which used 22 monosyllabic words

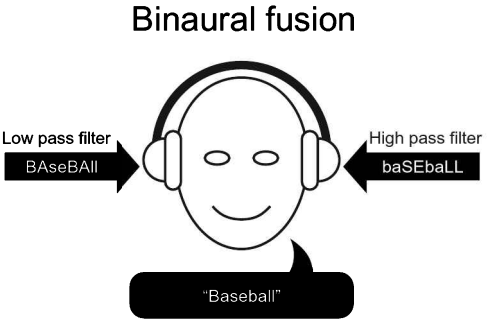

Figure 16–5. Illustration of the binaural fusion paradigm.

(low-pass filter with cutoff frequency of 500 Hz and high-pass filter with a cutoff frequency of 3000 Hz, both with a slope of 60 dB per octave). They observed both ceiling effects and a small but significant improvement on retest. This may indicate that it would be more appropriate to conduct the BFT after a training session.

Consonant–vowel nucleus–consonant (CVC) words have also been used as stimuli for BFTs, with the consonant segments presented to one ear and the vowel segment presented to the other ear (Wilson, Arcos, & Jones, 1984). Using a 50 dB HL presentation level, normal hearing subjects achieved a 10% correct word recognition when the vowel segment or the consonant segments of the words were presented monaurally in isolation, and 90% correct word recognition in the binaural condition (i.e., with the vowel segment presented to one ear and the consonant segments to the other ear).

Effects of Stimulus Parameters and Test Procedures

Presentation level affects the low-band-pass in a different manner relative to the high-band-pass for the monaural condition, with a rapid rise in intelligibility of the high-band-pass and a shallower slope for the low-band-pass segment with increasing intensity (Katz & Ivey, 1994). A presentation level of 25 to 30 dB SL results in low intelligibility for the band-pass segments in the monaural condition, but high intelligibility for the dichotic condition (i.e., the true measure of binaural fusion) (Katz & Ivey, 1994). Using his CVC BFT with 120 adults with normal hearing sensitivity at various presentation levels, Wilson (1994) reported that with the monaural vowel segments there was minor improvement of about 8% when the presentation level was increased from 20 to 40 dB HL, and that the psychometric function was constant at 15 to 20% correct over the 20 to 70 dB HL range. Recognition performance improved substantially for the consonant segments in the monaural condition when the presentation level increased from 40 to 60 dB HL (from 17% to 67%), with the psychometric function asymptoting around 65 to 70% over the 55 to 70 dB HL range. He attributed this improvement in performance to the relation between the presentation level and the level of energy in the segments of the CVCs, with the low frequency energy in the vowel speech spectrum peaking at 0 dB HL and the high frequency energy in the consonant speech spectrum peaking at –30 dB HL. Therefore, presentation level should be considered when administering a test of binaural fusion.

Other considerations include word familiarity (Katz & Ivey, 1994; Windham, Parks, & Mitchener-Colston, 1986) and auditory closure abilities (Neijenhuis, Snik, & van den Broek, 2003). Furthermore, it has been suggested that children in particular should be familiarized with more difficult words (Windham et al., 1986) to eliminate the potential language confound. In addition to word familiarity, researchers have found auditory closure abilities to be a factor as well. Neijenhuis and colleagues (2003) reported a correlation between BFT results and filtered-speech test results in a principal component factor analysis (Neijenhuis et al., 2003).

Effects of Age and Peripheral Hearing Loss

Matzker (1959) validated binaural fusion on more than 1000 patients using a test similar to Willeford's. The patient

groups included individuals with brain tumors, cerebral atrophy, multiple sclerosis, severe skull trauma, hypertension, or psychosis. Three conditions were administered using 41 words: (1) low-band-pass segment presented to one ear (BF_lo); (2) high-band-pass presented to one ear (BF_hi); and (3) simultaneous (i.e., dichotic) presentation of low-band-pass to one ear and high-band-pass to the other ear (BF_lohi). He reported that the scores in the binaural fusion (BF_lohi) condition were equal to or higher than the scores in the monaural conditions in high proportions of all the patient groups except the patients with psychosis. He interpreted the finding of poorer binaural fusion than monaural scores as evidence of a lack of auditory integration in this group. Post mortem histopathology in some of the patient groups with abnormal BFT identified pathology in the brainstem, predominantly in the olivary region.

More recently researchers have investigated the effects of aging and hearing loss on BFTs. Not unlike other measures of speech recognition, the BFT is affected by the presence of peripheral hearing loss. When peripheral hearing loss has been taken into account, however, aging per se does not affect binaural fusion test results (Grady et al., 1984; Kelly-Ballweber & Dobie, 1984). Individuals with relatively flat mild hearing losses performed significantly poorer on the BFT task than individuals with normal hearing, even though the test stimuli were presented at an adjusted level (i.e., SL), and despite correction of BFT scores on the basis of a pure-tone average (Neijenhuis, Tschur, & Snik, 2004). Thus, peripheral hearing sensitivity must be considered when administering tests of binaural fusion.

Binaural Fusion Tests in Special Populations

Researchers have investigated the effects of both brainstem and cortical lesions on tests of binaural fusion. Smith and Resnick (1972) reported different binaural fusion abilities in subjects with temporal lobe lesions and subjects with brainstem lesions using a procedure similar to Matzker (1959). Word recognition for their normal hearing subjects hovered around 20% when either band-pass was presented alone and at 60% when the low-band-pass (360–890 Hz) was presented to one ear and the high-band-pass (1750–2220 Hz) to the other ear. They also compared scores for the task with one band to each ear (dichotic) relative to when both bands were presented to both ears (diotic) in three patients with lesions of the temporal cortex. These subjects demonstrated no difference between the dichotic versus diotic presentations, similar to normal subjects and subjects with bilateral sensorineural hearing loss. Conversely, they found abnormal results (i.e., diotic scores better than the dichotic scores) by 18 to 24%, for four subjects with brainstem lesions indicating that these subjects had poor binaural interaction abilities. In addition, Noffsinger and colleagues (1972) reported abnormal BFT performance in 8 out of 36 subjects with MS. Abnormal performance occurred more commonly in subjects with lesions above the lower brainstem and abnormalities were more frequently displayed for the diotic rather than dichotic presentation, in contrast to the results of Smith and Resnick (1972).

In addition to evaluating binaural fusion abilities in individuals with neurological lesions, researchers have investigated binaural fusion abilities in indi-

viduals with dyslexia (Welsh et al., 1980), specific language impairment (SLI) (Stollman, van Velzen, Simkens, Snik, & van den Broek, 2003), CAPD (Neijenhuis et al., 2003), as well as children with a history of OME (Schilder, Snik, Straatman, & van den Broek, 1994). Welsh and colleagues (1980) employed the BFT designed by Matzker (1959) to assess central auditory function in 77 students with dyslexia (age range 7–18 years) and found abnormal results in three-quarters of this population. They interpreted their findings as indicative of the presence of a multifocal abnormality associated with dyslexia, also encompassing the brainstem. Stollman et al. (2003) used a Dutch BFT as part of a central auditory test battery to assess central auditory function in 20 six-year-old children with SLI and a group of 20 age-matched controls. The children with SLI obtained scores significantly lower than those of the control group, with an approximately zero difference between the binaural and summed monaural conditions for the SLI group, but significantly higher than zero differences in the normal control group. These findings suggest binaural interaction deficits in individuals with SLI. However, Neijenhuis et al. (2003) reported that a binaural fusion task (described in Neijenhuis et al., 2001; see above for brief description) had low sensitivity in identifying children and adults with CAPD, with only 40% of their sample falling below the 25th percentile, which they attributed to the presence of a specific auditory decoding deficit in this subgroup of patients. The presence of an auditory decoding deficit may underlie and explain why BFT results may be abnormal in the presence of normal brainstem responses (Musiek & Geurkink, 1982; Welsh, Welsh, Healy & Cooper, 1982). Finally, similar to other binaural interaction tests, children with an early history of OME (at age 2–4 years) present normal BFT performance when tested at a later age (age of 8 years) indicating that early history of conductive hearing loss may not have long-term negative effects on binaural fusion (Schilder et al., 1994).

Listening in Spatialized Noise (LisN)

Test Description

Binaural interaction may also be assessed using the recently developed Listening in Spatialized Noise–Continuous Discourse Test (LISN-CD; Cameron, Dillon, & Newall, 2006) and subsequent version of Listening in Spatialized Noise–Sentences Test (LISN-S; Cameron, & Dillon, 2007), which shows a good correlation with MLDs (Cameron et al., 2006). The test represents a new and different approach to assessing binaural interaction and is administered by special computer software via headphones and produces a virtual three-dimensional auditory environment. The three-dimensional effect is achieved by processing the speech stimuli with head-related transfer functions (HRTFs), so that the target sentences in LiSN-S are perceived as coming from directly in front of the listener (0° azimuth), while the competing speech (children's stories) is manipulated in respect to its location (0° versus ± 90° azimuth). In addition, the vocal quality of the speaker(s) is manipulated (same as, or different to, the speaker of the target sentences). Thus, four listening conditions exist, same voice target and competing speech at 0° azimuth (SV0), same voice at ± 90° azimuth (SV90), different voices at

0° azimuth (DV0), and different voices at ± 90° azimuth (DV90). The listener's performance in each of the listening conditions is measured in dB (signal-to-noise-ratio; SNR) and the speech reception threshold is thus adaptively determined. Furthermore, there are three derived "advantage" measures, that include talker advantage, spatial advantage, and total advantage, that represent the benefit in dB gained when the maskers contain either vocal (DV0), spatial (SV90), or both vocal and spatial cues (DV90), compared with the baseline (SV0) condition (Cameron & Dillon, 2007) (Figure 16–6).

LiSN in Special Populations

The LiSN-S is proposed to measure skills that relate to auditory stream segregation, that is, the process of extracting meaningful incoming acoustic signals from the nontarget background auditory signals that arrive simultaneously at the ears, by making use of various auditory cues such as spatial location or speakers'

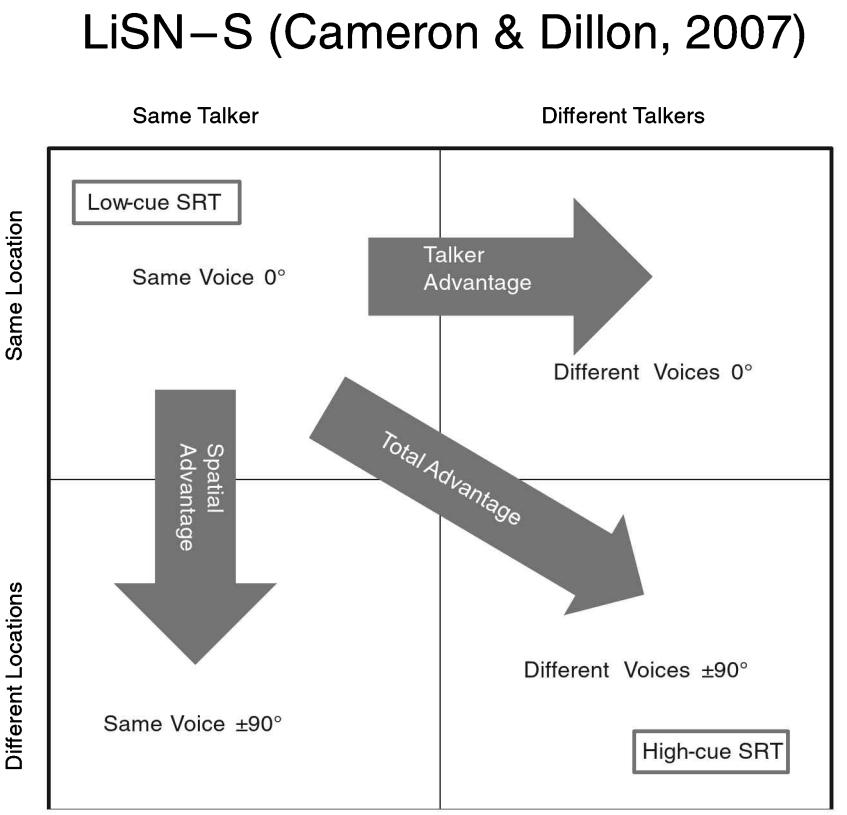

Figure 16–6. Illustration of the four listening conditions and derived "advantages" (spatial, talker, and total) of the LiSN-S. The listening conditions are: same voice target and competing speech at 0° azimuth (SV0), same voice at ± 90° azimuth (SV90), different voices at 0° azimuth (DV0), and different voices at ± 90° azimuth (DV90). From *Ear and Hearing*, by Cameron, S. & Dillon, H. 2007, Vol 28, Issue 2. Reproduced with permission from Wolters Kluwer Health.

voice (Cameron & Dillon, 2008). Cameron et al. (2006) proposed that by subtracting the thresholds for same talker versus different talkers, and for same direction versus different directions, a measure for the ability to use different cues to suppress noise can be obtained that is minimally affected by higher order language, learning, and communication factors (Cameron & Dillon, 2007). Their initial study reported that 9 of ten children with presumed CAPD failed the "spatial advantage" measure of the prototype LiSN-CD (Cameron et al., 2006). Furthermore, a group of 9 children with suspected CAPD were found to have a deficit in the derived measure of spatial advantage in the LiSN-S, while a group of 11 children with attention or learning disorder did not demonstrate such a deficit, when compared with normative data of 70 children (Cameron & Dillon, 2008). These results suggest that the LiSN measures may be used to evaluate auditory stream segregation abilities in children that may be related to binaural interaction abilities. See Chapter 8 of Volume 2 of this Handbook for elaboration of the LiSN test and treatment program.

[redacted]nsequences of Poor [redacted]naural Interaction

Some of the most challenging acoustic environments are associated with multiple fluctuating sound sources (e.g., more than one simultaneous voice emanating from different speakers with additional degradation due to room reflections). In a normal-sized room, normal listeners perceive the leading sound signal and its immediate reflections as a single fused "auditory image." The apparent direction of this image is determined by the interaural cues associated with the earlier-arriving direct sound with suppression of the later-arriving reflections (Wallach et al., 1949; Zurek, 1980). In addition, the human brain has the remarkable ability to segregate the object of interest, such as the voice of an attended speaker, in a complex auditory scene, such as a cocktail party (Cherry & Taylor, 1954; Sayers & Cherry, 1957). Binaural listening cues underpinned by interaural time and intensity differences, as well as other auditory processes (e.g., temporal processing, auditory discrimination) may help in resolving the degraded signals in these environments (Bronkhorst & Plomp, 1992; Chen, 2005). If the different speakers are separated in space, it is easier for a listener to understand the target speech signal. This is due to both monaural advantages from improvements of speech-to-noise ratio at the better ear and to binaural advantages resulting from binaural unmasking of the low-frequency parts of the speech signal, which is facilitated by the ITD between the competing sources (Hawley, Litovsky, & Culling, 2003). It has been proposed that some of the synapses involved in auditory sensory perception in complex acoustic environments might be hardwired due to long-term evolution, thus reducing the time necessary for the complex brain computation to occur (Chen, 2005).

Deficits in binaural interaction (e.g., in the presence of MS in adults or OME in children) would therefore result in listening difficulties in complex acoustic environments and would manifest with deficits in at least two auditory processes: sound localization/lateralization and auditory performance in backgrounds of competing acoustic signals. Deficits in either or both of these processes would

be consistent with the diagnosis of CAPD (ASHA, 2005). It has been proposed that, to some extent, age-related difficulties in understanding speech in reverberant environments can be attributed to a decreased ability to perceive the precedence effect (Cranford & Romereim, 1992). In addition, it has been suggested that small MLDs in children with OME may be reflected in difficulties detecting and attending to signals in noisy environments (Moore et al., 1991). Due to the long-term maturation of the various mechanisms that underpin binaural hearing, even normally developing children have greater difficulties than adults in complex fluctuating auditory backgrounds (e.g., Hall et al., 2004). This may be particularly relevant in the classroom environment, where there may be multiple sources of noise, such as from a heating/ventilation unit, activity in an adjacent classroom or corridor, traffic or aircraft noise, student activity within the classroom, or any combination of these. Children with deficits in binaural interaction are at greater risk of having difficulties in localizing the voice of the teacher (and other students), and of having difficulties processing the speech signal of the teacher in background noise.

Primary Intervention Strategies

Human listeners may be trained to change their responses to cues of sound-source position by exposure to altered sound cues, and they may also improve their performance in sound localization with training by using normal cues, although the degree of adaptation varies across subjects (Wright & Zhang, 2006). Indeed, practice during formal testing leads to improved test performance in both ITD and IID discrimination tasks, although training results differ for the two tasks and depend on a number of factors, such as stimuli used for training, amount of training, and initial levels of performance, as well as increments of training (Linkehoker & Knudsen, 2002; Wright & Fitzgerald, 2001; Wright & Zhang, 2006).

There is a substantial capacity for improvement of binaural interaction, both in animals and in humans (Linkehoker & Knudsen 2002; Wright & Fitzgerald, 2001; Wright & Zhang, 2006). Sound localization training in a reverberant room may lead to small, albeit significant improvements for a number of spatial judgments (Shinn-Cunningham, 2000), and these improvements are both stimulus and vision dependent (Abel & Paik, 2004). This processing plasticity may well be underpinned by the special properties of the SOC at a neurochemical level, since the SOC nuclei express molecules such as GAP-43 mRNA and subunits of integrin that are known to be involved in development, plasticity, and learning (Illing, Kraus & Michler, 2000). For example, SOC neurons may respond to hearing impairment with the expression of these substances, indicating changes in neural connectivity (Illing et al., 2000). The reader is referred to Chapters 3, 4, and 5 in Volume 1 of the Handbook and to Chapters 1 and 7 in Volume 2 of the Handbook for discussions of neuroplasticity.

Auditory training (AT) should lead to improved binaural interaction. Intervention for CAPD should be deficit-focused; therefore, if test results and functional deficits reveal a binaural interaction deficit, training should target those skills. Specific formal AT could include: ITD and IID detection or discrimination tasks,

and localization and lateralization training in the free field, both in quiet and in noise and at various azimuths. Informal AT of binaural interaction also could be incorporated in everyday activities (e.g., taking a walk in the park) or could be exercised as part of a game such as "Blind Man's Bluff' and "Marco Polo." Another option is the recently developed auditory training program that targets binaural interaction called the *LiSN & Learn* program (Cameron & Dillon, 2011). This computer-based auditory training program incorporates spatial cues in the auditory training paradigm and presents sentences as the target, and distracter stories as the masker. A pilot study showed significant improvements after training in 9 children with CAPD and spatial processing deficits in the LiSN-S measures that reflect spatial processing, but not in other measures (Cameron & Dillon, 2011). The study, however, did not include any untrained controls so that maturational or practice effects cannot be excluded. In addition the same female voice was used as the target voice for the training paradigm and the outcome measure and it is thus unclear if the observed improvements were the result of task familiarity. Although these results may suggest that the *LiSN & Learn* training holds promise for the remediation of spatial processing deficits, more research is required to further investigate its efficacy.

In addition to direct remediation, environmental adaptations to minimize reverberation in the classroom and provision of an FM system to improve signal-to-noise ratios would be of paramount importance for listeners with binaural interaction deficits. Central resources (compensatory) training might also relieve listening difficulties and might include auditory closure training (Bamiou, Campbell, & Sirimanna, 2006; Musiek, 1999). The reader is referred to Volume 2 of this Handbook for extensive discussion of the range of treatment and management approaches.

Summary and Concluding Comments

Binaural interaction at the brainstem level underpins sound localization/lateralization and auditory performance in backgrounds of competing acoustic signals. Binaural interaction tests should, therefore, be included in the central auditory battery if the referring complaint (e.g., difficulties in localization or difficulties with understanding speech in noise), history (such as history of OME or MS) or the findings from other assessments (such as the binaural interaction component of the ABR) raise the suspicion that aspects of binaural hearing may be impaired or that maturation of this process is delayed in a specific individual. The currently available tests include various forms of the MLD and the BFT, and the more recently developed LiSN. It is hoped that localization/lateralization tasks will soon transfer i nto clinical practice, since they are easy and enjoyable to perform, and because these provide a measure of auditory performance in response to precisely controlled acoustic stimuli and may allow the clinician to make inferences for processing at different levels of the auditory system. In contrast, the RASP has not received much clinical acceptance because of the low sensitivity of this test in identifying patients with CAPD, as well as in differentiating between different sites of brain pathology. In

general, the clinician ought to be aware that, with the exception of the MLD, binaural interaction tests can be affected by brain pathology outside the brainstem, thus poor scores in these tests may reflect deficits in higher order processing. In addition, results should be interpreted with caution in the presence of hearing loss. However, careful application of these tests may help identify the level of the auditory pathway at which impaired processing occurs, as well as the auditory deficit(s) that need to be targeted in rehabilitation.

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CHAPTER 17

ELECTROACOUSTIC AND ELECTROPHYSIOLOGICAL AUDITORY MEASURES

In the Assessment and Management of Central Auditory Processing Disorder

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Diagnosis of central auditory processing disorder (CAPD) requires demonstration of a deficit in the perceptual (i.e., neural) processing of auditory stimuli in the central nervous system that is not due to higher order language, cognitive, or related factors, which may, however, coexist with auditory perceptual deficits (AAA, 2010; ASHA, 2005). CAPD can be associated with or lead to difficulties in learning, speech, language, attention, and social and related functions (AAA, 2010; ASHA, 2005). The potential for these frequent comorbidities underscores the need for comprehensive assessment and diagnostic procedures to fully explore the nature of the presenting difficulties of each individual suspected of having CAPD (AAA, 2010; ASHA, 2005).

The search for objective and effective methods to evaluate the integrity of the central auditory nervous system (CANS) is ongoing. Integrity of the auditory pathways is a necessary condition for the normal development of auditory abilities. Tests and procedures that reflect the auditory processes at different regions/levels within the CANS must be used to fully assess the neural processing of auditory stimuli. The two most used approaches to assess the CANS are: (a) behavioral assessment and (b) electrophysiologic assessment. Recently, electroacoustic evaluation has been used as well, based on reports of the potential of otoacoustic emissions to document the function of the efferent auditory pathways, to rule out peripheral disorders as

the cause of observed auditory processing deficits, and to strengthen differential diagnosis (e.g., auditory neuropathy spectrum disorder vs. CAPD) (Jerger & Musiek, 2000; see Chapter 19).

Voltage changes in the brain can be recorded with the use of electrodes attached to the scalp and visualized on a computer screen after appropriate amplification. This electrical activity from a large number of neurons is called an electroencephalogram (EEG) (Burkard, Don, & Eggermont, 2007). The normal EEG shows typical changes that are related to changes in the sound stimulation. These changes are called auditory-evoked potentials (AEPs). Davis was the first to report an auditory-evoked response in 1939. Over the years our knowledge about AEPs has grown substantially, due to increases in computing power and enhanced signal processing strategies, as well as the urgency of developing an objective measure of hearing (McPherson, 1996). Audiologists searching for an objective measure of hearing first focused on auditory brainstem responses (ABRs). In contrast, psychologists, psychiatrists, and neurologists focused on the late potentials because these evoked response potentials (ERPs) are associated with both perceptual and cognitive processes (McPherson, 1996).

In this chapter, we review the use of electrophysiologic and electroacoustic measures in the assessment of central auditory processing and for the diagnosis of CAPD. The role of these measures to gauge treatment outcomes is discussed. We also review current research on procedures that are evolving and may hold promise for clinical application. (See details about recording and interpreting AEPs in Burkard, Don, & Eggermont, 2007; Hall, 1992; Musiek & Rintelmann, 1999).

Electroacoustic Evaluation

Acoustic Reflex Findings

Acoustic reflex measurements are clinically useful in the identification and localization of peripheral and central auditory pathology. Acoustic reflexes may be absent, present at elevated thresholds, reduced in amplitude, or absent. Acoustic reflex latency and amplitude decay may also provide insights regarding the nature and locus of pathology. Individuals with brainstem auditory dysfunction usually present acoustic reflex abnormalities in the contralateral (crossed) measurement condition, but not in the ipsilateral (uncrossed) condition, although some studies showed acoustic reflex abnormalities in both the ipsilateral and contralateral signal conditions in individuals with CAPD (Hall & Johnson, 2007). There also are reports of excessive decay of the acoustic reflex amplitude in intra-axial brainstem pathology (Borg, 1982; Jerger & Jerger, 1977). In contrast to the above findings, Downs and Crum (1980) reported better than expected acoustic reflex thresholds in four children with CAPD, which they attributed to "decreased central inhibition of peripheral auditory system with CAPD" (p. 401). Demonstrating the range of acoustic reflex findings in CAPD, Jerger, Jerger, and Loiselle (1988) reported normal acoustic reflex findings in a group of young children with CAPD. As noted by Hall and Johnson (2007), the range of acoustic reflex findings may be due to the different regions of dysfunction across the individuals examined in these studies. For example, acoustic reflex abnormalities would be unexpected in

individuals with CAPD related to cortical or interhemispheric involvement with normal lower brainstem pathways.

Otoacoustic Emissions

The processing of auditory signals begins in the periphery, with considerable transformation of the signal in the cochlea. Individuals with cochlear dysfunction typically show decreased speech recognition in noise performance, as well as deficits in temporal processing skills, such as recognition of compressed speech (Lorenzi, Gilbert, Carn, Garnier, & Moore, 2006; Moreira & Junior, 2004; Schochat, 1997). A compromised cochlear compression mechanism caused by outer hair cell motility dysfunction (and evidenced in otoacoustic emissions, discussed in this section) may compromise temporal processing. Clearly, when cochlear function is preserved, central auditory performance is more efficient (Sanches, Sanchez, & Carvallo, 2010).

Otoacoustic emissions (OAEs) provide information relative to the integrity of the outer hair cells of the cochlea that may be not detected by other procedures, such as conventional pure-tone audiometry. Damage or dysfunction at the level of the cochlea can impact processing across the entire auditory pathway, causing, for example, difficulties in auditory temporal integration and gap detection (Gorga, Niely, Dorn, & Konrad-Martin, 2002; Smurzynski & Probst, 1999). It is important to note that peripheral auditory dysfunction can lead to transsynaptic degeneration of central auditory structures (Hardie & Shepherd, 1999). Auditory thresholds, recruitment, and frequency selectivity issues, which impact temporal processing, can result from the loss of outer hair cells, which can be assessed by OAEs (Gorga et al., 2002; Neely, Gorga, & Dorn, 2003; Oxenham & Bacon, 2003; Williams & Bacon, 2005).

In the CANS, in each nucleus of the auditory pathway, the neuronal responses change in the afferent and efferent pathway, indicating a progressive and sequential signal processing. At several points of the auditory pathway, there are efferent projections, suggesting that nuclei are not independent and are influenced by more rostral nuclei of the pathway (Rouiller, 1992).

Contralateral suppression of transient evoked otoacoustic emissions (TEOAEs) provides a direct exploration of the uncrossed (efferent) medial olivocochlear pathway. The TEOAEs are reduced by introducing a noise in the ear contralateral to that in which the TEOAE is measured. It is inferred that this effect, known as the suppression effect, results from the influence of the efferent olivocochlear pathway on the movement activity of the outer hair cells (Zhao & Dhar, 2011).

Olivocochlear neurons form the caudal aspect of the efferent system that originates in the superior olivary complex of the brainstem and projects to the cochlea. This structure is composed of a medial and a lateral portion. The lateral portion projects to dendrites of cochlear nerve fibers innervating inner hair cells. They are thin and unmyelinated, and they originate from the lateral superior olivary complex. The medial olivocochlear bundle (MOCB) is composed of large myelinated neurons and originates from the medial portion of the superior olive, projecting mainly contralaterally directly to outer hair cells (Sahley, Nodar & Musiek, 1997 for a review; Warr & Guinan, 1979). The activation of the MOCB

influences the electromotile properties of the cochlear outer hair cells. In general, the medial olivocochlear system has an inhibitory effect on the peripheral auditory system (Warren & Liberman, 1989). It has been hypothesized that due to its inhibitory effects, the efferent system plays a protective role in the auditory system (Rajan, 1990). In addition, considerable research has implicated the olivocochlear bundle as playing a role in enhancing hearing in noise, which is of course a critical factor in CAPD (see Sahley et al., 1997 for a review, but see de Boer, Thornton, & Krumbholz, 2012 for a conflicting finding).

Assessment of the integrity of the MOCB in children and adults diagnosed with CAPD (using the TEOAE suppression effect) has provided insights regarding the relationship between the MOCB and auditory processing. Muchnik et al. (2004) reported decreased MOCB activity in children diagnosed with CAPD, as evidenced by less suppression of TEOAEs evoked with nonlinear stimuli. A less active MOCB in children with CAPD, perhaps more specifically those children who experience difficulties listening in the presence of noise, may indicate reduced inhibitory function as seen in reduced TEOAE suppression (Muchnik et al., 2004).

To analyze the efferent auditory system activity by OAE suppression and acoustic reflex sensitization in human subjects with CAPD, a prospective study was conducted on 50 children with CAPD (study group) and a control group of 38 children without CAPD, obtaining OAEs with and without contralateral noise and measuring the acoustic reflex with and without contralateral facilitating stimuli. Sensitization occurs when acoustic reflex thresholds are reduced as a result of presentation of a high frequency facilitating stimulus presented prior to or simultaneously with the reflex activating tone (Kumar & Barman, 2002). The study group had lower OAE suppression values and higher acoustic reflex sensitization values when compared with the control group. The higher acoustic reflex sensitization values provide evidence of less cochlear protective function against intense sounds (Burguetti & Carvallo, 2008).

Elgeti et al. (2008) measured spontaneous OAEs (SOAEs) in 27 children with reduced speech-in-noise recognition and in a control group matched by gender and age. The presence of SOAEs was significantly higher (85%) in the affected group relative to the control group (44%). An abnormally functioning MOCB with reduced inhibition could lead to an increase in SOAEs (Elgeti et al., 2008). Measuring SOAEs in a central auditory test battery may be helpful as it provides an objective measure for the assessment process.

Though there is laboratory evidence that OAE suppression might provide insights into the integrity of the efferent pathway (MOCB), clinical utility of the procedure remains relatively low. This low acceptance in the clinical domain is likely due to the small and variable differences between OAE levels in control versus suppression conditions.

Electrophysiologic Auditory Measures

Speech and language processing can be defined as neural operations responsible for transforming the acoustic characteristics of speech into a linguistic representation (Kraus, McGee, Carrell & Sharma, 1995). These characteristics

must be processed efficiently, relying on the coordinated and simultaneous activation of a large number of neurons from the eighth nerve to the auditory cortex (Nicol & Kraus, 2004). The encoding of the acoustic properties of speech sounds occurs at all levels of the auditory system (Blackburn & Sachs, 1990), with evidence that encoding is modified and properties represented differently at each level of the auditory pathway (Creutzfeldt, Hellweg & Schreiner, 1980). In individuals with CAPD and with documented central lesions, the perception of acoustic contrast is not totally affected, with some sounds more vulnerable to disruption than others (Kraus & Cheour, 2000). (See Chapter 5 for a review of neural processing of acoustic stimuli.)

As defined by the AAA (2010), CAPD is a disorder of the CANS; therefore, obtaining information regarding the integrity of the CANS should improve the diagnosis of the disorder, assist in intervention programming, and contribute to monitoring treatment outcomes. Given the frequent comorbidity of CAPD with language, learning, and attention deficits, the inclusion of electrophysiologic measures in clinical assessments is gaining traction in clinical practice.

AEPs can be good indicators of how well the central auditory system functions and to some degree how well this system processes sound (Schochat & Musiek, 2006). Compared with behavioral tasks, many but not all electrophysiologic measures do not require the listener's active participation, and are therefore more objective and can be reliably recorded from infants and very young children. In addition, unlike psychophysical measures, electrophysiologic procedures are less affected by confounds such as memory, language, motivation, and task, and response criteria and strategies. Electrophysiologic measures, therefore, can provide a nonbehavioral means of investigating the auditory processing of sound and the underlying physiologic mechanisms (He, Grose, & Buchman, 2012).

Using these potentials, auditory processing from the level of the eighth nerve to the auditory cortex has been investigated, using ABRs and middle latency response (MLR), as well as the late cortical response, P300 response, and mismatch negativity response (MMN) (Jirsa, 1992; Kraus, McGee, Zecker, Nicol, & Koch 1996; Musiek, Gollegly, Kibbe, & Verkest, 1988; Purdy, Kelly, & Davies, 2002).

ABR and Complex Sound ABR

The conventional ABR has a strong track record of being sensitive and specific to confirmed central auditory lesions of the brainstem (see Musiek & Lee, 1995; Starr & Achor, 1975; for review, Musiek, Shinn, & Jirsa, 2007). This test efficiency varies across differing kinds of neurological involvement, but overall ABR can provide accurate insight as to central auditory involvement in the brainstem pathways. The conventional ABR has not shown high hit rates in children with learning problems, though in some cases it has been revealing diagnostically (Josey, 1985; Marosi, Harmony, & Becker, 1990). Most ABR assessment employed click or tonal stimuli; however, recently, there has been an effort to use more complex stimuli (e.g., speech segments) as stimulus, in an attempt to measure subtle processes in the brainstem related to more complex sound processing.

Complex sound ABR was first used by Nina Kraus's group at Northwestern University in a study of children with

learning problems and speech sound perception deficits (Cunningham, Nicol, Zecker, Bradlow, & Kraus, 2001) The complex sound (i.e., speech signal) has an elaborate spectral-temporal structure formed by harmonic elements that change rapidly in frequency over time and therefore require a synchronized neural response to enable accurate decoding (Kraus & Nicol, 2003; Nicol & Kraus, 2004). The complex ABR (cABR) reflects the neural synchrony and encoding of temporal characteristics of speech sounds at the level of the brainstem, providing information relevant to investigations of the neural basis of speech perception (Kraus & Nicol, 2003) (Figure 17–1).

Brainstem responses to complex sound stimuli are altered (i.e., latency delay and lower amplitudes) in children with learning disabilities and CAPD (Abrams, Nicol, Zecker, & Kraus 2006; Fillippini & Schochat, 2009; Hays, Warrier, Nicol, Zecker, & Kraus, 2003; Johnson, Nicol, & Kraus, 2005; Russo, Trent, Musacchia, & Kraus 2004). Complex sound ABR responses may be related to cortical processing (King, Warrier, Hayes, & Kraus, 2003; Wible, Nicol, & Kraus, 2005) and the efficiency of auditory training in the rehabilitation of individuals with speech perception deficits (Hayes et al., 2003; Russo, Nicol, Zecker, Hayes, & Kraus, 2005).

In a study of children with learning disabilities, Filippini and Schochat (2009) concluded that complex sound stimuli are more sensitive than click stimuli for the evaluation of CAPD, since only the speech stimuli revealed alterations in synchronicity and speech processing neural input speed. They suggested that children with CAPD may have deficits in the response generator synchronization (as evidenced by amplitude differences) and/or in the neural signaling transmission velocity (as evidenced by latency differences) (Wible, Nicol, & Kraus, 2004). Delays in the latencies of brainstem responses for complex sound stimuli have a negative impact on the ability to rapidly process acoustic signals by specialized cortical structures (Abrams et al., 2006). In fact, an impairment of speech processing at the brainstem and cortical levels may be responsible for abnormal perception of speech and thus compromise language abilities (Wible et al., 2005).

Speech perception deficits in CAPD may be related to the complexity of acoustic signals, which demands much from the auditory system. The auditory system must be sensitive to quick spectrum and level changes, to unfavorable signal-to-noise ratios, and to the simultaneous reception of many stimuli in a short time span. Much of basic acoustical processing of speech is handled automatically at the level of the brainstem, independent of higher level cognitive and language functions. Some have noted that complex sound ABRs provide important, objective, quantifiable information about the neural encoding of speech, regardless of the attention of the individual (Rocha, Filippini, Moreira, Neves, & Schochat, 2010; Russo et al., 2004). Nonetheless, a lesion or dysfunction in this caudal region of the auditory pathways could lead to difficulties understanding speech (Kraus & Nicol, 2003).

The cABR may assist in differential diagnosis as well. Rocha-Muniz, Befi-Lopes, and Schochat (2012) obtained the cABR in 75 children (6 to 12 years old) divided in three equal groups (control, CAPD, and specific language impairment [SLI]) .They reported abnormal neural encoding for different acoustic features (temporal and spectral) in the CAPD (just temporal) and SLI groups (temporal and

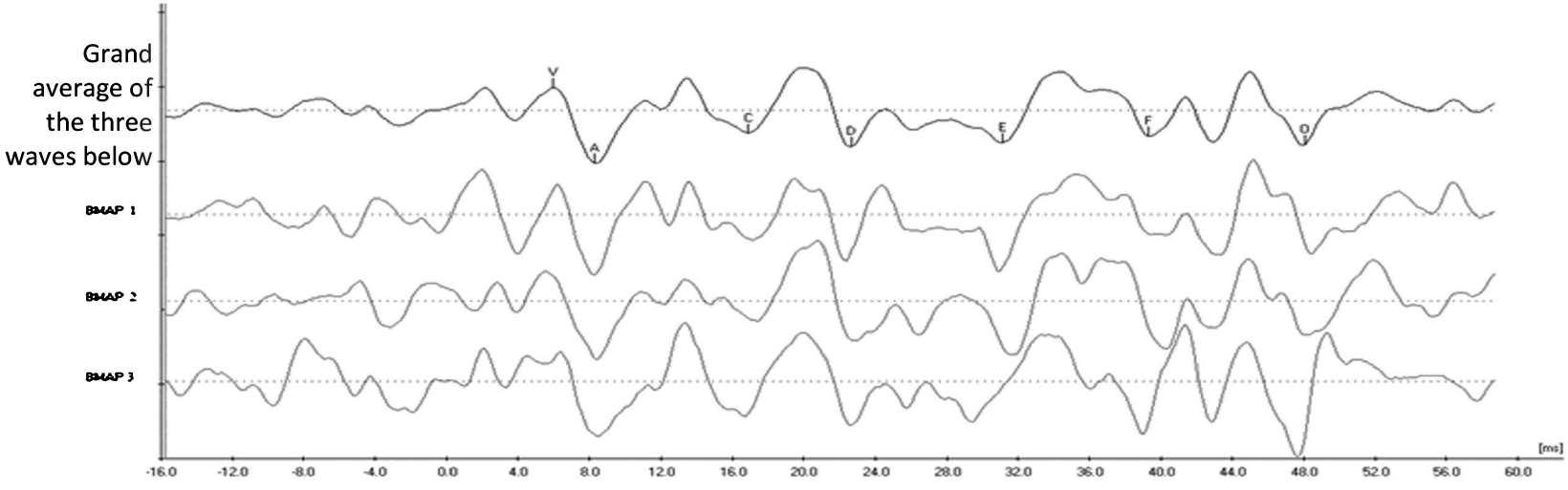

Figure 17–1. An example of three individual averages (*bottom*) of the cABR and the grand average (*top*).

spectral) relative to the controls. The SLI group exhibited poorer responses relative to the CAPD group. (See Chapter 7 for additional discussion of the complex or speech ABR.)

Presently, few reports examining cABR in individuals with various central auditory pathologies have been published. More research is needed on this measure to demonstrate its potential in assessment and diagnosis of CAPD, particularly its sensitivity and specificity to CANS involvement.

Auditory Middle Latency Response

The MLR, as is true of most AEPs, typically is evoked by clicks or tone bursts depending on the purpose of the evaluation: for neurodiagnosis or audiometry (i.e., for threshold evaluation and identification of malingering) (Hall, 1992). The MLR waves occur between 10 and 80 ms after the stimulus onset. The Pa is the most reliable wave because it is the most visible and robust of this auditory potential (Figure 17–2). Although the MLR has relatively good within-subject reliability there is variability for intersubject measures of amplitude, and this may be why MLR is still not commonly used clinically.

There is considerable evidence that the MLR is of value for evaluating lesions or dysfunctions of the CANS, and is especially sensitive to lesions of the auditory cortex and thalamocortical connections (Kileny, Paccioretti, & Wilson, 1987; Musiek, Charette, Kelly, Lee, & Musiek; Musiek & Lee, 1995; Scherg & von Cramon, 1986; Schochat, Rabelo, & Loreti, 2004; 1999; Shehata-Dieler, Shimizu, Soliman, & Tusa, 1991). Hit rates in the mid-70% range or better have been reported (Musiek et al., 1999). The amplitudes of the MLR are reduced when the auditory cortex has been damaged; however, MLR amplitude is preserved in the presence of lesions in other areas of the cortex,

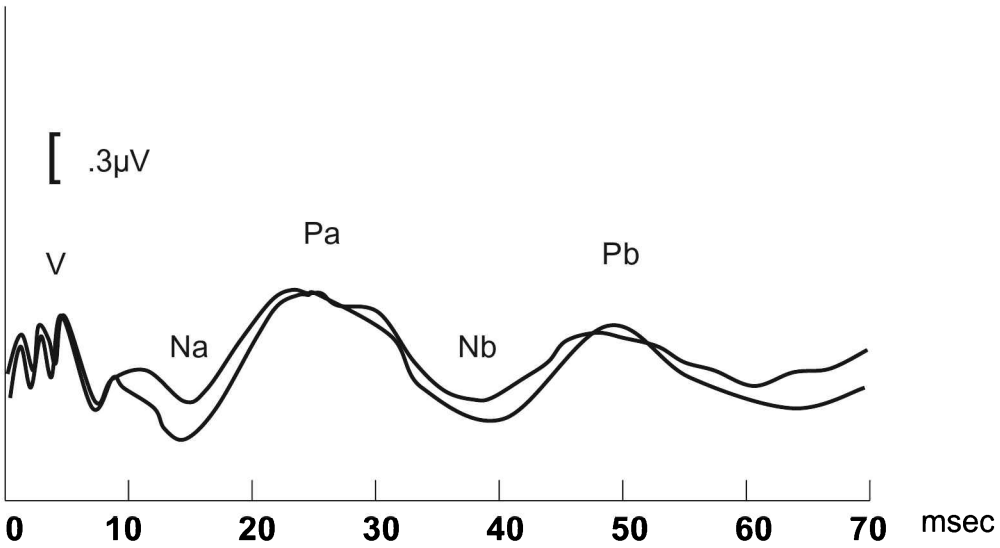

Figure 17–2. An example of a normal MLR waveform labeled with wave V from the ABR and subsequent MLR waves.

which speaks to the differential capability of this measure (Kileny et al., 1987; Shehata-Dieler et al., 1991). The MLR can be combined with the conventional ABR and can yield important information in regard to the comparison of brainstem and thalamocortical auditory function. Unlike the ABR, the MLR's more sensitive diagnostic index is amplitude rather than latencies (see Musiek & Lee, 1999).

Clearly, the MLR has the advantage of providing information regarding the integrity of the CANS through the level of the primary cortex. MLR testing is typically reserved for those individuals with normal peripheral hearing sensitivity who present with deficits that suggest involvement of the CANS beyond the generator sites for the ABR. MLR testing has an important role in diagnostic audiology as well as in the rehabilitation of individuals with neuroaudiologic disorders (Musiek, Baran, & Shinn, 2004). This electrophysiologic response does not depend on an individual's linguistic skills or cognitive processing of sound and can be used as an objective, noninvasive tool to investigate auditory processing and the plasticity of auditory function in humans.

Weihing, Schochat, and Musiek (2012) assessed 155 children with normal peripheral and central hearing to establish normative amplitude values for different measurements of MLR and to determine whether these measurements provided a significant reduction of within group variability when compared with raw, absolute amplitude measures. They defined a relative amplitude difference as the difference in Na–Pa amplitude between two electrodes or between the two ears. In contrast, absolute amplitude was defined as a single Na–Pa measurement made at one electrode for stimulation of one ear. Weihing et al. (2012) reported that within-group variability was significantly smaller for relative differences compared with absolute amplitude measures, and the electrode effects showed significantly less variability than ear effects. They concluded that relative differences may provide better utility in the clinical diagnosis of central auditory deficits in children when compared with absolute amplitude measures because these difference measures show significantly lower variability when examined across subjects (Weihing et al., 2012).

In an effort to enhance the efficiency of evaluation of the CANS, Weihing and Musiek (2008) examined binaural enhancement (BE) and binaural interaction (BI) in different levels of noise during concurrent measurement of the ABR and MLR in 15 normal-hearing adults. Subjects were exposed to 0, 20, and 35 dB effective masking (EM) of white noise during monotic and dichotic click stimulation while AEPs were simultaneously acquired. The ABR showed BE at 0 and 20 dB EM, but not at 35 dB EM; MLR showed BE at all noise levels, but the degree of BE decreased with increasing noise level. BI was seen for both ABR and MLR at all noise levels; however, BI decreased with increasing noise level. The authors concluded that ABR neural generators may respond to noise differently than MLR generators; that BE can be measured while concurrently recording the ABR and MLR in the presence of up to 20 dB of EM noise; and that BI can be similarly recorded in the presence of up to 35 dB EM of noise.

Recent studies of the MLR have aimed to find better ways to evaluate the CANS and correlate the findings with behavioral central auditory test results. Schochat, Musiek, Alonso, and Ogata (2010)

studied the MLR characteristics (latency and amplitude) in children with CAPD, who were categorized as such by their performance on a behavioral central auditory test battery. The MLR was obtained from 30 children with CAPD (ages 8 to 14) and a control group of 22 children without CAPD. The CAPD group exhibited lower C3–A1 and C3–A2 (C3 electrode was positioned over left hemisphere and C4 was over right hemisphere) wave amplitudes in comparison with the control group (C3–A1, 0.84 μV [mean], 0.39 (standard deviation [SD] for the CAPD group; 1.18 μV [mean], 0.65 SD for the control group; C3–A2, 0.69 μV [mean], 0.31 SD for the CAPD group and 1.00 μV [mean], 0.46 SD for the control group). These findings demonstrate the ability of the MLR to differentiate children behaviorally diagnosed with CAPD from matched controls.

Recently, Mancini, Durrant, Starling, and Iório (2012) conducted a study using basic audiological and electrophysiologic evaluations to compare the peripheral and central auditory pathways of 25 children with early diagnosed and treated phenylketonuria (PKU) and an age-matched control group of 35 children, aged 5 to 16 years. All participants underwent auditory evaluations, including otoscopy, pure tone and speech audiometry, immittance testing (i.e., tympanometry and assessment of contralateral stapedial reflex thresholds), and evaluations of the auditory brainstem (ABR) and MLR. Audiometric evaluation revealed pure-tone thresholds within normal limits for both groups; however, children with PKU had poorer average speech recognition scores and higher stapedial reflex thresholds at 4000 Hz. The ABR of children with PKU showed longer average latencies for waves III and V, greater interaural differences for wave V, and longer average latencies for the interpeak interval I–V compared with the control group. No significant differences between groups were seen for the latencies of the Na and Pa waves or the Na-Pa amplitude; however, electrode or ear effects were present in 87.5% of the experimental group. Mancini et al. (2012) concluded that the pontine auditory pathway may be disordered in children with PKU even when the condition is diagnosed and treated early, and despite the appropriateness of their diet.

Auditory Late Potentials

Unlike the ABR and MLR, which are considered exogenous potentials, the late potentials (i.e., event-related potentials) are considered to be both endogenous and exogenous potentials, as they are influenced both by the state of the nervous system when stimuli are presented, as well as by the physical properties of the stimuli themselves. In this discussion, the "late potentials" will include the P1–N1–P2 complex, P3 (P300) and MMN, the latter two being more endogenous than the former. Also discussed will be the acoustic change complex (ACC). The P1–N1–P2 complex is an obligatory component believed to reflect neural activation originating from adjacent generator sites in the supratemporal plane in or near the primary auditory cortex (Tremblay, Kraus, McGee, Ponton, & Otis, 2001). For instance, the P1–N1–P2 complex is considered to be sensory-evoked potentials. However, no classification system is above criticism, since it is believed that this potential (P1–N1–P2) reflects the preattentive sensory encoding of the auditory stimulus attributes, including spec-

tral and temporal cues critical to speech perception (McPherson, 1996). Studying the P1–N1–P2–N2 complex, Polich, Aung, and Dalessio (1988) concluded that intensity and interstimulus interval affect the complex's amplitude and latency values, but that repeated stimulus presentations do not, suggesting that there is an exogenous component in this late potentials. Temporal cues are crucial to the differential sensitivity for intensity, frequency, and duration, and N1–P2 components may provide a neurophysiologic means of not only identifying dysfunction in this area, but also provide an objective means of monitoring therapeutic progress in an intervention program (Musiek & Berge, 1998). Some researchers also use the N2 measurement as an obligatory response, and it is believed to be an endogenous potential when it is used in an oddball paradigm (Martin, Tremblay, & Stapells, 2007).

The P300 is an endogenous potential with an exogenous component that is triggered by the use of the "oddball" paradigm (i.e., an experimental method in which a target infrequent or "odd" stimulus is presented among more frequent standard stimuli), which requires the listener to detect the infrequent stimulus (Figure 17–3). McCullagh, Weihing, and Musiek (2009) compared the amplitude, latency, morphology, and threshold of the auditory P300 using standard oddball and omitted paradigms (i.e., the target "stimulus" is a silent gap) in 15 normal hearing adults and reported that there were lower amplitude, poorer morphology, and higher thresholds for the omitted

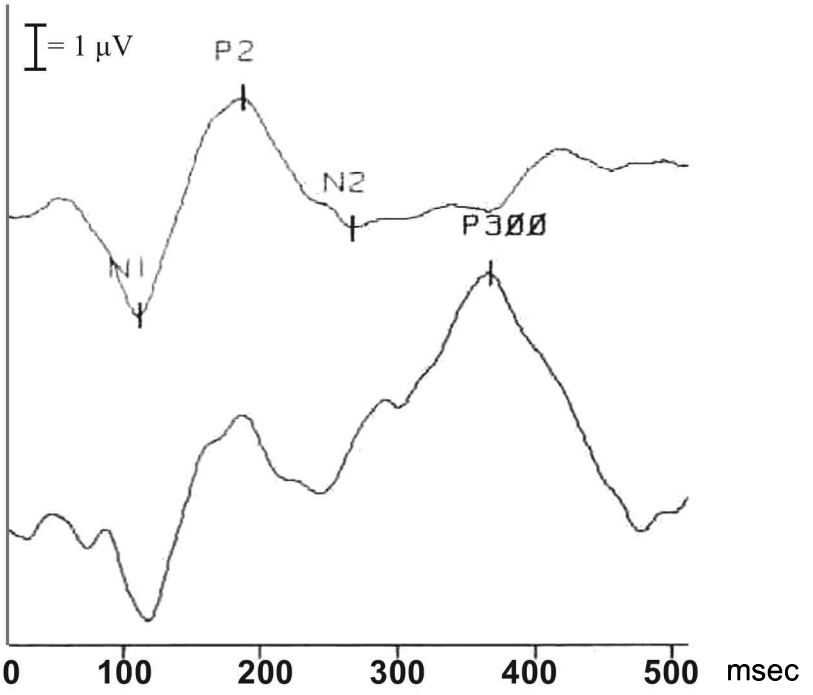

Figure 17–3. An example of a P300 waveform showing the responses from the frequent stimulus on top and from the infrequent or rare stimulus on the bottom.

paradigm compared with the standard, suggesting that P300 might have a larger exogenous component than was previously thought.

The P300 is highly dependent on attention as well as sensory processing. Generators of the P300 arise within the medial temporal lobe, limbic system, thalamus, and frontoparietal cortex. P300 is governed by attention but also engages the higher CANS. Although nonauditory regions contribute to the P300, there is evidence that lesions in auditory cortical regions (i.e., temporoparietal junction) compromise the latency and amplitude of the P300 (Knight, Scabini, Woods, & Clayworth, 1989; Musiek, Baran, & Pinheiro, 1992).

The N1 and P2 late potentials can also be recorded concurrently with P300 using the oddball paradigm. Ostroff, Martin, and Boothroyd (2008) suggested that the N1–P2 acoustic change complex (ACC) response might indicate auditory discrimination capacity. It has been suggested that the ACC comprises overlapping slow cortical responses, which reflect auditory discrimination capacity in the absence of attention. It is thought that the AAC represents a change detection response rather than a simple onset response, even though the P1–N1–P2 complex elicited by a brief acoustic stimulus and the ACC response elicited by speech tokens (e.g. /sei/) show similar general characteristics (Martin, Tremblay, & Korczak, 2008).

P1–N1–P2 Complex

As one might expect, given that their generator sites are at or near the primary auditory cortex, the P1–N1–P2 complex often is affected by confirmed involvement of the auditory cortex. Historically, lesion studies have shown latency and/or amplitude affects related to central auditory involvement of the auditory cortex and related areas (Knight, Hillyard, Woods, & Neville, 1980; Knight et al., 1988). In fact these late potentials have been shown to be compromised when auditory regions were involved, but not so for nonauditory areas of the brain (e.g., frontal lobe) (Knight et al., 1980). Concerning the N1–P2–N2 complex, one study found a significant difference in the latency of wave N1 and the amplitude of wave P2 in children with CAPD relative to normal children (Zalcman & Schochat, 2007). In 2001, Tremblay and colleagues investigated the effects of training subjects to differentiate novel temporal cues in the N1–P2 complex. They found that as the subjects learned to differentiate the temporal cues, there was a significant increase in N1–P2 amplitude, which they felt resulted from an enhancement of the N1–P2 neural generators subsequent to the training program.

Acoustic Change Complex

The ACC might provide a useful tool in populations for whom the reliability of behavioral responses is questionable, such as in infants and very young children. However, the N1–P2 ACC has not yet been recorded in infants. Because the ACC is a large response, it may be useful as an index of discrimination for infants at both the individual and group level. This gives the ACC a clinical advantage compared with MMN, another AEP that reflects discrimination of a change in a stimulus, because MMN is based on difference waves (requiring a more complicated paradigm) and is most sensitive

to group rather than individual effects. Despite the ACC's potential clinical application, research on the relationship between the ACC response and auditory discrimination abilities is sparse, and the studies conducted thus far have used different measures and procedures, which can affect the interpretation of results (He et al., 2012). Nonetheless, ACC appears to be a promising tool for the objective evaluation of auditory discrimination abilities (Small & Werker, 2012).

He et al. (2012) investigated the relationship between the ACC and perceptual measures of auditory discrimination and the effect of the magnitude of the acoustic change on the amplitude and latency of the ACC. Their findings showed that ACC thresholds were more variable than behavioral thresholds, as indicated by large standard deviations of the means for all acoustic dimensions, which could have been due to the larger step-sizes that were used in the electrophysiologic measures than those used for behavioral measures. They concluded that ACC can be used as an objective index of auditory discrimination for frequency and intensity, and that ACC amplitude, which was more consistent across acoustic dimensions, is a better indicator of auditory processing than ACC latency (He et al., 2012).

Future studies must confirm that ACCs can be recorded in response to a variety of stimuli, and determine how much of a change is needed before an ACC can be elicited in infants, as well as determine the optimal stimulus parameters for measuring ACCs in infants of different ages.

P300

Various studies have shown P300 abnormalities in individuals with neurological central auditory involvement, including those with dementia (Goodin, Squires, & Starr, 1978; Michaelewski, Rosenberg, & Starr, 1986; Musiek et al., 1992). Both latencies and amplitudes of the P300 complex have been shown to be affected by central auditory disorders in the auditory areas of the cerebrum. Knight et al. (1989) reported that individuals with lesions of the temporoparietal junction yielded reduce P300 amplitudes compared with individuals with lesions limited to only the parietal lobe. This study speaks to the underlying neuroanatomy key to the P300 recording. Of special note, the P300 has successfully identified those with acute and long-standing auditory effects of traumatic brain injury (TBI) (Musiek, Baran, Shinn & Jones, 2012; Segalowitz, Bernstein, & Lawson, 2001).

CAPD often is comorbidly presented with learning disorders in children. Wiemes, Kozlowski, Mocellin, Hamerschmidt and Schuch (2012) conducted a study to examine whether children with reading and writing disorders and prolonged P300 latencies also presented altered Staggered Spondaic Word (SSW) test (Katz, 1962) and speech-in-noise test results suggestive of CAPD. All subjects presented normal peripheral hearing and normal ABRs. They divided their subjects (7 to 14 years old) into two groups based on the P300 latency: group A, with latencies above 335 msec and group B, with latencies below 335 msec. Using this cutoff, they reported that children with P300s exceeding 335 msec presented abnormal results on the SSW and the speech-in-noise test, suggestive of CAPD.

Jirsa and Clontz (1990) demonstrated the use of the late potentials, specifically the P300 in diagnosing children with CAPD. In their study, the latency of the P300 was greater for a control group compared with the children with CAPD.

Though N1 and P2 trended to be later in latency in the CAPD group, these differences did not reach statistical significance. Interestingly, Krishnamurti (2001) showed that P300 latencies were delayed for an adult group with CAPD (as diagnosed using behavioral central auditory tests) compared with a control group for binaural (tone) stimulation and for a competing noise condition. The latencies were significantly later in the noise condition than the binaural condition for the CAPD group, but not for the control group.

Mismatch Negativity Component

The MMN response is an event-related component of the larger auditory evoked response that reflects the detection of acoustic change by mechanisms within the central auditory system. It is believed that the neurophysiologic change that precedes behavioral change can be evaluated via MMN. The MMN is elicited using an oddball paradigm in which a repetitive string of standard stimuli is occasionally interrupted by a deviant (i.e. "oddball") stimulus. The paradigm is based on the premise that a neural trace or template is formed to represent the standard stimulus and held in short term memory (Näätanen, 1995), and unlike P300 can be elicited even when subjects ignore the sounds presented to them (Martin et al., 2007).

In a study of the reliability of the MMN in an adult population, Dalebout and Fox (2001) found that the replicability of the derived difference waveforms was poor and the identification rate of the MMN was too low (29%). However, the MMN waveform can be robust with good recordability (Escera, Yago, Polo & Grau, 2000; Pekkonen et al., 1995) even in the absence of the participant's attention or performance of a behavioral task (e.g., in sleeping infants) (Ruusuvirta Huotilainen, Fellman, & Näätänen, 2009), as well as in stroke patients, comatose patients (Fischer et al., 1999; Fischer & Luauté, 2005), and patients in persistent vegetative states (Wijnen, van Boxtel, Eilander, & de Gelder, 2007).

In order to verify whether MMN is suitable to add information to the behavioral auditory central tests, Bauer et al. (2009) assessed 32 children with CAPD and 13 healthy controls (mean age 6 years, range 5–7). They used three different types of sound deviants presented in a multi-deviant MMN design. The incidence of MMN was always higher in the healthy children, and they had peak latency that occurred significantly earlier in frontal, central, and temporal electrodes sites. They concluded that the MMN can supplement behavioral auditory central data.

Neuhoff et al. (2012) studied the MMN as an endophenotype in three groups of children (n = 225): those diagnosed with dyslexia, a second group who were the siblings of the participants with dyslexia who had average reading and spelling skills, and a third group composed of control children—nonrelated and presenting average reading and spelling skills. The results showed that all children showed a good MMN activity to /ba/ and /da/ contrasts; only the late MMN component (300–700 ms) revealed significant group differences. Both groups affected by the dyslexia gene showed attenuated response for this late MMN response. The authors concluded that the MMN could be a potential endophenotype for dyslexia showing an analogous

alteration of neurophysiologic processes in children with dyslexia and the normal siblings with a genetic risk for dyslexia.

Another promise for the use of MMN is the investigation of prosodic abilities of infants and as an early predictor of SLI, which is commonly diagnosed at a later age. Weber, Hahne, Friedrich, and Friederici (2005) hypothesized that the prosodic abilities of infants at risk for SLI are less elaborated than those of controls because of the less efficient processing of the relevant acoustic cues. One of the most critical prosodic cues for word segmentation is stress pattern, so the researchers used a passive oddball design. Infants (5 months old) were grouped retrospectively based on their production performance at the ages of 12 and 24 months. In contrast to matched controls, infants with very low word production showed an MMN with significantly reduced amplitude of the discrimination response. This amplitude difference indicates impaired prosodic processing of word stress during early development and may thus be taken as an early marker of risk for SLI.

It has already been demonstrated that the MMN is a good potential to be used for large groups (Hall & Johnson, 2007). Additional studies are necessary to determine whether this potential will be useful in clinical settings, in terms of time required for administration, and as importantly, whether recordings obtained from individual patients will provide useful insights.

Multiple Auditory Potentials

The effects of contralateral noise on the CANS have been investigated using multiple auditory potentials in an effort to improve diagnostic capability for central auditory disorders. For example, Cranford and Martin (1991) showed that contralateral speech noise did not influence the ABR or MLR, but did compromise the N1, P2, and P300 (especially in older individuals). Schochat, Matas, Samelli, and Carvallo (2012) verified the effects of contralateral (white) noise on OAEs and AEPs in 25 normal hearing subjects, aged 18 to 30 years. In general, the latencies of the various auditory potentials were increased and the amplitudes were reduced in noise. The amplitude of OAEs decreased significantly in the presence of contralateral noise. These results suggest that the efferent system acts to inhibit responses at both caudal and rostral portions of the CANS. The calculated estimate of the inhibition probabilities (using a multinomial model via weighted least squares—see Koch et al., 1985 or Agresti, 2002 for details) was higher in the caudal portion of the auditory system (ABR: 96%) and lower in rostral portions (P300: 80%), as can be seen in Table 17–1. This may reflect the role of attention, as rostral structures are more susceptible to the influence of attention (Schochat et al., 2012).

Table 17–1. Estimates and Standard Errors of the Inhibition Probabilities

Test	Estimate	Standard Error
ABR	96%	4%
MLR	92%	5%
N1P2	88%	7%
P300	80%	8%

Comparison of Different Auditory Evoked Responses in the Diagnosis of CAPD

Our literature review showed some advantages and some disadvantages (Table 17–2) for the use of AEPs in the diagnosis of CAPD. Below, we summarize the principal findings.

Use of Auditory Evoked Potentials in Intervention

In addition to their use in diagnosis, AEPs can be used to monitor progress and outcomes of interventions, in particular auditory interventions or auditory training (Jirsa, 1992; Musiek & Berge, 1998). Auditory training may lead to the alteration of the neural activity that provides the necessary coding for speech sound processing, and changes in neural activity may then be integrated into changes in functional behavior (Tremblay, Kraus, & McGee, 1998).

Given their objective assessment of the CANS and because neurophysiologic change often precedes behavioral change (Dalebout & Stack, 1999; Kraus et al., 1995; Tremblay et al.,1998), electrophysiologic measures may offer significant advantages over the traditional behavioral measures to assess progress in therapeutic programs. Some studies used commercial auditory processing training software to study the plasticity and the neural timing in the central auditory nervous system of children diagnosed with learning disabilities (Hayes et al., 2003; Russo et al. 2005) and/or attention deficit disorder (Hayes et al., 2003). Prior to and after three months of the auditory training, they were evaluated using complex sound ABR in silent and in noise and cortical responses to speech stimuli. They concluded that the children who were trained exhibited plasticity of neural encoding of speech sounds by improving neural synchrony in the auditory brainstem, demonstrating that training programs affect both the perception and the cortical representation of sound. (See Chapters 3, 7, 8, and 9 in Volume 2 of the Handbook for discussion of auditory training.)

Zalcman and Schochat (2008) sought to verify the N1–P2–N2 complex characteristics (latency and amplitude) in children with CAPD and also to verify the evolution of such characteristics after auditory training (AT). Thirty individuals with CAPD (SG) and 22 individuals without auditory processing disorder (CG) were selected, ranging in age from 8 to 16 years old. The SG was subjected to an auditory training program in an acoustic booth during eight sessions and was reevaluated later by both behavioral and electrophysiologic tests. Between the pre- and post-auditory training situations, there was a significant difference in the latency of wave P2 and the amplitudes of waves N1 and P2, showing that there was an increase of neural connections, providing an increased amplitude of waves N1 and P2 as well as decreased latency of P2. In the CG, there was no significant difference between the initial and the second evaluation three months later, as they were not subjected to any training. This fact emphasizes the stability of this AEP. The authors concluded that the electrophysiologic measures of the N1–P2–N2 complex seem to be a good instrument for assisting in the diagnosis and the therapy monitoring of children with auditory processing disorder.

Table 17–2. Advantages and Disadvantages of Different Auditory Evoked Responses in the Diagnosis of Central Auditory Processing Disorders

Potential	Advantage	Disadvantage
ABR	Anatomy/physiology known; Neurodiagnosis of auditory brainstem dysfunction; Assesses neural temporal function; Highly reliable in young children.	Low sensitivity for CAPD (usually normal); No information on auditory function for pathways rostral to brainstem.
c-ABR	Investigation of complex sounds processing in throughout auditory pathway.	Absence of sensitivity and specificity data; Influenced by stimuli characteristics of complex sounds.
MLR	Closer relation to behavioral findings and high correlation with CAPD; Good for intragroup analysis; The best AEP for evaluation and monitoring changes after AT; Measurable in young children; Information on primary auditory cortex.	High response variability for intergroup analysis; Susceptibility to effects of sleep, sedation and other drugs; Not well defined yet which is the best parameter to analyze in CAPD latency and/or amplitude and ear and/or electrode effect.
N1–P2–N2	Origin in auditory cortex; Good for attention/memory assessment; May provide information on effectiveness of intervention.	Influence of sleep and drugs (e.g., sedatives); Not well defined yet protocols and parameters of analysis in CAPD patients.
P300	Assessment of higher level auditory processing; Elicited with complex stimuli (e.g., speech); High level origin in auditory system (e.g. cortex and hippocampus); Relation to behavioral findings; Information on effectiveness of intervention.	Affected by other alterations as memory and/or attention. Some subject cooperation required; Not well defined yet protocols and parameters of analysis in CAPD patients.

continues

Table 17–2. *continued*

Potential	Advantage	Disadvantage
MMN	Cortical origin; Can be recorded even in the absence of the subject attention or behavioral task, e.g., in sleeping infants, stroke patients and in comatose and persistent–vegetative-state patient; Complex stimulus can be used (e.g., speech); Important relation to behavioral findings; Information on effectiveness of intervention	Reliability in individual subjects is questionable caused by low identification rate of MMN; Poor replicability waveforms; Not an established clinical technique due to the time required and poor individual value for diagnostic.

To detect changes in CANS after AT, Alonso and Schochat (2009) conducted a study on P300 in young individuals. Their comparison between the evaluations made before and after the AT showed that there was a statistically significant difference among P300 latency values, and they also found decreased latency and increased amplitude of wave P300 after AT. P300 appears to be a useful tool to monitor CANS, as is the N1–P2 complex.

Another study of the N1–P2 complex showed that this complex responded differently to listening training. Significant changes in P1 and N1 amplitude were recorded over the right hemisphere. In contrast, increases in P2 were observed bilaterally. The authors suggested that these results indicate that training-related changes in neural activity are reflected in far-field aggregate neural responses and that distinct patterns of neural change, perhaps reflecting hemispheric specialization, likely represent different aspects of auditory function (Tremblay & Kraus, 2002).

The effect of auditory training on MLRs was investigated in children with CAPD (Schochat et al., 2010), who were categorized as such by their performance on the central auditory test battery. The authors also investigated the effects of auditory training on test performance. The 30 children with CAPD were 8 to 14 years of age were enrolled in an 8-week auditory training program and then retested at the completion of the program.. After training, the MLR C3–A1 and C3–A2 wave amplitudes of the CAPD group significantly increased, so that there was no longer a significant difference in MLR amplitude between the CAPD and control groups. These findings suggest progress in the use of electrophysiologic measurements for the diagnosis and treatment of CAPD.

To verify the effectiveness of formal AT in children with hearing and language disorders through behavioral assessment and cABR, with and without background noise, Filippini et al. (2012) evaluated 30 children (aged 7–13 years), divided into

four groups: normal development (ND, n = 7), CAPD (n = 9), SLI undergoing AT (SLIa, n = 6), and SLI not undergoing AT (SLIb, n = 8). All underwent behavioral assessment of auditory processing and cABR with and without background noise and all children were reevaluated 12 weeks after the initial assessment. Only the groups that underwent AT showed improvements at the final assessment. The CAPD group presented smaller amplitudes of the transient portion of the response and altered VA complex duration and slope, which were stable after the AT. Regarding cABR with background noise, the SLIa group presented significant improvements in wave latencies after AT (which were delayed before AT), becoming similar to the responses presented among the other studied groups. The authors concluded that the cABR with background noise may be an effective tool to monitor the effects of AT. Table 17–3 lists several studies with their AEP results in individuals subjected to formal AT.

Conclusions and Future Directions

As discussed in this chapter, electroacoustic measures (OAEs and acoustic reflex) can provide some insights as to how the central auditory system is functioning, especially if noise is used to suppress efferent activity. Likewise, AEPs can be useful for clinical assessment (especially ABR, MLR, and P300) across multiple

Table 17–3. Selected Auditory Training Studies Using Auditory Evoked Potentials As Outcome Measures

Auditory-Evoked Potential	Reference	Results After AT
N1–P2–N2 complex	Tremblay & Kraus (2002)	Changes in N1P1 amplitude in the right hemisphere Increase in P2 amplitude bilaterally
N1–P2–N2 complex	Zalcman & Schochat (2008)	Increase in amplitudes of waves N1 and P2
P300	Jirsa (1992)	Decrease in latency and increase in amplitude of wave P300
P300	Alonso & Schochat (2009)	Decrease in latency and increase in neural connections
MLR	Schochat et al. (2010)	Decrease in MLR latency
Complex ABR	Filippini et al. (2012)	Decrease in wave latencies with background noise

levels and regions of the auditory system, from brainstem to cortex. The cABR and the ACC may be useful tools for investigating CAPD; however, additional research on these evoked responses is needed, especially with individuals with defined lesion sites. This would not only provide valuable information as to the possible generator sites for the latter segments of the ACC evoked response, but also might provide insight as to its sensitivity to central auditory disorders with a neurological basis.

New strategies and techniques are being developed to probe central auditory processing, the CANS, and intersections with higher order processing. Evoked potentials that reflect auditory processing beyond basic perceptual levels may provide electrophysiologic insights into the bridge between processing in the CANS and more global language and executive processes. One such potential is the N400, a negative-going voltage deflection related to semantic processing and higher level decisions, which is elicited by any potentially meaningful stimulus, such as a word (Kutas & Hillyard, 1984). It occurs from approximately 200 to 600 msec, peaking around 400 msec after stimulus onset. The specific generators are not yet known; however, it is generally accepted that it has multiple endogenous sources. Its amplitude is reduced by factors thought to increase the activation of stored conceptual information associated with the stimulus (e.g., stimulus repetition, word frequency, and the expectancy of a word in a sentence context). (See Kutas & Federmeier, 2000 for a review.) Among the new approaches emerging in the literature are the recording of AEPs while the individual listens to a behavioral stimulus but is not required to respond to that stimulus. For example, Palmer and Musiek (2013) recorded N1-P2 in response to gaps in broadband noise and examined the correlation between the electrophysiologic response to the gap and the previously obtained behavioral gap deteciton threshold. Their finding that waveform amplitude is a good index of gap detection suggests that this AEP might someday provide an objective measure of temporal resolution that could be used with patients who are not able to participate in the behavioral task. Another interesting strategy to augment the potential of AEPs in the diagnostic process is to examine the microstructure of not only onset but also offset evoked potential responses (Gonzalez & Musiek, 2012).

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SECTION 4

[redacted]rential Diagnosis

CHAPTER 18

AGING OF THE AUDITORY SYSTEM AND DIFFERENTIAL DIAGNOSIS OF CENTRAL AUDITORY PROCESSING DISORDER IN OLDER LISTENERS

TERI JAMES BELLIS and LINDSEY E. JORGENSEN

Much of the focus in central auditory processing disorder (CAPD) has been on the relationship between central auditory dysfunction and learning, language, listening, communication, and related function in school-aged children. However, in recent years, attention has turned to the relative contribution of central auditory nervous system (CANS) function to the listening and speech understanding abilities of older adults. It is generally accepted that elderly individuals have difficulty with understanding spoken language, particularly in situations of competing background messages. What is less clear, however, is the underlying cause(s) of the speech perceptual difficulties of aging listeners.

Although it is generally accepted that older adults have difficulty with speech perception in difficult listening situations, the ways in which underlying changes in physiology associated with the aging central nervous system (CNS) affect the auditory processing of older adults are less understood. The majority of the literature pertaining to aging listeners has focused primarily on the effects of aging on the peripheral auditory system. Many investigators have attributed the difficulties experienced by aging listeners in the processing and understanding of speech to age-related changes in the auditory periphery (e.g., Cooper & Gates, 1991, 1992; Divenyi & Haupt, 1997; Humes, 1996; Humes & Christopherson, 1991; Humes & Roberts, 1991; van Rooij & Plomp, 1991; Wiley, Cruickshanks, Nondahl, Tweed, Klein, & Klein, 1998). Others have focused on the impact of cognition on speech perception and understanding (e.g., Gates, Cobb, Linn,

Rees, Wolf, & D'Agostino, 1996; Gordon-Salant & Fitzgibbons, 1997; Hallgren, Larsby, Lyxell, & Arlinger, 2001; Sommers, 1996, 1997). Some studies have even found that changes in audibility associated with the peripheral system can, conversely, impact performance on cognitive tests (Jorgensen, 2012; Moore, Voytask, Kowalski, & Maddens, 2002; Oron, Zwecker-Lazar, Levy, Kereitler & Roth, 2011).

Notwithstanding the importance of both the auditory periphery and higher-level cognitive function to speech perception, several studies have focused on the impact of age-related changes in the CANS on auditory perceptual abilities (e.g., Bellis & Wilber, 2001; Golding, Mitchell, & Cupples, 2005; Jerger, 1992; Jerger & Chmiel, 1997; Jerger, Jerger, Oliver, & Pirrozzolo, 1989; Otto & McCandlis, 1982; Stach, Spretnjak, & Jerger, 1990; Strause, Hall, & Burger, 1995). Although studies of the effects of biological aging and peripheral auditory dysfunction on neural encoding in the animal model may be found in the literature (e.g., Malmo & Malmo, 1982; Rose et al 1967; Spoendlin, 1971; Willott, 1986; Willott, Parkham, & Hunter, 1988a, 1988b, 1988c; Wolf, Ryan & Bone, 1981), the majority of earlier human studies failed to control for peripheral auditory dysfunction, including very subtle dysfunction that may not readily be apparent on behavioral audiometric testing. Nonetheless, more recent research has begun to investigate in a systematic manner the relative contribution of peripheral auditory, central auditory, and cognitive factors to listening and speech recognition in aging adults. In 2012, The American Academy of Audiology Task Force on Central Presbycusis concluded that there is sufficient evidence at the present time to conclude that there is an impact of aging on the CANS, although they caution that such effects likely interact in a multifactorial manner with age-related changes in the auditory periphery and in cognitive function (Humes et al., 2012). Furthermore, problems with central auditory processing, hearing loss, and cognitive decline not only overlap and coexist but may exacerbate one another, as discussed later in this chapter.

In this chapter, the factors contributing to listening and auditory processing difficulties in older listeners will be explored. Particular attention will be paid to methods of disentangling peripheral auditory and cognitive deficits from central auditory dysfunction for purposes of differential diagnosis of CAPD in older listeners. Finally, key intervention strategies for older listeners who exhibit central auditory dysfunction with and without concomitant peripheral hearing will be discussed.

Prevalence of and Risk Factors for Auditory Processing Difficulties in Aging Listeners

Examination of the CANS in the elderly population has shown degenerative anatomical changes on postmortem inspection, although a majority of studies have analyzed a small sample of the brain (Brody, 1955; Ellis, 1920; Hansen & Reske-Nielsen, 1965; Kirikae, Sato, & Shitara, 1964; Konigsmark & Murphy, 1974). This leads one to believe that these changes in physiology could have an impact on the function of the entire auditory system and suggests further that aging may lead to CAPD. Various figures have been offered for the prevalence of CAPD in

older listeners. Stach et al. (1990) were perhaps the first to investigate what they termed "central presbycusis" in the older population, at the same time attempting to control for confounding effects of peripheral hearing loss and general cognitive decline. They found that 70% of adults aged >60 years in their clinical population exhibited some degree of central auditory dysfunction. CAPD prevalence increased with increasing age, with 17% prevalence in the 50 to 54 year age group, but a remarkable 95% prevalence in the clinical population aged 80 years and older. In their nonclinical sample, they found a lower, yet still significant, prevalence, with 61% of those aged 75 to 79 years and 72% of those aged 80 years and older demonstrating some degree of CAPD.

In contrast, Cooper and Gates (1991) reported a much lower prevalence of central auditory disorder in older listeners. Using the Framingham cohort, the authors found a 22.6% overall prevalence in their subject aged 65 to 93 years, with no significant effect of gender on CAPD prevalence. Cooper and Gates (1991) suggested that the prevalence of CAPD increased with increasing age, but that age alone accounted for only 15% of the total variability. Other estimates of the prevalence of CAPD in the aging population have been offered (e.g., Jerger et al., 1989; Rodriguez, DiSarno, & Hardiman, 1990). The degree to which peripheral auditory dysfunction and cognitive factors were controlled, the use of clinical or (community) based populations, and the definition and diagnosis of CAPD in these studies could explain the differences in estimates of prevalence.

In a more recent, population-based study of the prevalence of CAPD in 2015 Australian adults aged 55 years and older, Golding, Carter, Mitchell, and Hood (2004) also found that the prevalence of central auditory disorder increased with increasing age. More specifically, they reported that older listeners are more likely to perform poorly on a greater number of tests of auditory function. Additionally, depending on how the disorder was defined, the prevalence figures could range from 2%, using a strict criterion of failure on all seven measures of auditory function employed in the study, up to 76.4% when a lax criterion of failure on one or more measures was used. Neither peripheral hearing loss nor cognitive factors could account for their findings. In line with previously reported studies (e.g., Dubno, Lee, Matthews, & Mills, 1997; Jerger, Chmiel, Allen, & Wilson, 1994; Wiley et al., 1998), Golding et al. (2004) reported that central auditory dysfunction was more prevalent in men than in women. The authors concluded, as did most investigators before them, that the prevalence of CAPD in the aging population is of sufficient magnitude that its impact cannot be ignored.

Further examining the previously mentioned Australian cohort, Golding et al. (2005) attempted to delineate the risk factors associated with central auditory dysfunction in older listeners. They defined differing degrees of CAPD and reported that moderate or severe CAPD was more prevalent with increasing age and resulted in increased self-perceived hearing handicap. The self-perception of hearing difficulty is not surprising given the deficits experienced by these adults. The authors explained further that severe CAPD was more common in men than in women; however, gender did not appear to be a predictive factor for mild or moderate CAPD. Decreased cognitive function was a significant predictor for

central auditory dysfunction of all severities. Finally, general physiologic and life-related factors such as cardiovascular health, diabetes, alcohol consumption, presence of a social network, and living arrangements (e.g., alone or with others) were not associated with increased CAPD prevalence.

It has been suggested that hearing loss, in general, is twice as likely in individuals with dementia or other mental disorders as compared with those with normally aging cognitive function (Hodkinson, 1973; Kay, Beamish, & Roth, 1964; Uhlmann, Larson, Rees, Koepsell, & Duckert, 1989). As the CNS is compromised in those with dementia or cognitive dysfunction, it is not surprising that there is a higher incidence of auditory processing difficulties in this population (Gates et al., 1995, 1996). More specifically, Gates et al. (1995) reported that CAPD is significantly more likely even in those with mild "probable" Alzheimer's disease than in age-matched controls without dementia, even though their peripheral auditory findings were similar. This would lead one to conclude that the prevalence of CAPD within the population of aging adults with dementia and other cognitive impairments is significant.

Taken together, the results of these studies suggest that there is a significant impact of aging on the central auditory system and that there is a sufficient prevalence of CAPD in the aging population to be of clinical concern. Potential impact of the disorder in older adults includes increased self-assessment of hearing handicap (e.g., Chmiel & Jerger, 1996; Golding et al., 2005; Jerger, Oliver, & Pirozzolo, 1990). In addition, when central auditory dysfunction occurs comorbidly with peripheral hearing loss, there may be an adverse impact on an individual's ability to realize benefit from the use of amplification (e.g., Chmiel & Jerger, 1996; Chmiel, Jerger, Murphy, Pirozzolo, & Tooley-Young, 1997; Jerger et al., 1990). Although men appear to be more at risk for age-related central auditory dysfunction than women, the relationship between gender and CAPD in aging is less than clear, and may differ by gender depending on the nature of the CAPD and the point-of-time in the life span (Bellis & Wilber, 2001), with some forms of central auditory dysfunction occurring during the middle-aged years (e.g., Bellis, Nicol, & Kraus, 2000; Bellis & Wilber, 2001; Jaaskelainen, Varonen, Naatanen, & Pekkonen, 1999). While aging adults may be at risk for developing CAPD, those with significant cognitive changes, such as those with dementia, are at significantly higher risk for developing difficulty with central auditory processing. Furthermore, both peripheral hearing loss and cognitive decline interact in a complex manner with central auditory dysfunction, and the presence of CAPD may influence results of psychiatric assessment of older patients and may be an early marker for the onset of dementia (e.g., Gates et al., 1996, 2002, 2008, 2011; Strouse et al., 1995). See Chapter 15 in Volume 2 of the Handbook for additional discussion of auditory and nonauditory changes in aging listeners.

Factors Affecting Auditory Processing and Speech Recognition in Older Listeners

When classifying hearing loss associated with aging, Otto and McCandlis (1982) concluded that there is evidence of changes in both behavioral and elec-

trophysiological auditory measures that accompany senescence. While changes due to peripheral hearing loss were noted, there were also changes in electrophysiologic responses (auditory brainstem response) that occurred with increasing age and that could not be explained by peripheral hearing loss alone. They concluded that both peripheral hearing loss and central changes due to age affected the responses.

It is accepted that difficulties with spoken message recognition, especially in difficult listening situations, are more difficult in the aging adult. There are three primary hypotheses that have been offered to quantify the decreased speech perceptual abilities of older listeners (CHABA, 1988; Humes, 1996; Humes & Dubno, 2010): The peripheral hypothesis attributes the speech perceptual difficulties experienced by aging individuals to age-related dysfunction in the cochlea and VIII nerve. The central hypothesis suggests that age-related changes in the CANS, independent of peripheral auditory dysfunction, may lead to decreased speech perceptual abilities in the elderly. Finally, the cognitive hypothesis suggests that general age-related cognitive deterioration leads to processing difficulties in a variety of modalities, including those that are specific to listening and speech perception. The Committee on Hearing, Bioacoustics, and Biomechanics for the National Academy of Sciences (CHABA) (1988) suggested that the impact of changes in the auditory periphery may be further divided into the impact of audibility and the impact of cochlear pathology. Recent evidence suggests, however, that all of these factors interact in a complex and multiplicative manner, and the relative contribution of each to listening and communicative function is highly individualized (Humes et al., 2012).

Peripheral Auditory Factors

It has been well accepted for many years that there are significant changes in the peripheral auditory system associated with aging (Schuknecht, 1955, 1964). As discussed by CHABA (1988), aging can affect the peripheral system in two different manners. First, age-related peripheral pathology can impact the audibility of the signal. Second, effects beyond simple audibility of the signal can occur secondary to cochlear pathology. These factors will be discussed briefly as they impact the signal that is sent to the CANS.

Audibility

Lack of audibility is the initial confound for signal perception for those with hearing loss. If the signal is not loud enough to be "heard" by the CANS, it will not be perceived accurately. The most important factor for speech understanding is an audible signal (Humes, 1991, 2007; Kamm et al., 1978; Otto & McCandlis, 1982). The overall decrease in intensity caused by a hearing loss alone is a disadvantage, even if no recruitment, nonlinearity, or other cochlear distortions exist. However, it is the variability in intensity and frequency that occurs in speech that makes parts of speech inaudible and thus makes speech understanding difficult. A number of investigators have concluded that the single most important factor affecting speech perceptual abilities in the elderly is peripheral hearing sensitivity and signal audibility (e.g., Bess & Townsend, 1977; Humes & Christopherson, 1991; Humes & Roberts, 1990; Marshall & Bacon, 1981; van Rooij & Plomp, 1991). Cooper and Gates (1991), in their study of 1,026 older adults, demonstrated that pure-tone average (PTA) accounted for 30% of the variability in

performance on a test of central auditory function and that it was the PTA, rather than aging alone, that correlated most significantly with central auditory performance. Similarly, Divenyi and Haupt (1997) emphasized that peripheral hearing loss was a primary factor affecting auditory performance in both elderly and young listeners. As a result, they cautioned that even very mild pure-tone threshold elevations may influence performance on any auditory test. Niejenhus, Tschur, and Snik (2004) also found that mild hearing loss can impact even those central auditory measures (e.g., Dichotic Digits) previously thought to be resistant to peripheral hearing impairment, underscoring the need to consider carefully the relative influence of peripheral factors on "central" auditory function. Jerger and Chmiel (1997) reported that both low frequency and high frequency hearing sensitivity were significant factors contributing to overall auditory impairment in elderly individuals.

Signal audibility may be corrected, at least to some degree, with the use of appropriate amplification. However, as is noted by many researchers, this does not necessarily improve speech perception performance. Instead, factors beyond mere audibility contribute to the difficulties encountered by aging listeners with peripheral hearing loss, factors that are associated with the nature of cochlear pathology itself.

Cochlear Pathology

With the loss of outer hair cell function associated with age-related sensorineural hearing loss, there is a disruption of many of the normal functions of the cochlea. This includes the typical nonlinearity of the cochlea. In normal cochlear function, the movement of the basilar membrane is distinctly nonlinear and compressive (Kamm et al., 1978). Damage to the outer hair cells associated with sensorineural hearing loss results in loss of this normal nonlinear functionality of the basilar membrane (Otto & McCandlis, 1982). This loss of nonlinearity explains the abnormally rapid growth in loudness resulting in reduced frequency selectivity (Florentine et al., 1980; Glasberg & Moore, 1986; Grose, 1996), reduced dynamic range (Grose, 1996), and abnormal nonlinear growth of masking (Wingfield, 1996). These changes in cochlear function, in turn, impact speech recognition.

Changes in cochlear function also can distort the temporal and spectral cues necessary to understand speech, especially in difficult listening situations. Several studies have investigated patterns of temporal resolution in persons with hearing loss (e.g., Fitzgibbons & Gordon-Salant, 1987; Fogerty, Kewley-Port, & Humes, 2012; Maddens & Feth, 1992; Tyler, Summerfield, Wood, & Fernandes, 1982). Listeners with cochlear hearing loss perform more poorly than normally hearing listeners at tasks that are thought to depend heavily on information, such as low-rate frequency modulation (Lacher-Fougere & Demany, 1998) and interaural phase difference discrimination (Lacher-Fougere & Demany, 2005).

Given the multiplicative effects of peripheral hearing loss on speech recognition, it may be impossible to parse out the relative effects of audibility versus other effects of cochlear pathology on auditory perception in older listeners. Even with correction of audibility through the use of amplification, the effects of outer hair cell loss on frequency and related cues important for speech perception remain. In addition, sensory hearing loss leads to alterations in neural circuitry *upstream*, that is, central effects

of peripheral pathology. As such, peripheral pathology can have an impact well beyond the cochlea.

Central Effects of Peripheral Pathology

There is substantial evidence to support the existence of changes in the cochlear nucleus through the auditory portions of the cortex that occur secondary to the presence of a peripheral hearing loss. Studies of the effects of peripheral auditory dysfunction and biological aging in the animal model have demonstrated that the presence of high frequency hearing loss not only results in decreased audibility of certain spectral components of the acoustic signal, but also leads to a disruption in the tonotopic organization of the CANS (Willott, 1986; Willott & Lu, 1982; Willott et al., 1988a, 1988b, 1988c). For example, it has been found that neurons at the level of the inferior colliculus that may have responded previously to high frequency acoustic signals exhibit lower characteristic frequencies (CFs) following the advent of cochlear hearing loss, resulting in a disproportionately greater representation of low frequency information in the CANS. It may be predicted that this would lead to degraded encoding of high frequency spectral components of the speech signal.

Furthermore, because low frequency auditory channels exhibit inherently poorer temporal resolving abilities (Phillips, Rappaport, & Gulliver, 1994; Stuart & Phillips, 1996; Stuart, Phillips, & Green, 1995), it is likely that such tonotopic reorganization secondary to peripheral auditory dysfunction would result in degradation of temporal encoding of the acoustic signal. These factors, combined with reduced audibility of certain spectral components of the acoustic signal, may be predicted to lead to significant disruptions in auditory processes that rely on precise temporal and frequency encoding, including speech perception. As such, the neural systems underlying speech perception can be compromised by the presence of peripheral hearing loss (e.g., Peelle, Troiani, Grossman, & Wingfield, 2011). Additional brainstem and thalamic central auditory structures also have been shown to demonstrate transsynaptic degeneration as a direct result of cochlear pathology (e.g., Feng, Bendiske, & Morest, 2012; Morest & Bohne, 1983). A comprehensive review of the effects of peripheral pathology on CANS structure and function is outside the scope of this chapter. The reader is referred to Gordon-Salant, Frisina, Popper, and Fay (2010) and Ison, Tremblay, and Allen (2010) for more information.

In summary, it is clear from the studies reviewed that the integrity of the auditory periphery is a key factor contributing to listening and related auditory function in aging adults, particularly given the high prevalence of peripheral hearing loss in this population. Furthermore, it is apparent that peripheral pathology can lead to secondary alterations in structure and function of the CANS that will further impact auditory perceptual abilities. However, it is equally clear that peripheral hearing loss alone cannot account for all of the listening difficulties exhibited by older listeners.

Cognitive Factors

Age-related changes in cognitive functions such as working memory and information processing speed have been hypothesized to be responsible for some of the speech perception difficulties of

older adults (e.g., Birren & Fisher, 1995; CHABA, 1988; Humes, Lee, & Coughlin, 2006; see Humes et al., 2012 for a review). Relationships between cognitive function and performance on central auditory tests have been reported in the literature (e.g., Gates et al., 1996, 2008; Golding et al., 2005, Gordon-Salant & Fitzgibbons, 1997; Strouse et al., 1995). Hallgren et al. (2001) found that dichotic listening performance correlated with measures of general cognitive ability, including working memory and processing speed, particularly when the listener is forced to attend to and report stimuli presented to the left ear. They attributed these findings to a decrease in working memory in older adults and argued that such findings support the cognitive explanation of auditory deficits in aging. Others also have found a significant relationship between cognitive performance and directed report of stimuli to the left ear during dichotic listening performance (e.g., Cowell & Hugdahl, 2000), and suggest that the forced-left report condition may reflect additional attentional and cognitive mechanisms rather than true perceptual asymmetries. These findings hold implications for the use of directed-report versus free-report conditions during dichotic listening testing, as discussed in a subsequent section of this chapter.

In these and similar studies, it might be assumed that cognitive decline affects older adults' performance on central auditory tests. However, an alternative explanation is equally, perhaps even more, likely. There is a great deal of evidence to suggest that the presence of perceptual deficits results in the need for greater resources to be allocated to auditory processing, thus leaving fewer resources available "upstream" for central cognitive processes required for storage and retrieval of auditory information (e.g., Pichora-Fuller, Schneider, & Daneman, 1995; McCoy, Tun, Cox, Colangelo, Stewart, & Wingfield, 2005). In this resource allocation model, apparent "cognitive" changes are actually a consequence of a decline in auditory abilities and are exacerbated by both perceptual and cognitive stressors (McCoy et al., 2005; Murphy, Craik, Li, & Schneider, 2000; Pichora-Fuller, 2003; Pichora-Fuller et al., 1995; Rabbitt, 1991; Schneider, Daneman, & Pichora-Fuller, 2002; Schneider, Daneman, & Murphy, 2005). Recent support for this hypothesis has been provided by Jorgensen (2012), who found that the presence of even mild, previously undiagnosed peripheral hearing loss resulted in a misdiagnosis of mild dementia.

That the central auditory and speech recognition difficulties of aging adults cannot be explained entirely or even primarily by cognitive decline is bolstered further by the finding that degree of cognitive deficit, unlike presence of central auditory dysfunction, does not correlate with perceived self-assessment of hearing handicap in older listeners (Jerger et al., 1990), and that measures of working memory and sequence-learning ability do not demonstrate a significant association with various measures of speech recognition in elderly listeners (e.g., Humes & Floyd, 2005). Indeed, Strouse et al. (1995) cautioned that the presence of auditory processing deficits may lead to poorer performance on cognitive tests and, thus, may influence results of psychiatric assessment in older adults. Finally, although it has been demonstrated that older adults exhibit poorer performance than younger listeners on speech recognition tasks in the presence of meaningful competition, which has been taken

as evidence that elderly listeners exhibit reduced attentional control (e.g., Tun, O'Kane, & Wingfield, 2002), neurophysiologic studies, discussed in the next section, suggest that it appears to be the processing of the signal in competition itself, rather than the ability to selectively attend, that is impaired in older adults (DeChicchis et al., 2002; Fisher et al., 2000; Hymel et al., 1998).

In contrast, research indicates that the linguistic experience of older adults actually facilitates their ability to compensate for deficits in sensory and perceptual processing. This is evidenced by studies demonstrating that older adults are better than younger listeners at using context to recognize words (Cohen & Faulkner, 1983; Jerger & Martin, 2005), and are better able to rely on previous linguistic knowledge and short-term conceptual memory when recognizing accelerated speech (e.g., Gordon-Salant & Fitzgibbons, 2001; Wingfield, Tun, Koh, & Rosen, 1999). Furthermore, the ability to make use of prosodic information, including clausal structure, to facilitate spoken word recognition is preserved in normal aging (Fallon, Kuchinsky, & Wingfield, 2004; Wingfield, Lindfield, & Goodglass, 2000). In short, due to well-developed top-down abilities, older listeners appear better able to compensate for reduced redundancy in speech signals than younger listeners (e.g., Pichora-Fuller, 2008; Pichora-Fuller et al., 1995).

[redacted]entral Auditory Factors

Notwithstanding the importance of both the auditory periphery and cognition to listening and speech recognition in aging, there is compelling evidence that neither of these factors can account for all of the auditory performance decrements seen in older listeners. Instead, even when peripheral hearing loss and cognitive status are taken into account and/or corrected for, or when participants are free of peripheral auditory or cognitive dysfunction, older listeners tend to perform more poorly on a variety of auditory measures. Moreover, these performance decrements are accompanied by age-related changes in the neurophysiologic representation of acoustic stimuli that cannot be attributed to changes in the auditory periphery or to cognitive factors alone.

Age-Related Changes in Auditory Neurophysiology

The majority of studies that investigate the underlying neurophysiologic changes associated with aging have used small samples of human brain tissue. In 1955, Brody investigated postmortem human samples of the cerebral cortex and concluded that there was a decrease in neurons with age. He particularly noted changes in the superior temporal gyri, precentral gyri, and area striata, areas that are specifically important for speech processing. Koningmark and Murphy (1970, 1974) reported a significant decrease in volume of the ventral cochlear nucleus beyond the fifth decade of life. This decrease in volume was due to decreased number of glial cells, loss of axis cylinders, loss of neuronal processes including dendrites, decrease in the size or number of blood vessels, and decrease in the extracellular space. Furthermore, when comparing middle-aged adults with older adults, the neurons were robust and well myelinated in middle-aged individuals;

however, there was a significant decrease in the number and myelination of these fibers in the older adults. This decrease in volume within the ventral cochlear nucleus could reasonably be hypothesized to decrease the efficiency or accuracy of the transmission of auditory signals within the central auditory system, resulting in changes in the ability of elderly adults to process auditory signals such as speech.

Other neuroanatomical changes in the aging CNS have been reported, including age-related changes in the size and degree of myelination of the corpus callosum (e.g., Hanyu et al., 1997; Janowsky, Kaye, & Carper, 1997; Silver, Barker, McManus, Tofts, & Miller, 1997). These anatomical changes in corpus callosum are of particular note as such changes are unlikely to be the result of peripheral hearing loss and, yet, may have an impact on central auditory—particularly interhemispheric—processing.

Age-Related Changes in Neurophysiologic Representation of Acoustic Signals

Animal studies of the effects of biological aging in the absence of peripheral auditory dysfunction have suggested that the effects of aging, per se, on the neural encoding of acoustic input may be relatively subtle. However, studies have demonstrated that an increase in spontaneous neural activity, possibly resulting from a decrease in inhibitory gamma-aminobutyric acid (GABA), accompanies advancing age and may be indicative of increased "neural noise" in the aging CANS (Caspary, Raza, Lawhorn Armour, Pippin, & Arneric, 1990). It has been postulated that this increase in neural noise may underlie the speech perceptual difficulties of elderly listeners in competing acoustic backgrounds (Gregory, 1975; Novak & Anderson, 1982; Salthouse & Lichty, 1985). Furthermore, it has been shown that the number of neurons that exhibit precise temporal encoding is reduced in the inferior colliculus of aging mice, accompanied by a higher proportion of neurons that display sluggish temporal response properties (Willott et al., 1988a).

Human studies of the effects of aging on neurophysiologic encoding of auditory stimuli have revealed differences between young and elderly subjects. For example, studies have shown that the absolute latencies of auditory brainstem response (ABR) waves demonstrate a slight increase and that wave amplitudes diminish with advancing age (Harkins, 1981; Jerger & Hall, 1980; Patterson, Michalewski, Thompson, Bowman, & Litzelman, 1981; Rosenhamer, Lindstron, & Lundborg, 1980; Soucek, & Mason, 1990). Likewise, an increase in latency of peak components of the middle latency response (MLR) (Lenzi, Chiarelli, & Sambataro, 1989; Ryan, 1989; Woods & Clayworth, 1986), as well as in late components of the auditory evoked response (Goodin, Squires, Henderson, & Starr, 1978; Pfefferbaum, Ford, Roth, & Kopell, 1980; Pfefferbaum, Ford, Roth, Hopkins, & Kopell, 1979; Picton, Stuss, Champagne, & Nelson, 1984), has been documented in elderly subjects. However, the underlying cause(s) of these changes in neurophysiologic measures are difficult to interpret, as such changes may be due to peripheral (e.g., decrease in the short-latency response from the basal

end of the cochlea secondary to high frequency hearing loss) or central (e.g., decreased neural synchrony/increased neural noise) factors. Finally, the finding that peak Pa of the MLR demonstrates an increase in amplitude with advancing age (e.g., Chambers & Griffiths, 1991; Jerger, Oliver, & Chmiel, 1988; Woods & Clayworth, 1986) suggests a possible impairment in inhibition in the aging organism, which is consistent with the findings of decreased GABA levels in animals discussed previously. Bellis et al. (2000) also postulated that a decrease in inhibition of right-hemisphere neural activity may underlie the lack of typical hemispheric asymmetry in the neural representation of speech sounds in older, normally hearing women.

More recent studies of the neurophysiologic representation of acoustic signals in aging populations have suggested that age effects may be more pronounced when complex speech signals and/or challenging tasks paradigms are employed than when simple, nonverbal acoustic stimuli are utilized to elicit neural responses (e.g., Fisher, Hymel, Cranford, & DeChicchis, 2000; Jerger & Lew, 2004; Tremblay, Billings, & Rohila, 2004). Tremblay, Piskosz, and Souza (2002, 2003) reported that the cortical event-related potentials elicited by speech signals differing in voice-onset time (VOT) were different in older listeners when compared with younger controls. These neurophysiologic differences were accompanied by concomitant difficulty in psychophysical discrimination of the same VOT contrasts in the older adults, with performance decrements more pronounced in older adults who also exhibited peripheral hearing loss. The authors concluded that at least some of the speech perceptual difficulties exhibited by aging adults may be due to age-related changes in excitatory and inhibitory neural factors affecting neural synchrony, leading to poorer temporal precision in the central auditory pathways of older listeners. The fact that these difficulties were compounded in listeners with peripheral hearing loss may hold implications for hearing aid success in older adults with comorbid central and peripheral auditory dysfunction. In a subsequent study, Tremblay et al. (2004) demonstrated that the abnormalities seen in the neural responses of aging adults were not attributable merely to presentation rate of the stimuli, alone, but were due to signal complexity, as well. That is, both the speed of presentation/interstimulus intervals of consecutive stimulus onsets as well as stimulus complexity (i.e., speech versus nonspeech signals) affected various components of the cortical responses in older listeners, providing additional evidence for age-related temporal processing deficits, especially for complex signals such as speech.

Bertoli, Smurzynski, and Probst (2005) also found that the presence of peripheral hearing loss affected frequency discrimination abilities in older listeners, especially in backgrounds of competing noise, and that the psychophysical findings were accompanied by age-related differences in cortical auditory event-related potentials. Although they found that their normally hearing older adults exhibited preserved discrimination abilities for simple frequency contrasts, the age-related differences in neural representation of the stimuli suggested that the aging adults used different processing strategies than did the younger controls and that behavioral maintenance of

discrimination abilities in older listeners was more effortful even in the absence of peripheral hearing loss.

Other studies have focused on age-related changes in interhemispheric asymmetries in the neural representation of auditory signals. These studies, in combination, have demonstrated that the pattern of hemispheric asymmetry in the neural representation of auditory signals is different in older adults when compared with younger listeners. In a study of whole-head auditory evoked magnetic fields in young versus elderly subjects, Pekkonen and colleagues (1995) found that the interhemispheric latency difference of the N100m component to simple tone pips was significantly increased in older adults, suggesting that age affects signal processing in the ipsilateral auditory cortex. Bellis et al. (2000) demonstrated that the obligatory P1–N1 complex elicited by synthetic speech syllables did not exhibit the expected left-hemisphere dominance in older, normally hearing women and that this decrease in typical interhemispheric asymmetry was accompanied by deficits in the behavioral discrimination of the same speech syllables. These findings provided evidence for a biological, age-related change in the basic sensory representation of elemental speech stimuli that cannot be attributed to either peripheral hearing loss or to cognitive decline.

Jerger and colleagues (e.g., Greenwald & Jerger, 2001; Jerger, Alford, Lew, Rivera, & Chmiel, 1995; Jerger, Moncrieff, Greenwald, Wamtacq, & Seipel, 2000), in a series of studies combining behavioral and electrophysiologic measures of dichotic listening, demonstrated that aging adults exhibit increased interaural asymmetry (i.e., left-ear deficit) for dichotic speech stimuli and that this pattern is reflected in the electrophysiologic representation of the same stimuli. Moreover, a reversed pattern is seen when nonverbal signals are used. The patterns observed both behaviorally and electrophysiologically were strikingly similar to those seen in individuals with corpus callosum dysfunction, leading to a hypothesis of decreased interhemispheric transfer of auditory information in aging adults. Further supporting this hypothesis is the finding of decreased interhemispheric coherence of electroencephalographic (EEG) activity with advanced age (Duffy, Mcanulty, & Albert, 1996).

Finally, in an attempt to disentangle possible age-related difficulties in processing speech in competing backgrounds from higher level selective attention deficits, Hymel and colleagues (DeChicchis, Carpenter, Cranford, & Hymel, 2002; Fisher et al., 2000; Hymel, Cranford, & Stuart, 1998) demonstrated that aging affects the electrophysiologic correlate of stimulus competition (e.g., electrophysiologic representation of signals presented in a background of noise). In contrast, the biologic mechanisms underlying the ability to attend selectively to target stimuli appear to be more resistant to the aging process, as evidenced by preserved higher level selective-attention abilities. These findings provide further evidence of a fundamental signal-in-competition deficit in older listeners that cannot be attributed to higher level attention or other cognitive factors.

When taken together, studies of the neurophysiologic representation of auditory signals in older adults demonstrate decreased temporal precision and neural synchrony, atypical interhemispheric asymmetry, poorer interhemispheric communication and transfer of auditory information, and changes in the neural

representation of stimulus competition with aging. These findings are most pronounced for speech signals, especially those involving rapid spectrotemporal acoustic changes. Although these effects are compounded by the presence of peripheral hearing loss, they cannot be accounted for by peripheral factors or by higher level attention or cognitive factors. Therefore, these studies provide compelling evidence of age-related changes in the central auditory mechanisms underlying binaural listening and speech perception, especially in backgrounds of competition.

On a final note, the auditory P300 event-related potential has been demonstrated to be instrumental in evaluating cognitive effects of aging and in the clinical assessment of dementia (e.g., Katado, Sato, Ojika, & Ueda, 2004; Knott et al., 2003). Thus, the combined use of brainstem, cortical, and cognitive auditory electrophysiologic responses may provide useful information regarding sensory encoding and cognitive processing of auditory stimuli in aging adults (see Olichney & Hillert, 2004, for a review).

Age-Related Changes in Psychophysical Measures of Central Auditory Function

Several studies have examined the effects of aging on a variety of psychophysical measures of fundamental auditory skills. Most models of speech perception maintain similar underlying mechanisms that rely on a central system for auditory and cognitive processing of speech (Fitzgibbons & Gordon-Salant, 1996; Ohenham & Plack, 1997; Plack et al., 2004). The effects of age on frequency resolution have been documented to decline with each decade of life (Glasberg, Moore, Patterson, & Nimmo-Smith, 1984; Glasberg & Moore, 1986; Grose, 1996; Lutman, Gatehouse, & Worthington, 1991; Matschke, 1991; Patterson, Nimmo-Smith, Weber, & Milroy, 1982). Temporal processing abilities in elderly listeners are decreased compared with younger listeners on tasks involvinging frequency and duration discrimination (Abel, Krever, & Alberti, 1990; Fitzgibbons & Gordon-Salant, 1994, 1998; Konig, 1957; Maddens & Feth, 1992; Marshall, 1981; Meurman, 1954; Phillips, Gordon-Salant, Fitzgibbons, & Yeni-Komishian, 1994; Tyler, Summerfield, Wood, & Fernandes, 1982), gap detection (Harris, Eckert, Ahlstrom, & Dubno, 2010; Ludlow, Cudahy, & Bassich, 1982; Lutman, 1991; Moore, Peters, & Glasberg, 1992; Schneider, Speranza, & Pichora-Fuller, 1998; Snell & Frisina, 2000; Strouse, Ashmead, Ohde, & Grantham, 1998; Walton, Frisina, & O'Neill, 1998; Zendell, 2011), precedence effect (Cranford, Boose, & Moore, 1990), segregation of simultaneously occurring vowel sounds (Snyder & Alain, 2005; Vongpaisal & Pichora-Fuller, 2007), and backward masking (Cobb, Jacobson, Newman, Kretschmer, & Donnelly, 1993). It should be noted that the effects of aging on gap detection, in particular, may be complex, with evidence suggesting that it is influenced by condition (e.g., across-channel versus within-channel gap detection; Roberts & Lister, 2004), distance between the gap and the onset or offset of the signal (He, Horwitz, Dubno, & Mills, 1999), marker duration (Schneider & Hamstra, 1999), and other factors, including the presence of competing noise (Snell, 1997). Furthermore, age effects on gap detection performance may not be apparent with some gap detection paradigms when attention is directed toward the task, despite being

evident during electrophysiologic measures of pre-attentive temporal processing (e.g., Bertoli, Smurzynski, & Probst, 2002). In addition, animal studies suggest that stimulus level also may play a role in aging effects on gap detection performance (Allen, Burkard, Ison, & Walton, 2003).

A plethora of studies have investigated the ability of elderly subjects to process speech stimuli, both in quiet and under conditions of background noise and/or various forms of signal distortion. Results of these investigations have suggested that older individuals have more difficulty than younger listeners in noisy or reverberant conditions (e.g., Dubno, Dirks, & Morgan, 1984; Dubno, Lee, Mathews & Mills, 1997; Harris, & Reitz, 1985; Nabalek, 1988; Nabalek, & Robinson, 1982; Wiley et al., 1998), and with temporally distorted speech (Gordon-Salant & Fitzgibbons, 1993a,b, 2001; Rastatter, Watson, & Strauss-Simmons, 1989). In the absence of significant hearing loss, the speech perceptual abilities of elderly listeners do not appear to be greatly affected in quiet listening conditions (Dubno et al., 1984; Holmes, Kricos, & Kessler, 1988; Surr, 1977; Townsend & Bess, 1980). However, when the listening condition is made difficult either through the addition of competing noise or via introduction of some form of distortion in the speech signal, even normally hearing elderly listeners exhibit reduced speech perceptual abilities as compared with younger listeners (e.g., Helfer & Wilber, 1990; Rodriguez et al., 1990).

Many investigations of speech perception and aging have used either presbycusic listeners or listeners with "normal hearing for age/minimal hearing loss." Because a decline in peripheral hearing sensitivity typically accompanies aging, it is difficult to determine what proportion of the difficulties exhibited by elderly subjects in these studies may be due to central effects of biological aging alone versus central effects of subtle peripheral dysfunction. Even minimal peripheral auditory dysfunction may degrade the neural input to the CANS, resulting in frequency, intensity, and temporal distortions of the acoustic signal; therefore, one cannot underestimate the possible detrimental effect of even very subtle peripheral dysfunction on those "central" processes presumed to be important for speech perception (Humes et al., 2012; Willott, 1991). In fact, findings by Wilson and Weakley (2005) suggested that word recognition performance in a background of multitalker babble was influenced more by the degree of peripheral hearing loss than by the age of the subjects per se. Nonetheless, and as discussed previously in this chapter, other authors have demonstrated that the speech perceptual difficulties exhibited by aging listeners are independent of and/or cannot be accounted for entirely by peripheral hearing loss (e.g., Golding et al., 2005; Gordon-Salant & Fitzgibbons, 1993a, 1993b; Jerger & Chmiel, 1997; Wiley et al., 1998).

One way to separate the effects of peripheral hearing loss from central auditory deficits is to examine ear differences in performance on central auditory tests (e.g., Bellis, Billiet, & Ross, 2011). That is, the presence of a significant ear effect despite symmetrical hearing sensitivity or, alternatively, the presence of an ear deficit in the ear with *better* peripheral hearing sensitivity in cases of asymmetrical hearing loss, can be taken as evidence in favor of a central auditory deficit rather than of a peripheral explanation. Studies of dichotic listening performance in elderly listeners have been particularly useful in this regard. The presence of

a significant left-ear deficit (or greater than normal right ear advantage, REA) on dichotic speech tests in aging adults has been reported in numerous studies (e.g., Bellis & Wilber, 2001; Chmiel & Jerger, 1996; Cowell & Hugdahl, 2000; Jerger & Chmiel, 1997; Jerger et al., 1994, 1995; Hallgren et al., 2001).

Bellis and Wilber (2001) demonstrated that the effects of aging on auditory and visual-motor indices of interhemispheric function, including dichotic listening and temporal patterning as well as visual-motor interhemispheric transfer time (IHTT), differed depending upon gender and point-of-time in the adult life span. In their study of 120 adults with 15 females and 15 males in each of four discrete age groups from 20 to 75 years of age, they demonstrated that males exhibited maximum interhemispheric auditory function in the early adult years, followed by a gradual and linear decline in interhemispheric abilities with increased age. Women, on the other hand, demonstrated preserved function throughout the child-rearing years up to approximately age 55, at which point a rapid decline in function occurred. No gender differences were observed in the oldest or youngest age groups. These findings mirrored previously reported anatomical changes in corpus callosum structure as a function of gender and point-of-time in the life span (Cowell, Allen, Zaltimo, & Dennenberg, 1992). This complex interaction between gender and point-of-time in the life span may explain, at least in part, previous contradictory findings regarding gender, aging, and dichotic listening performance. An additional, rather serendipitous, finding of Bellis and Wilber's (2001) study was that women tended also to exhibit a deficit in right-hemisphere-based central auditory function presumed to underlie perception of rhythm, stress, and intonational prosodic cues of speech during the immediate postmenopausal years that subsequently improved in later years. It was hypothesized that this latter finding may provide insight into a biological mechanism underlying, at least in part, the oft-reported difficulty in interpreting tone-of-voice cues in postmenopausal women.

In summary, studies of aging and central auditory function indicate that older adults exhibit difficulties in a variety of fundamental auditory processes as well as in speech recognition in backgrounds of competition and binaural listening abilities. Although peripheral auditory dysfunction compounds these difficulties, it cannot account for all of the auditory effects observed.

In conclusion, a review of the literature on aging and speech perception suggests that many of the speech perceptual difficulties experienced by the elderly may be attributed to peripheral auditory dysfunction and/or cognitive function. As a result, Humes et al. (2012) concluded that there is little "evidence for central presbycusis as an independent entity in the absence of hearing loss, cognitive, deficits, or both" (p. 663). However, it is crucial to note that the authors did not include in their comprehensive review the findings of Bellis and Wilber (2000), which demonstrated behavioral central auditory effects of aging that could not be accounted for by peripheral hearing loss or cognitive decline. Nor did they include in their review the electrophysiologic studies discussed herein that also support the presence of central effects of biological aging.

As such, age-related changes in neural synchrony and neural signal-to-noise ratios, along with declines in temporal

processing and inefficient interhemispheric transfer of auditory information, also may give rise to degradation in the neural encoding of acoustic stimuli, leading to speech perceptual difficulties in the absence of peripheral auditory or cognitive dysfunction, especially in backgrounds of competing noise. These central effects of biological aging may differ by gender and by point-of-time in the life span. Nonetheless, it does seem clear that the combination of peripheral auditory dysfunction, possible cognitive decline, and subtle age-related changes in auditory encoding result in a multiplicative effect, leading to significant speech perceptual difficulties in presbycusic listeners and/or listeners with cognitive dysfunction that cannot be predicted from the effects of biological aging or peripheral auditory/cognitive dysfunction alone (Humes et al., 2012).

Finally, the presence of peripheral and/or central auditory deficits may result in decreased resources available for storage and retrieval of auditory information. However, the ability to use linguistic, contextual, and prosodic knowledge to facilitate spoken language recognition may assist in offsetting the negative impact of peripheral and central auditory decline in older listeners.

Implications of Research for Auditory Assessment and Intervention in Older Adults

Several clinical implications can be drawn from the research regarding speech recognition, auditory processing, and aging. First, because the presence of peripheral auditory dysfunction has a deleterious effect on speech recognition and related abilities, particularly in backgrounds of noise, it is clear that a complete evaluation of peripheral auditory function is indicated in any case of reported listening or related difficulty, including physiologic measures to rule out subtle cochlear dysfunction that may not be apparent from the pure-tone audiogram. Furthermore, even when results of peripheral auditory evaluation suggest normal hearing, one should not assume that the entire auditory system is, therefore, normal. Evaluation of peripheral auditory function is merely the first step in the assessment process. Finally, clinicians should be cautious in interpreting traditional tests of speech recognition in quiet as it has been demonstrated clearly, first, that the speech perceptual difficulties of older adults often are apparent only in conditions of competition and/or signal distortion and, second, that auditory processing abilities cannot be predicted from traditional word-recognition-in-quiet scores. Therefore, more thorough evaluation of auditory function is indicated, particularly for those cases in which reported listening and related difficulties are greater than would be expected on the basis of the audiogram alone.

Second, based on findings related to prevalence of CAPD in the aging population, beginning as early as the middle-age years, as well as the potential impact of central auditory dysfunction on self-perceived hearing handicap, benefit from amplification, and other factors, it may not be unreasonable to suggest that some type of central auditory assessment or screening be included as part of every audiological evaluation involving older adults whenever possible (e.g., Bellis,

2003b). The role of the audiologist is to evaluate hearing, and the act of hearing involves mechanisms beyond just the peripheral auditory system. Given the significant adverse effects of central auditory dysfunction on listening and communication function, it behooves clinicians to obtain as much information as possible regarding auditory function so that intervention efforts can be directed appropriately. Issues related to diagnosing CAPD and principles of intervention are discussed later in this chapter. Further, because both gender and point-of-time in the life span may affect central auditory function differentially, intervention likely should include cross-gender counseling of spouses, family members, and other communicative partners.

Finally, there is evidence to suggest that evaluation of peripheral and central auditory function may be important in cases of suspected dementia or other cognitive disorder in older adults (e.g., Gates et al., 1996, 2002, 2008, 2011; Jorgensen, 2012; Strouse et al., 1995). Because the presence of sensory or perceptual deficit can result in "upstream" effects on memory and related cognitive abilities due to insufficient processing resources (McCoy, Tun, Cox, Colangelo, Stewart, & Wingfield, 2005; Pichora-Fuller, Schneider, & Daneman, 1995), it is critical that audiologists work as team members with psychiatrists, speech-language pathologists, and other professionals in the evaluation of older adults in an effort to disentangle the relative effects of peripheral and central auditory dysfunction from higher level cognitive, language, and other deficits. It should also be recognized, however, that the presence of peripheral hearing loss may, conversely, lead to an unjustified diagnosis of cognitive impairment (Jorgensen, 2012). Thus, as with children, a multidisciplinary team approach is critical to the accurate and effective diagnosis of and intervention for CAPD in the older population.

Differential Diagnosis of CAPD in Older Listeners

Because many older adults exhibit peripheral hearing loss, and because even mild peripheral auditory dysfunction may affect performance on many tests of central auditory function (Divenvi & Haupt, 1997; Humes, Coughlin, & Talley, 1996; Musiek, Baran, & Pinheiro, 1990; Musiek, Gollegly, Kibbe, & Vekest-Lenz, 1991; Neijenhius, Tschur, & Snik, 2004), behavioral central auditory assessment in this population must be undertaken with caution. Nonetheless, it is possible to separate the relative effects of peripheral and central auditory dysfunction in many cases. However, clinicians must be familiar with the research underlying the available test procedures and select those that have been shown to be relatively resistant to peripheral hearing impairment. Furthermore, clinicians should be cautious in interpreting any test of central auditory function when peripheral hearing loss is present, even if previous research indicates that the test is relatively unaffected by peripheral disorder (Niejenhus et al., 2004). (See Chapter 11.)

Based on research discussed previously in this chapter, dichotic speech tests may provide the most useful information regarding central auditory function in older listeners. However, not all dichotic speech tests are equal in terms of resistance to peripheral hearing loss. As a

general rule, those dichotic speech tests that also require fine-grained discrimination of minimal-pair speech-sound contrasts, such as dichotic consonant-vowel (CV) tests, also are most likely to be influenced adversely by peripheral hearing loss (e.g., Speaks, Niccum, & Van Tassell, 1985). In contrast, dichotic speech stimuli that carry a light linguistic load and that do not require fine-grained auditory discrimination, such as the Dichotic Digits Test (Musiek, 1983) appear to be most resistant to cochlear dysfunction (Musiek, Gollegly, Kibbe, & Verkest-Lenz, 1991). An additional advantage of the Dichotic Digits Test is its ease and rapidity of administration (Musiek, 1983; Musiek et al., 1991), rendering it particularly appropriate as a screening tool for inclusion in standard audiologic test batteries (Bellis, 2003b). Moreover, the Dichotic Digits Test has been shown to have good test-retest reliability even in elderly listeners with Alzheimer's disease, as well as in elderly listeners with no evidence of dementia (Strouse & Hall, 1995). Other dichotic speech tests that have been demonstrated to be relatively resistant to peripheral hearing loss include the Dichotic Sentence Identification (DSI) Test (Fifer, Jerger, Berlin, Tobey, & Campbell, 1983) and the Staggered Spondaic Word Test (SSW; Arnst, 1982; Katz, 1962).

When administering any dichotic speech test, clinicians first should ensure that the test does not exceed the patient's working memory capacity. Thus, for example, if a test requires a patient to repeat four digits, as is the case with the Dichotic Digits Test, clinicians first should ensure that the patient's digit span well exceeds the threshold level of four in a noncompeting condition prior to presenting the stimuli in the dichotic, competing condition. This provides at least one within-patient control for possible confounding effects of cognition and memory on central auditory test performance and assists in ensuring that dichotic speech deficits are due more likely to the introduction of the stimulus competition rather than to generalized memory problems.

Additional consideration should be given to the report condition required by tests of dichotic listening. When testing dichotically, two report conditions are possible. In the free report (FR) condition, listeners are instructed to repeat stimuli directed to both ears, usually in any order. This assesses the process of binaural integration (e.g., Bellis, 2003a; Bellis & Ferre, 1999; Chermak & Musiek, 1997). In the directed report (DR) condition, listeners typically are instructed to attend to and report the stimuli delivered to the target ear only, thus assessing the process of binaural separation (Bellis, 2003a; Bellis & Ferre, 1999; Chermak & Musiek, 1997). Although it may appear at the outset that the FR condition is more susceptible to cognitive and/or memory confounds as it requires the listener to report a greater number of stimuli, previous research indicates that it is the DR condition that correlates most closely with cognitive function (e.g., Cowell & Hugdahl, 2000; Hallgren et al., 2001; see Hugdahl, Westerhausen, Alho, Medvedev, Laine, & Hamalainen, 2009 for a review), as this condition requires the listener to recruit additional cognitive and attentional mechanisms. These studies, therefore, concluded that performance decrements in the FR condition are more likely to be a consequence of true auditory perceptual asymmetries than are performance decrements in the DR condition. Therefore, clinicians should be very careful in assigning central auditory versus cognitive explanations to differences in per-

formance on dichotic speech tests based upon report condition. Instead, because the two report conditions tap into different underlying auditory processes, the use of both FR and DR conditions may, when administered and interpreted appropriately, provide useful insight into the specific auditory processes that are impacted by a given central auditory disorder. This information then may be used in designing a deficit-specific intervention plan to address the presenting auditory deficit profile. (See Chapters 6 and 14 for reviews of research and clinical application of dichotic listening, respectively.)

An additional control that assists in disentangling the effects of peripheral and/or cognitive confounds from true central auditory dysfunction is the use of within-test comparisons of ear performance (AAA, 2010; Bellis et al., 2011). Thus, and as previously discussed, the presence of a significant ear-specific deficit on dichotic speech tasks in the presence of normal or symmetrical hearing sensitivity or, alternatively, the presence of a deficit in the ear with the better hearing thresholds in the case of asymmetrical hearing sensitivity, may be taken as evidence of central auditory involvement.

Other behavioral tests of central auditory function also have been suggested to be somewhat resistant to peripheral hearing loss as long as the signals are audible to the listener, including temporal patterning tests such as Frequency Patterns and Duration Patterns (Musiek, Baran, & Pinheiro, 1990; Musiek & Pinheiro, 1987). Again, ensuring that listeners can perform the task using live-voice modeling and employing within-test comparisons of contrasting report conditions (i.e., linguistic labeling of the stimuli versus nonverbal or humming report) can assist in controlling for potential cognitive and/or memory confounds and ensure that performance decrements observed are due to central auditory disorder.

A final control that assists in differentiating true central auditory dysfunction from more generalized cognitive, motivational, hearing, or other confounds is the use of intertest and multidisciplinary test analysis. Specifically, measures are examined for *patterns* of performance across tests that conform to well-established neuroscience tenets drawn from cases of known central auditory dysfunction. For example, the finding of left-ear deficits on dichotic speech tasks combined with a deficit on temporal patterning tests in the linguistic labeling condition only has been demonstrated in cases of known interhemispheric dysfunction involving the corpus callosum (e.g., Baran, Musiek, & Reeves, 1986; Musiek, Kibbe, & Baran, 1984). The emergence of this same pattern has been observed in aging adults and correlates with anatomical changes in corpus callosum structure with aging (Bellis & Wilber, 2001; Cowell et al., 1992). Additional decrements in visual-motor interhemispheric transfer time, bimanual and/or bipedal difficulties, and other multidisciplinary findings indicative of inefficient interhemispheric function also may be seen on multidisciplinary tests (Bellis & Wilber, 2001).

In contrast, absence of a clear pattern of performance across tests, inconsistency in test performance, poor performance on all measures in all report conditions, or contradictory test findings (e.g., a left-ear deficit on one dichotic speech task accompanied by a right-ear deficit on another) argues for a nonauditory explanation and may be due to a host of factors, including generalized cognitive difficulty, motivational issues, lack of understanding of the test procedures, or

other confounds. In these cases, diagnosis of a CAPD would not be supported.

Although several other behavioral tests of central auditory function are available, the majority of them are affected adversely by cochlear pathology. Therefore, unless the listener presents with normal peripheral hearing status, these tests may not be appropriate for use in differential diagnosis of CAPD in older listeners. For a detailed discussion of effects of cochlear, brainstem, and cortical (including corpus callosum) pathology on behavioral tests of central auditory function, as well as for information regarding administration and interpretation of these tests, readers are referred to Bellis (2003a).

As has been seen earlier in this chapter, electrophysiologic measures of central auditory function also hold great promise for differential diagnosis of CAPD in older listeners, particularly when complex (e.g., speech) stimuli are used. Although many of the electrophysiologic paradigms discussed previously are not in general clinical use at this time, there is no doubt that they can be a useful addition to the clinician's differential diagnostic armamentarium (e.g., Jerger & Lew, 2004).

Finally, the use of speech-in-noise testing for CAPD diagnosis should be mentioned. Although tests of speech in noise may provide a great deal of information about how an individual functions in backgrounds of competition, they may be impacted by peripheral and central auditory disorders as well as other factors, such as attention, language, and cognition, and normative values will necessarily be different depending on the stimuli, type and signal-to-noise levels of the competing signal, degree and configuration of hearing loss, and a number of other factors. Therefore, the authors do not recommend their use for the diagnosis of CAPD per se. Nonetheless, they can assist clinicians both in determination of functional impact of auditory disorders as well as in investigating posttreatment outcomes. Several speech-in-noise tests have been developed for use with hearing-impaired populations, including the Quick-SIN (Etymotic Research, 2001) and the Hearing in Noise Test (HINT, Nillson, Soli, & Sullivan, 1994). Similarly, the use of self-assessments of hearing handicap such as the Hearing Handicap Inventory for the Elderly (HHIE; Ventry & Weinstein, 1982), Hearing Handicap Inventory for Adults (HHIA; Newman, Weinstein, Jacobson, & Hug, 1990), or the Communication Scale for Older Adults (CSOA; Kaplan, Bally, Brandt, Busacco, & Pray, 1997) may provide invaluable information regarding impact of the individual's auditory disorder on daily life and also may be useful in assessing posttreatment outcomes.

To obtain information regarding cognitive function, processing speed, working memory, and other factors that might impact performance on central auditory tests, clinicians also may wish to include general measures of these abilities. Some useful tools include the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975), a test of processing speed and interference such as the Stroop Color-Word Test (Uttl & Graf, 1997), and formal or informal listening span tests (e.g., Pichora-Fuller et al., 1995). Although none of these tests should be considered diagnostic for central auditory or cognitive disorder, they may provide important insight into the listener's overall levels of functioning across a variety of domains. Nonetheless, it should be emphasized that, as with any

case of suspected CAPD, a multidisciplinary approach is important to understanding fully the overall capabilities of the individual, and all efforts should be made to include physicians, psychologists, speech-language pathologists, and other professionals in the overall assessment process when indicated.

General Principles of Intervention for CAPD in Older Adults

As with any CAPD, it is important that intervention for older adults incorporate both bottom-up and top-down approaches. That is, intervention should focus both on the acoustic signal itself using environmental modifications (bottom-up) and on the deployment of higher level language, cognitive, and related strategies (top-down) to buttress deficient auditory skills. Furthermore, the precise nature of the intervention approaches will be dependent upon the individual's presenting deficit profile, lifestyle demands and communicative needs, and the presence of comorbid conditions, if applicable. As such, a multidisciplinary approach to intervention for CAPD in older adults is just as critical as it is when considering the pediatric population. Although it is not within the scope of this chapter to provide detailed information regarding intervention approaches for adults with CAPD, readers are referred to Chapter 15 among others in Volume 2 of this Handbook, as well as to Bellis (2002, 2003a,b, 2007), for a more in-depth discussion of treatment and management activities applicable to adults with CAPD.

Research indicates that older listeners are able to use their linguistic knowledge and other top-down skills to assist in mitigating the effects of auditory deficits. To this end, compensatory strategies, or central resources training is critical to assist patients in making optimum use of language, metalanguage, cognitive, metacognitive, and related skills during listening. Specific details regarding central resources training is provided in Chapter 10 of Volume 2 of this Handbook. In addition, particular attention should be paid to the listening and communicative environment of older adults. For those adults who are still in the workplace, or who regularly attend functions that require the ability to hear a speaker well (e.g., church, Bingo, the theater), consideration may be given to hearing assistive technology (HAT) to improve the signal-to-noise ratio in these environments. Even older adults who spend a great deal of time at home may benefit from HAT to assist in hearing the television or radio. Communication repair strategies and auditory-visual speech perception training also is an integral part of intervention for older adults with auditory disorders, including CAPD and peripheral hearing loss. It is critical that family members, spouses, and other communicative partners be involved in the intervention and counseling process, and that mutually agreed upon solutions to communicative dilemmas be developed. See Chapter 12 in Volume 2 of this Handbook for detailed discussion of HAT.

The use of clear speech to enhance acoustic cues has been demonstrated to improve signal clarity and speech recognition for listeners with auditory disorders (Bradlow, Kraus, & Hayes, 2003). Clear speech consists of several acoustic modifications, including a reduction in speaking rate, increase in duration of consonants and vowels, complete

release of stop consonants, and increased consonant-to-vowel intensity ratio (e.g., Bradlow et al., 2003; Ferguson & Kewley-Port, 2002; Pichenyi, Durlach, & Braida, 1986). Although research has shown that simply asking communicative partners to speak clearly is effective in achieving these acoustic modifications, resulting in improved speech recognition on the part of the listener, there is recent evidence to indicate that overtly *training* communicative partners in clear speech production results in greater speech recognition benefits (Caissie, Campbell, Frenette, Scott, Howell, & Roy, 2005). Furthermore, as discussed previously in this chapter, cross-gender counseling also may be of benefit for life partners to assist in understanding the opposite gender's listening and related difficulties (Bellis & Wilber, 2001).

Because so many older adults also present with peripheral hearing loss, it is important to remember that there is some evidence to suggest that the presence of CAPD may impact the ability to receive optimum benefit and/or satisfaction from binaural amplification (e.g., Chmiel & Jerger, 1996; Chmiel et al., 1997). Although this does not by any means indicate that binaural amplification should not be considered for patients with peripheral hearing loss and comorbid CAPD, it does indicate that special care may need to be taken to ensure optimum adjustment to binaural amplification in these cases.

In addition, evidence is emerging that individualized, intensive auditory training may improve hearing aid outcomes in general (e.g., Henderson-Sabes & Sweetow, 2007; Sweetow, 2005; Sweetow & Henderson-Sabes, 2004; Sweetow & Palmer, 2005; Sweetow & Sabes, 2006). This is consistent with research indicating that auditory plasticity extends throughout the life span and that adults demonstrate both stimulation- and deprivation-induced plasticity in the central auditory pathways (e.g., Alain & Synder, 2008; Blake, Strata, Churchland, & Merzenich, 2002; Silman & Silverman, 1993; Tremblay, Kraus, Carrell, & McGee, 1997; Tremblay, Kraus, & McGee, 1998; Tremblay, Kraus, McGee, Ponton, & Otis, 2001). Thus, although greater attention has been given historically to methods of assisting older adults in compensating for auditory deficits through acoustic signal enhancement and communication strategy training approaches, the role of deficit-specific, intensive auditory training for adults should not be overlooked. See Chapters 7 and 9 in Volume 2 of this Handbook for in-depth discussion of auditory training.

In conclusion, intervention for CAPD in older adults, as with children, should focus on three primary areas: environmental modifications (bottom-up) to improve acoustic signal clarity and enhance access to the auditory signal, compensatory strategies (top-down) to strengthen and recruit central resources to assist in buttressing deficient auditory skills, and direct auditory training (bottom-up) to address specifically the auditory deficits present.

Summary

This chapter has focused on the various factors that contribute to the well-recognized speech recognition difficulties of older adults. Specifically, peripheral hearing loss, changes in central auditory function, and cognitive factors often interact multiplicatively to impact adversely an adult's ability to listen and communicate

effectively. Methods of differentially diagnosing CAPD in older adults must take into account all three of these factors, as well as the lifestyle and communicative needs of the individual patient. Intervention for CAPD in older adults should focus on improving the acoustic and communicative environment and utilizing central resources to compensate for auditory deficits. Finally, the potential importance of deficit-specific auditory training for maximizing listening and communication success in older listeners should not be discounted. Speech perception requires decoding and cognitive effort to process the sounds of language into a meaningful message. It is "what we do with what we hear" (Katz, Stecker, & Henderson, 1992, p. 5) that matters.

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CHAPTER 19

DIFFERENTIAL DIAGNOSIS OF CENTRAL AUDITORY PROCESSING DISORDER AND AUDITORY NEUROPATHY SPECTRUM DISORDER

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Auditory neuropathy spectrum disorder (ANSD) is a condition characterized by normal otoacoustic emissions (OAEs), a recordable cochlear microphonic (CM), and an absent or grossly abnormal auditory brainstem response (ABR) (Sininger & Oba, 2005). The primary complaint of a person diagnosed with ANSD is difficulty understanding speech, especially in background noise. Similarly, difficulty understanding speech in background noise is a hallmark symptom of individuals with central auditory processing disorder (CAPD) (Chermak & Musiek, 1997). "CAPD refers to the difficulties in the perceptual processing of auditory information in the central nervous system and the neurobiologic activity that underlies that processing and gives rise to the electrophysiologic auditory potentials" (ASHA, 2005). It is a "true" clinical disorder, as demonstrated by numerous site of lesions along the central auditory nervous system (CANS), affecting behavioral and electrophysiologic measures (AAA, 2010). Although both ANSD and CAPD can present with some of the same auditory problems and overlapping symptomatology, ANSD can be distinguished from CAPD through careful assessment with an appropriate test battery.

There is little question that a comprehensive test battery is the best approach to confirm a CAPD diagnosis (AAA, 2010; ASHA, 2005; Jerger & Musiek, 2000). In addition, interpretation of the CAPD test battery may provide insight into the anatomical site of the auditory dysfunction (Bellis, 2003). Recent advances in diagnostic testing have also enabled

clinicians to gain a better understanding of the dysfunctional site(s) responsible for ANSD.

Other terminology has been used to describe ANSD, including auditory neuropathy and later avoid the implication of an anatomical diagnosis, the term auditory neuropathy/auditory dyssynchrony was used (Berlin, Hood, & Rose, 2002). Recently, the term ANSD was suggested, as it may include several possible sites of lesion and includes a range of communicative deficits (Northern, 2008).

Rapin and Gravel (2003) contend ANSD has been overused to describe a heterogeneous group of disorders without regard to the anatomical site, and the term auditory neuropathy (AN) should only be used when the peripheral auditory system (i.e., spiral ganglion cells, their processes, and the VIIIth nerve fibers) is affected. Comprehensive testing may lead to an anatomically based diagnosis of ANSD and may be used in management decisions (Rapin & Gravel, 2003; McMahon, Patuzzi, Gibson, & Sanli, 2008).

ANSD is not a new disorder, as reports that predate the use of the ANSD term have described patients who have abnormal or absent ABR recordings but have normal pure tone audiograms (Davis & Hirsh, 1979; Worthington & Peters, 1980). For many years, these patients have been poorly understood, misdiagnosed, improperly managed, and sometimes dismissed as not having an organically based hearing loss (Kraus, 2000). Some patients, diagnosed with a sensorineural hearing loss (SNHL) at a time before OAE testing became routine may have rejected hearing aids (Berlin, Hood, Hurley, & Wen, 1996) or had a history of responding inconsistently to sound (Kraus, 2000) resulting from undiagnosed ANSD.

Incidence and Prevalence

Kraus, Ozdamar, Stein, and Reed (1984) first reported what is now known and described as ANSD. In their retrospective study, these investigators reported that 49 of 543 children who were evaluated for hearing loss had absent ABRs. Of these 49 patients, 42 had severe to profound hearing loss that was later confirmed by behavioral thresholds. Seven of the 49 patients with absent ABRs had behavioral thresholds that were no greater than a moderate hearing loss and would most likely, with OAE data, have been diagnosed with ANSD. Thus, the ANSD incidence that is the number of newly diagnosed cases in a given year would be 1.3% (Kraus et al., 1984).

ANSD prevalence (i.e., the total number of individuals with the disorder) is generally higher than the prevalence of CAPD in the general population; however, the reported prevalence of ANSD may be inflated due to the use of an inappropriate diagnostic criterion that requires only an abnormal ABR for diagnosis versus the more stringent requirement of an absent ABR and in different populations such as infants "at risk" or infants with permanent hearing loss. Rance et al. (1999) found 12 of 5199 (0.23%) at-risk babies were diagnosed with ANSD, compared with 2% diagnosed with a SNHL. Ngo, Ran, Balakrishan, Lim, and Lazaroo (2006) reported nine cases of ANSD in 14,807 babies as part of a universal hearing screening (0.6 per 100 screened). Dowley et al. (2009) reported that of 45,050 infants in a neonatal intensive care unit, 30 infants had severe or profound hearing loss and 12

of the 30 had ANSD. Reviewing multiple studies, Rance (2005) reported ANSD occurs in approximately 7% of children with permanent hearing loss. Many of these children had associated risk factors of anoxia and hyperbilirubinemia.

Chermak and Musiek (1997) estimated that CAPD affects approximately 2% to 3% of the school-age population, whereas Bamiou, Musiek, and Luxon (2001) maintained that auditory processing deficits impacted 7% of children.

Risk Factors for ANSD and CAPD

Just as there is no single cause for hearing loss, there is no single cause for ANSD or CAPD. Sininger and Oba (2001) reported that 80% of 25 patients with ANSD who had symptoms before age 2 had neonatal risk factors for hearing impairment. Berlin et al. (2010) reported that of 153 pediatric patients with a diagnosis of ANSD, only 18% had normal birth history and 20% reported a normal pregnancy. Berg, Spitzer, Towers, Bartosiewicz, and Diamond (2005) reviewed risk factors for 9419 newborns screened for hearing loss and ANSD and could not find a statistical model for predicting ANSD. The predisposing risk factors associated with hearing loss, ANSD, and CAPD are listed in Table 19–1.

Interestingly, several risk factors, such as hyperbilirubinemia and a positive family history, are associated with hearing loss, ANSD, and CAPD. The central auditory system is highly sensitive to bilirubin toxicity. Damage occurs in the auditory nuclei of the brainstem, with the neurons in the cochlear nuclei being severely affected or completely destroyed resulting in a central pathology (Dublin, 1985). In contrast, bilirubin does not appear toxic to the peripheral auditory structures, including the VIIIth nerve or hair cells (Dublin, 1985). As noted above, Rapin and Gravel (2003) suggested that ANSD should not be diagnosed in the absence of involvement of the spiral ganglion cells, their processes, and the VIIIth nerve. It is important to note that individuals with ANSD can recover over time (Attias & Raveh, 2007; Worley, Erwin, Goldstein, Provenzale & Ware, 1996). Psarommatis et al. (2006) reported improved ABRs in 13 of 20 babies one month after initial testing, suggesting that multiple assessments are necessary.

Just as many siblings are diagnosed with CAPD, investigators have reported positive sibling histories of ANSD (Starr et al., 1998). For example, Sininger and Oba (2001) reported 46% of 59 subjects had a positive family history of ANSD. In the case of CAPD, there are postnatal factors that may contribute to CAPD, such as frequent bouts of otitis media, meningitis, high fever, and exposure to lead or other toxic substances. In addition, any type of neurological disorder or head injury that may be associated with ANSD also can cause CAPD (Barr, 1976; Brown, 1994; Chedru, Bastard, & Efron, 1978; Gravel & Wallace, 1992; Hall & Grose, 1993; Musiek, Baran, & Shinn, 2004; Willeford & Burleigh, 1985). Some patients with ANSD will have other neurological abnormalities or other peripheral neuropathies that may be recognized only during a neurological examination. ANSD can be part of a syndrome, occur with other medical conditions, or occur with nonauditory neuropathies such as Friedrich ataxia (Taylor, McMenamin,

Table 19–1. Associated Risk Factors for Hearing Loss, ANSD, and CAPD

Hearing Loss	ANSD	CAPD
Family History	Family History	Family History
Infections: (Toxoplasmosis, Rubella, Cytomegalovirus [CMV], Herpes Virus, Syphilis)	Infections: (Toxoplasmosis, Rubella, Cytomegalovirus [CMV], Herpes Virus, Syphilis)	Infections: (Toxoplasmosis, Rubella, Cytomegalovirus [CMV], Herpes Virus, Syphilis)
Hyperbilirubinemia	Hyperbilirubinemia	Hyperbilirubinemia
Craniofacial Anomalies	Immune Disorders (Type 1 Diabetes)	RH Incompatibility
Low Birth Weight	Uremia	Difficulty During Birth
Other Syndromes	Genetic/Syndrome	Toxic Exposures
Ototoxic Medications	Ototoxic drugs	Ototoxic Medications
Prematurity	Prematurity	Prematurity
Anoxia	Anoxia	Anoxia
Infections after Birth		Infections after Birth
Mechanical Ventilation	Mechanical Ventilation	Head Trauma
Bacterial Meningitis		Cerebrovascular Disorders
		Metabolic Disorders
		Epilepsy
		Recurrent Otitis Media
		Meningitis/Encephalitis
		Developmental Disorders (e.g., Dyslexia, Learning Disability, Language Impairment, Attention Deficit Hyperactivity Disorder)

Andermann, & Walters, 1982), Stevens-Johnson syndrome, Ehrlers-Danlos syndrome, and Charcot-Marie-Tooth syndrome (Berlin, Hood, Cecola, Jackson, & Szabo, 1993; Deltenre, Mansbach, Bozet, Clercx, & Hecox, 1997). ANSD may be unilateral or bilateral, with unilateral cases occurring in less than 10% of ANSD patients (Buchman et al., 2011); Berlin et al. (2010) reported that in their sample, 93% (241) were bilateral as compared with 7% (19) being unilateral cases of

ANSD (Berlin et al., 2010). See Chapters 4 and 8 for additional discussion of risk factors and etiologies of CAPD.

[redacted]te of ANSD Dysfunction

The underlying physiologic site of auditory dysfunction that results in ANSD may be determined using an extensive electrophysiologic test battery. There are several hypothesized anatomical sites for this disorder and there may be more than one site of physiologic dysfunction that results in ANSD. Investigators have reported the underlying physiologic site of ANSD could possibly be a mechanical dysfunction of the inner hair cells (Harrison, 1988), the synapse between the inner hair cells and type I auditory nerve fibers, or a dysfunction of the VIIIth nerve axons, cell bodies, and/or myelin sheath (Starr, Picton, Sininger, Hood, & Berlin, 1996). Rapin and Gravel (2003) reported site-specific test results for ANSD. For example, an inner hair cell etiology would result in recordable OAEs and CM, but an absent ABR, and abnormal psychoacoustic tests (e.g., speech in noise, masking level difference, temporal resolution, frequency resolution, intensity processing [loudness scaling and intensity discrimination]) and would classify this as an isolated site-specific ANSD. This is in contrast to an isolated brainstem lesion, which would result in absent acoustic reflexes (either ipsilaterally, contralaterally, or both, depending on the size and site of the lesion, recordable OAEs and CM, recordable waves I and II of the ABR, but absent, or delayed, waves III and V, and abnormal psychoacoustic tests, for example, speech in noise, masking level difference, temporal resolution, frequency resolution, and intensity processing [loudness scaling and intensity discrimination]). Recent investigations have classified ANSD into presynaptic and postsynaptic lesions (McMahon et al., 2008). This distinction, as reviewed in a following section, may be useful in determining management options. Furthermore, subtype profiles have been proposed to better understand the anatomical site(s) of auditory dysfunction and its behavioral manifestations for both ANSD and CAPD (Bellis & Ferre, 1999; Katz, 1992; Musiek & Gollegly, 1988; Rapin & Gravel, 2003; Starr, Picton, & Kim, 2001).

Determining the precise site of dysfunction is important for understanding the physiologic mechanisms and may be very useful in determining the remediation plan for these patients. Loss of inner hair cells has been a hypothesized model for ANSD. Starr et al. (2003) reported temporal bone histology of an individual with Charcot-Marie-Tooth disease, showing loss of inner hair cells, normal outer cells, and a significant loss of spiral ganglion cells.

Amatuzzi et al. (2001) reported histological temporal bone studies in 15 nonsurviving infants of a neonatal intensive care unit. Of the 15 babies born prematurely, 12 had failed an ABR screen bilaterally, one had failed the ABR unilaterally, and two had passed, bilaterally. Two patients had loss of both inner and outer hair cells, two with only outer hair cell loss, and three with selective inner hair cell loss. No hair cell loss was seen in either patient who had passed the ABR screen, bilaterally. Many of these infants had other hearing loss risk factors such as respiratory failure that may be consistent with prolonged hypoxia that may have a greater effect on inner hair cell

than outer hair cell survival (Shirane & Harrison, 1987).

To further investigate the effects of prematurity on inner hair cell loss, Amatuzzi, Liberman, and Northrop (2011) reported histopathological studies of 54 temporal bones from premature infants and a matched control group of 46 temporal bones from full-term, nonsurviving infants of a neonatal intensive care unit. Both groups shared common risk factors, including respiratory insufficiency, sepsis, and neurological problems. Six patients in the premature group also had hyperbilirubinemia. Because of postmortem cellular disintegration, results from only 37 of the 54 premature temporal bones and from 36 of the 46 full-term temporal bones were reported. Normal inner and outer hair cell distribution was found in 22 of 37 (59%) preterm ears and in 26 of 36 (72%) term ears. Outer hair cell loss was seen in four of 37 (11%) preterm ears and in 6 of 36 (17%) term ears. Both inner and outer hair cell loss was seen in one of 37 (3%) premature ears and in three of 36 (8%) term ears. Inner hair cell loss was seen in 10 of 37 (27%) preterm ears and in only one of 36 (3%) term ear. The authors concluded that one possible etiology of ANSD is selective inner hair cell loss.

Another possible site of lesion leading to ANSD is the synapse between the inner hair cells (Starr et al., 20011). Neurotransmitters are stored at the base of the hair cells. A presynaptic dysfunction may prevent the release of neurotransmitters and a postsynaptic dysfunction may inhibit the auditory receptor dendrite from responding to the chemical exchange (Starr et al., 2000).

Finally, the dysfunction may be the auditory nerve. In reviewing MRI studies of 51 children with ANSD, Buchman et al. (2006) reported 9 of 51 ANSD patients had small or absent cochlear nerves. Starr et al. (1996) reported eight of 10 subjects in this early paper had evidence of other peripheral nerve abnormalities.

Starr (2001) proposed four types of ANSD that may affect the cochlear nerve. In the first type, demyelinating neuropathies affect the Schwann cells that compose the myelin sheaths and affect the rapid transmission of information exchange from one neuron to another (Starr, Picton, & Kim, 2001). In the second type, axonal neuropathies result in muscular weakness and muscular atrophy (Starr et al., 2001). In the third type, sensory axonal neuropathies cause impaired sensation (Starr et al., 2001). In the fourth type, mixed neuropathies affect both the myelin sheath and neuron axon (Starr et al., 2001) and may occur as a progressive degeneration from axonal to demyelinating neuropathy or vice versa (Rapin & Gravel, 2003). If the pathology extends to the brainstem, however, this lesion should be characterized as a central lesion, not ANSD (Rapin & Gravel, 2003).

CAPD Dysfunction

Although no CAPD subtypes have been universally accepted, investigators have attempted to document the heterogeneous nature of CAPD by describing the characteristics in terms of commonalities (Bellis & Ferre, 1999; Katz, 1992; Musiek & Gollegly, 1988). For example, Musiek and Gollegly (1988) reported three causes of CAPD in children with learning disabilities: one based upon neuromaturational delay, a second resulting from a neuromorphological disorder, and a third arising from neurologic disease or insult.

More recently, research has documented the neurobiological underpinnings of CAPD in children, pointing out the neurological bases of inefficient interhemispheric transfer of auditory information and/or lack of appropriate hemispheric lateralization, and atypical hemispheric asymmetries experienced by patients with CAPD. A much less frequent etiology of CAPD is a neurologic disorder, insult, or abnormality in children (Jerger et al., 2002; Kraus et al., 1996; Musiek, Baran, & Pinheiro, 1994). In adults, CAPD may result from accumulated damage or deterioration to the CANS due to neurological/neurodegenerative diseases, disorders, or insults, brainstem abnormalities, and the aging process itself, which leads to poorer neural synchrony, slower refractory periods, decreased central inhibition, and interhemispheric transfer asymmetry/deficits (Bellis, Nicol, & Kraus, 2000; Bellis & Wilber, 2001; Jerger, Moncrieff, Greenwald, Wambacq, & Seipel, 2000; Pichora-Fuller & Souza, 2003; Tremblay, Piskosz, & Souza, 2003; Willott, 1996; Woods & Clayworth, 1986). Nonetheless, generally, it is not yet possible to directly and definitively determine the etiology of CAPD in certain populations (e.g., certain neuromorphological abnormalities like ectopic cells can only be visualized postmortem) using today's clinical tools.

[redacted]ferential Diagnosis

Given the overlapping symptomatology, etiology, and similar anatomical sites that may contribute to both CAPD and ANSD, it is necessary to administer a complete test battery to differentially diagnose these two disorders. Rapin and Gravel (2003) commented that it is unfortunate that few patients have received the thorough test battery required, which has resulted in the current confusion in the audiology literature regarding physiologic and behavioral test results in patients with presumed ANSD. Performance differences across a number of behavioral tests, and electrophysiologic and electroacoustic procedures are reviewed in the following sections.

Pure-Tone Audiometry

Patients with ANSD may present with normal hearing or any degree of hearing loss from mild to profound and with various configurations such as flat, reverse slope, or high frequency (Berlin et al., 2010; Sininger & Oba, 2001). Most importantly, hearing thresholds for patients with ANSD may also fluctuate, as reported by some investigators (Sininger & Oba, 2001). Specifically, hearing thresholds for some patients with ANSD may deteriorate over time but remain stable in other patients (Sininger & Oba, 2001). Investigators have also reported a "temperature-sensitive" ANSD in which pure-tone thresholds change from normal to profound hearing loss as a function of internal temperature (Gorga, Stelmachowitz, Barlow, & Brookhouser, 1995; Starr et al., 1998). In short, there is a great deal of variance in the audiometric threshold results for ANSD patients. Unlike ANSD, pure tone audiometric thresholds for patients with CAPD are by definition normal, although individuals with CAPD can also present comorbid hearing impairment, particularly among older adults (Stach, Spretnjak, & Jerger, 1990). However, typically pure-tone thresholds are normal and do not fluctuate.

Immittance Testing

Generally, tympanometry is normal for patients with ANSD; thus, indicating normal middle ear pressure and compliance. These measures are also generally normal in patients with CAPD; although children with CAPD may experience otitis media and resulting abnormal tympanometry (Willeford & Burleigh, 1985). Acoustic reflexes can provide valuable information about the integrity of the middle ear, cochlea, VIIIth nerve, and lower brainstem. Acoustic reflexes are generally abnormal or elevated in patients with ANSD (Berlin, 1993; Berlin et al., 2010; Hood, 1998a; Sininger & Oba, 2001), while generally acoustic reflexes are present at normal levels in patients with CAPD, unless there is a lower brainstem lesion (Jerger & Jerger 1974). Hall and Johnson (2007) reported abnormal contralateral reflexes in 30% and abnormal ipsilateral reflexes in 20% of 196 children undergoing CAPD assessment. In some patients with CAPD, reflexes may be slightly elevated or absent perhaps due to histories of persistent otitis media. Welch and Dawes (2006) reported elevated acoustic reflexes in a group of 631 children with histories of protracted otitis media, with no elevation of audiometric thresholds.

Otoacoustic Emissions

OAEs are expected to be present in patients with normal middle and inner ear function; however, a diagnosis of ANSD should be suspected if a person has recordable (normal) OAEs with an audiogram that shows an SNHL greater than 35 dB HL (Starr, 2001). Present OAEs and a SNHL may, however, be a confounding factor in some patients with ANSD due to the fact that OAEs may deteriorate over time (Deltenere et al., 1997; Starr et al., 1996) or can be absent (Berlin et al., 2010). Individuals with CAPD are expected to have normal OAEs. The obvious exceptions in either group are the patients who have histories of protracted otitis media, resulting in absent or abnormal OAEs, which reflects a mild impairment of sound transmission through the middle ear (Rappaport & Provencal, 2002).

Speech Audiometry

One of the most common complaints of individuals with ANSD is difficulty understanding speech, especially in background noise (Sininger & Oba, 2001). Word recognition ability in quiet in suspected ANSD patients is usually poorer than expected based on behavioral pure tone thresholds (Sininger & Oba, 2001). Furthermore, patients with ANSD may often have histories of reporting that speech is not clear, often sounding distorted (Berlin, Hood, & Rose, 2002). Likewise, many patients with CAPD present this auditory complaint of difficulty hearing in background noise (Bellis, 2003).

Electrophysiologic Assessment

Cochlear Microphonic

It is believed that the CM response is generated primarily by the outer hair cells (Dallos, 1973); however, the inner hair cells may likewise contribute to this response (Dallos, 1997). The CM is expected to be present in ANSD patients, albeit at an amplitude that may be larger

than expected (Sininger & Oba, 2001; Starr, Sininger, & Pratt, 2000). In CAPD, the CM is expected to be of normal amplitude. To ensure accurate interpretation, clinicians are encouraged to obtain CM recordings using both rarefaction and condensation clicks that allow ABR waves to be separated from the CM response (Berlin et al., 1998).

Electrocochleography

Electrocochleography (ECochG) may be useful in differentiating between a presynaptic etiology involving the inner hair cells and a postsynaptic etiology involving the afferent neurons (McMahon et al., 2008; Santarelli & Arslan, 2002; Santarelli, Starr, Michalewski, & Arslan, 2008). ECochG investigations are promising and offer diagnostic information as to the underlying anatomical site of lesion. This is very useful for management decisions. For example, recording ECochGs to clicks and an 8000 Hz tone from a round window electrode, McMahon et al. (2008) found two distinct patterns of abnormality relating to presynaptic and postsynaptic sites of dysfunction in 14 patients with ANSD. The presynaptic abnormalities showed a delayed summating potential (SP), with no dendritic potential (DP), but with a delayed or distorted compound action potential (CAP). Six of seven patients demonstrating this presynaptic site of lesion also demonstrated good electrically evoked ABR (EABR). The postsynaptic findings demonstrated by six patients showed a normal latency SP, possible DP, but no CAP. All six patients demonstrating a postsynaptic site of lesion had absent EABR.

Santarelli and Arslan (2002) reported ECochG recorded from the promontory in five patients with absent ABRs and present OAEs. All recordings showed CM responses; however, three showed CAP recordings, even though the ABR was absent. The presence of the CAP may be an example of "temporal smearing of spike activity or neural dyssynchrony during ABR collection" (Berlin et al., 2001).

Santarelli et al. (2008) reported site of lesion results from EcochG responses in patients with ANSD. They reported inner hair cell loss to be consistent with a present SP, but absent CAP. A proximal nerve disorder was associated with the presence of both SP and CAP. A postsynaptic nerve terminal abnormality would be characterized by the presence of a prolonged neural response and a substantial decrease in amplitude of the CAP at high stimulation rates.

There is a paucity of investigations reporting ECochG recordings in patients with CAPD. Most likely, one would assume this recording would be normal; however, abnormal recordings may reflect inner hair cell abnormalities.

Auditory Brainstem Response

An ABR is a short latency response, less than 10 msec poststimulus onset, which provides objective evidence of the integrity of the auditory brainstem. The ABR consists of a waveform complex that reflects synchronous firing of the auditory nerve through the brainstem; it is a test of neurosynchrony (Hood, 1998b). Historically, the ABR has also been used in the diagnosis of central nervous system disorders such as demyelinating diseases (Jerger, Oliver, Chmiel, & Rivera, 1986), degenerative diseases (Harkins 1981), and asynchronous disorders such as ANSD (Hood, 1998b; Starr et al., 1996). Unlike imaging techniques that have

superior spatial resolution and are useful in identifying structural defects, the ABR has excellent temporal resolution and is useful in detecting CANS disorders (Hall, 1992). In short, the ABR is a test of physiology, whereas most imaging techniques examine anatomical structure.

One of the primary criteria for the diagnosis of ANSD is an absent ABR (Hood & Berlin, 2001; Sininger & Oba, 2001; Zeng, Oba, Garde, Sininger, & Starr 2001). Other investigators have broadened the diagnosis of ANSD to include abnormal ABRs (Starr, 2001). These expanded criteria may lead to a diagnosis of ANSD when there is instead a central auditory deficit (Rapin & Gravel, 2003). The ABR abnormality begins with an absent wave I, which implies asynchronous firing at the synapse between the hair cells and auditory nerve (Figure 19–1). Sininger and Oba (2001) reported absent ABRs in 70% of their 59 subjects with confirmed ANSD, abnormal ABRs in 6% of these subjects, with 19% of these subjects presenting only wave V in the ABR recording, implying asynchrony in the lower brainstem. Thus, an absent ABR is most indicative of ANSD. In contrast, most investigations report normal ABRs in children with CAPD (Hall & Johnston, 2007; Hall & Mueller, 1997; Hurley, 2004; Mason & Mellor, 1984; Roush & Tait, 1984). However, reports of compromised ABR in patients with CANS dysfunction demonstrate the importance of this electrophysiologic response in the central auditory processing battery (Musiek, Charette, Morse, & Baran, 2004). ABR is a very valuable tool in CAPD assessment, as it may provide objective evidence of any brainstem involvement, especially in patients with protracted histories of otitis media or hyperbilirubinemia (Dublin, 1985; Musiek et al, 1994; Musiek & Lee, 1995).

Auditory Middle Latency Response

The underlying auditory generators of the auditory middle latency response (AMLR) include the thalamocortical pathway (Kileny, Paccioretti, & Wilson, 1987; Kraus, Ozdamar, Hier, & Stein, 1982; Ozdamar & Kraus, 1983), the reticular formation (Kraus, Kileny, & McGee, 1994), and the inferior colliculus (McGee, Kraus, Comperatore, & Nicole, 1991). Specifically, the early AMLR components, waves Na and Pa, might arise from the medial geniculate and thalamus, whereas the association areas of the cortex might be responsible for waves Nb and Pb (Geisler, Frishkopt, & Rosenblith, 1958).

There is no consistent pattern for the AMLR in patients with ANSD. Starr et al. (1998) reported absent AMLRs in four patients: Abnormal AMLR recordings were obtained in two patients, and a normal AMLR recording was obtained in one patient. Absent AMLR recordings have also been reported in other published case reports (Hood & Berlin, 2001). The presence or absence of the AMLR is dependent on where the site of auditory dysfunction lies along the central auditory pathway. Thus, the inclusion of the AMLR response may provide valuable information regarding the site of the auditory dysfunction.

The AMLR recording also is a valuable tool in assessing maturity of the central auditory pathway, with a multiple electrode montage being recommended in CAPD assessment (Chermak & Musiek, 1997). Possibly because the intersubject variability of the AMLR is great in the

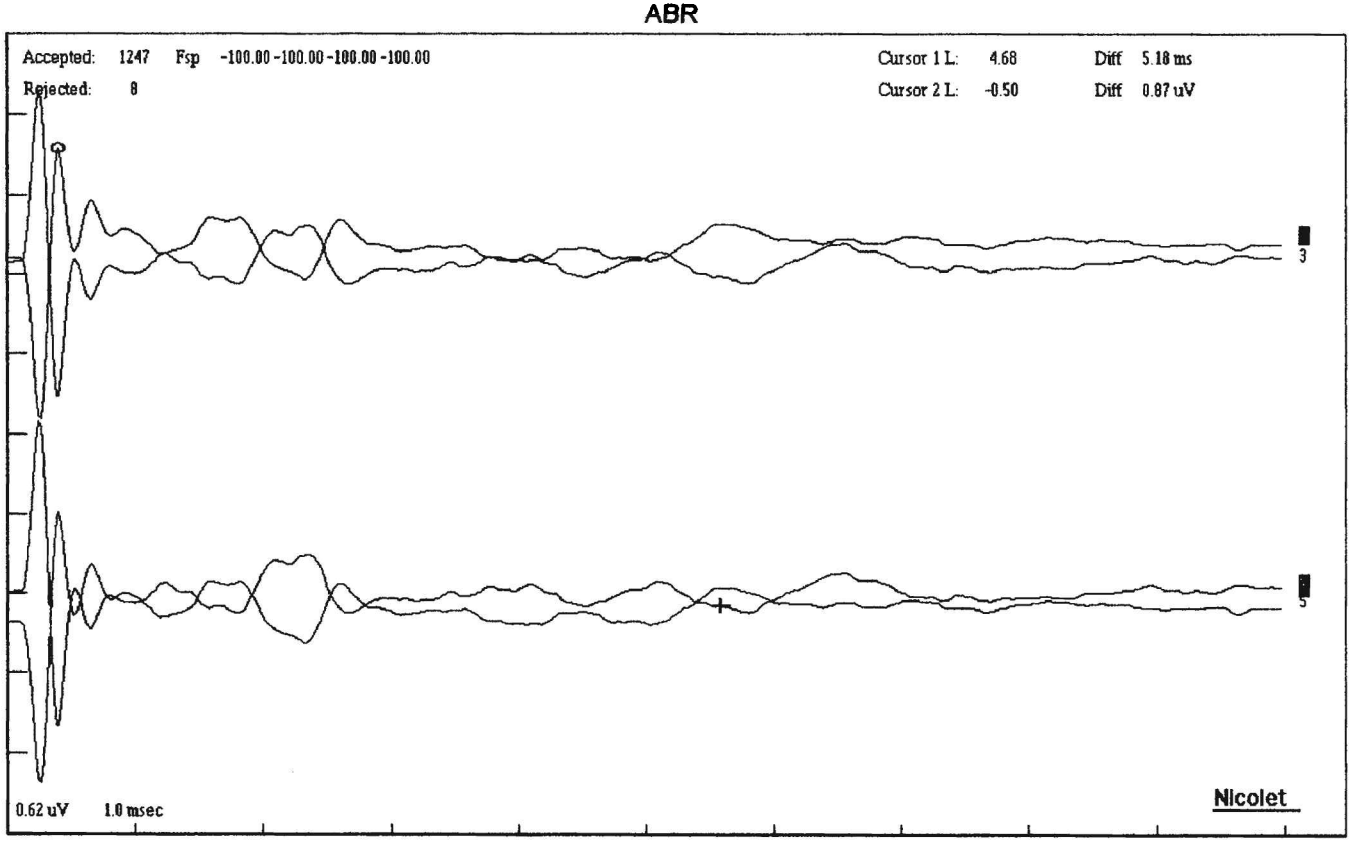

Figure 19–1. The auditory brainstem response (ABR) recording from a 15-year-old female that illustrates the common finding of a biphasic response in ANSD cases. Also note a complete inversion of elements even beyond 2 msec. This patient presented a flat, bilateral sensorineural hearing loss of moderate degree; however, distortion product otoacoustic emissions for most of the 1000 to 8000 Hz test range were within normal limits.

pediatric population, there have been no attempts to establish normative latency and amplitude data for very young children. It is important to note that the AMLR does not reach adult maturity until approximately age 8 to 12 years. However, it is a significant finding if the AMLR is absent in this age population (Chermak & Musiek, 1997).

Unlike the standard ABR, amplitude measures may be a more sensitive measure than latency in CAPD assessment using the AMLR (Kraus et al., 1982; Scherg & von Cramon, 1986). The multisite amplitude measures are compared over hemispheres to determine if there is an electrode effect (Musiek, Baran, & Pinheiro, 1994) or ear effect (Musiek et al., 1999). Pre- and post-AMLR recordings may also be useful to assess the efficacy of auditory training programs for individuals with ANSD or CAPD, and to study auditory maturation in young children (Musiek et al., 2004; Schochat, Musiek, Alonso, & Ogata, 2010).

Previous investigations have reported abnormal AMLR recordings in children with learning and speech and/or language disabilities (Jerger & Johnson, 1988; Milicic et al., 1998), in adults diagnosed with CAPD (Marvel, Jerger, & Lew, 1992), and in patients with cortical lesions (Musiek, Baran, & Schochat, 1999). Although the AMLR may be an objective indicator of CANS disorder, there is still a lack of data on the AMLR in children with CAPD.

Auditory Long Latency Response

The auditory long latency response (ALLR) is a set of cortical responses that can be divided into sensory potentials (auditory late response [ALR]) and processing contingent potentials (P300) (Davis, 1976; Steinschneider, Kurtzberg, & Vaughan, 1992). These potentials have generators that are located in the auditory cortex and are believed to reflect higher order processing in individuals (Steinschneider et al., 1992). Waves N1 and P2 of the ALR, also referred to as N100 and P200, as they occur at approximately 100 and 200 msec poststimulus, occur in response to clicks, tones, or speech. These sensory potentials are sometimes referred to as exogenous potentials (stimulus-related). Specifically, the generator of the N1 and P2 components is believed to be the auditory cortex (Naatanen & Picton, 1987), although the P2 component may also reflect activity in the reticular formation (Naatanen & Picton, 1987).

The P300 response is a processing-contingent potential and is also referred to as an endogenous response (subject-related) (Squires & Hecox, 1983). This potential is usually elicited by an oddball paradigm in which the subject actively attends to an occasional rare stimulus as opposed to the standard stimuli (Sutton, Braren, Zubin, & John, 1965). Thus, attention, discrimination, and memory are reflected in the P300 recording (Picton & Hillyard, 1988). The underlying generators are not completely defined, but evidence shows that the hippocampus, auditory cortex, and temporal lobe contribute to this response (Buchwald, 1990; McPherson, 1996).

Similar to the AMLR, a multiple electrode montage is recommended when recording ALLRs (Chermak & Musiek, 1997; Peronnet & Mickel, 1977), as this will provide amplitude and latency measures to objectively determine hemispheric involvement for ear or electrode effects. Chermak and Musiek (1997) recommended recording N1 and P2 from electrode sites C3, C4, and Cz and the

P300 from Fz, Pz, and Cz and additionally C3 and C4. As with AMLR recordings, the ALR and P300 studies may be useful in longitudinal studies to monitor or assess the effectiveness of an auditory training program.

Neural synchrony is represented differently in the ALLR than in the early ABR, as disrupted neural synchrony in the earlier auditory evoked potentials does not lead to asynchronous ALLRs. Starr et al. (1996) reported differing results in a group of patients with ANSD. All patients had abnormal ABRs, yet two patients had normal ALRs, two had abnormal ALRs, and two had absent ALR recordings. Berlin and Hood (2001) also reported absent ALRs in one patient with ANSD. Again, the presence or absence of these potentials appears to be dependent on the site of disorder in the central auditory pathway. ALLRs may also be recorded because they are less dependent upon neural synchrony and more resistant to fluctuations in the timing of individual responses.

Jirsa and Clontz (1990) reported prolonged P300 latencies in a group of children with CAPD. Similar, prolonged P300 latencies have also been reported in a group of patients with cerebral lesions (Musiek, Baran, & Pinhero, 1992). It is important to note that nonauditory factors may affect the P300 recording (Knight, 1990); thus, an absent or abnormal ALR and P300 recording does not warrant a diagnosis for CAPD. However, inclusion of the ALR and P300 recordings in the CAPD battery may provide objective evidence to support the diagnosis of a CAPD (Jerger, Chmiel, Tonini, Murphy, & Kent, 1999; Purdy, Kelly, & Davies, 2002; Wible, Nicol, & Kraus, 2005). (See Chapter 17 for discussion of auditory evoked potentials in CAPD diagnosis.)

Behavioral Tests of Temporal Processing

Temporal processing requires synchronous discharge of neurons of the peripheral and central auditory pathway (Phillips, 1995). Sound undergoes complex processing by intricate neural mechanisms and neural networks that are composed of structures located in the brainstem, subcortex, primary and association areas of the auditory cortex, and the corpus callosum (Phillips, 1995). In addition, these mechanisms are responsible for transmitting, enhancing/inhibiting, reshaping, refining, and assigning recognition and meaning to sound. (See Chapters 3 and 5 for discussion of neurorepresentation of time in the CANS.)

Temporal processing is critical in speech perception, since all acoustic signals, such as speech, vary over time (Tallal, 1985). In order to extract meaning from these varying acoustic signals, the listener must be able to detect very small and rapid time variations. Physiologic changes such as axonal loss and demyelination can easily disrupt temporal pattern coding in the auditory system. Although temporal processes are critical in a number of auditory behaviors, there are a limited number of clinical tests available to assess temporal processing abilities (e.g., pitch and duration pattern tests, gap detection tests, and masking level differences). (See Chapter 15 for discussion of behavioral tests of temporal processing.)

Much of the difficulty with speech perception may stem from impaired temporal processing (Starr et al., 1991; Zeng, Kong, Michalewski, & Starr, 2005). Zeng et al. (2001, 2005) showed gap detection ability was impaired in all subjects with ANSD, as was temporal modulation

transfer function performance (i.e., the ability to detect an amplitude fluctuation in a steady-state noise). Zeng et al. (2001) also assessed the performance of ANSD patients on other psychoacoustic tasks and reported abnormal loudness growth measures in one patient with ANSD, normal intensity discrimination in four subjects with ANSD, and poorer frequency discrimination for frequencies less than 2000 Hz for all patients with ANSD. Interestingly, as the test frequency increased, the ANSD patient's frequency discrimination for higher frequencies improved. Abnormal forward and backward masking results were also found in patients with ANSD (Kraus et al., 2000; Zeng et al., 2001). Localization deficits have also been reported; Zeng et al. (2005) reported localization deficits on the bases of impaired interaural timing differences.

Temporal processing is a key component of auditory function; therefore, tests of temporal processing (e.g., temporal ordering or sequencing and temporal resolution) should be included in the behavioral central auditory test battery. Impaired temporal processing disrupts the normal development of an efficient phonological system that may result in language and/or reading disorders (Tallal, Miller, & Fitch, 1995), and poor temporal processing is one characteristic of CAPD (Chermak & Musiek, 1997). Temporal processes are critical in a number of auditory functions "including auditory discrimination, binaural interaction, pattern recognition, localization/ lateralization, monaural low-redundancy speech recognition, and binaural integration" (Schow, Seikel, Chermak, & Berent, 2000, p. 67). Thus, it is likely that many individuals with CAPD will have impaired temporal processing.

Audiological Summary

Table 19–2 lists many of the expected outcomes for the tests that may be used in ANSD assessment. One of the most important distinguishable differences in ANSD and CAPD is hearing loss. Although there is no distinct audiometric configuration and pure-tone thresholds may fluctuate, there is documented SNHL in most patients with ANSD; however, patients with CAPD generally have normal hearing. Acoustic reflexes are generally absent or elevated in patients with ANSD; whereas, the acoustic reflexes are generally normal in patients with CAPD. Also important is the finding that the standard ABR is expected to be absent in patients with ANSD, but typically is normal in most patients with CAPD (Mason & Mellor, 1984; Roush & Tait, 1984; Hurley, 2004) unless there is brainstem involvement (Musiek et al., 1995).

Intervention

Accurate differential diagnosis should lead to more targeted, efficient, and effective intervention. Clearly, there are challenges in managing patients with ANSD and CAPD. As with CAPD, children with ANSD should have an appropriate individual intervention and educational plan. Each case of ANSD is unique and may require modifications to the habilitation plan over time. The habilitation plan should include therapy that is directed toward the improvement of cognitive, language, and auditory skills (Bellis, 2003; Chermak & Musiek, 1997). There is evidence to support the use of assistive technologies, that is, hearing aids (bilateral or

Table 19–2. Expected Test Results For Patients With ANSD and CAPD

Audiometric Test/Procedure	Auditory Neuropathy	CAPD
Pure-Tone Thresholds	Various degrees of hearing loss and configurations	Usually within normal limits
Tympanometry	Normal	Normal
Acoustic Reflexes	Elevated or Absent	Usually within normal limits depending on site(s) of central auditory nervous system (CANS) dysfunction
Speech Recognition in Noise	Poor	Variable, depending on site(s) of CANS dysfunction
Otoacoustic Emissions	Present	Present
Gap Detection	Abnormal	Often abnormal, depending on site(s) of CANS dysfunction
ECochG	Variable depending on site of dysfunction	Normal
ABR	Absent	Usually normal
MLR	Questionable	Variable, depending on site(s) of CANS dysfunction
ALLR/P300	Questionable	Variable, depending on site(s) of CANS dysfunction

unilateral), FM systems, and/or cochlear implants for patients with ANSD.

The use of amplification with ANSD patients remains controversial. Starr et al. (1996) reported no benefit for patients with ANSD with the use of wearable amplification. Conversely, Rance et al. (1999) reported benefit from wearable amplification in half of their patients with ANSD. Berlin et al. (2010) reported no benefit in approximately 52/85 (61%) of patients with ANSD who were fitted with hearing aids. However, other ANSD patients, 12 of 85 (14%), were reported as receiving some or good benefit. Difficulties with the uncertainty of benefit are limited sample sizes and lack of standardization of how benefit is defined.

The differing results with amplification may reflect the heterogeneous makeup of the ANSD patient group.

Assistive listening devices are often recommended for patients with ANSD. An FM system increases the signal-to-noise ratio at the listener's ear, to aid communication. Patients with ANSD have reported benefit from FM systems (Hood, 1998b). Recently, Teagle et al. (2010) reported offering FM systems to all families of patients with ANSD by the child's first birthday.

Cochlear implants have also been recommended for patients with ANSD after a trial period of amplification (Hood, 1998; Northern, 2008; Rance et al., 1999; Shallop et al., 2001; Trautwein, Sininger, & Nelson, 2000). Investigators have suggested that although a cochlear implant may appear counterintuitive given possible peripheral anatomical sites of ANSD (i.e., inner hair cells) (Harrison, 1988), patients have shown benefit (Hood, 1998; Shallop et al., 2001; Trautwein et al., 2000). Berlin et al. (2010) reported successful use in 42 of 49 implanted cases of ANSD.

Teagle et al. (2010) report outcomes in 52 (37%) of 140 children with ANSD who received cochlear implants. Among these, 26 patients (50%) were able to achieve open-set recognition. Results from 11 (21%) children with ANSD who had been implanted less than six months were not obtained. Significant auditory progress was noted on the Infant-Toddler Meaningful Auditory Integration Scale for 15 (29%) children with ANSD who could not be tested formally due to age or developmental delays. Additionally, these authors also report abnormal central nervous system pathologies shown by MRI in 38% of children with ANSD, and absent or abnormal electrical ABRs were found in 41% of children with ANSD. Further, these authors reported no child with abnormal central nervous system pathology such as cochlear nerve deficiency was able to achieve open-set speech recognition. Additionally, children with open-set recognition had good electrical compound action potential (ECAP).

Recently, Breneman, Gifford, and DeJong (2012) reported that 32 of 35 (91%) children with ANSD who received cochlear implants had some degree of open-set recognition. Furthermore, there was no statistical difference between this group and their age-matched peers with SNHL. All 35 children had normal electrical compound action potential. The authors also reported outcomes of 13 children with ANSD who received cochlear implants but were not included in the pair-matched study because of comorbid conditions that limited formal speech testing. Parents of these 13 children reported an improved awareness of sound and improved quality of life.

Teagle et al.'s (2001) report that open-set speech recognition was not obtained in approximately 29% of children with ANSD is similar to other reports. Miyamoto, Kirk Renshaw and Hussain (1999) reported a child with ANSD secondary to Friedreich ataxia who could not achieve open-set speech recognition with a cochlear implant. Rance et al. (1999) reported improved lip reading ability in a child who was implanted at age 3 yr, 9 mos; however, open-set recognition remained at chance level, and this child's electrical ABR did not indicate synchronous recordings.

One important goal for children with ANSD is the development and acquisition of usable language, promoted through auditory and visual methods. The extent to which a child depends

on visual communication is related to the extent to which that child benefits from auditory input. If the child can process auditory information, there will be less dependence on visual information. There are several visual communication approaches that have been incorporated into training programs for children with ANSD, including speech reading, English-based signs, and Cued Speech (Berlin et al., 1998, 2010; Hood, 1998b).

The present situation suggests that there does not appear to be one and only one correct or successful management plan for ANSD treatment, which gives further support to the contention that the ANSD group is heterogeneous; it is not one disorder, but rather a family of disorders. Recent investigations by McMahon et al. (2008), Santarelli and Arslan (2002), and Santarelli et al. (2008) are promising and may add new diagnostic information about CI benefits.

Similarly, management for CAPD must be appropriate for the specific auditory deficits. A comprehensive management plan includes three approaches: (1) environmental modifications to improve access to the auditory signal (e.g., FM system); (2) direct therapy to enhance perceptual auditory skills (i.e., auditory training); and (3) central resources training to develop compensatory language, cognitive, and metacognitive skills and strategies (Bellis, 2003; Chermak & Musiek, 1997). The particular emphases within and across these three approaches depends upon the specific clinical deficit profile (ASHA, 2005; Bellis, 2003). It is important to note that although some of these management approaches may be considered for both patients with CAPD and ANSD (i.e., FM systems and auditory training), CAPD and ANSD are distinct disorders, and management must be tailored to the behavioral deficits with recognition of the underlying physiologic differences that characterize these two disorders. See Volume 2 of the Handbook for extensive discussion of intervention for CAPD.

Summary

Although differential diagnosis of ANSD and CAPD is possible using current behavioral and electrophysiologic techniques, additional research is needed to distinguish more clearly the underlying physiologic deficit(s), perceptual correlates, and functional deficits of ANSD. Although patients with ANSD and CAPD present some similar complaints, these are distinct auditory disorders that can be distinguished by a comprehensive behavioral, electrophysiologic, and psychoacoustic test battery. The results of Zeng et al.'s (2001) work indicate the value of including psychoacoustic tests in the test battery that might enable the clinician to better understand a patient's communication difficulties. Furthermore, interpretation of a comprehensive battery can lead to inferences regarding the gross anatomical site and physiology of the auditory dysfunction and will provide valuable information to tailor auditory interventions. The effectiveness of treatment and management for these complex disorders depends on accurate and thorough diagnosis and assessment.

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CHAPTER 20

DIFFERENTIAL DIAGNOSIS OF CENTRAL AUDITORY PROCESSING DISORDER AND ATTENTION-DEFICIT/ HYPERACTIVITY DISORDER

GAIL D. CHERMAK and TERI JAMES BELLIS

Individuals diagnosed with attention-deficit/hyperactivity disorder (ADHD) frequently present with difficulties performing tasks that challenge the central auditory nervous system (CANS). Individuals diagnosed with central auditory processing disorder (CAPD) demonstrate central auditory dysfunction that often presents comorbidly with other valid diagnoses. In children, CAPD frequently co-occurs with ADHD, language impairment, and learning disability (Chermak, Hall, & Musiek, 1999). Tests with documented sensitivity and specificity for CANS dysfunction are *necessary* for a diagnosis of CAPD. Likewise, accurate diagnosis of ADHD depends on the sensitivity and specificity of the rating scales and/or continuous performance tests commonly used to infer the presence of ADHD. Multidisciplinary assessment, evaluating the role of higher order global, supramodal or pansensory, cognitive, attention, language, and related disorders, is essential for *differential* diagnosis (Bellis, 2003; Chermak & Musiek, 1997; Musiek, Bellis, & Chermak, 2005). Accurate knowledge of an individual's problems derived from multidisciplinary evaluations leads to the most effective intervention programs. In cases of comorbidity, CAPD and ADHD must be diagnosed fully and accurately and a treatment program must be developed and implemented by a team of professionals to address all significant functional deficits.

Spectrum of Comorbid Disorders

The association observed between attention deficits and performance on central auditory tests (Campbell & McNeil, 1985;

Cook, Mausbach, & Burd, 1993; Gascon, Johnson, & Burd, 1986; Pillsbury, Grose, Coleman, Conners, & Hall, 1995) has elicited suggestions of linkage between ADHD and CAPD. Some have questioned whether CAPD is a manifestation of impaired attention (Burd & Fisher, 1986; DeMarco, Harbour, Hume, & Givens, 1989; Robin, Tomblin, Kearney, & Hug, 1989) and whether CAPD and ADHD reflect a single developmental disorder (Cook et al., 1993; Gascon et al., 1986). Others have interpreted central auditory performance deficits among children with ADHD as a reflection of the co-occurrence or comorbidity of CAPD and ADHD (Keith & Engineer, 1991; Riccio, Cohen, Garrison, & Smith, 2005; Riccio, Hynd, Cohen, & Gonzales, 1993; Riccio, Hynd, Cohen, & Molt, 1996). For example, finding low correlations between performance on the Staggered Spondaic Word Test (SSW) (Katz, 1977) and behaviors characteristic of ADHD (i.e., inattention, hyperactivity, and impulsivity), Riccio et al. (1996) concluded that ADHD and CAPD are distinct entities that may nonetheless both involve deficits in auditory processing. Extending their earlier findings, Riccio et al. (2005) found no significant correlations between measures of attention (i.e., continuous performance test and rating scales for attention problems and hyperactivity) and measures of central auditory processing (i.e., the SSW and the Screening Test for Auditory Processing Disorders [SCAN; Keith, 1986]). Riccio and colleagues concluded that deficits in auditory processing may not necessarily be associated with ADHD or attention deficits. Despite overlapping clinical profiles and comorbidity, converging lines of evidence indicate that CAPD and ADHD are clinically distinctive entities (Chermak, Hall, & Musiek, 1999). The two disorders can be differentially diagnosed, leading to distinctive treatment and management strategies.

That central auditory performance deficits among children with ADHD may reflect the presence of CAPD rather than the ADHD per se is supported further by the frequently reported history of chronic otitis media in children with ADHD (Adesman, Altshuler, Lipkin, & Walco, 1990; Feagans, Sanyal, Henderson, Collier, & Appelbaum, 1987; Pillsbury et al., 1995; Roberts, Burchinal, Collier, Ramey, Koch, & Henderson, 1989; Silva, Kirkland, Simpson, Stewart, & Williams, 1982). The association between chronic otitis media and CAPD, with persistence of central auditory processing deficits even after resolution of the otitis media and return to normal hearing levels (Adesman et al., 1990; Brown, 1994; Ferguson, Cook, Hall, Grose, & Pillsbury, 1998; Gravel & Wallace, 1992; Hall & Grose, 1993, 1994; Hall, Grose, & Pillsbury, 1994, 1995; Hutchings, Meyer, & Moore, 1992; Jerger, Jerger, Alford, & Abrams, 1983; Moore, Hutchings, & Meyer, 1991; Pillsbury, Grose, & Hall, 1991; Silva, Chalmers, & Stewart, 1986) suggests that children with ADHD may experience central auditory performance deficits subsequent to chronic otitis media. Indeed, the frequently observed co-occurrence of CAPD and learning disability (Breedin, Martin, & Jerger, 1989; Chermak, Vonhof, & Bendel, 1989; Elliott & Hammer, 1988; Ferre & Wilber, 1986; Jerger, Martin, & Jerger, 1987; King, Warrier, Hayes, & Kraus, 2002; Kraus, McGee, Carrell, Zecker, Nicol, & Koch,1996; Purdy, Kelly, & Davies, 2002; Warrier, Johnson, Hayes, Nicol, & Kraus, 2004) and CAPD and language impairment

(Lubert, 1981; Marler, Champlin, Gillam, 2002; Sloan, 1980; Tallal, 1980a, 1980b; Tallal & Piercy, 1973a; Tallal, Stark, & Mellits, 1985; Tallal et al., 1996) have led to speculation that these deficits also may be causally related (Katz & Illmer, 1972; Keith, 1981; Knox & Roeser, 1980; Lubert, 1981; Miller, Kail, Leonard, & Tomblin, 2001; Sloan, 1980; Merzenich et al.1996; Rey, De Martino, Espesser, & Habib, 2002; Tallal, 1980a; Tallal & Piercy, 1973a; Tallal, Stark, & Mellits, 1985; Tallal et al., 1996). Temporal processing deficits have been linked to language and learning problems; however, this purported linkage is controversial (see, for example, Studdert-Kennedy & Mody, 1995; Bishop et al., 1999; Nittrouer, 1999). Finding perceptual deficits cutting across diagnostic categories, including children with diagnoses of learning disability, ADHD, and dyslexia, Kraus (2001) concluded that there is a common perceptual deficit in a subset of children with various clinical diagnoses.

[redacted]rain Organization [redacted]oerlies Comorbidity

The literature is replete with reports of individuals with concurrent diagnoses of CAPD, attention deficits, and learning disabilities (Cunningham, Nicol, Zecker, Bradlow, & Kraus, 2001; Katz, 1992; Keith, 1986; Keller, 1992; King, Warrier, Hayes, & Kraus, 2002; Kraus, 2001; Kraus, McGee, Carrell, Zecker, Nicol, & Koch, 1996; Musiek, Charette, Kelly, Lee, & Musiek, 1999; Newhoff, Cohen, Hynd, Gonzalez, & Riccio, 1992; Pillsbury et al., 1995; Purdy et al., 2002; Riccio et al., 1993, 1996; Warrier, Johnson, Hayes, Nicol, & Kraus, 2004; Wible, Nicol, & Kraus, 2002). The relationships among these comorbid conditions are complex and not completely understood. Tremendous gains in our understanding of brain organization and function, however, have provided insights regarding linkages and distinctions.

Comorbidity is the result of the complex organization of the brain that is temporally coupled across the cortex, modalities, and hemispheres (Merzenich, Shreiner, Jenkins, & Wang 1993). Although there may be some brain regions that are auditory specific, the brain's organization is predominantly nonmodular and nonexclusively segregated (Streitfeld, 1980). Neurons in so-called *auditory areas* may respond *primarily*, although not exclusively to auditory stimuli (Musiek, Bellis, & Chermak, 2005). Moreover, auditory neurons in the cerebrum exhibit interconnectedness with a variety of neurons in other nonauditory areas of the brain, including the limbic system, cingulate gyrus, hippocampus, and the frontal lobe (Streitfeld, 1980).

Additional areas of the brain that have been identified as auditory responsive include the amygdala, striatum, and frontal lobe (Bayazit, Oniz,Hahn, Gunturkun, & Ozgoren, 2009; Petacchi, Kaernbach, Ratnam, & Bower, 2011; Poldrack et al., 2001; Poremba, Saunders, Crane, Cook, Sokoloff, & Mishkin, 2003; Salvi, Lockwood, Frisina, Coad, Wack, & Frisina, 2002; Wong et al., 2009). Many of the auditory responsive and interconnected areas support attention, executive control, and motor regulation and are implicated in the underlying pathophysiology of ADHD (Castellanos, 1997; Sowell, Thompson, Welcome, Henkenius, Toga, & Peterson, 2003). In addition, central auditory lesions often extend beyond artificial

boundaries that are increasingly recognized as inaccurate reflections of true brain organization (Musiek et al., 2005). Even if auditory lesions were relatively circumscribed, the fact that most brain regions are not modality specific likely results in comorbid dysfunction in other systems due to shared neurophysiological substrates. Hence, the often reported comorbidity of CAPD and ADHD may be explained by shared physiologic and neurologic networks.

Neurobiological Correlates

Brain imaging studies and postmortem examinations of individuals with dyslexia, learning disabilities, ADHD, and normal controls have revealed morphologic and structural differences in auditory areas of the brain that are activated when listening to simple tonal complexes, language, and music (i.e., superior temporal gyrus, Heschl's gyrus, planum temporale, posterior portion of the insula, sulcus of the corpus callosum) (Galaburda & Kemper, 1978; Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990; Hynd, Semrud-Clikeman, Lorys, Novey, Eliopulos, & Lyytinen 1991). The accumulating data suggest a neurobiological basis for the often observed co-occurrence of CAPD, auditory attention deficits, dyslexia, and learning disabilities. At the same time, a number of studies also reveal activation patterns that may help distinguish CAPD from ADHD (e.g., Barry et al., 2008; Tannock, 1998; Tsai, Hung, & Lu, 2012).

Postmortem studies have documented brain abnormalities (e.g., nests of ectopic [misplaced] and underdeveloped cells) involving auditory regions of the brain in children with learning disabilities and dyslexia (Galaburda & Eidelberg, 1982; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Galaburda & Kemper, 1978). Brain imaging studies have revealed morphologic and structural differences in auditory areas, as well as motor regulation/behavioral inhibition areas (prefrontal lobes and striatum) of the brains of children with ADHD, as compared with the brains of normal children, implicating some deviation in normal brain development (Hynd & Semrud-Clikeman, 1989; Hynd et al., 1990; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Tannock, 1998; Voeller, 1991; Zametkin et al., 1990).

The corpus callosum has been reported to be smaller in children with ADHD relative to normal controls. These differences in corpus callosa between children with ADHD and normal controls appear to reflect both macro- and microstructural abnormalities, particularly in the posterior regions (Cao et al., 2010). In contrast, adults with ADHD may exhibit macrostructural abnormalities only (Dramsdahl, Westerhausen, Haavik, Hughdahl, & Plessen, 2012). It is important to note that the posterior regions of the corpus callosum have been shown to be critical for interhemispheric transfer of auditory information (Musiek & Reeves, 1986).

The morphology of Heschl's gyrus may also differ in children with ADHD, as compared with normal controls (Baumgardner et al., 1996; Castellanos et al., 1996; Giedd et al., 1994; Hynd et al., 1991; Semrud-Clikeman et al., 1994). The planum temporale has been found to be shorter in the left hemisphere, resulting in a reversed asymmetry (R>L), in subjects with dyslexia relative to normal controls (Hynd et al., 1990). Similarly,

the insular region of the brains of children with dyslexia is smaller bilaterally compared with normal controls (Hynd et al., 1990). Using high-resolution magnetic resonance imaging, Sowell et al. (2003) reported abnormal morphology and reduced size of the frontal cortices of children and adolescents with ADHD relative to normal controls. Prominent increases in gray matter in large portions of the posterior temporal and inferior parietal cortices of subjects with ADHD were also reported.

Morphological differences and possible dysfunction in areas of the brain associated with motor regulation and self-control (e.g., frontal region, caudate nucleus) suggest a neurobiological basis for co-occurring central auditory deficits and behavioral regulation problems in ADHD (Gallagher & Schoenbaum, 1999; Paus, 2000). Lou et al. (1989) reported decreased metabolism in the caudate nucleus associated with ADHD. Mann et al. (1992) found increased slow wave activity in the frontal regions and decreased beta activity in the temporal regions in boys with ADHD, compared with normal control subjects. Hynd et al. (1990) reported bilaterally smaller anterior cortexes in children with ADHD and dyslexia relative to a control group of children, reflecting significantly decreased right frontal lobe width. Positron emission tomography (PET) studies revealed widespread and bilateral reduction in glucose metabolism, in the premotor and superior frontal cortexes, as well as in the striatum and the thalamus (Zametkin et al., 1990). In addition, the children with dyslexia showed hemispheric symmetry in this region in contrast to the typical pattern of the right frontal lobe being larger than the left (Hynd et al., 1990). Giedd et al. (1994) concluded that anatomical differences in several regions of the corpus callosum support theories of abnormal frontal lobe development and function in ADHD. Sowell et al. (2003) suggested that abnormal morphology of the frontal cortices in children and adolescents with ADHD may underlie attention and behavioral inhibition problems.

Finally, event-related potential (ERP) studies document the neurobiological basis for the often-observed comorbidity between CAPD and ADHD. Compared with normal controls, individuals with ADHD have been shown to exhibit smaller amplitudes and prolonged latencies in the auditory P300, possibly reflecting the longer time and greater difficulty required to complete stimulus evaluation (Klorman, 1991). Also suggesting impairment in processing of auditory stimuli in ADHD are findings of abnormal auditory brainstem evoked responses (ABR) and abnormalities in the N1, N2 of the auditory late response (ALR) and the auditory P300 (Jonkman et al., 1997; Lahat et al., 1995). Tsai et al. (2012) found increased latency of P300 suggesting slower processing speed, and decreased P300 amplitude, which they interpreted as disruption of inhibitory control, in children with ADHD. Similarly, children and adults diagnosed with CAPD and/or lesions of the CANS present prolonged latencies and decreased amplitudes in the auditory P300 (Jirsa & Clontz, 1990; Krishnamurti, 2001; Musiek, Baran, & Pinheiro, 1992), increased ABR latencies (Musiek & Lee, 1995), and other evoked potential abnormalities (e.g., middle-latency response [MLR]; (Musiek, Baran, & Pinheiro, 1994). (See Maerlender and Heath's Chapter 18 in Volume 2 of the Handbook for additional review of the neurobiology of ADHD.)

Central Auditory Processing Disorder: An Overview

CAPD results from difficulties in the perceptual processing of auditory information in the central nervous system and the associated changes in the neurobiologic activity that underlies those processes and gives rise to the electrophysiologic auditory potentials (AAA, 2010; ASHA, 2005). In some cases, neurobiologic dysfunction may involve interhemispheric transfer deficits, lack of appropriate hemispheric lateralization, reversed hemispheric asymmetries, or imprecise synchrony of neural firing (Bellis & Wilber, 2001; Bellis, Nicol, & Kraus, 2001; Jerger et al., 2002; Kraus, McGee, Carrell, Zecker, Nicol, & Koch, 1996; Moncrieff, Jerger, Wambacq, Greenwald, & Black, 2004). CAPD also may coexist with more global dysfunction that affects performance across modalities (e.g., attention deficit, neural timing deficit, language representation deficit) (AAA, 2010; ASHA, 2005; Chermak & Musiek, 1997). CAPD has been observed in diverse clinical populations, including those where central nervous system (CNS) pathology or neurodevelopmental disorder is suspected (e.g., developmental language disorder, dyslexia, learning disability, attention deficit disorder) and those where evidence of CNS pathology is clear [e.g., aphasia, multiple sclerosis, epilepsy, traumatic brain injury, tumor, and Alzheimer's disease] (ASHA, 1996). CAPD has also been observed in older adults, presumably due to nonpathologic neurologic changes associated with aging (Committee on Hearing, Bioacoustics and Biomechnaics [CHABA] Working Group on Speech Understanding and Aging, 1988; Gulya, 1991; Stach, Spretnjak, & Jerger, 1990). Atypical interhemispheric transfer of auditory information may be a factor contributing to the listening difficulties seen in some children and in aging adults (Bellis, Nicol, & Kraus, 2000; Bellis & Wilber, 2001; Chmiel & Jerger, 1996; Chmiel, Jerger, Murphy, Prozzolo, & Tooley-Young, 1997; Jerger, 1997; Jerger, Moncrieff, Greenwald, Wambacq, & Seipel, 2000; Jerger et al., 2002; Musiek, Gollegly, & Baran, 1984, Musiek, Pinheiro, & Wilson, 1980). Additional age-related changes in the CANS include less synchrony and time-locking, slower refractory periods, and decreased inhibition (Pichora-Fuller & Souza, 2003; Tremblay, Piskosz, & Souza, 2003; Willot, 1999; Woods & Clayworth, 1986).

A CAPD manifests as a deficit in one or more of the following behaviors: sound localization and lateralization; auditory discrimination; auditory pattern recognition; temporal processing (e.g., temporal resolution, temporal masking, temporal integration, and temporal ordering); auditory performance with competing acoustic signals; and auditory performance with degraded acoustic signals (AAA, 2010; ASHA, 2005; Chermak & Musiek, 1997). Characteristically, patients with CAPD have difficulty comprehending spoken language in competing speech or noise backgrounds and in reverberation (Chermak & Musiek, 1997; Hornickel & Kraus, 2011; Song, Skoe, Bania, & Kraus, 2011; Wible, Nicol, & Kraus, 2002). Children with CAPD ask frequently for repetitions, often misunderstand messages, have difficulty paying attention, have trouble following complex auditory directions or commands, and difficulty localizing sound (Bellis, 2003; Chermak & Musiek, 1997).

CAPD is diagnosed on the basis of performance on a battery of auditory tests,

which may include electrophysiological as well as behavioral procedures, administered under acoustically controlled conditions (AAA, 2010; ASHA 2005; Chermak & Musiek, 1997; Jerger & Musiek, 2000). The sensitivity and specificity of behavioral and electrophysiologic central auditory tests and procedures recommended for inclusion in the test battery (see Chapter 12) have been established on patients with known lesions of the CANS (e.g., Aharonson, Furst, Levine, Chaigrecht, & Korczyn, 1998; Bamiou et al., 2006; Baran, Musiek, & Reeves, 1986; de Bode, Sininger, Healy, Mathern, & Zaidel, 2007; Furst et al., 2000; Hendler, Squires, & Emmerich, 1990; Jerger et al., 2002; Karlsson & Rosenhall, 1995; Meyers, Roberts, Bayless, Volkert, & Evitts, 2002; Musiek, Shinn, Jirsa, Bamiou, Baran, & Zaidan, 2005b; Rappaport Gulliver, Phillips, van Dorpe, Maxner, & Bhan, 1994).

Prevalence data for CAPD are sparse, particularly for children. Estimates of CAPD in children range from 2% to 7%, with a 2:1 ratio between boys and girls (Bamiou, Musiek, & Luxon, 2001; Chermak & Musiek, 1997; Musiek, Gollegly, Lamb, & Lamb, 1990). Estimates of CAPD in older adults range from 23% to 76% for community-based samples (Cooper & Gates,1991; Golding, Carter, Mitchell, & Hood, 2004) and 70% in a clinical sample of patients over age 60 years (Stach et al., 1990).

Attention Deficit Hyperactivity Disorder: An Overview

ADHD is characterized as the most common neurobehavioral disorder of childhood (Barkley, 1998). ADHD consists of a persistent pattern of inattention and/or hyperactivity and impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development and manifests in multiple settings (e.g., school, home); interferes with developmentally appropriate social, academic, or occupational functions; and presents several symptoms prior to age 12 years (APA, 2013).

Patterns of inattention, hyperactivity, and impulsivity are used to differentiate ADHD into three subtypes. The predominantly inattentive type presents primary symptoms of inattention (APA, 2013). The predominantly hyperactive-impulsive type is considered a behavioral regulation disorder (APA, 2013; Barkley, 1994, 1998). The combined type is characterized by hyperactivity-impulsivity (i.e., behavioral regulation disorder) and inattention (APA, 2013). Different neuroanatomical loci are posited for the different subtypes. The combined and predominantly hyperactive-impulsive types might arise from problems in the prefrontal-limbic pathways, particularly the striatum (Lou et al., 1989). The predominantly inattentive type might involve more posterior associative cortical areas and/or cortical and subcoortical feedback loops, perhaps involving the hippocampal system (Heilman, Voeller, & Nadeau, 1991; Hynd, Lorys, Semrud-Clikeman, Nieves, Huettner, & Lahey, 1991).

According to the *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. (DSM-5), impulsivity is characterized by blurting out answers, failing to take turns, and interrupting or intruding on others. Hyperactivity is characterized by fidgeting with hands or feet or squirming in seat, difficulty remaining seated, running or climbing excessively in inappropriate contexts, difficulty engaging

in quiet activity, constantly moving or engaging in activity, and talking excessively. Inattention in ADHD is marked by difficulties maintaining focus, sustaining and shifting attention, concentrating, listening when spoken to directly even in the absence of any obvious distraction (e.g., competing noise), following through and completing tasks, engaging in tasks requiring sustained mental effort and persistence, organizing tasks, and ignoring extraneous stimuli, and is associated with careless mistakes in school work, losing things, and forgetfulness.

There are no empirical markers that identify ADHD. Diagnosis of ADHD is based on observational criteria defined as a cluster of behaviors involving impaired attention and distractibility, impulsivity, and hyperactivity (AAP, 2000; APA, 2013). The limited sensitivity and specificity of the rating scales used to identify ADHD can lead to misdiagnosis and overdiagnosis, possibly identifying some children with ADHD when in fact their primary deficit is CAPD (Stein, 2001).

Many epidemiologic studies have been conducted to determine the prevalence of ADHD in children and adolescents, with estimates ranging from 2% to 14%. Based on their review of these studies, Scahill and Schwab-Stone (2000) concluded that the best estimate appears to be 5 to 10%. The prevalence of ADHD is higher among boys, with estimates ranging between 2:1 and 9:1, varying as a function of the age range sampled (Scahill & Schwab-Stone, 2000). The prevalence of ADHD is stable in girls across the range of 10 to 20 years; however, an age-related decline in ADHD is seen among boys (Scahill & Schwab-Stone, 2000). Nonetheless, the DSM-5 notes that a significant number of individuals diagnosed with this disorder as children continue to experience this disorder as adults.

Reconceptualization of ADHD

The recent shift in conceptualizing ADHD as a behavioral regulation disorder rather than a primary attention disorder differentiates CAPD and ADHD (Chermak & Musiek, 1997). Symptoms of impulsivity and behavioral disinhibition are considered the result of neurologically based developmental deficiencies in the regulation and maintenance of behavior by rules and consequences (Barkley, 1998). Deficits in rule-governed behavior, perhaps resulting from elevated arousal thresholds (Zentall, 1985) or elevated reinforcement thresholds (Haenlein & Caul, 1987), lead to problems initiating, inhibiting, or sustaining responses to tasks or stimuli (Barkley, 1998), which heretofore had been considered characteristics of attention deficits. Deficits in rule-governed behavior lead to problems in executive functioning and self-regulation (Barkley, 1998). Consistent with this reconceptualization, ADHD is seen, essentially, as a motivational deficit, rather than an attention deficit (Barkley, 1994, 1998). This reconceptualization of ADHD as one of poor rule-governed behavior may also explain the self-control problems, social skill deficits, and language disorders (e.g., difficulty topic switching, turn taking, and sustaining dialogue) so frequently observed in ADHD (Augustine & Damico, 1995).

Differentiating CAPD and ADHD

Modeling Information Processing

Insofar as attention is essential to higher level processing, poor attention can com-

promise listening. Musiek and Chermak (1995) proposed that viewing the relationship between attention and auditory processing within the top-down and bottom-up information processing models provides a theoretical framework that clarifies the nature of the relationship between ADHD and CAPD. The inability to sustain sufficient attention to auditory stimuli might cause (i.e., top-down) auditory processing deficits; conversely, deficient auditory processing (i.e., bottom-up) might impair attention (Chermak & Musiek, 1997). Understanding the relationship between the attention deficits of ADHD and CAPD hinges on the interaction between perception and higher level cognitive processing (Chermak & Musiek, 1997). Most germane is whether an auditory processing deficit causes some attention deficit (as occurs in CAPD) or whether a more global attention deficit impedes auditory processing (as occurs in ADHD).

Consistent with a bottom-up model, attention is driven by incoming sensory stimulation and garnered by properly integrated and processed sensory stimuli (Chermak & Musiek, 1997; Musiek & Chermak, 1995). If acoustic stimuli are not properly processed, as occurs in CAPD, then optimal attention cannot be focused on these stimuli in a timely manner (Phillips, 1990). Attention deficits are seen as secondary to auditory perceptual processing deficits within the framework of a bottom-up model (Chermak & Musiek, 1997). In contrast, CAPD would be seen as a manifestation of a global attention deficit within a top-down information processing model (Chermak & Musiek, 1997). The literature indicates, however, that a bidirectional interaction between central auditory processing and attention is necessary for optimal listening comprehension. Attention modulates auditory processing, as is seen in the classical cocktail-party phenomenon whereby selective attention improves perception of high-priority stimuli in the environment at expense of other less relevant stimuli. Focusing attention to a given acoustic feature not only increases neural activity level, it also enhances neuronal selectivity to that feature in the particular part of the auditory cortex specialized in processing it (Kauramaki, Jaaskelainen, & Sams, 2007). In addition to modulating auditory processing at cortical levels, attention also modulates auditory processing at subcortical levels. For example, attention influences that cue in the time/intensity trading relationship receives greater weighting for localization (Lang & Buchner, 2008). However, sensory stimulation also drives attention in that the auditory sensory system imposes bottom-up environmental constraints on attention (e.g., loud noises or one's name garners attention) (Broadbent, 1978; Cherry, 1953). Moreover, central changes are contingent on sensory function and listening experience (Moore, 1993). For example, cortical reorganization has been observed in young and adult mammals following induced cochlear lesions (Harrison et al., 1992; Irvine et al., 1992; Kilgard et al., 2001; Rajan & Irvine, 1998; Rajan, Irvine, Wise, & Heil, 1993; Robertson & Irvine, 1989; Schwaber, Garraghty, & Kaas, 1993; Willott, Aitkin, & McFadden, 1993). The known interactions between the auditory pathway and the reticular activating system at the brainstem level provide support for the bottom-up view of attention deficits (Koch, 1999). Therefore, one can conceive of the bidirectional relationship as top-down, attentional processes imposing constraints in *deciding* what is relevant and deserving of attention, whereas bottom-up perceptual pro-

cesses drive the processing required to resolve the perceptual and/or language detail required for optimal listening comprehension.

Consistent with a bottom-up perspective, listening difficulties seen in CAPD result from primarily auditory perceptual deficiencies rather than global attention deficits (ADHD predominantly inattention subtype) or behavioral regulation deficits (combined ADHD and predominantly hyperactive-impulsive ADHD subtypes) (Phillips, 1990, 1995; Tallal et al., 1996). As outlined in Table 20–1, CAPD is considered an input disorder that impedes selective and divided auditory attention. The combined and predominantly hyperactive-impulsive ADHD subtypes are seen as output disorders in response programming and execution that indirectly cause sustained attention deficits across modalities. Dual diagnoses of both CAPD and ADHD may result, therefore, from comorbid attention deficits at different levels and primacy of sensory and global dysfunction (Chermak & Musiek, 1997).

Defining Attention and Types of Attention Deficits

Inattention is symptomatic of many psychiatric and medical conditions (Riccio, Reynolds, & Lowe, 2001). Symptoms of inattention unrelated to poor self-control (e.g., ADHD predominantly inattentive subtype) poses a particular diagnostic challenge (Chermak, Hall, & Musiek,

Table 20–1. Differentiating Attention Deficits in Attention Deficit Hyperactivity (ADHD) Disorder and Central Auditory Processing Disorder (CAPD)

ADHD Combined and Predominantly Hyperactive-Impulsive Subtypes
• Output (Behavioral Regulation) Disorder
• Sustained Attention (Vigilance) Deficit Secondary to Behavioral Disinhibition and Self-Regulation Deficit
• Executive Dysfunction Primary Source of Disorder
ADHD Predominantly Inattentive Subtype
• Input (Processing) Disorder
• Global (Supramodal) Attention Deficit
• Selective (Focused) Attention Deficit
• Reduced Speed of Information Processing Primary Source of Disorder
CAPD
• Input (Processing) Disorder
• Primarily Auditory Modality Specific Perceptual Deficit
• Binaural Separation and Binaural Integration Deficits
• Executive Dysfunction as Secondary Source of Listening Problems

1999). In such cases, clinicians must rule out a range of possible disorders, including anxiety, depression, obsessive-compulsive disorder, learning disabilities, and CAPD. Although attention deficits may be seen to characterize CAPD and ADHD (particularly the combined and predominantly inattentive subtypes), distinctions can be drawn regarding the *nature* of the inattention observed in the two disorders.

Consistent with the reconceptualization of ADHD as a behavioral regulation disorder, inattention in individuals with the hyperactive-impulsive and combined ADHD subtypes is reflected in behaviors of disorganization, distractibility, and lacking persistence (Barkley, 1994, 1998). Inattention attributed to individuals with the predominantly inattentive subtype of ADHD is reflected in their passive, sluggish, and daydreamy behavior (Barkley, 1998). They seem not to listen when spoken to even in the absence of any obvious distraction (e.g., competing noise). In contrast, individuals with CAPD are not truly inattentive. Rather, their auditory deficits result primarily from difficulties in binaural separation in the presence of competition (related to auditory selective [focused] attention) and binaural integration (related to auditory divided attention) (Chermak & Musiek, 1997; Musiek et al., 2005). These auditory perceptual deficits lead to difficulties understanding spoken language in competing noise and reverberant backgrounds, misunderstanding messages, and difficulty following directions in individuals with CAPD (Chermak & Musiek, 1997). As outlined in Table 20–1, the attention deficits of ADHD typically are pervasive and supramodal, impacting more than one sensory modality (AAP, 2000; APA, 2013; Keller, 1992). In contrast, individuals with CAPD experience attention deficits that may be restricted to the auditory modality or are at least more pronounced in the auditory modality (ASHA, 2005; Chermak & Musiek, 1997; Musiek et al., 2005).

Different types of attention deficits may be seen in ADHD and CAPD (Chermak & Musiek, 1997). Although the neural mechanisms underlying the different behaviors associated with various attention tasks are not fully known, research suggests that attention deficits associated with the combined and predominantly hyperactive-impulsive ADHD subtypes may be restricted to sustained attention, albeit in multiple modalities (Barkley, 1997a, 1997b; Hooks, Milich, & Lorch, 1994; Seidel & Joschko, 1990). Selective (multimodal) attention and speed of information processing deficits may characterize the predominantly inattentive ADHD subtype (Barkley, 1997a). Selective (focused) and divided auditory attention deficits characterize CAPD (Cherry, 1980; Jerger & Jerger, 1984; Katz & Illmer, 1972; Keith, 1986; Lasky & Tobin, 1973) (see Table 20–1). Inclusion of tests of sustained attention (vigilance) in the central auditory test battery should prove helpful in substantiating the distinction discussed above regarding types of attention deficits (i.e., sustained, focused, and divided attention), aiding the differential diagnosis of CAPD and ADHD (Chermak, 2004; McPherson & Salamat, 2004; Riccio et al., 2001; Salamat & McPherson, 1999).

Differentiating Behavioral Profiles

Despite some overlapping symptomatology, clinicians seem able to distinguish behavioral profiles for CAPD and ADHD. Chermak, Somers, and Seikel (1998)

found that pediatricians and audiologists view the predominant symptoms of ADHD and CAPD as being rather distinct, with only two (i.e., inattention and distractibility) of the eleven most frequently cited behaviors reported as common to both conditions (Table 20–2). Inattention and distractibility were ranked as the first and second most typical behaviors characterizing ADHD. Audiologists ranked these same behaviors as seventh and sixth, respectively, in cases of CAPD. CAPD was characterized by a selective attention deficit and associated language processing and academic difficulties; ADHD was characterized by inappropriate motor activity, restlessness, and socially inappropriate interaction patterns. In a follow-up study, Chermak, Tucker, and Seikel (2002) found that pediatricians and audiologists can distinguish the primary symptoms of the predominantly inattentive subtype of ADHD and CAPD. None of the four behaviors ranked two standard deviations above the grand means (i.e., inattention, academic difficulties, asking for things to be repeated, and poor listening skills) was ranked in common (Table 20–3).

Other investigators have reported that behavior problems, such as difficulty waiting one's turn and playing quietly and excessive talking, more often characterize children with ADHD than CAPD (Newhoff et al., 1992). Similarly, severe socioemotional sequelae (i.e., conduct disorders, juvenile delinquency) are more common among children with ADHD (Newhoff et al., 1992). Interestingly, the predominantly inattentive ADHD subtype shares little if any comorbidity with disruptive behavior disorders, in contrast to the predominantly hyperactive-impulsive and combined ADHD subtypes (Barkley,

Table 20–2. Rank Order of Behavioral Means Greater Than One Standard Deviation Above the Respective Grand Mean

ADHD		CAPD	
1. Inattentive	4.36	1. Difficulty hearing in background noise	4.40
2. Distracted	4.27	2. Difficulty following oral instructions	4.20
3. Hyperactive	4.14	3. Poor listening skills	4.10
4. Fidgety or restless	4.14	4. Academic difficulties	4.00
5. Hasty or impulsive	4.14	5. Poor auditory association skills	3.75
6. Interrupts or intrudes	3.86	6. Distracted	3.70
		7. Inattentive*	3.55
Grand mean	3.25	Grand mean	2.90
Standard deviation	0.55	Standard deviation	0.66

*Note that inattentive was included based on "evens-down/odds up" rounding rule; the standard deviation was .01 points below the criteria of +1 SD of the grand mean.

Source: "Behavioral signs of central auditory processing disorder and attention deficit hyperactivity disorder," by G. D. Chermak, E. K. Somers, and J. A. Seikel, 1998, *Journal of the American Academy of Audiology*, *9*, 78–84. Copyright 1998 American Academy of Audiology. Reproduced with permission.

1997a). Wilens, Biederman, and Spencer (2002) noted that individuals with ADHD combined subtype are most impaired, presenting more comorbid psychiatric diagnoses and more substance abuse disorders. Individuals with ADHD combined subtype and ADHD predominantly inattentive subtype experience greater academic problems, while individuals with ADHD predominantly inattentive subtype experience fewer emotional and behavioral problems than other subtypes (Millstein, Wilens, Biederman, & Spencer, 1997; Wilens et al., 2002).

The distributed nature of information processing and underlying brain activation explains overlapping behavioral deficits; however, overlapping behavioral profiles do not necessarily implicate common antecedents. The sustained attention problems observed in ADHD probably result from a supramodal, cognitive deficit, reflecting deficiencies in behavioral regulation rather than attention

Table 20–3. Rank Order of Behavioral Means Greater Than One and Two (*) Standard Deviations Above the Respective Grand Mean

ADHD-PI	AVG.	CAPD	AVG
Inattentive	4.45*	Asks for things to be repeated	4.39*
Academic difficulties	4.22*	Poor listening skills	4.39*
Daydreams	4.05	Difficulty following instructions given orally	4.33
Distracted	4.04	Difficulty hearing in background/ambient noise	4.28
Poor listening skills	3.86	Academic difficulties	4.22
Disorganized	3.82	Distracted	3.78
Asks for things to be repeated	3.70	Reduced rate of information processing	3.78
Auditory divided attention deficit	3.67	Auditory divided attention deficit	3.76
Difficulty hearing in background/ambient noise	3.62	Auditory selective attention deficit	3.76
		Auditory sustained attention deficit	3.71
		Poor memory	3.67
		Difficulty discriminating speech	3.65
Grand mean	3.11	Grand mean	2.93
Standard deviation	0.50	Standard deviation	0.72

Source: Reprinted with permission from "Behavioral characteristics of auditory processing disorder and attention deficit disorder," by G. D. Chermak, E. Tucker, and J. A. Seikel, 2002. *Journal of the American Academy of Audiology, 13,* 332–338. Copyright 2002 American Academy of Audiology.

per se. In contrast, the selective and divided auditory attention deficits of CAPD result from deficient auditory perceptual processing. Similarly, difficulty following directions is commonly observed among individuals with ADHD and CAPD; however, deficiencies in rule-governed behavior may underlie these difficulties in ADHD, whereas deficient central auditory processing of auditory signals may underlie the same performance deficit in CAPD. Executive dysfunction, as discussed below, may also underlie the resemblance in clinical profiles seen across CAPD and ADHD.

Summary

While additional research is needed to clarify differences in the nature and type of attention deficits observed in ADHD and CAPD, it is clear that the clinical inattention profiles differ significantly. The inattention profile of ADHD involves difficulty initiating, tracking, and remembering tasks (APA, 2013), in addition to sustaining allocation of attentional resources. The focused and divided attention deficits that characterize CAPD impact monaural and binaural separation (i.e., selective attention), and binaural integration (i.e., divided attention) tasks (Chermak & Musiek, 1997). Most important, the inattentiveness seen in CAPD is a primary deficit resulting from an input or information processing deficit. In contrast, the hyperactive-impulsive and combined ADHD subtypes are characterized as output or response programming and execution disorders (Barkley, 1997a, 1997b). Behavioral disinhibition ultimately results in poor goal-directed persistence and defective resistance to distraction subsequent to poor self-regulation and executive control of behavior (Barkley, 1997a, 1997b; Goodyear & Hynd, 1992). Consistent with this conceptualization, inattention is a secondary deficit in the combined and predominantly hyperactive-impulsive ADHD subtypes (Barkley, 1997a, 1997b). As elaborated above, the ADHD inattention profile may implicate a primary executive control deficit, rather than an attention deficit per se. Differentiating the predominantly inattentive ADHD subtype from CAPD is more challenging, since the inattention in both disorders is considered to be a primary input or information processing deficit. Notwithstanding the challenge, differential diagnosis of CAPD and the supramodal, predominantly inattentive ADHD subtype can be accomplished using a sensitized test battery of the CANS in combination with a comprehensive multidisciplinary evaluation. (See Test Batteries and Testing Strategies.)

Executive Function

Executive functioning provides a construct useful in understanding a wide range of symptoms observed across many disorders with overlapping clinical profiles (Pennington, Bennetto, McAleer, & Roberts, 1996), including ADHD and CAPD. Because executive functions place significant demands on attention (both *sustained* and *selective attention* to enable sensory and perceptual processing of events) and memory to register, store, and make knowledge and experience available to the individual (Barkley, 1996; Butterfield & Albertson, 1995; Pennington, Bennetto, McAleer, & Roberts, 1996),

executive dysfunction may be related to deficits characterizing ADHD and CAPD.

Executive function is a component of metacognition that refers to a set of general control processes that ensure that an individual's behavior is adaptive, consistent with some goal, and beneficial to the individual (Borkowski, Milstead, & Hale, 1988; Brown, Bransford, Ferrara, & Campione, 1983; Denckla, 1996; Sternberg, 1985; Torgesen, 1996). Executive function involves judgment, planning, and organization to self-regulate, self-control, and accomplish goals to maximize future outcomes. Executive control processes coordinate knowledge (i.e., cognition) and metacognitive knowledge in support of task analyses, planning, and reflective decision making, ultimately transforming this knowledge into behavioral strategies (Barkley, 1996; Butterfield & Albertson, 1995). They are crucial to the execution of novel behavioral sequences; learning and problem solving; psychosocial function, including self-image and self-regulation of emotion and motivation; and goal-directed behaviors, including listening (Borkowski & Burke, 1996; Grattan, Bloomer, Archambault, & Eslinger, 1994; Grattan & Eslinger, 1992). Similar to the role working memory serves in supporting central auditory processing, so too, working memory and executive function may be inextricably interdependent, perhaps sharing a common underlying component (McCabe, Roediger, McDaniel, Balota, & Hambrick, 2010). Executive function may be assessed by a variety of procedures and tests, as noted later in the chapter.

Synchronized activation across multiple cortical and subcortical regions, including the frontal lobe, temporal lobe, parietal lobe, basal ganglia, and thalamus, subserves executive functioning (Eslinger & Grattan, 1993; Goldenberg, Oder, Spatt, & Podreka, 1992). Many of the neural networks thought to underlie executive function follow a prolonged course of postnatal development, extending into adolescence and perhaps continuing into adulthood (St. James-Roberts, 1979; Thatcher, 1991; Yakovlev & Lecours, 1967); therefore, the system is highly vulnerable to disruption from a variety of causes, including neurobiological stressors, as well as environmental deprivation (Barkley, 1996).

Executive function deficits have been described in a wide variety of clinical populations, often in association with brain disease or injury, and may underlie childhood neurological disorders, in particular the academic problems experienced by children with learning disabilities or ADHD (Denckla, 1996; Fletcher, Taylor, Levin, & Satz, 1995; Graham & Harris, 1996; Pennington, 1991; Stanovich, 1986; Torgesen, 1994). Executive function deficits also have been identified in children who do not meet eligibility criteria for learning disabilities or ADHD but experience significant difficulties in school (Denckla, 1989). The prevalence of CAPD in the latter group of children has not been determined (Chermak & Musiek, 1997).

Linkages among executive function, rule-governed behavior, and self-control (Hayes, Gifford, & Ruckstuhl, 1996) have led to suggestions that executive dysfunction is the source of the behavioral regulation and inattention problems, as well as the language problems exhibited in ADHD (Barkley, 1994; Denckla & Reader, 1993; Smith, Gould, Marsh, & Nichols, 1995; Tannock & Schachar, 1996). Recognizing that pragmatic and metacogni-

tive behaviors associated with communication are both language based and rule governed, Westby and Cutler (1994) reasoned that executive dysfunction also may explain language deficits in ADHD, such as poor topic maintenance, inappropriate topic switching, poor problem solving, and difficulty producing coherent extended discourse such as stories and expository texts (Heyer, 1995), as well as contribute to the pragmatic problems observed in individuals with ADHD, which include excessive talking, interrupting others, blurting out answers, difficulty waiting one's turn, and difficulty negotiating peer interactions.

Although executive dysfunction in CAPD has not been examined fully, working memory deficits, a key component of executive function, have been reported in children with CAPD due to PAX6 mutations with abnormalities of the interhemispheric pathway and central auditory processing deficits indicative of reduced auditory interhemispheric transfer (Bamiou et al., 2007). Moreover, working memory supports auditory processing, including localization, pattern processing, dichotic listening, and speech recognition in noise (Akeroyd, 2008; Jancke & Shah, 2002; Martinkauppi et al., 2000; Salvi et al., 2002; Wong et al., 2009; Zatorre, Belin, & Benhune, 2002); therefore, it is reasonable to expect that auditory perceptual deficits impede operation of executive functions (Chermak & Musiek, 1997). Difficulty organizing, monitoring, and understanding acoustic signals may reflect limited use of executive function. In contrast to the proposed causal role of executive dysfunction in ADHD (Barkley, 1994; Denckla & Reader, 1993; Smith et al., 1995), and consistent with a bottom-up processing model, executive dysfunction in CAPD would be considered a secondary feature, not a primary cause, of listening difficulties (Chermak & Musiek, 1997). These secondary deficits could compound auditory processing deficits, impede generalization of strategic listening behaviors across settings, and thereby jeopardize treatment effectiveness and efficacy (Borkowski & Burke, 1996; Chermak & Musiek, 1997).

Differential Diagnosis of ADHD and CAPD

The Challenge

All auditory tasks, from pure tone detection to spoken language processing, are influenced by higher order, nonmodality-specific factors such as attention, memory, motivation, and decision processes, and the underlying multimodal, cross-modal, and supramodal neural interfaces supporting performance of these behavioral tasks (Chermak, Hall, & Musiek, 1999; Chermak & Musiek,1997; Musiek et al., 2005). For example, listening in noise activates auditory and nonauditory areas of the brain, including areas involved in attention, executive control, working memory, language processing, and motor planning (Salvi et al., 2002; Wong et al., 2009). Moreover, cognitive processes undergird basic perceptual events, as demonstrated by the integral role of working memory in numerous auditory processes, including localization, temporal resolution, and pattern recognition (Jancke & Shah, 2002; Marler, Champlin, Gillam, 2002; Martinkauppi, Rama, Aronen, Korvenoja, & Carolson, 2002; Wong et al. 2009; Zattore et al., 2002).

Indeed, the complex organization of the brain, involving interactive and interfacing sensory, cognitive, and linguistic networks, requires testing methods and careful interpretation of test outcomes that reduce the potential confound of factors not under direct examination in behavioral testing for CAPD.

The differential diagnostic challenge is to distinguish between two or more conditions presenting with similar symptoms or attributes and to disentangle impairments due to comorbid conditions. In the context of ADHD and CAPD, the clinician must determine whether there is an auditory component to the ADHD profile (or conversely whether there is an attention component to the CAPD profile). Furthermore, one must ask whether one condition is responsible for the symptoms seen in the other (i.e., diagnostic overshadowing). Clearly, differential diagnosis is exceedingly important, can be quite challenging, and requires multidisciplinary and comprehensive assessment.

McFarland and Cacace (1995; Cacace & McFarland, 2005) discussed three categories of individuals who perform poorly on tests of auditory function: (1) those who represent CAPD in its *purest* form and perform poorly solely on auditory tests; (2) those who exhibit auditory perceptual problems that coexist with other specific processing problems and, thus, present with a mixed or comorbid pattern of deficits; and (3) those who perform poorly on auditory tests because of a global, supramodal problem involving, cognition, attention, language, memory, or related skills, and who perform poorly on both auditory and visual tasks. CAPD is a deficit in neural processing of acoustic stimuli that *is not the result of* dysfunction in other modalities (AAA, 2010; ASHA 2005). It would be inappropriate to apply the label of CAPD to listening difficulties exhibited by individuals with higher order, global, supramodal, or pansensory disorders (e.g., ADHD, autism, cognitive delay) *unless* a comorbid deficit in the CANS is documented. By combining multidisciplinary evaluation along with tests of central auditory function that have been demonstrated to be both sensitive and specific for disorders of the CANS, it is possible to differentiate the auditory deficits present in individuals falling into the first two categories while, at the same time, *ruling out* CAPD in those individuals who fall into the third category (Musiek, Bellis, & Chermak, 2005).

Assessing Central Auditory Processing in Children With ADHD

Audiologic assessment of children with ADHD is clinically challenging. Valid behavioral measurement of auditory status requires that the child willingly cooperate in the assessment, understand the instructions, and attend to the task. Each of these requirements may be compromised in the ADHD population. The likelihood of successfully assessing CANS function in children with ADHD or suspected ADHD is enhanced by several practical modifications in the test strategy. However, when behavioral audiometric findings remain incomplete, inconclusive, or invalid despite the implementation of these modifications, one must rely more on electrophysiologic techniques (Chermak, Hall, & Musiek, 1999; Chermak & Musiek, 1997). Peripheral auditory function should be evaluated thoroughly prior to the central auditory evaluation.

Perhaps the single most important step in successful audiologic assessment of the child with diagnosed and medically managed ADHD is to ensure that the child received an effective dose of medication immediately before the test session (Chermak, Hall, & Musiek, 1999).[1] Although this statement seems obvious, it is important to specifically instruct caregivers to follow the typical school day routine for medication on the day of the audiologic assessment. Some might argue that audiologic assessment should be conducted with the child in his or her natural state, for example, without medication. Others might express concerns about the possible confounding effects of the medication on test performance. Clinical experience suggests at least three responses to these arguments. First, if the child regularly is given medication on school days, then following the prescribed medication schedule will result in a typical state. Second, there is no evidence that the medications used in management of ADHD (e.g., Adderall, Ritalin, Strattera) have any influence on the peripheral or central auditory nervous system functioning. Finally, for children with diagnosed ADHD who are treated medically, valid audiologic assessment would rarely be possible without medication. Therefore, it is advisable to verify that the child received appropriate medication on the test day. In addition, the central auditory diagnostic session is best scheduled to begin first thing in the morning (e.g., 8:30 or 9:00 AM) to avoid late-day fatigue and/or decreasing effects of nonmedications that do not provide a long-term, steady release. Frequent breaks during which the child is given the opportunity to move about should be provided during the test session. The child should be asked to repeat or paraphrase instructions and be given practice items. Furthermore, the child should be offered a verbal contract to discourage hyperactive and impulsive behavior and the child should be given positive reinforcement (e.g., frequent praise for his or her efforts) to enhance motivation (Keller & Tillery, 2002).

Test Batteries and Testing Strategies

The overall objective of the central auditory test battery is to determine whether the individual has a deficit in one or more of the central auditory processes. Demonstration of distinctive patterns across multidisciplinary tests helps distinguish CAPD from supramodal cognitive, language-based, and/or supramodal attention deficits. These patterns are derived from comparison of performance on behavioral tests of central auditory function and neurophysiologic results from auditory-evoked potentials with behavioral and neurophysiologic measures of other sensory, language, and cognitive systems (Bellis, 2003; Bellis & Ferre, 1999; Chermak, 2004; Chermak & Musiek, 1997; Chermak, Hall, & Musiek, 1999; Musiek, Bellis, & Chermak, 2005).

[1]Certainly, one can imagine exceptions to this recommendation. For example, if questions arise regarding the effectiveness of medication, or if medication is not taken regularly, the audiologist might be asked to compare a child's auditory performance with and without medication (for purposes of determining both medication effectiveness and relative contribution of CAPD versus attention disorder to presenting complaints).

Additional consideration needs to be given to the potential influence of attention and/or executive control factors on the tasks required of the listener during central auditory testing, particularly when using dichotic speech tests. Dichotic speech tests may be administered in a forced-report or free-report paradigm. Through the use of imaging and behavioral studies, Hugdahl and colleagues have demonstrated that, although the free-report condition appears to reflect basic sensory perceptual processes, forced-report paradigms may also reflect attentional (forced-right) or executive control (forced-left) neural mechanisms and behaviors in addition to auditory perceptual factors (see Hugdahl et al., 2009 for review). Furthermore, forced-left conditions appear to activate neural circuits (i.e., prefrontal gyrus) that are not activated under forced-right or free-report conditions (Kompus et al., 2012). While this does not suggest that forced-report dichotic speech tasks cannot be used in the differential diagnostic test battery, it does underscore the need to look across all measures of central auditory function for consistent patterns that conform to neuroscience tenets. As such, an isolated abnormal finding in a directed-report dichotic listening test may be more indicative of an attention or executive control disorder rather than CAPD. See Chapters 6 and 16 for additional discussion of dichotic listening.

Much debate has also centered around the possible need to include nonauditory analogs of central auditory diagnostic tests for purposes of differentiating CAPD from higher order, pansensory, global disorders such as CAPD (Cacace & McFarland, 2005; McFarland & Cacace, 1995; Musiek et al., 2005). In a series of studies examining performance of children with CAPD, children with ADHD, and normal child and adult controls, Bellis and colleagues found that the addition of visual analogs of central auditory tests in common clinical use demonstrated limited clinical utility in the differential diagnosis of CAPD and ADHD (Bellis, Billiet, & Ross, 2008, 2011, Bellis & Ross, 2011). Specifically, the authors found that children with ADHD—as would be predicted—performed more poorly overall and not significantly different from those with CAPD on both visual and auditory tasks compared with normal controls. However, when intratest comparison measures were employed (i.e., ear differences on dichotic/dichoptic tasks, response condition differences on visual and auditory patterns tests), the performance of children with ADHD did not differ from that of typically developing children. Most important, the children with CAPD exhibited intratest performance patterns (e.g., significantly larger right-ear advantage for dichotic digits) that were not seen in any of the other participant groups and that were significantly more pronounced in the auditory modality. As such, the use of intratest comparisons of performance on central auditory tests alone was sufficient to differentiate the children with CAPD from those with ADHD and from normal controls. These studies suggest that the addition of visual or multimodal analogs of central auditory tests hold little clinical utility in the differential diagnosis of CAPD and ADHD. Furthermore, these studies underscore the assertion that, when interpreted appropriately, currently available tests of central auditory function can be used to differentially diagnose CAPD.

Noting the limitations of multimodal (analog) tests that differ only in sensory stimulus, Musiek et al. (2005) encouraged the development of other approaches to examining multimodality function. Among the limitations they identified are: (1) questions of the equivalence and comparability of multimodal (analog) tests; (2) absence of data supporting the sensitivity and specificity of any multimodal analog tests to differentially identify known central auditory versus supramodal dysfunction; (3) practical and professional scope of practice issues (e.g., few professionals have the clinical education and training to assess multiple sensory modalities); and (4) many proposed multimodal tests are not currently available to clinicians engaged in diagnosing and treating CAPD. These limitations suggest that it is more reasonable to adopt a multidisciplinary approach to differential diagnosis of CAPD in which professionals with the relevant expertise and scope of practice (e.g., neuropsychologists, educational diagnosticians, speech-language pathologists) obtain validated measures of other modalities and of higher order cognitive, language, attention, and related function. Comprehensive multidisciplinary evaluation in combination with the sensitized test battery of the CANS will lead to accurate diagnoses that will guide treatment and management of CAPD and ADHD. The reader is referred to other chapters in this volume for detailed discussion of CAPD assessment procedures and protocols, as well as the analysis and interpretation of central auditory test findings. In addition to the use of intratest comparisons with an individual patient, a number of other testing methods and interpretation strategies of test outcomes can reduce the potential confound of factors not under direct examination in behavioral testing for CAPD. These methods and strategies include: (1) use of nonverbal stimuli or, alternatively, stimuli that carry a light linguistic load; (2) intertest and cross-discipline (multidisciplinary) analysis to explore supramodal effects; (3) use of binaural separation/integration tasks during dichotic listening to examine the consistency of ear deficits (e.g., a consistent left-ear deficit in both conditions, given symmetrical hearing sensitivity, is unlikely to result from a supramodal deficit, whereas an isolated deficit in binaural separation only may indicate difficulties with attention and/or executive function); and (4) use of a simple response mode (ASHA, 2005; Bellis, 2003; Bellis & Ferre, 1999; Chermak et al., 1999; Chermak & Musiek, 1997; Jerger & Musiek, 2000; Musiek et al., 2005).

Findings of asymmetrical speech recognition deficits and/or speech recognition deficits seen only in the presence of either ipsilateral or contralateral competition would suggest a CAPD rather than a supramodal attention deficit (Baran & Musiek, 1995), whereas poor performance across all measures suggests a more global deficit (e.g., attention, memory, anxiety, or motivation, among others). Phoneme processing difficulties in quiet are unlikely to be the result of a pervasive attention deficit (Jerger, Martin, & Jerger, 1987). Similarly, word recognition deficits in noise for phonemically similar words, but not for semantically similar words argues against a pervasive attention deficit as the cause of the abnormal performance (Jerger et al., 1987). Poor response consistency or reliability across trials also suggests a more global deficit, as do deficits that *resolve*

with reinforcement. In addition to the basic audiologic examination and central auditory test battery, Chermak (2003) recommended assessment of executive function to evaluate more clearly supramodal or pansensory status versus multimodality function. (Electrophysiologic measures may even differentiate performance of individuals with ADHD better than behavioral measures [McPherson & Salamat, 2004].) Measures of executive function assess the ability to sustain, focus, and shift attention (e.g., continuous performance tests, measures of verbal fluency, Matching Familiar Figures Test, Stroop tasks, Trail Making Test, and the Wisconsin Card Sort, among others) (Denckla, 1989). Finally, certain test paradigms and stimuli may be more resistant to alterations in laterality due to attention (e.g., dichotic fusion/Dichotic Rhyme Test) and particular dichotic test administration modes (i.e., free recall) may minimize attention effects and thereby maximize the validity of observed laterality effects (Hugdahl et al., 2009; Moncrieff & Musiek, 2002; Musiek et al., 2005b; Shinn, Baran, Moncrieff, & Musiek, 2005).

Conclusions

Attention deficits, listening deficits, and poor academic achievement associated with both ADHD and CAPD (AAA, 2010; APA, 2013; ASHA, 2005; Bellis, 2003; Chermak & Musiek, 1997) render differential diagnosis especially challenging and underscore the importance of thorough, multidisciplinary assessment with individuals suspected of these disorders. Differences in auditory areas of the brain, relative to normal controls, suggest a neuroanatomical basis for the frequently observed central auditory performance deficits among individuals diagnosed with ADHD. Listening difficulties seen in CAPD result from primarily auditory perceptual deficiencies rather than global attention deficits (ADHD predominantly inattention subtype) or behavioral regulation deficits (combined ADHD and predominantly hyperactive-impulsive ADHD subtypes) (Phillips, 1990, 1995; Tallal et al., 1996). ADHD and CAPD may differ in the extent of the attention deficit (i.e., cognitive and supramodal versus sensory and restricted to the auditory modality), as well as the type of attention deficit (i.e., sustained versus selective and divided attention). Within an information processing framework, the sustained attention problems observed in ADHD are seen as the consequence of a supramodal cognitive deficit whereas CAPD results from a specific auditory perceptual deficit. Inattention in ADHD may reflect deficiencies in behavioral regulation rather than attention per se. In contrast, the selective auditory attention deficits of CAPD result from deficient auditory perceptual processing.

Clinicians must use sensitive measures to evaluate the integrity of underlying perceptual and supramodal systems to determine the predominant and primary deficits, as well as secondary problems that may underlie these deficits (McFarland & Cacace, 1995, 2005). Such an approach requires the multidisciplinary efforts of audiologists, speech-language pathologists, educators, psychologists, and physicians for assessment, differential diagnosis, and for intervention. Effective intervention for ADHD and CAPD hinges on accurate diagnosis of these conditions and is detailed in Volume 2 of this Handbook.

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SECTION 5

CHAPTER 21

CASE STUDIES: DIAGNOSIS

ANNETTE E. HURLEY

Case studies connect theory and practice. The following case studies, focused on diagnosis, illustrate the heterogeneous nature of central auditory processing disorder (CAPD). Case studies reflect *real world* situations in which assessment information from other professionals often is not available at the time the audiologist sees the patient. Often in case review, analysis raises additional questions, elicits reflection, and exposes missed opportunities. Following each case is a "debriefing" section in which strengths and weaknesses of the process described in the case are discussed, and suggestions, posed in a question and answer format, are used to encourage the reader to explore additional aspects of the case.

Case 1

Background

Mary is a 6-year-old female referred for a CAPD screening by her speech-language pathologist (SLP) due to lack of progress in speech-language therapy. Significant medical history includes three sets of pressure equalization tubes, positive history of jaundice at birth, and history of speech delay. Previous audiological evaluations reported hearing sensitivity within normal limits. Earlier in the academic year, Mary *passed* a hearing screening. Excessive cerumen was noted upon otoscopy, but normal (type A) tympanograms were obtained, bilaterally.

Mary attends a private school and is having difficulty with reading and spelling.

Assessment

Otoscopic examination revealed impacted cerumen bilaterally. Audiometric thresholds suggested a conductive hearing loss, bilaterally. Pure-tone thresholds are shown in Table 21–1. Mary's mother reported that she has previously voiced concern about Mary's predisposition for excessive buildup and has requested cerumen removal by the pediatrician. Mary's pediatrician ignored her concerns stating that wax was normal and did not need to be removed. Because of the pediatrician's indifference, Mary was referred to an ear, nose, and throat (ENT) physician for cerumen removal.

Two weeks later, Mary returned to the audiology clinic and normal peripheral hearing was established bilaterally. Behavioral results obtained from the SCAN 3C: Test for Auditory Processing in Children (SCAN-3:C) (Keith, 2009) are shown in Table 21–2, indicating normal central auditory processing ability on four diagnostic subtests (i.e., two tests of monaural low redundancy and two dichotic speech tests). Normal auditory brainstem responses (ABRs) to clicks and to speech (cABR) were also obtained bilaterally. (See Chapters 7 and 17 for discussion of electrophysiological measures.)

Results and Recommendations

Behavioral and electrophysiological measures were within normal limits and no

Table 21–1. Audiometric Thresholds in dB HL for Case 1 (Mary)

Frequency (Hz)	250	500	1000	2000	4000	8000
Right air conduction	35	25	25	30	30	40
Left air conduction	35	30	35	30	30	35
Unmasked bone	0	0	–5	–5	0	

Table 21–2. Screening for Auditory Processing in Children (SCAN-3:C) Results for Case 1 (Mary)

	Raw Score	Standard Score	Percentile Rank
Auditory Figure-Ground	35	10	50
Filtered Words	32	13	84
Competing Words	48	17	99
Competing Sentences	67	17	99
SCAN-3C Composite	NA	133	99

additional audiological recommendations were made. It was recommended, however, that Mary continue speech-language therapy. If progress is not seen, additional central auditory processing testing in one year is recommended when Mary will be 7 years old. Because of Mary's predisposition to excessive cerumen accumulation, Mary's mother was advised to maintain vigilance about cerumen removal with Mary's pediatrician or ENT.

Debriefing

This case reinforces the importance of establishing peripheral auditory function before central auditory testing. A report of "normal" from another facility at a previous time does not ensure normal peripheral hearing at a later time when central auditory processing testing is scheduled.

- What effect would a hearing loss have on behavioral central auditory test results? Would one type of central auditory process be influenced to a greater degree by hearing loss (e.g., monaural low redundancy, temporal patterning, or dichotic testing)? Would you accept a referral for a central auditory processing evaluation for someone with a hearing impairment?
  A person with hearing loss can be tested for CAPD; however, tests should be carefully selected and interpretation may still necessarily be qualified or guarded. Distinct patterns of performance on the central auditory test battery can suggest the likelihood of a CAPD, regardless of degree of hearing loss. Moreover, there are low linguistically loaded tests (e.g., Dichotic Digits Test) or nonspeech tests (e.g., Frequency Pattern Test) that are less affected by the presence of a hearing loss. Temporal processing tests (e.g., duration patterns, frequency patterns) may be less influenced by peripheral hearing loss. The Frequency Pattern Test (Musiek & Pinherio, 1987) consists of low frequency tonal stimuli (880 and 1122 Hz) and may be particularly useful if the hearing loss is seen outside these frequencies. Electrophysiologic recordings can be obtained using broadband stimuli, such as clicks, or low frequency tone bursts where normal hearing may be present. See Chapter 11 for discussion of strategies for testing central auditory processing in individuals with peripheral hearing loss.

- What CAPD risk factors were reported?
  History of protracted otitis media, hyperbilirubinemia (jaundice), speech delay, and poor academic performance in reading and spelling

- Why would central auditory processing testing be considered at a later age?
  CAPD testing is rigorous and places considerable demands on the child for focused attention, physical and mental endurance, and the ability to process challenging tasks. For the most part, normative data for many central auditory processing tests before the age of 7 are not available; moreover, due to the challenge associated with these tasks, normal, young, children present considerable variability in performance. Additional testing may be recommended if Mary does not make progress in speech-language therapy.

Case 2

Background

Hannah is a 10-year-old female referred by her SLP. Her history indicates a normal pregnancy. With the exception of speech, developmental milestones were achieved at appropriate ages. A negative history of otitis media was reported. Hannah has been in speech language therapy since age 4. She has been successfully medically managed for attention deficit hyperactivity disorder (ADHD). Hannah is in the fourth grade. She has difficulty with reading (decoding, word attack, reading fluency, and reading comprehension), and presents deficits in short-term and working memory. Hannah has previously completed one *Fast ForWord Language* program. She receives speech-language therapy and reading resource services at her public school. Additionally, she receives private reading therapy by a SLP. A family history of dyslexia (brother and maternal uncle) was reported. Despite the ADHD diagnosis, Hannah's mother reports that she is unsure whether Hannah has a hearing problem or a memory problem. She reports Hannah frequently misunderstands or misinterprets multistep directions.

Assessment

Normal peripheral hearing was established bilaterally. Hannah's performance on the behavioral tests of the central auditory processing battery is shown in Table 21–3. Results indicated a left ear deficit on dichotic speech tests and difficulty linguistically labeling the tonal stimuli on the Frequency Pattern Test. A normal ABR and cABR were obtained, bilaterally. (Readers are referred to Chapters 13 to 17 for additional description and discussion of each test, and to Chapter 20 for discussion of central auditory testing of individuals with ADHD.)

Recommendations

A list of metacognitive management strategies was provided for Hannah's SLP. Hannah will begin a new multisensory reading program (Orton-Gillingham) provided by her school. She will also be working with this program privately with her SLP. Additionally, Hannah qualifies to continue the *Fast ForWord Language to Reading* and *Reading Assistant* software programs and she will begin these programs at her school. Dichotic listening therapy was recommended; however, Hannah's mother decided to wait until after the school year to begin dichotic listening therapy. Additional follow-up in one year is recommended.

Debriefing

- What CAPD risk factors are reported?
  Developmental disorders including speech delay, reading difficulty, familial history of dyslexia, and ADHD

- Which central auditory processes are impaired? Which neurological structures may be implicated?
  Left ear deficits on dichotic speech tests reveal difficulty with binaural integration. This finding coupled with frequency pattern results suggest interhemispheric transfer deficit. See Chapter 14 for discussion of dichotic listening and testing.

Table 21–3. Behavioral CAPD Test Results for Case 2 (Hannah)

Speech-in-Noise (+5 Signal to noise Ratio)		Low-Pass Filtered Words		Competing Sentences		Dichotic Digits		Frequency Patterns		Gap Detection Threshold	Masking Level Difference
Left	Right	Left	Right	Left	Right	Left	Right	Left verbal	Left hummed	4 msec	10 dB HL
94%	94%	88%	90%	40%	100%	64%	98%	30%	90%		
Normal		Normal		Abnormal Left Ear Deficit		Abnormal Left Ear Deficit		Reflects Interhemispheric Dysfunction		Normal	Normal

- What is the theory behind dichotic listening therapy?
  In normal listening conditions, auditory information is transmitted to the auditory cortex by both ipsilateral and contralateral auditory pathways. Kimura (1961) was first to note the dominant contralateral pathway and theorized that during dichotic listening the ipsilateral pathway is suppressed by the dominant contralateral pathway. Dichotic listening training is an innovative therapy for the remediation of the compromised central auditory pathway (Musiek, Chermak, & Weihing, 2007). This is accomplished via dichotic listening in which the signal level to the impaired pathway or deficit ear (in this case the left ear) remains at a comfortable level. The intensity to the stronger, dominant ear (in this case the right ear) is reduced significantly at the beginning of training. During training, the intensity to the right ear is increased slowly as the weaker, impaired pathway grows stronger. See Chapter 9 of Volume 2 of this Handbook for detailed description of dichotic listening training.

- Would you recommend follow-up before one year?
  It is important to continue to follow Hannah to ensure progress and to ensure auditory maturation (Musiek, Gollegly, & Baran, 1984). It would be prudent to reassess after Hannah completes the Fast ForWord Language to Reading and Reading Assistant programs. Positive changes in behavioral and electrophysiological recordings would reflect training-induced plasticity and/or maturation of the central auditory nervous system (CANS).

Case Study 3

Background

Sofia is an 8-year-old second-grade female referred for additional testing after failing the SCAN: 3C administered by a psychologist. Sofia was diagnosed with a "reading disorder" and is currently receiving reading resources at school. She has not had a speech-language assessment. Normal birth history and developmental milestones were reported at age-appropriate times. There was no history of ear infections or family history of dyslexia, learning disorders, or ADHD. Sophia is the youngest of four children. At the time of audiologic testing, she was taking medication for anxiety and ADHD. Academic concerns include reading, spelling, listening, and following directions in group settings. She attends a private school.

Assessment

Pure tone audiometry established normal hearing sensitivity, bilaterally. Normal tympanograms and acoustic reflexes were obtained, bilaterally, indicating normal middle ear function. Sophia's test results are shown in Table 21–4. Results of the central auditory processing test battery indicated abnormal performance for monaural low-redundancy speech tests and tests of temporal resolution and binaural interaction, bilaterally. A normal click ABR and cABR were obtained bilaterally.

Table 21–4. CAPD Test Results for Case 4 (Sophia)

Speech-in-Noise		Low-Pass Filtered Words		Time Compressed Speech		Competing Sentences		Dichotic Digits		Frequency Patterns	Gaps-in-Noise Threshold	Masking Level Difference	Phonemic Synthesis Test
Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left verbal	14 msec	6 dB HL	Quantitative Score = 10
58%	62%	48%	52%	48%	64%	80%	100%	80%	80%	80%			
Abnormal		Abnormal		Abnormal		Normal		Normal		Normal	Abnormal	Abnormal	Abnormal

Recommendations

The following recommendations were made: (1) environmental modifications to include a trial of an FM system for classroom listening and strategic classroom seating, (2) referral for speech-language evaluation, (3) formal auditory training to address temporal resolution and binaural interaction deficits, and speech-in-noise training, (4) phonemic awareness training, and (5) metacognitive strategies training to help Sophia anticipate difficult listening situations and begin to problem solve to improve these situations. (See Chapter 10 for elaboration of metacognitive strategies, Chapter 12 for discussion of FM systems, and Chapter 7 for elaboration of auditory training in Volume 2 of this Handbook.)

Debriefing

- What CAPD risk factors were reported?
  No predisposing medical risk factors were noted in Sophia's case history. ADHD and poor academic performance in reading and spelling were reported.

- Which central auditory processes are impaired?
  Deficits on tests of monaural low-redundancy speech tests (speech-in-noise, low-pass filtered speech, and time-compressed speech) suggest deficits of auditory closure. Poor gap detection suggests a temporal resolution deficit. Abnormal masking level difference threshold suggests a deficit in binaural interaction. See Chapters 13, 15, and 16 for elaboration of these diagnostic procedures.

- What additional information would be helpful for your diagnosis and remediation plan?
  A CAPD was diagnosed, based upon Sophia's behavioral test results. A speech-language assessment was recommended. Collaboration with a multidisciplinary team including Sophia's psychologist and speech-language pathologist will maximize the potential for successful management and academic success

- Can a psychologist diagnose CAPD?
  CAPD is an auditory diagnosis and an audiologist is the professional qualified to make the diagnosis (ASHA, 2005). A psychologist may be part of the multidisciplinary team involved in differential diagnosis, identification of comorbid conditions, and remediation planning.

- When would you suggest CAPD follow-up?
  Annual assessments are recommended to assess progress of auditory training and maturation of the CANS.

- Discuss how binaural interaction may be assessed by behavioral and electrophysiological measures.
  Binaural interaction refers to how the two ears work together. Tests of binaural interaction assess how the ability of the CANS uses information from the two ears (Bellis, 2003). Behavioral tests of binaural interaction include Rapidly Alternating Speech Perception, MLD, Binaural Fusion, and interaural time or intensity tests. Because of the concerns of sensitivity of these tests and ease of administration, many of these behavioral tests, with exception of the MLD, are not routinely used.

  Objective measures for auditory processing are promising. The

Binaural Interaction Component (BIC) has been of interest for many researchers, but has not been embraced clinically. Central neural interaction of the ABR potentials to binaural stimulation was first reported by Jewett (1970). In theory, the amplitude of the right recording added to the amplitude of the left recording should equal the amplitude of the binaural recording. However, this is not the case. Generally speaking, there is a difference waveform that can be derived by subtracting the binaural response recording from the summed monaural recording, or vice versa. In humans, this difference waveform usually occurs at a latency in the approximate range of wave V, and this difference waveform is thought to show objective evidence for binaural interaction. The BIC may be useful tool in evaluating binaural processes such as localization, lateralization and fusion, and may also be used to investigate some of the specialized brainstem processing for binaural hearing (Berlin et al., 1984; Hall, 2007; Wrege & Starr, 1981) and in the future become a promising clinical tool in central auditory assessment (Delb, Strauss, Hohenberg, & Plinkert, 2003; Gopal & Pierel, 1999).

Case Study 4

Background

Abe is a 17-year-old male who was identified with auditory neuropathy spectrum disorder (ANSD) shortly after birth. Abe was bilaterally fitted with hearing aids at 2 months of age, but currently wears only one hearing aid in the right ear, and only while attending school. He reports that he stopped wearing his hearing aid in his left ear during upper elementary school as he did not receive any benefit from the device and because the hearing aid "sounded like loud noise." He reports experiencing difficulty in background noise and in situations with multiple talkers. Although he reported improvement with his FM system, he does not use this device. His resistance to using the device is self-reported as "peer- related." Abe attends a private school with small classes. He reported that his teachers know about his hearing difficulty and are very helpful when he needs clarification or accommodations. He expects to graduate from high school with honors and enter college in a few months. The purpose of the audiological assessment was to evaluate Abe's auditory processing ability, offer any possible recommendations, and provide documentation for Abe to receive services through the university's disability services office. (See Chapter 19 for discussion of ANSD and Chapter 14 of Volume 2 of the Handbook for discussion of management for adolescents and adults.)

Assessment

Otoscopy was unremarkable. Otoacoustic emissions (OAEs; transient) were absent in both ears. Immittance testing revealed normal (type A) tympanograms. Ipsilateral and contralateral acoustic reflexes were absent bilaterally at 110 dB HL. Pure-tone audiometry revealed a mild to moderate hearing loss bilaterally, as shown in Table 21–5. Speech recognition thresholds were consistent with pure-tone averages. Word recognition scores in quiet of 88% and 80%

were obtained in the right and left ears at most comfortable listening presentation levels of 75 and 85 dB HL, respectively. With the introduction of ipsilateral noise presented at +10 signal-to-noise ratio, Abe's word recognition scores decreased to 48% and 24% for the right and left ears, respectively. Abe's results for selected behavioral central auditory tests are shown in Table 21–6. Abnormal temporal resolution was revealed by the abnormal gap detection threshold in his left ear and binaural interaction deficits were revealed by abnormal masking level differences (MLD). (See Chapter 11 for discussion of central auditory testing in the presence of peripheral hearing loss.)

Electrophysiological recordings were also obtained. The ABR (shown in Figure 21–1) was recorded to 100 μsec condensation and rarefaction click stimuli presented at a level of 85 dB nHL, at a rate of 27.7 per second. His ABR displays the classic out-of-phase ringing diagnostically characteristic of ANSD. Interestingly, wave V absolute latency was delayed, with a longer absolute latency in the left ear of 6.49 ms compared to 5.82 ms for the right ear.

A two-channel auditory middle latency response (AMLR) was obtained. Latencies and amplitude values are listed in Table 21–7. The AMLR latency was delayed, but symmetrical between ears. The Na–Pa amplitude was within normal limits. Clinical analysis of the AMLR is often more concerned with the amplitude than latency (Chermak & Musiek, 1997). The summed responses from two individual recordings are shown in Figure 21–2. Auditory late evoked responses (ALERs), including the P300 recordings, were also obtained and the summed responses from two individual recordings are shown in Figure 21–3. This recording was elicited by an "oddball" paradigm, in which the listener hears a low frequency tone with an occasional high frequency tone that the listener must count. Latency and

Table 21–5. Audiometric Thresholds in dB HL for Case Study 4 (Abe)

Frequency (Hz)	250	500	1000	2000	4000	8000
Right air conduction	30	40	40	60	65	50
Left air conduction	30	40	45	60	70	55
Unmasked bone	20	35	40	55	65	

Table 21–6. Behavioral CAPD Test Results for Case 4 (Abe)

Gaps-in-Noise Threshold		Dichotic Digits		Frequency Patterns		Masking Level Difference
Left 20 mec	Right 6 msec	Left 90%	Right 98%	Left 100%	Right 100%	6 dB HL
Abnormal	Normal	Normal		Normal		Abnormal

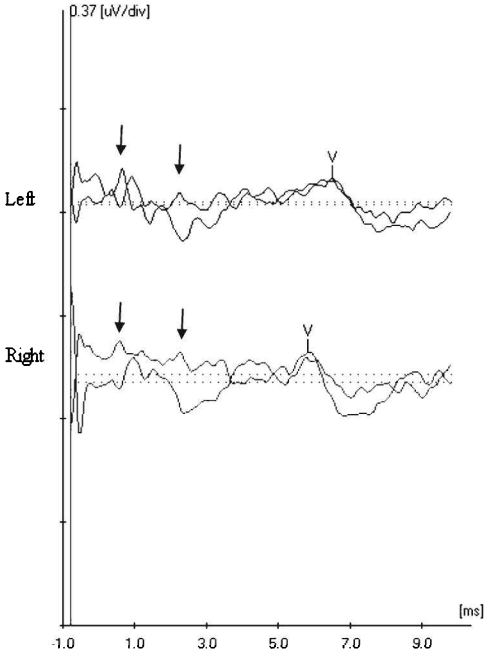

Figure 21–1. A ringing ABR is characteristic of a patient with AN/AD.

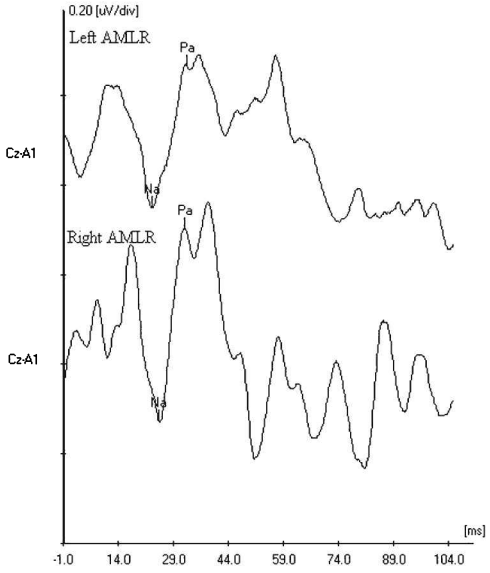

Figure 21–2. Depicted is the summed response from two individual recordings for the right and left AMLR.

Table 21–7. AMLR Obtained for Case 4 (Abe)

Electrode	Stimulus Ear	Na latency in msec	Pa latency in msec	Na-Pa amplitude
Cz	Left	25.64	38.55	0.49 μV
Cz	Right	23.35	35.84	0.35 μV

amplitude values are provided in Table 21–8. Latencies and amplitudes of the P300 were symmetrical and within normal clinical range.

Recommendations

Abe prefers to wear only one hearing aid in his right ear and does not feel that a hearing aid in the left ear is helpful. We theorize there is greater dyssynchrony in the left ear than in the right, as reflected behaviorally in the abnormal gap detection thresholds obtained for the left ear, as well as by the prolonged left ABR wave V absolute latency, which was .67 msec later than the right ear. (This interaural latency difference had been seen on Abe's ABR recordings obtained during infancy.) The abnormal MLD also is evidence of abnormal binaural interaction.

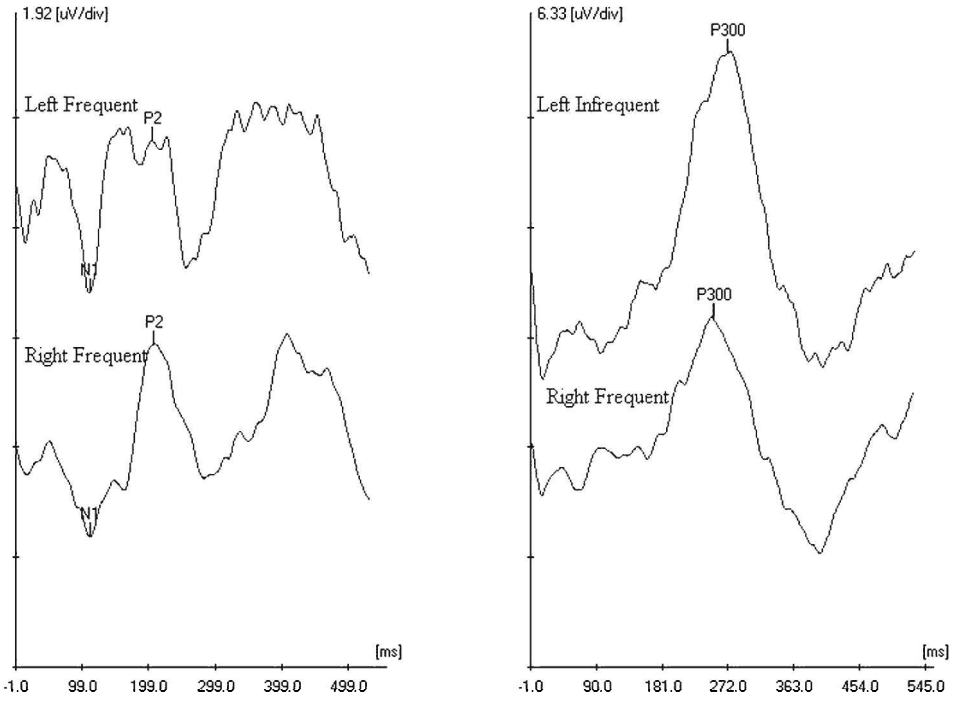

Figure 21–3. A normal, symmetrical P300 is recorded for the right and left ears. Shown are the summed responses from two individual recordings.

Table 21–8. ALER Obtained for Case 4 (Abe)

Ear	N1 latency in msec	P2 latency in msec	N1/P2 amplitude	P300 latency in msec	P300 amplitude
Left	110.59	204.28	2.64 μV	271.95	12.39 μV
Right	110.59	206.36	2.02 μV	252.17	7.39 μV

Abe recognizes his need for accommodations in college, including the use of an FM system in the classroom. It is also recommended that he use the college's note-taking services. Abe should have his hearing tested annually to monitor any changes in hearing sensitivity.

Debriefing

- Do all patients with ANSD have CAPD?

Patients with ANSD clinically present with impaired speech recognition scores, especially in the presence of background noise. Temporal processing deficits are also reported in patients with ANSD. Early electrophysiologic responses are absent or grossly abnormal. In some cases, patients with ANSD also present with CAPD due to the underlying anatomical disorder. (See Chapter 14

for additional information about ANSD.)

- Are gap detection thresholds normally obtained for individual right and left ears? Why or why not?
  Previous investigations (Baker, Rosen, & Goodrich, 2000; Efron, Yund, Nicholls, & Crandell, 1985; Musiek et al., 2005) reported similar gap detection thresholds for the right and left ears. Nonetheless, obtaining monaural gap detection thresholds is best practice and may yield useful findings especially in cases of ANSD, acquired CAPD as a result of traumatic brain injury, or other unusual cases.

- Should annual central auditory processing evaluations be included as part of annual hearing evaluations? Why or why not? Would the patient's age influence your rationale?
  Abe's hearing impairment has not changed since identification shortly after birth. Annual audiograms are routinely recommended for children over 5 years even if hearing thresholds have remained unchanged since identification. Annual central auditory processing assessments are also recommended. Abe is very cognizant of his hearing status and will return to the clinic sooner if he detects any change in his hearing sensitivity.

Case Study 5

Background

Pat is a 28-year-old female who self-referred for a central auditory processing assessment. She reported having numerous hearing tests which all indicated normal hearing; however, she reported great difficulty hearing and understanding in noisy environments or in group situations. She reported this difficulty has been present all of her life, but that she has effectively compensated for this difficulty by employing such strategies as sitting near the front of a classroom, borrowing notes from friends, and studying in quiet areas. Her difficulty in hearing has now negatively impacted her career, as her once private office at work has been changed to a cubicle setting with many colleagues nearby, which causes great auditory interference. Pat holds a graduate degree in social work and is working as a health administrator where she counsels clients and supervises other employees. She reports that she "had to work harder than her siblings for her grades," but does not feel that she has a learning disability. She reported her communication difficulty has also made social situations very difficult and describes herself as "becoming a recluse." Pat reported a history of depression and anxiety, had been in counseling in the past, and was previously treated with medication by a psychiatrist. She reported that she is considering therapy and pharmacological management once again.

Assessment

Normal middle ear function was established bilaterally, with type A tympanograms and acoustic reflexes obtained at normal levels. Pure-tone thresholds were screened at 20 dB HL. Results of the central auditory processing assessment, shown in Table 21–9, indicated difficulty understanding in degraded acoustic environments.

Table 21–9. Behavioral CAPD Test Results for Case 5 (Pat)

Speech-in-Noise		Filtered Words		Time Compressed Speech		Competing Sentences		Dichotic Digits		Frequency Patterns	Duration Patterns	Gap Detection Threshold	Masking Level Difference
Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left verbal	Left verbal	4 msec	10 dB HL
48%	52%	40%	48%	60%	62%	90%	90%	90%	90%	80%	100%		
Abnormal		Abnormal		Abnormal		Normal		Normal		Normal	Normal	Normal	Normal

Recommendations

The Listening and Communication Enhancement (LACE) program was recommended for speech-in-noise training. Additionally, environmental modifications for work were suggested. These included the use of noise-canceling earphones at her cubicle. Additional work modifications were explored. It was recommended that Pat utilize a private conference room as needed for meetings and telephone conference calls at her office. Metacognitive strategies to help identify and problem solve difficult noise environments and social situations were discussed. Pat reported noticeable improvement as LACE training continued. She also reported beginning counseling with a life coach and was receiving medical management for depression and anxiety.

Debriefing

- What additional information would you like to have?
  Pat commented that she struggles with information presented auditorily. Although she holds a graduate degree, she reports she struggled and worked very hard throughout her school years. She has never had any formal psychoeducational testing. Results from such an assessment could reveal specific areas of strengths and weaknesses. This information might help Pat better understand her learning abilities and use this information to her benefit.

- What auditory processes are affected?
  Pat presents auditory closure deficits, as demonstrated by deficits on monaural low-redundancy speech tests (speech-in-noise, low-pass filtered speech, and time compressed speech).

- What other activities might be appropriate to include in the rehabilitation plan?
  A list of informal listening activities was provided (e.g., websites with listening games activities, and short stories, essays and conversations with questions for the listener).

- Could this patient have King-Kopetzky syndrome?
  King-Kopetzky syndrome is an "obscure auditory stress disorder" (Hinchcliffe, 1992; Saunders & Haggard, 1989) in which a patient has normal hearing but reports difficulty listening in the presence of background noise. Zhao and Stephens (2000) proposed the "impairment" may not be auditory, but psychological or psychologically amplified. Assessing Pat's "hearing handicap" may provide further information about the impact of Pat's listening difficulty and provide information for possible medical referral.

[redacted] Case Study 6

Background

Danielle is a 9-year-old female referred for a central auditory assessment by her SLP who reported a moderate expressive and receptive language delays. A normal birth history was reported. Developmental milestones were achieved at appropriate ages. There is a negative history of

middle ear infections. Results of a recent psychoeducational evaluation reported average intelligence, deficits in short-term and working memory, and deficits in auditory comprehension. Danielle began speech-language therapy at age 7 after academic difficulties were noted. She is the youngest of four children and her academic difficulties are extremely puzzling to her parents, both physicians, who reported that Danielle's siblings have had no difficulty in school. Danielle also experiences difficulties with written language, including reading, writing, and spelling. She attends a private school and receives speech-language therapy and tutoring.

Assessment

Normal middle ear function and normal peripheral hearing were established bilaterally. Central auditory processing test results, reported in Table 21–10, indicate abnormal performance on monaural low-redundancy speech tests bilaterally and an abnormal left ear deficit on dichotic speech tests. A normal ABR was obtained for the right and left ears, as shown in Figure 21–4. Abnormal cABR for the right and left ears were obtained, as shown in Figure 21–5, providing objective evidence of abnormal neural encoding of speech stimuli. (See Chapter 7 for discussion of cABR and neural encoding.)

Recommendations

A list of environmental and metacognitive strategies was given to Danielle and her SLP. A personal FM system for classroom use was recommended, as was continued speech-language therapy. Danielle's school provides Fast ForWord, for which Danielle has qualified and will begin within the next month. Danielle was scheduled to return for follow-up in six months for reassessment, at which time additional therapy options, such as dichotic listening therapy, will be explored.

Debriefing

- What are the CAPD risk factors? *Speech and language delays and deficits in short-term and working memory and auditory comprehension are noted in the case history.*

- What effect does memory have on CAPD tests? How can the clinician rule out memory as a factor in central auditory test battery interpretation? *Memory deficits can affect CAPD results. For example, a child who cannot repeat four numbers will not perform well on the Dichotic Digits Test, because of memory, not necessarily because of a CAPD. Ensuring that a child can repeat a four-number digit span or simple sentence when presented monaurally prior to administering the dichotic digit test will maximize the chances that deficits seen on the dichotic test are processing deficits, and are not due to a higher global deficit. Auditory memory tests (digit span forward to address short term memory and digit span backward to address working memory) are also included in the CAPD test battery in other countries. See Chapter 18 in Volume 2 for discussion digit span testing in differential diagnosis of CAPD and ADHD.*

- Would the habilitation plan be changed if the cABR recording was normal? Discuss how electrophysiological recordings affect diagnosis and

Table 21–10. Behavioral CAPD Test Results for Case 6 (Danielle)

SCAN-3:C Auditory Figure Ground	SCAN-3:C Filtered Words	SCAN-3:C Competing Words	SCAN-3:C Competing Sentences	Dichotic Digits		Time Compressed Speech NU 6		Frequency Patterns		Gap Discrimination (3-interval forced choice)
Standard Score	Standard Score	Standard Score	Standard Score	Left	Right	Left	Right	Left Verbal	Left Hummed	Right
1	1	7	3	28%	82%	20%	20%	30%	100%	3 msec
Abnormal	Abnormal	Abnormal left ear deficit noted, normal Standard Score	Abnormal Left ear deficit noted	Abnormal, Left ear deficit noted		Abnormal		Indicates interhemispheric deficit		Normal

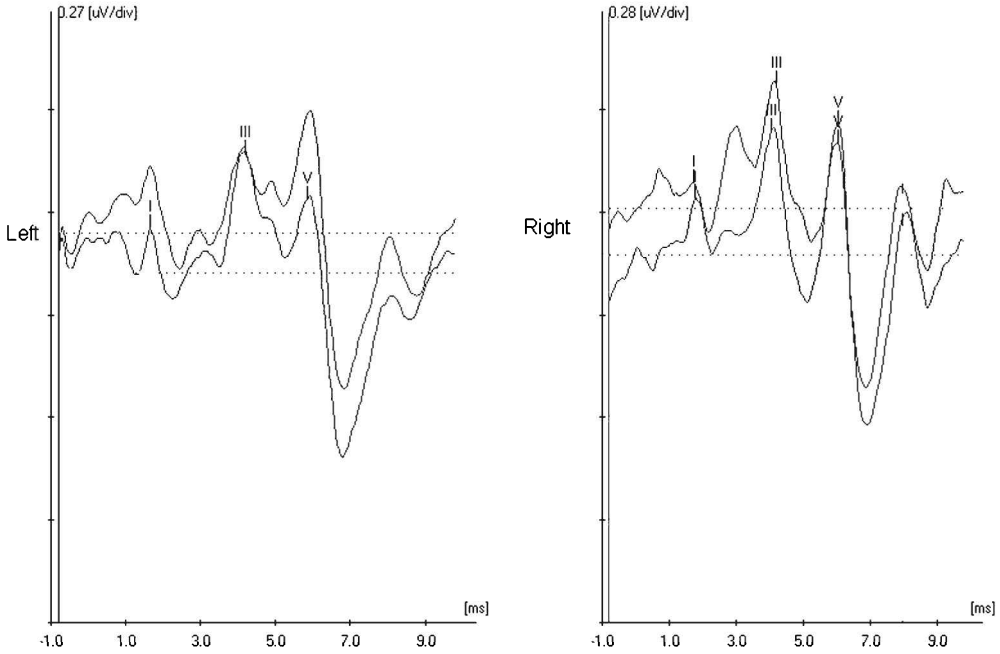

Figure 21–4. A normal ABR to clicks was obtained for the right and left ear.

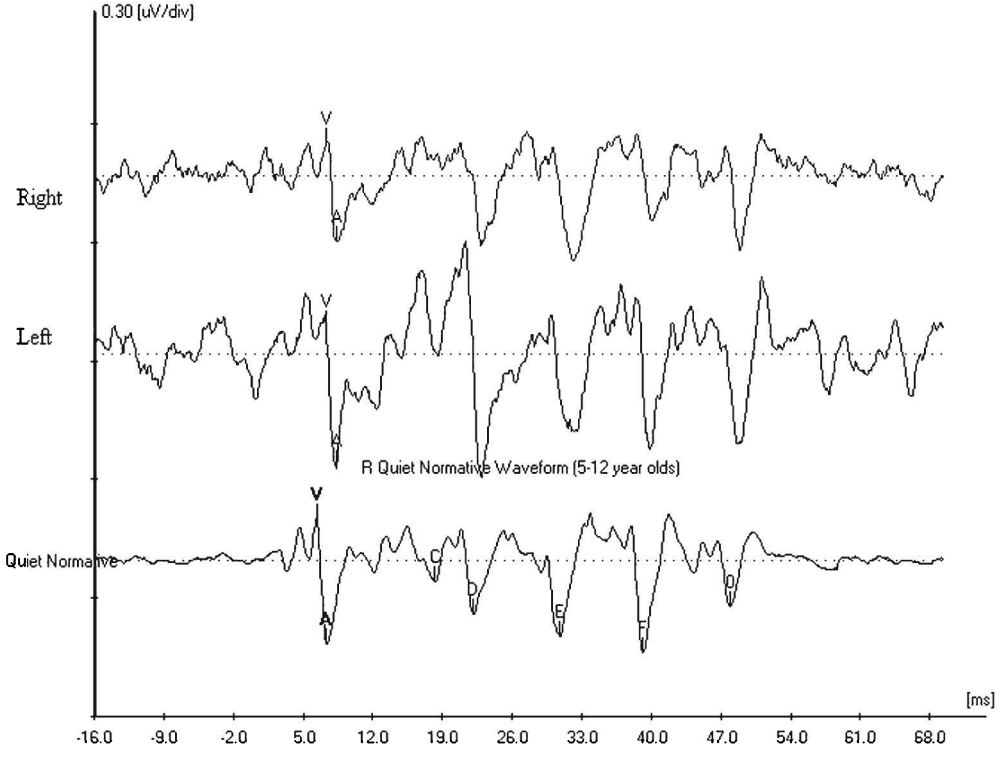

Figure 21–5. An abnormal cABR was obtained for both the right and left ears. Also shown is a normative cABR waveform.

may be useful in treatment/ follow-up?
Approximately 30% of children with language-based learning disabilities have abnormal cABR recordings. Although the habilitation plan would not have differed if the cABR had been normal, the cABR provides valuable information about neural encoding of speech for each patient. Previous research has reported improvements of the cABR after auditory training (Russo, Hornickel, Nicol, Zecker, & Kraus, 2010), suggesting training-induced plasticity of the CANS.

Case Study 7

Background

Alexandra, an 8-year-old female, was referred for audiological evaluation by her school SLP. She presented with a diagnosis of an expressive and receptive language disorder. A normal pregnancy was reported; however, Alexandra contracted meningitis as an infant. An abnormal magnetic resonance imaging (MRI) scan revealed frontal and temporal lobe damage in the right hemisphere. Developmental milestones were slightly delayed. Alexandra has received occupational, speech, and physical therapy since birth, and is currently receiving occupational and speech-language therapy. Previous audiological tests indicated normal peripheral hearing. There is a negative history of middle ear infections. Alexandra is medically managed for ADHD. She is in second grade at a private school. Normal cognitive ability is reported. Alexandra's teachers reported that she does not hear well in group settings, is not able to follow multistep directions, and presents with reading comprehension difficulties. Also noted is a growing concern for social interactions with peers. Alexandra's mother feels that Alexandra cannot keep up with the flow of conversation.

Assessment

Normal peripheral hearing and normal middle ear function were established bilaterally. Results of the central auditory processing assessment are shown in Table 21–11. Alexandra performed within normal limits on all tests, except for the dichotic listening tests and on the labeling condition of the Frequency Pattern Test (Musiek & Pinheiro, 1987). A normal ABR was obtained bilaterally, as shown in Figure 21–6. Latency and amplitude values are shown in Table 21–12. An abnormal cABR was obtained for the right ear, as shown in Figure 21–7. A normal AMLR also was recorded for the right and left ears; the summed responses from two individual recordings shown in Figure 21–8. Latency and amplitude values are shown in Table 21–12. A decrease in amplitude was seen over the temporal lobes, but was symmetrical between C3 and C4 and interpreted as normal. The P300, recorded from Cz with right and left stimulation, revealed some interaural differences, as shown in Figure 21–9. Latency and amplitude values are reported in Table 21–12. Interestingly, the right-ear stimulation produced greater amplitudes for N1–P2 and P300s.

Table 21–11. Behavioral Test Battery Results for Case 7 (Alexandra)

SCAN-3:C Auditory Figure Ground	SCAN-3:C Filtered Words	SCAN-3:C Competing Words	SCAN-3:C Competing Sentences	SCAN-3:C Time Compressed Speech	Dichotic Digits		Frequency Patterns		Gap Detection Threshold
Standard Score	Standard Score	Standard Score	Standard Score	Standard Score	Left	Right	Left Verbal	Left Hummed	Right
10	12	6	6	9	20%	96%	40%	80%	3 msec
Normal	Normal	Abnormal Left ear deficit	Abnormal Left ear deficit	Normal	Abnormal Left ear deficit		Anecdotal of interhemispheric dysfunction		Normal

Table 21–12. Amplitude and Latencies of the Electrophysiological Recordings for Case 7 (Alexandra)

	ABR (click)			ABR (speech)		AMLR			ALER				
	I in msec	III in msec	V in msec	V in msec	A in msec	Na in msec	Pa in msec	Na–Pa' in μV	N1 in msec	P2 in msec	N1–P2' in μV	P300 in msec	P300 Amplitude in μV
Left (Cz)	1.41	4.16	5.95	7.20	8.03	19.60	38.97	1.33	106.42	192.83	4.57	248.00	5.99
Left (C3)								.61					
Right	1.45	4.11	4.95	7.12	7.87	19.60	41.26	1.28	98.10	204.28	13.42	270.91	9.45
Right (C4)								.74					

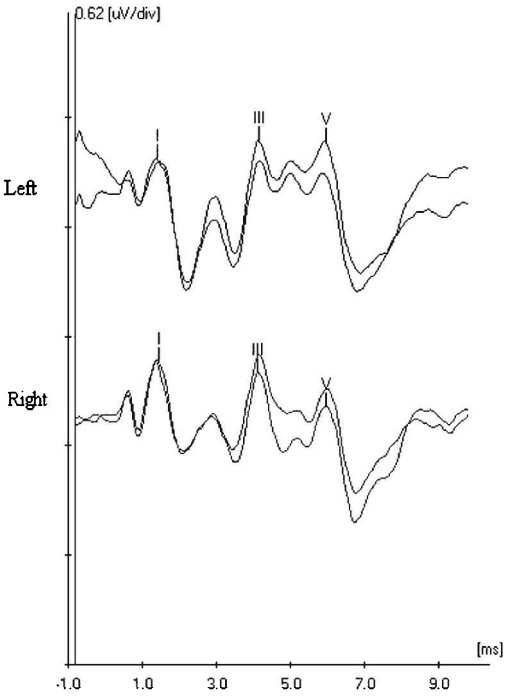

Figure 21–6. A normal ABR to clicks was obtained for the right and left ears.

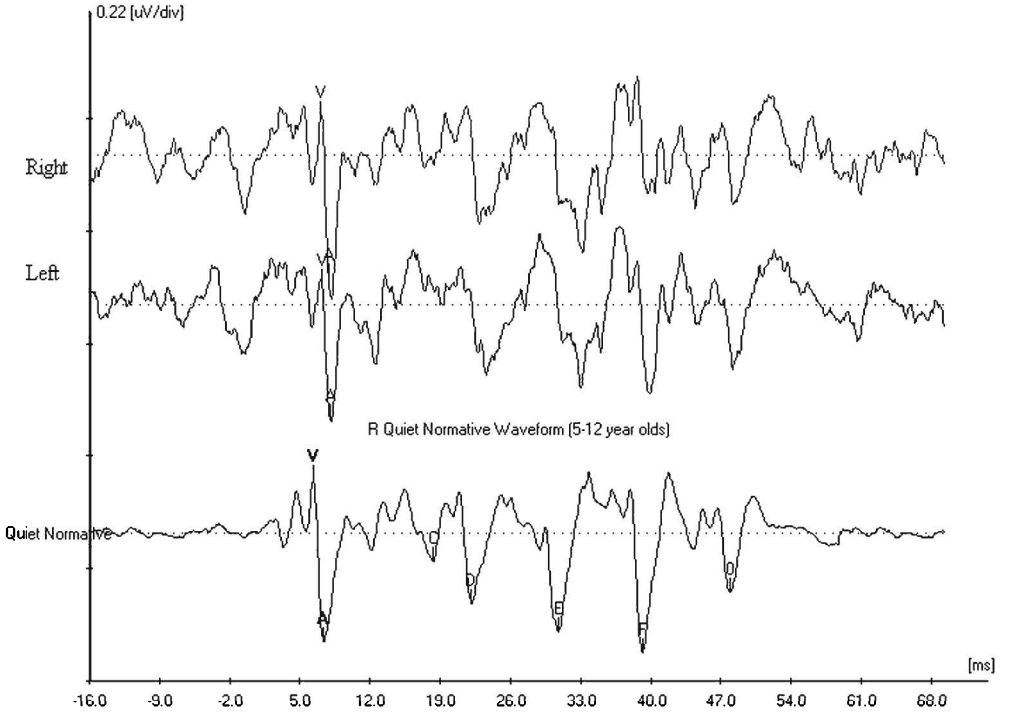

Figure 21–7. An abnormal cABR was obtained for the right and left ears. Also depicted is a normative cABR waveform.

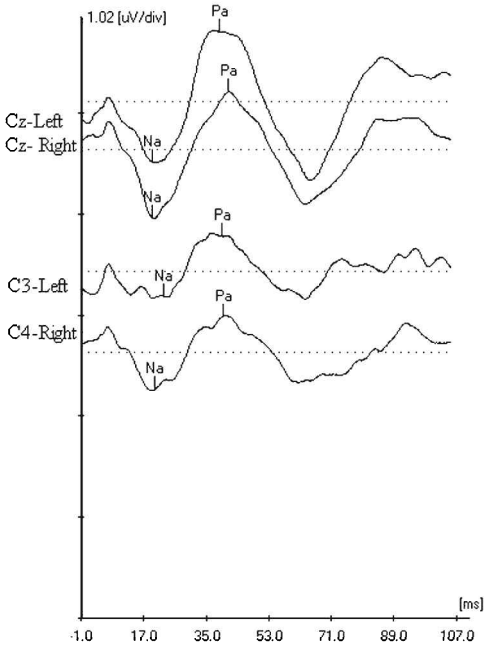

Figure 21–8. The AMLR recorded from Cz to ipsilateral right and left ears and C3 and C4 referenced to the ipsilateral ear are depicted. Each waveform is the summed response of two individual recordings for each condition.

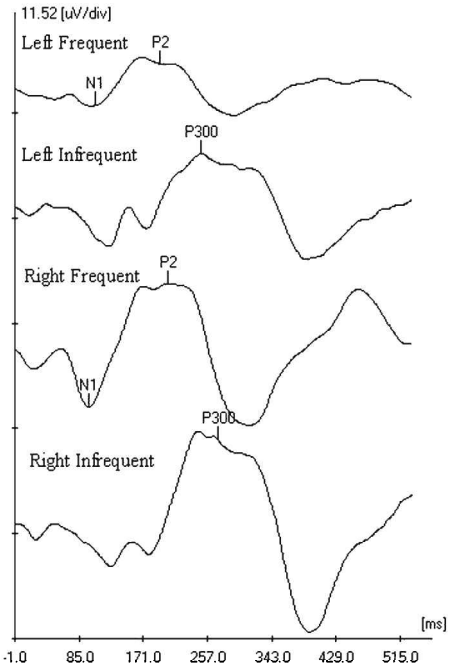

Figure 21–9. ALER recordings from Cz to the ipsilateral right and left ears are shown. Each waveform is the summed response of two individual recordings for each condition.

Recommendations

Alexandra will continue speech-language therapy. Formal dichotic listening training was recommended. Alexandra will be followed annually for repeat behavioral and electrophysiological testing.

Debriefing

- What CAPD risk factors were present?
  Developmental delays in expressive and receptive language development gross and fine motor development, and ADHD. Evidence of damage to right temporal lobe was noted on the MRI.

- Discuss Alexandra's performance on dichotic listening tests in relation to her MRI.
  A left ear deficit was noted on dichotic speech tests. This is in agreement with Kimura's (1961) reports of decreased scores in the ear contralateral to the anatomical lesion.

- When would follow-up be recommended?
  We would like to follow this patient's progress and auditory maturation annually through age 12. We will reassess her central auditory processing ability as she completes dichotic listening therapy.

- What is the significance of the addition of electrophysiological recordings in this case?
  Electrophysiological recordings can corroborate behavioral central auditory test results. The abnormal cABR is objective evidence of the abnormal neural encoding of speech at the level of the brainstem. Symmetrical AMLRs were recorded and interpreted as normal. Additional recording sites of the P300 may provide laterality information. Amplitudes of the N1–P2 and P300 response were greater for right ear stimulation than for left ear stimulation, consistent with the MRI and behavioral test data. (See Musiek, Baran, & Pinheiro, 1992 and Knight, Scabini, Woods, & Clayworth, 1989 for additional information on the P300 recording with lesions of the auditory cortex.)

Case Study 8

Background

Dylan is an 11-year-old fifth-grade boy who has been diagnosed with dyslexia and expressive and written language disorder. A normal pregnancy was reported and developmental milestones were achieved at appropriate ages. There is no history of otitis media or jaundice. A familial history of dyslexia (father, older half-brother, and maternal uncle) was reported. Dylan attends a private school, where he has received resource services since third grade. Currently, Dylan receives private reading tutoring three times per week, and is working through a multisensory reading program, Orton-Gillingham. He has previously completed Fast ForWord Language and Fast ForWord Language-to-Reading programs. Dylan recently completed a psychoeducational assessment with intelligence and memory reported to be in "superior" range. With accommodations, he maintains an A-B average. Dylan is extremely active and enjoys athletics. He plays baseball, basketball, and tennis, participates in karate, and plays piano. Dylan's mother reports he has no hearing difficulties, but he has difficulty listening and understanding language.

Assessment

Normal peripheral hearing was established bilaterally prior to the central auditory processing assessment. Test results, shown in Table 21–13, revealed performance within normal limits on all central auditory tests administered. To further explore decoding skills, the Phonemic Synthesis Test (Katz & Harmon, 1982) was administered and results indicated poor phonological processing skills. A normal ABR was recorded for the right and left ears, as shown in Figure 21–10. An abnormal cABR, shown in Figure 21–11, was recorded bilaterally, providing objective evidence of abnormal neural encoding of speech.

Recommendations

No audiological recommendations were offered. Dylan has previously completed several computer-mediated auditory training programs in the past and yet his cABR remains abnormal. Dylan will continue to receive reading tutoring and resource services. He is working through a multisensory reading program which may help with his dyslexia.

Table 21–13. Behavioral CAPD Test Results for Case 8 (Dylan)

SCAN-3:C Auditory Figure Ground	SCAN-3:C Filtered Words	SCAN-3:C Competing Words	SCAN-3:C Competing Sentences	SCAN-3:C Time Compressed Speech	Dichotic Digits		Frequency Patterns	Gap Detection Threshold	Phonemic Synthesis Test
Standard Score	Standard Score	Standard Score	Standard Score	Standard Score	Left	Right	Left Verbal	Right	Binaural
12	12	10	10	10	92	96	100%	3 msec	Quantitative score = 12 Abnormal

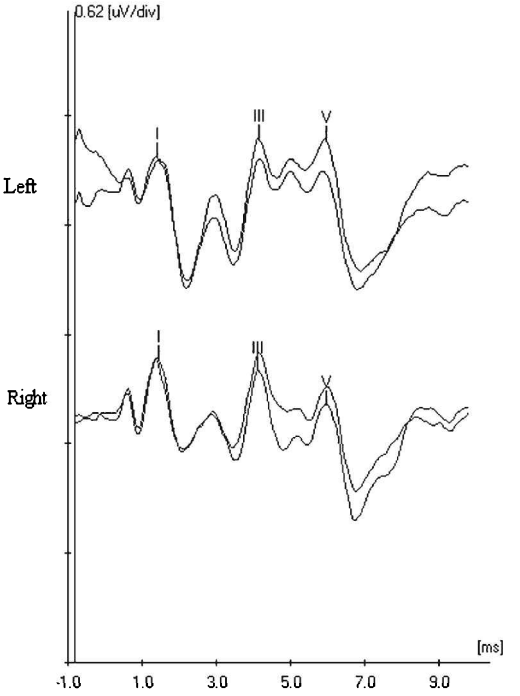

Figure 21–10. A normal ABR was obtained for the right and left ears.

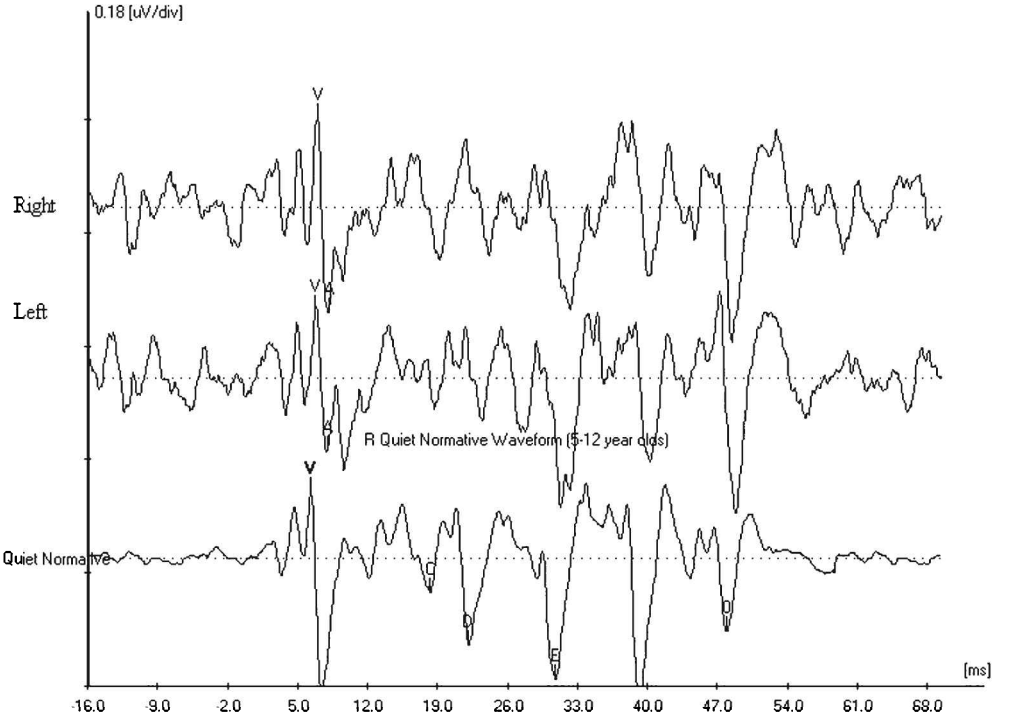

Figure 21–11. An abnormal cABR was obtained for the right and left ears. A normative cABR is also shown.

Debriefing

- What risk factors for CAPD are present?
  Dylan has been diagnosed with dyslexia. A familial history of dyslexia is present. No other medical risk factors are reported.
- Would you continue to follow this patient? Why or why not?
  Dylan presents with normal central auditory processing function based on performance on the behavioral central auditory processing test battery. His cABR was abnormal, indicating abnormal encoding of speech stimuli. Just as a normal cABR will not change the diagnosis of CAPD for a patient who presents with abnormal performance on a behavioral central auditory processing test battery, an abnormal cABR recorded for a patient with a normal behavioral central auditory processing test battery does not warrant a diagnosis of CAPD. The abnormal cABR is not characteristic of a specific type of learning disorder, such as CAPD, but is found in approximately 30% of patients with a language-based learning disability (Johnson, Nicol, & Kraus, 2005; Russo et al., 2010; Wible, Nicol, & Kraus, 2004).
- What is significant about Dylan's music training?
  Musicians are thought to have enhanced working memories and auditory processing skills, leading to the conclusion that early musical training may be advantageous. Readers are referred to the work of Kraus and colleagues at brainvolts .northwestern.edu and Chapter 7 for discussion of music training and brain function.

Case Study 9

Background

Lilly, an 8-year-old female, was referred for central auditory processing assessment. Lilly's mother, an audiologist, referred her for "the official diagnosis of CAPD" and for additional audiological recommendations. Lilly was born three weeks premature. Developmental milestones were achieved at the upper range of normal. She received occupational therapy for development of fine motor skills and was diagnosed with *sensory integration deficits* by an occupational therapist. She has received speech-language therapy since age 3. Lilly currently is receiving vision therapy. She is in second grade at a Montessori school. Lilly's mother reports no academic concerns at the present time, but feels there are some underlying auditory processing concerns, such as slow response to auditory stimuli, and misunderstanding words and song lyrics. Her mother reports that she has provided Lilly with many years of informal and formal auditory training, including listening activities, computer-mediated auditory-language training programs, and language enrichment programs.

Assessment

Results of individual tests, shown in Table 21–14, reveal a left ear deficit on dichotic speech tests. Interhemispheric dysfunction was inferred given the left ear dichotic deficit coupled with abnormal performance on the labeling condition of the Frequency Pattern Test. A normal ABR and cABR were recorded for the right and left ears.

Table 21–14. CAPD Behavioral Test Results for Case 9 (Lilly)

SCAN-3:C Auditory Figure Ground	SCAN-3:C Filtered Words	SCAN-3:C Competing Words	SCAN-3:C Competing Sentences	SCAN-3:C Time Compressed Speech	Dichotic Digits		Frequency Patterns		Gap Detection Threshold
Standard Score	Standard Score	Standard Score	Standard Score	Standard Score	Left	Right	Left Verbal	Left Hummed	Right
12	12	4	5	11	48%	90%	60%	100%	3 msec
Normal	Normal	Abnormal Left ear deficit	Abnormal Left ear Deficit	Normal	Abnormal Left ear Deficit		Abnormal Indicates interhemispheric dysfunction		Normal

Recommendations

Lilly will continue to receive occupational therapy, speech-language therapy, and vision therapy. Lilly was diagnosed with CAPD. Dichotic listening therapy was implemented to improve left ear performance, as well as exercise interhemispheric function. We plan to follow Lilly's progress in six months.

Debriefing

- What risk factors for CAPD are present?
  Risk factors include prematurity, fine motor delay, and diagnosis of sensory integration disorder. Lilly also is receiving visual integration therapy.

- When would you suggest follow-up?
  Lilly will begin dichotic listening therapy. She will be retested in six months. We routinely recommend reassessing central auditory processing function every year to assess progress and monitor normal maturation of the CANS.

Case Study 10

James, an 8-year-old third-grade boy, was referred by his psychologist for central auditory processing assessment. James was delivered after a term pregnancy by C-section with the umbilical cord wrapped around his neck; emergency oxygen was administered. He has a diagnosis of ADHD-inattentive type and anxiety, for which he is medically managed. James presents a receptive language disorder and written language disorder and is currently in private speech-language therapy. He also has poor motor coordination and has been receiving occupational therapy since age 3.

Assessment

Normal peripheral hearing testing was established bilaterally prior to administration of the central auditory test battery. Results are shown in Table 21–15. James performed within normal limits for all behavioral tests of central auditory processing. A normal ABR and abnormal cABR also were obtained.

Recommendations

James will continue to receive speech-language therapy. There are no audiological recommendations. The abnormal cABR is objective evidence of differences in the neural encoding of speech but does not in itself provide for the diagnosis of CAPD.

Debriefing

- What risk factors for CAPD are present?
  James has a history of anoxia and diagnosis of receptive language disorder and written language disorder.

- Why is the cABR abnormal in this case?
  The underlying assumption of an abnormal cABR is that the response represents abnormal neural encoding of the temporal and spectral characteristics of the acoustic signal presented. If the signals are not represented correctly at the level of brainstem, this can lead to or be associated with difficulty in speech perception abilities. The cABR may

Table 21–15. Behavioral CAPD Test Battery Results for Case 10 (James)

Speech-in-Noise		Filtered Words		Competing Sentences		Dichotic Digits		Frequency Patterns	Duration Patterns	Gap Detection Threshold	Masking Level Difference
Left	Right	Left	Right	Left	Right	Left	Right	Left verbal	Left Verbal	4 msec	10 dB HL
94%	94%	88%	90%	90%	80%	78%	80%	90%	90%		
Normal		Normal		Normal		Normal		Normal	Normal	Normal	Normal

be able to isolate temporal and spectral encoding problems in some children with CAPD or language-based learning deficits (Johnson et al., 2005; Russo et al., 2010).

Case Study 11

Background

Casey is an 11-year-old female fifth-grade student with an interesting case history. Although pregnancy and birth history were unremarkable, it was later discovered that Casey had suffered a left temporal-parietal infarct in utero. This resulted in the limited use of her right hand and intractable epilepsy. Ongoing seizures continued and increased in severity, requiring emergency cardiopulmonary resuscitation even though pharmaceutical management was followed. A functional modified left hemispherectomy was performed when Casey was 9 years old.

Psychological assessments obtained pre- and posthemispherectomy reported Casey to be within the average range of intellectual abilities on the following measures: verbal comprehension, perceptual reasoning, and working memory. Casey's academic success has been in part attributed to support services including speech-language therapy and private tutoring two times per week, as well as weekly, private occupational and physical therapy. The purpose of the audiological referral was to determine if there were any auditory processing problems, and if so, provide recommendations that might further support Casey's academic success.

Assessment

Normal peripheral hearing was established bilaterally, prior to administration of the central auditory test battery. Results of Casey's test results are shown in Table 21–16. Abnormal performance for the right ear for dichotic listening tasks is consistent with the anatomical left site of lesion. Casey performed within normal limits on monaural low-redundancy speech tests. The ABR was recorded from binaural and monaural stimulation and is shown in Figure 21–12. Amplitude and latency values, shown in Table 21–17, are within clinical normative limits. A normal cABR also was recorded and is shown in Figure 21–13. Wave V and A latency and algorithm scores are shown in Table 21–18. An electrode effect for the left temporal lobe C3 was seen on the AMLR (Figure 21–14). AMLR latency and amplitude values are shown in Table 21–19. The ALER was within normal latency values and is shown in Figure 21–15. Latency and amplitude values are shown in Table 21–20.

Recommendations

Casey should continue to receive academic support services. Dichotic listening therapy was recommended; however, Casey's mother declined therapy at the present time, but may consider this in the future.

Debriefing

- Are the dichotic scores consistent with the left functional hemispherectomy?
  Yes. Kimura (1961) reported depressed dichotic scores for the ear contralateral to the site of lesion.

Table 21–16. CAPD Test Battery Results for Case 11 (Casey)

Speech-in-Noise		Filtered Words		Time Compressed Speech		Dichotic Digits		Competing Sentences		Frequency Patterns		Duration Patterns		Gap Discrimination (3-interval forced choice)	Masking Level Difference
Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	2 msec	10 dB HL
90%	94%	98%	98%	90%	88%	98%	24%	100%	0%	80%	80%	80%	82%		
Normal		Normal		Normal		Abnormal Right Ear Deficit		Abnormal Right Ear Deficit		Normal		Normal		Normal	Normal

Therefore, a left hemispherectomy would indicate abnormal right scores.

- Discuss the inclusion of electrophysiological recordings in this case?
  Including electrophysiological measures in the central auditory evaluation may aid in the diagnosis and/or in validating the results of the behavioral test battery. The addition of electrophysiologic tests may provide useful information pointing to possible gross site of CANS dysfunction. A normal ABR response was obtained. This obligatory response is mediated by structures from the distal portion of the VIIIth nerve through the superior olivary complex, lateral lemniscus and inferior colliculus. These normal responses are not surprising as the generators from this response are in the brainstem and midbrain and not anatomically affected by the functional hemispherectomy. An electrode effect for the left temporal lobe C3 was indicated by the AMLR which is consistent with Kraus, Ozdamar, Hier, and Stein's (1982) report of diminished Pa amplitude over the lesioned area in 24 patients with temporal lobe lesions. The ALER was within normal latency values.

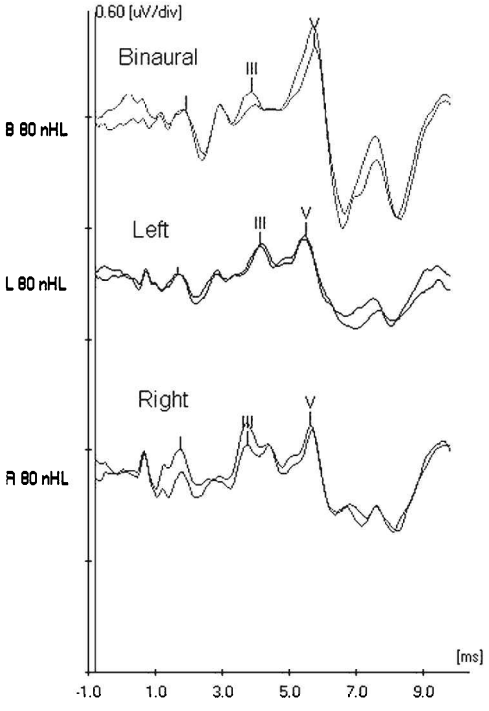

Figure 21–12. A normal ABR was obtained for binaural, left, and right stimulations.

- Would you continue to follow this patient?
  We will continue to follow Casey's progress and would like to retest her annually. It will also be important to monitor progress if Casey decides to begin dichotic listening therapy.

Table 21–17. ABR Latency and Wave V Amplitude Measures for Case 11 (Casey)

	I in msec	III in msec	V in msec	V Amplitude in μV
Binaural	1.91	3.86	5.70	.97
Right	1.74	3.74	5.70	.36
Left	1.66	4.11	5.49	.45

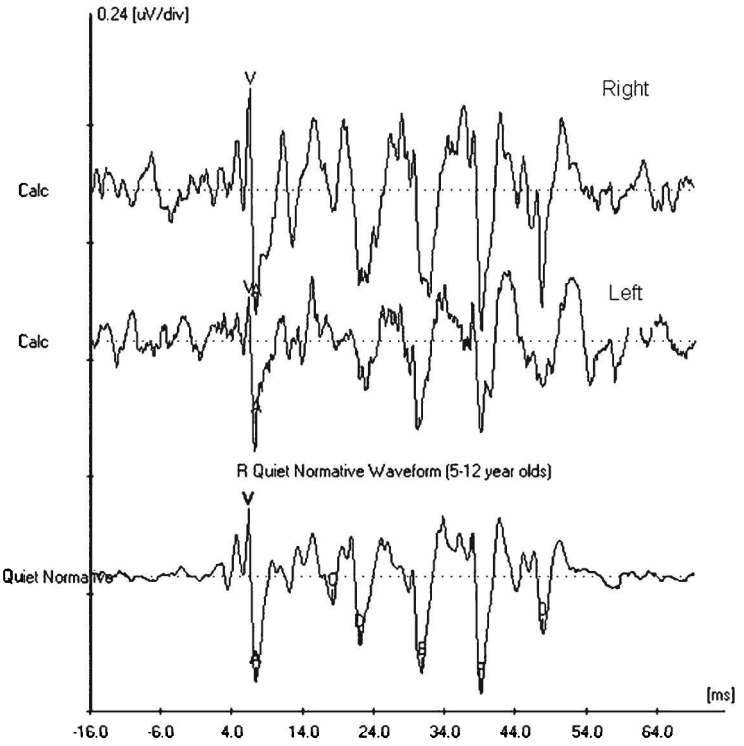

Figure 21–13. Normal cABR recordings for the right and ears are shown.

Table 21–18. Wave V and A Latency for the Speech ABR

	V in msec	A in msec
Right	6.53	7.45
Left	6.45	7.45

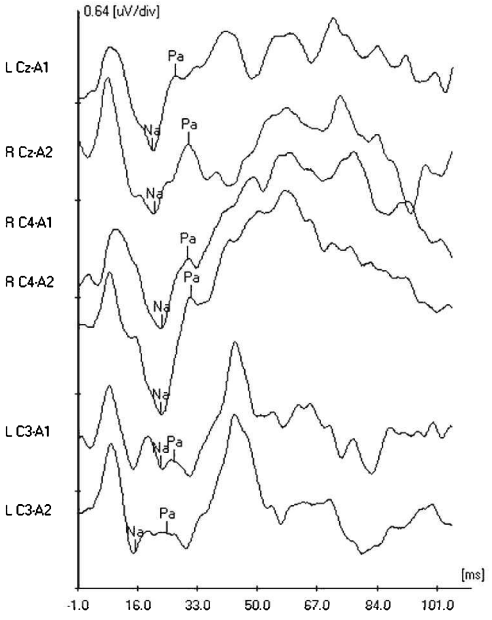

Figure 21–14. Left and right AMLR recordings at electrodes Cz, C3, and C4 are shown in the figure. Shown are the summed responses from two individual recordings. This is a fused Pa–Pb wave making interpretation difficult.

Table 21–19. Latency and Amplitudes for the AMLR. An electrode effect was evident for recording over the left temporal lobe (electrode site C3).

Electrode	Stimulus Ear	Na latency in msec	Pa latency in msec	Na-Pa amplitude in μV
Cz	Left	20.02	26.68	.49
Cz	Right	20.85	30.64	.45
C4	Left	22.94	30.22	.44
C4	Right	22.52	31.06	.76
C3	Left	22.52	26.47	.08
C3	Right	15.23	24.39	.12

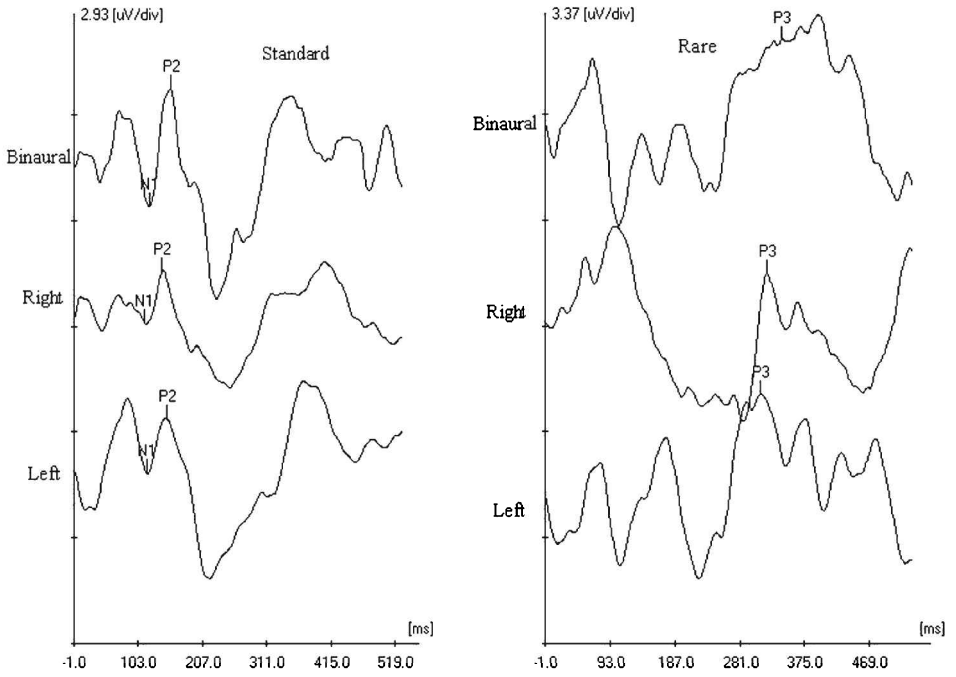

Figure 21–15. Binaural, right and left ALER and P300 tracings recorded at Cz. Displayed are the summed responses from two individual recordings.

Table 21–20. Latency and Amplitude of the P300 Recording

Ear	N1 latency in msec	P2 latency in msec	N1/P2 amplitude in μV	P300 latency in msec	P3 amplitude in μV
Binaural	121	155.35	3.25	304.22	1.83
Right	123.71	144.94	1.44	319.60	1.91
Left	116	144.94	1.41	311.50	2.97

Case Study 12

Background

Bob is a 10-year-old male in fourth grade, referred to our clinic as a medical/legal case to rule out ANSD. Bob was born at 36 weeks gestational age. He was discharged from the hospital after 48 hours, but was admitted two days later as his mother returned to the emergency room (ER) with reports of possible seizures. Bob remained in neonatal intensive care unit (NICU) for two weeks. He was diagnosed with kernicterus and received double volume transfusions and extensive phototherapy while in the NICU. An electroencephalogram (EEG) showed some seizure activity in the frontal and left temporal lobes as a result of elevated bilirubin levels. Developmental milestones were reported at appropriate times. Bob receives physical, occupational, and speech therapy at school. He has also received vision therapy at some time in the past. Previous hearing assessments have indicated normal peripheral hearing as indicated by present transient otoacousic emissions, bilaterally. His mother reported he has difficulty following multistep directions and keeping up with the flow of conversation. She also reported that Bob's teacher has described his behavior as disobedient, failing to consider that Bob might have a hearing problem. She reported that a psychoeducational assessment completed several years ago reported normal cognitive ability.

Assessment

Normal (type A) tympanograms were obtained bilaterally. A bilateral, mild low-frequency sensorineural hearing loss was indicated by pure-tone thresholds, as shown in Table 21–21. Ipsilateral and contralateral acoustic reflexes were absent bilaterally. Transient otoacousic emissions were absent, bilaterally. Behavioral test results, depicted in Table 21–22, show temporal processing deficits, as well as auditory deficits when listening to degraded speech, bilaterally.

Electrophysiological tests provided objective evidence of abnormal processing. The ABR waveform contained the sustained ringing that reflects the phase characteristic of the signal, which is a diagnostic marker of ANSD (Figure 21–16). Wave V was repeatable, and occurred at a normal latency. Interestingly, the left ABR was out of phase until wave III, occurring around 3.86 msec, whereas the right ABR was out of phase until wave V, occurring at approximately 5.65 msec.

An abnormal cABR was obtained, bilaterally. The cABR waveform for the right ear and a normative waveform are

Table 21–21. Audiometric Thresholds in dB HL for Case Study 12 (Bob)

Frequency (Hz)	250	500	1000	2000	4000	8000
Right air conduction	40	35	20	10	10	5
Left air conduction	40	35	20	10	10	5
Unmasked bone	25	35	15	5	5	

Table 21–22. CAPD Test Results for Case 12 (Bob)

Speech-in-Noise		Filtered Words		Time Compressed Speech		Dichotic Digits		Competing Sentences		Frequency Patterns	Duration Patterns	Gap Detection Threshold	Masking Level Difference
Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Left	20 msec Inconsistent Responses	4 dB HL
20%	34%	48%	52%	20%	28%	80%	88%	80%	80%	80%	80%		
Abnormal		Abnormal		Abnormal		Normal		Normal		Normal	Normal	Abnormal	Abnormal

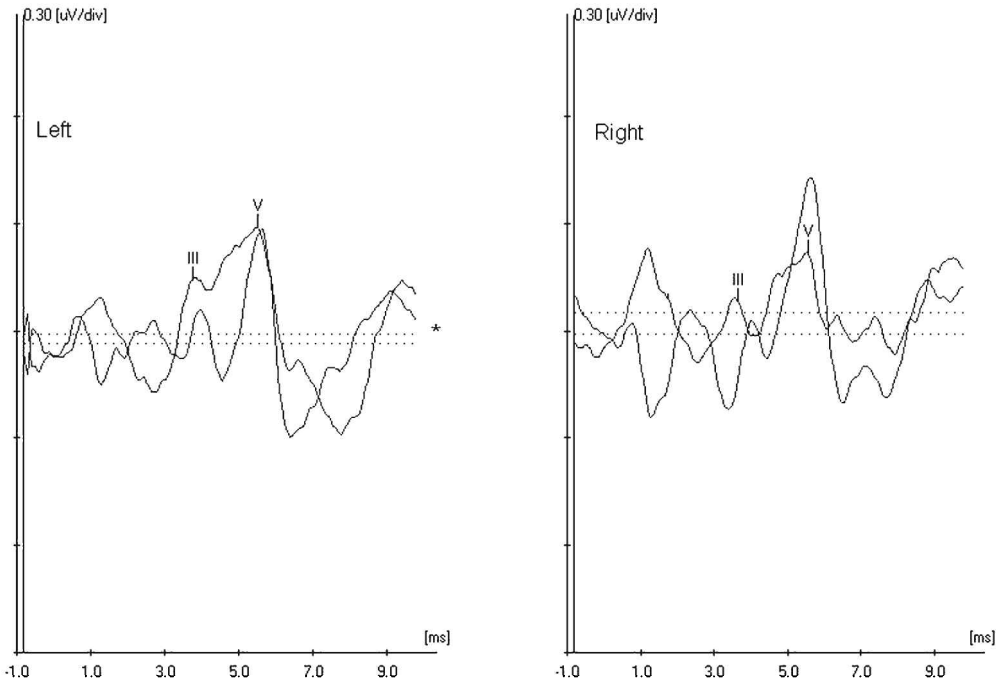

Figure 21–16. An ABR to condensation and rarefaction clicks for the right and left ears are shown.

displayed in Figure 21–17. Latencies and amplitudes are shown in Table 21–23. The AMLR was obtained for the right and left ear using a two-channel montage, recording from Cz to the ipsilateral ear. Latency and amplitude values are shown in Table 21–23. The AMLR was delayed. It is important to note Bob is 10 years old and this delay may reflect a slight delay in the maturation of the auditory system. Clinical analysis of the AMLR is more concerned with amplitude than latency (Chermak & Musiek, 1997). The Na–Pa amplitude was within normal limits and symmetrical between ears as seen in Figure 21–18.

The P300 response reflects electrical activity from multiple areas, including the medial temporal lobe, limbic structures, frontal and parietal lobe, and temporal-parietal junction (Hall, 2007; Knight et al., 1989). The P300 recorded for left and right ear stimulations is shown in Figure 21–19. The recording is symmetrical and interpreted as within normal limits. Amplitude and latency values are shown in Table 21–23.

Recommendations

The results of the audiological assessment indicate ANSD. This is evidenced by the characteristic out-of-phase ringing of the ABR. Although this was a medical-legal referral, there are additional educational recommendations to consider. First, environmental modifications should be employed, including strategic classroom seating and testing in a quiet area free of distractions. A trial with an FM system should begin to enhance the signal-to-noise ratio at Bob's ear. Visual

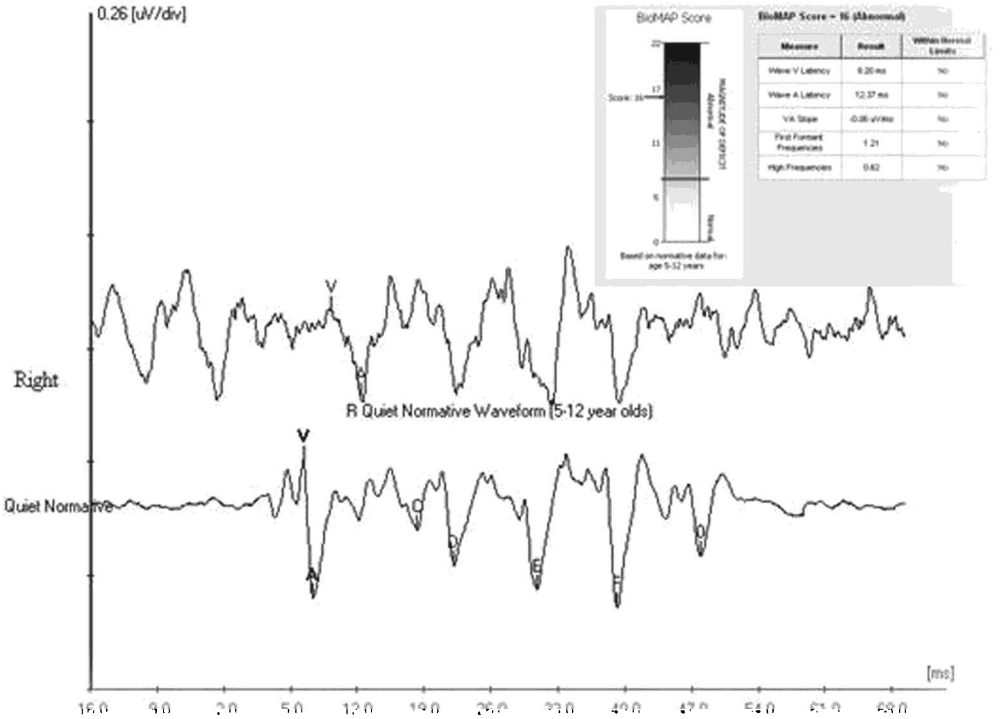

Figure 21–17. An abnormal cABR waveform and algorithm response from the right ear is shown.

aids should be given whenever possible. Gaining Bob's attention before giving directions is necessary. Moreover, if Bob does not follow a verbal direction, it is emphasized that this should not be attributed to defiant or disobedient behavior, but rather to his auditory deficits. Auditory therapy to address deficits in auditory closure and binaural interaction processes was recommended. A speech-language assessment is recommended. It is important that Bob's mother recognize that Bob should be informed of his auditory deficit so that he may self-advocate ad become active in his own treatment plan. At the present time, she does not want him to know "there is anything wrong with him." Metacognitive strategies are an important part of the rehabilitation plan and should include strategies such as recognizing difficult noise situations and problem solving, as well as self-advocacy skills. Bob will return to the clinic in six months for follow-up.

Debriefing

- Are the risk factors the same for CAPD and ANSD?
  CAPD and ANSD share many of the same risk factors such as hyperbilirubinemia, prematurity, anoxia, and so forth.

- What is the significance of the absent OAEs and the diagnosis of ANSD?
  ANSD is a condition characterized by normal OAEs; however, OAEs may deteriorate over time (Deltenere

Table 21–23. Amplitude and Latencies of the Electrophysiological Recordings for Case 12 (Bob)

	V in msec	V in msec	A in msec	Na in msec	Pa in msec	Na–Pa' in μV	N1 in msec	P2 in msec	N1–P2' in μV	P300 in msec	P300 in μV
Left	5.65	9.3	12.27	18.15	38.34	1.12	102.26	133	2.22	333.37	2.89
Right	5.49	9.2	12.23	22.73	38.97	1.25	96	139.74	3.16	333.37	3.92

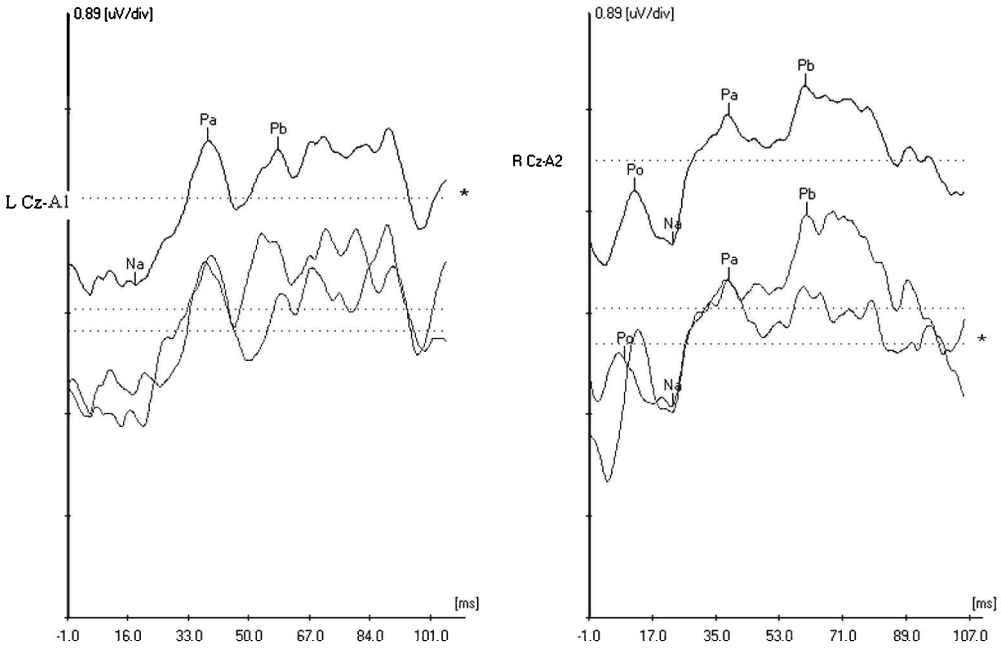

Figure 21–18. The AMLR recorded from Cz to the ipsilateral ear is shown. The summed response is displayed above the individual recordings.

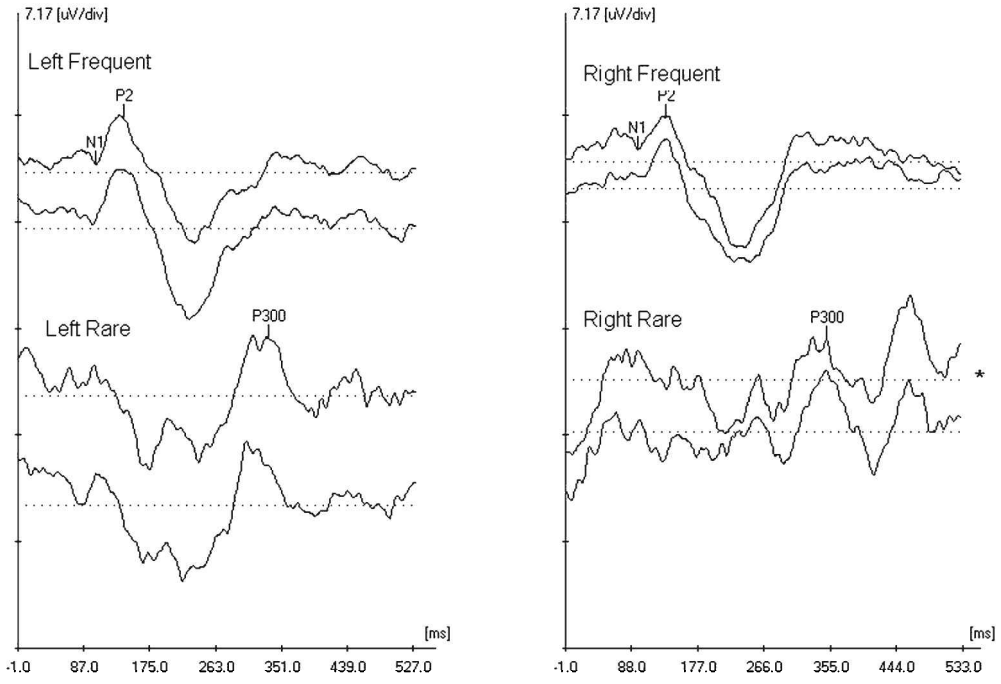

Figure 21–19. The ALER and P300 response for the right and left ears are shown.

et al., 1997; Starr et al., 1996). Although an audiological evaluation conducted when Bob was 3 years old reported "normal hearing" on the basis of present OAEs (ABR was not attempted at that time), OAEs were absent when examined at age 10 years.

- Which central auditory processing behaviors are negatively affected?
  Tests of monaural low-redundancy speech tests, as well as temporal resolution (gap detection thresholds) and binaural interaction tests were abnormal.

- Although this was a medical legal case, there were other educational recommendations to consider. What affect does a mild hearing loss have on listening and learning?
  A mild hearing loss can negatively impact learning. Furthermore, patients with ANSD have difficulty with speech recognition, especially in degraded acoustic environments. Environmental modifications such as an FM system are recommended.

- Would you recommend obtaining electrocochleography (ECochG) recordings?
  Research has shown that ECochG recordings may be useful in determining pre- or postsynaptic site of dysfunction. This may also be important when considering cochlear implant candidacy at a future time.

- Would you repeat any or all of the behavioral and electrophysiologic recordings in subsequent appointments?
  We recommend annual hearing assessments for any patient with hearing impairment. It is possible hearing thresholds may change. We also would like to monitor auditory maturation and would like to reassess central auditory function annually. For best practice we would continue to repeat all electrophysiological recordings and include an extratympanic ECochG recording.

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SECTION 6

[redacted]re Directions

CHAPTER 22

THE CANS AND CAPD: WHAT WE KNOW AND WHAT WE NEED TO LEARN

DENNIS P. PHILLIPS

Introduction

The second edition of this book is written at a particularly interesting time in the evolution of our understanding of central auditory processing disorder (CAPD). At least two intersecting factors contribute to this timeliness. One is a recent history of piercing and helpful inquiry about how we should conceptualize CAPD (e.g., Cacace & McFarland, 1998, 2005; Moore et al., 2010; Rosen, 2005). The second is a broadening of our conceptualization of what constitutes central auditory processing (CAP). The conclusions that the hearing science community draws from those inquiries will have significant impacts for how CAPD is to be diagnosed, and therefore for the choice and design of interventions for the disorder.

The purpose of this chapter is to survey both of those inquiries, and so provide a context for the other chapters in this book. For reasons of brevity, our review is necessarily somewhat selective, but we have tried within that constraint to be as broad in the ideas covered as we can. We hope to alert the reader to the range and depth of issues that could, and perhaps should, influence our conceptualization of CAP and therefore CAPD. It is perhaps inevitable that some of the authors' personal biases will flavor the particular issues we cover and the conclusions we draw from them. We encourage the reader to be open, curious, critical and mindful in their reading of what follows.

Central Auditory Processing

The auditory world unfolds over time. Surely, the most basic function of the central auditory nervous system (CANS) is to establish a neurophysiological representation ("neurological picture") of the stimulus that updates with the stimulus, and that by some means leads to the generation of our continuous, private, mental experience of the stimulus.

One historical approach to understanding central auditory processing, and therefore disorders of it, implicitly or explicitly treats the CANS in isolation. Much has been learned from neurophysiological studies in animals about the physiology (and therefore, the presumed behavioral function) of many of the core nuclei of the classical central auditory pathway (see chapters in the Auditory Neuroscience section of this volume). As an example, we now have a wealth of information about the roles of the medial and lateral superior olivary nuclei in encoding the interaural disparities in stimulus phase and level, respectively. We also know about some of the ways in which that encoding is modified at centers above the superior olive —notably the shift from a representation of the instantaneous interaural phase to one that is sensitive to the immediate acoustical history and thus to the direction of interaural phase change (Spitzer & Semple 1998). And we certainly know about the contralateral bias in spatial representation in CANS centers rostral to the outputs of the superior olivary nuclei (Middlebrooks & Pettigrew, 1981; Semple et al., 1983; Stecker et al., 2005).

These neurophysiological findings have behavioral correlates, and we cite the following examples. Animals with poorly developed medial superior olives have poor interaural phase sensitivity (Masterton, Thompson, Bechtold, & Robards, 1975). In man, demyelinating disease that affects the medial superior olive also affects perceptions based on interaural time (Hausler & Levine, 1980). Auditory neuropathy, which is characterized by impaired central neural timing (see Cowper-Smith et al., 2010) critical for the coding of interaural phase, also impairs interaural phase discriminations but not interaural level ones (Zeng et al., 2005). The consequences of unilateral lesions of CANS nuclei or the auditory cortex are typically restricted to impoverished sound localization performance in the acoustic hemifield contralateral to the lesion in both man (Sanchez-Longo and Forster, 1958; see also Dingle et al., 2012) and animals (ferrets: Kavanagh & Kelly, 1987; cats: Jenkins & Masterton, 1982; primates: Heffner, 1997).

A similar case might be made for the role of the primary auditory cortex in certain time-based discriminations. The neurophysiological data are that the input to the primary auditory cortex has preserved the precision of transient event timing seen in the cochlear nerve (Phillips and Hall, 1990) but has a much poorer temporal representation of stimulus periodicities than does the nerve (cf. Eggermont, 1991; Joris and Yin, 1992). In man, damage to the primary auditory cortex appears to have far more deleterious effects on the perception of transient event timing than on percepts based on periodicities (see Phillips and Farmer, 1990, for review; see also Stefanatos et al., 2007). This difference may be expected if the cortex has a relatively poor temporal representation of stimulus periodicities but has preserved acute timing of transients.

These two examples show an encouraging correspondence between the neural coding properties of the relevant CANS region(s) and the behavioral consequences of damage to those centers. Note, however, that all of this kind of evidence fails to address a crucial issue. The evidence tells us that there is good correspondence between the properties of the "neurological picture" and the properties of some auditory behaviors, but it says little or nothing about how the neurological picture comes to generate a conscious percept. If, as hearing scientists, we are concerned with what the listener "hears," then we have to address this issue. Recall that most of the studies of the neurophysiological coding of stimulus parameters have been conducted on animals under deep surgical anesthesia; these animals perceive nothing during the experiment.

In this regard, neural information from the cochlea probably reaches the auditory cortex in a matter of 10 to 20 ms (Celesia, 1976), and cochlear output is updated with better than millisecond precision. Nevertheless, it is easily shown, even using stimuli as simple as click trains, that conscious percepts (e.g., the spatial location of a particular click within a train) are profoundly influenced by events (clicks) within a temporal window at least a third of a second wide, including events that occur after the target click (e.g., the auditory saltation illusion; see: Phillips & Hall, 2001; Ishigami & Phillips, 2008; Phillips, 2012; see also Dennett, 1991). What perceptual processes are involved in this? Which brain regions mediate those processes? With over 300 ms to account for, one can well imagine that the brain regions involved extend far beyond the isolated, classical CANS. This is indeed the case (Posner, 1994; Hillyard et al., 1998).

This is our first hint that the CANS does not act in isolation. But the generation of a conscious percept is only one component of central auditory processing. It is likely that any operations executed on the auditory neural data stream will engage processes mediated by brain regions outside the classical CANS. Those operations are diverse: Sorting auditory sources that are concurrently active (i.e., "streaming," after Bregman, 1990), discriminating one sound as same or different from another, discriminating the temporal order of sounds, identifying a sound, recognizing the sound as the one the experimenter instructed you to listen for, establishing an emotional response to a sound—all of these operations involve brain regions in addition to those of the classical CANS. These assertions are supported by anatomical studies showing connectivity between the CANS and the limbic system (Sah et al., 2003), the motor system (Reale & Imig, 1983), spatial attention systems (Rauschecker, 1998), and prefrontal cortex (Romanski et al., 1999). Awareness of these points is perhaps one of the driving forces behind the wish to develop clinical auditory tests with the lowest possible cognitive "overhead," that is, clinical probes of CAP function that target the classical CANS as selectively as possible.

A second line of evidence on the interplay between the classical CANS and higher level processing in audition comes from studies of the descending auditory system. The auditory cortex is the origin of numerous and extensive, direct and indirect, descending pathways that target almost every nucleus in the classical CANS, including the cochlea (Winer, 2006). These pathways are often

conceptualized as topographically organized feedback loops through which ascending sensory neural information is modulated by descending control (e.g., Malmierca & Ryugo, 2011; Suga, 2012; Suga & Zhang, 1997). Broadly speaking, the modulation is expressed as an enhancement of responses to behaviorally relevant stimuli, and/or shifts of neural tuning toward the target stimulus and/or presumably through lateral inhibitory processes, a suppression of activity in topographically or functionally more remote loci (see especially Malmierca & Ryugo, 2011; Suga, 2012).

Now, there is no doubt that auditory cortical activity is under attentional control in humans (e.g., Petkov et al., 2004) and in animals (Lee & Middlebrooks, 2010; Walker & King, 2011). In practice at the single-neuron level, the modulations effected by behaviorally controlled attentional processes on the stimulus selectivity of cortical neurons is often a facilitation of responses to target stimuli and/or a suppression of activity at functionally adjacent or remote ones (e.g., Lee & Middlebrooks, 2010; see also Walker & King, 2011). If we construe these effects as the result of activity in the feedback loops between the auditory cortex and the brainstem, then it follows that any interference with ascending information flow in the auditory system also affects descending influences—either because the interference functionally impairs the descending pathway(s) at the site of interference or because the impaired corticopetal transmission deprives the feedback loops of their normal input. It is thus likely that data-driven and higher-level auditory/cognitive processing in the awake listener may not be as easily separable as one might have imagined.

Bottom-Up and Top-Down Processes in Auditory Perception

The preceding lines of argument prompt us to ask how top-down processing is expressed in human hearing. Let us address this in a couple of ways. Scharf and colleagues (1994, 1997) have developed a conceptually simple task requiring listeners to detect a low-amplitude tone of a target frequency against a background of continuous noise. On each trial, the listener is first cued to the target frequency by presentation of a tone of that frequency, delivered at a somewhat higher amplitude; the task of the listener is to specify in which of two subsequent intervals a threshold-level (probe) tone is presented. Intact listeners typically perform at high levels for probe tones of the target frequency, although the task probably requires considerable attentional resources because of the low amplitudes of the probe tones. Such listeners have poorer detection rates for probe tones of unexpected frequencies; their performance appears "tuned" to the target frequency. One interpretation of these findings is that they are an expression of selective attention in the frequency domain.

Scharf et al. (1994, 1997) extended their work to patients undergoing vestibular neurectomy to relieve intractable vertigo. What makes this so interesting is that the surgical section of the vestibular nerve also involves section of the efferent (olivocochlear) supply to the cochlea. Thus, a comparison of performance on the masked tone detection task before and after surgery provides data on the contribution of the descending,

olivocochlear system. Following the surgery, listeners retain good performance at detecting probe tones at the target frequency. However, they show higher performance at detection of probe tones of unexpected frequencies; indeed, it can match the performance at the target frequency. One way to interpret these findings is that the improved detection performance for unexpected-frequency tones reflects, in fact, a loss of selective attention in the frequency domain. This is a compelling demonstration of the influence of top-down processes on a task that might otherwise be deemed a heavily data-driven (bottom-up) one.

The role of top-down processing in auditory tasks cannot be understated. It is an axiom of psychological science that inattention on any task may impair performance, and since CAPD is so often comorbid with independently diagnosed attentional disorders (see below), there may be pressure on hearing scientists to try to isolate one from the other. We shall return to this point later in this chapter. But top-down processes in perception are far more pervasive than one might think. We begin this line of inquiry by appealing to phenomena in the visual system, because they are so vivid, and then make the case that roughly comparable phenomena occur in audition.

Every normal-seeing person has a blindspot in each retina. The blindspot is the point of exit from the retina of ganglion cell axons, and it is the point of entry and exit of blood vessels. The blindspot contains no photoreceptors, and for this reason, it provides no information to the central visual system about the presence or distribution of light falling on it. Nevertheless, we are completely unaware of any gap in our visual world. We have to contrive an experiment to reveal it. With one eye open at a time (so that the visual projection onto the blindspot in the open eye is not "filled in" by projection of the same visual field portion onto the other eye), we hold a pencil tip at arm's length and slowly move it to about 15 degrees lateral to the focal point; the pencil tip disappears and then reappears when the pencil is moved beyond the blindspot. The conventional interpretation of our unawareness of the blindspot, even monocularly viewed, is that the visual system "fills in" the representation of the visual scene based on the recent history of stimulation from that point in the visual field (see Goldstein, 2010).

Another way of saying this is that percepts apparently arising from stimulation of the blindspot are *constructed* by top-down processing. This counters our opening assumption in this chapter—that the way in which perceptual systems work is to establish a point-by-point, detailed neurological picture of the sensory world and to generate a veridical perceptual experience that is as detailed as the neurophysiological representation can provide. In practice, the visual system is able to fill in the visual scene when a portion of the input is absent or ambiguous. An arguably more dramatic addressing of our opening assumption is the phenomenon of "change blindness" (see Rensink, 2002 and Blackmore, 2004, for a general overview). The phenomenon is demonstrated by showing participants alternating images that appear identical but in one of which some feature has been manipulated or removed. The task of the participant is to identify the changed feature. If our percepts were actually based on an exhaustive, detailed,

point-by-point neural representation of the stimulus field, then the changed feature should be detectable very quickly. In practice, however, participants typically begin by being "blind" to the change, and only after many alternations of the images is the change eventually detected. Many are shocked by the magnitude of the change imposed on the image that they failed to detect for so many alternations. One general argument is that the brain simply does not have the processing power to supply consciousness with a fully detailed mapping of the stimulus. Nørretranders (1991) characterizes the problem as one of bandwidth. He argues that the sensory systems transmit information at rates in the range of millions of bits per second, while on the other hand, the bandwidth of conscious perception is orders of magnitude lower. The sensory systems therefore chunk information into objects, or hypotheses about objects, to reduce the transmission load on the processor mediating conscious perception. This is what makes us prone to change blindness.

Our question now concerns the extent to which auditory perception is subject to similar top-down phenomena. Warren (1970) was early to demonstrate that if a meaningful speech signal contains an acoustically ambiguous element (a phonetically meaningless sound substituted for an actual phoneme), then not only do listeners fail to notice the substitution, but they "restore" the missing phoneme on the basis of the semantic context in which the affected word is embedded (the phenomenon of "phonemic restoration"). Once again, this is a case in which part of our perceptual world is constructed with the aid of top-down processes rather than being purely data driven.

Human listeners are also prone to "change deafness," a loose homologue of visual change blindness (e.g., Eramudugolla et al., 2005). The rules that govern change deafness are slightly different from those that characterize change blindness (Pavani & Turatto, 2008), but the two phenomena do share significant similarities. Change deafness is typically demonstrated by the presentation of two successive constellations or scenes of multiple auditory objects, and the scenes differ in the presence or identity of one auditory object; the task of the listener is to identify the changed object. In practice, the task is very difficult unless the listener is cued to the change (Eramudugolla et al., 2005). This sensitivity to cueing suggests that conscious access to details of our perceptual world is subject to attentional filtering.

The reason for citing these phenomena is to bring home the point that our perception of the auditory world cannot be based solely on a detailed, moment-by-moment neural representation of the stimulus panorama. If it were so based, then listeners in a phonemic restoration study would detect the manipulated phoneme, and listeners in change deafness studies would not demonstrate the effect. The top-down processes that mediate these phenomena (and possibly many others) serve to enhance the computational efficiency of perceptual processing and therefore the efficiency of our interaction with the physical world, even if it means that the continuity of our perceptual world is rather illusory. This blend of bottom-up and top-down processes is a compromise that works well in the vast majority of instances. We often have to contrive experiments to reveal its failures. Perhaps this is why such efforts are

so effective as children's games ("Find the seven differences between these two pictures").

It is possible that the only features of the sensory world that actually *do* receive a detailed neurophysiological representation and veridical perceptual elaboration are those to which we are actively attending (Cohen et al., 2011; Simons & Chabris, 1999). Now, "attention" is a multicomponent process (Posner, 1994). With that in mind, what we mean here is that application of an attentional process mediates or facilitates the perceptual elaboration of the attended portion of the sensory neural data stream. To be sure, sensory information about objects that are not attended may well be "received," in the sense that it may be available to influence subsequent perceptual, reflexive, or affective responses, but there is little or no conscious awareness of the object, i.e., no detailed perceptual elaboration of the object or event (see also "inattentional blindness," according to Rock et al., 1992; see also Merikle & Joordens, 1997; Posner, 1994). The executive control of attention (i.e., the disciplined allocation of attention to a task or to regions of the sensory scene) and the state of vigilance (i.e., the maintenance of an alert state) are separable processes (Posner, 1994).

The Roles of Electrophysiology in Diagnosing CAPD

At least in part, the development of electrophysiological and other nonbehavioral measures of CAP was motivated by the desire to develop indices of CANS function that were objective and did not require the active participation of the listener. In some regards, this venture has been extraordinarily successful. In its early days, the development of the auditory brainstem response (ABR) and middle latency responses was immensely revealing of the locus of CANS pathology (Musiek et al., 1994). This remains true to the present day. It is thus the conjunction of very poor speech discrimination in the face of intact otoacoustic emissions but poor ABRs that has become almost pathognomic of auditory neuropathy (Cowper-Smith et al., 2010; Kraus et al., 2000; Starr et al., 1996; Zeng et al., 2005).

On the other hand, two lines of argument provide cautions to how these early electrophysiological measures should be interpreted. First, there are anecdotal reports of patients with confirmed brainstem lesions, absent ABRs, but in whom it is quite difficult to identify auditory perceptual deficits. For example, Rappaport et al. (1994) described a multiple sclerosis patient with radiologically confirmed damage to brainstem and forebrain auditory pathways, grossly abnormal ABRs and middle latency responses, but who self-reported no auditory perceptual difficulties and in whom we found a perceptual deficit only in responses to speech presented against an interrupted noise background. This kind of observation is instructive because it reminds us that the objective tests may tell us more about the ability of the CANS to support synchronous, stimulus-driven neural activity recordable at the scalp than it does about what the listener is actually hearing. This point is particularly important if one holds the view that there is no conscious perception without attention.

A second and related argument derives from earlier discussion in this chapter. If we construe the function of the CANS

as providing the listener with veridical auditory percepts, then by devising electrophysiological probes that explicitly do not require the listener's perceptual cooperation, we also restrict the level of the conclusions we can draw from those probes. We can, however, turn this to our advantage by seeking to measure the fidelity of the strictly sensory representation, stripped of attentional or cognitive overhead. This isolation of sensory processing from attentional processes potentially gives us measures of the discriminations of which the auditory sensory system is capable, even if that discriminative capacity is not necessarily incorporated into perceptual behavior.

In this regard, the mismatch negativity (MMN) response has been used to great advantage. The MMN is a largely preattentive cortical response (Alain & Tremblay, 2007; Näätänen et al., 1993) to the detection of a change ("deviant") in a train of more frequent, otherwise homogeneous signals ("standards"). In the first place, the strength of the MMN response is related to the discriminability of the deviants from the standards (Alain & Tremblay, 2007), even though the listener is not required to discriminate the sounds at the time of MMN recording. Second, behavioral training of normal listeners in a previously difficult or impossible discrimination results in the emergence of an MMN when the trained listeners are tested with stimulus pairs used in the discrimination (Kraus et al., 1995). This finding extends to children with learning difficulties (Kraus et al., 1996). Interestingly, the development of the MMN during trained discriminations can precede the behavioral expression of the training, suggesting that the neurophysiological underpinnings of the sensory discrimination may occur before their incorporation into behavior (Tremblay et al., 1998). The MMN is not alone in being sensitive to auditory training. The obligatory N1–P2 complex also becomes stronger with training of previously difficult discriminations, and the growth in the amplitude of the N1–P2 response tends to parallel behavioral performance (Alain et al., 2009; Tremblay et al., 2001). Taken together, these data suggest that cortical event-related potentials may be used not simply as some index of the intactness of auditory pathways, but as a measure of what discriminations the cortical auditory system can support, and how that support changes with experience or training.

One of the more interesting developments has come from Jerger and Reagor (2012). They adapted a dichotic words task for use in a paradigm that had three levels of "processing depth" and was amenable to electrophysiological monitoring. Briefly, the listener is provided with a diotic cue word, which is then followed by a pair of dichotic words, one lateralized to each side. In one condition, one of the dichotic words is in the same semantic category as the cue and is presented to the right ear, while a distractor word is presented to the left ear. In a second condition, the cue is followed again by a dichotic word pair, but with the semantic category match presented to the left ear. In the third condition, neither member of the dichotic pair is a semantic category match to the cue word. The task of the listener is simply to indicate whether either member of the dichotic pair is a semantic category match for the cue. These three conditions differ in the "processing depth" required to execute the task. The right ear match condition requires the least, because of the more direct or efficient transmission

of verbal material from the right ear to the left hemisphere language processor. The left ear match condition requires the further neurological "work" of interhemispheric transfer of the information. The no ear match condition requires the most work, because both words of the dichotic pair have to be analyzed to completion in order to conclude correctly that neither is a match.

Jerger and Reagor (2012) recorded the scalp-recorded "processing negativity," averaged across listeners, beginning with a latency of about 300 ms (and that likely is a correlate of a number of attentional, phonological, and semantic operations acting in series and parallel). They reported that the processing negativity had amplitude and latency values that were distinctly different for the three experimental conditions, and in the expected directions. They suggest that in principle, it should be possible to separate out a genuinely abnormal interaural symmetry (difference between right- and left-ear match conditions) from a more global attentional deficit (differences from norms for all three match conditions). Whether this paradigm will be helpful in individual listeners is as yet unknown, but certainly this seems to be a viable line of inquiry.

CAPD in Neurologically Normal Listeners

From the foregoing, it is abundantly clear that a neuropathology of the CANS can result in a CAPD. These pathologies may be in the form of tumors, demyelinating diseases, auditory neuropathy, and probably many others (see Musiek et al., 1994, for review; see also Chapter 4). The extent to which the perceptual processing deficit is modality-specific likely depends on whether the pathology itself is restricted to the CANS (however we construe that). Rosen (2005) notes that it would seem odd to say that a patient with pathology to the CANS, and correlated auditory perceptual dysfunctions, does not have a CAPD simply because the pathology also invaded nonauditory structures. This is a good point, but it raises an important distinction for how we conceptualize CAPD. It is clear that a disorder of central auditory processing (i.e., a CAPD) can result from neurological damage to the CANS. But the majority of listeners referred for CAPD assessment show no obvious neurological signs. How are we to conceptualize CAPD in such cases?

One possibility is that any neuropathology in these instances is sufficiently minor that it does not raise strictly medical issues (e.g., seizures) and is manifested only as an auditory processing disorder (see Musiek & Chermak, Chapter 4 in this volume). In the absence of overt medical symptoms, patients are perhaps unlikely to undergo detailed structural or functional brain imaging to reveal the source of the processing disorder, and even if they do, the pathology may not be of a kind that is readily imaged. Congenital amusia (see below) may be one expression of such a pathology.

A second approach to this question derives from the work of Moore et al. (2010). They surveyed a very large population of children in the United Kingdom for their performance on a battery of auditory processing tests, and independently assessed listening, communication, and speech-in-noise performance. The important thing about the auditory processing tests was that some of them were designed in pairs (e.g., backward

masking with and without a 50-ms delay between the target tone and the subsequent noise masker); subtraction of the score for one of them (masking without the delay) from the other (masking with the delay) was argued to provide a "pure" (derived) measure of temporal resolution. This was because the tasks were otherwise identical, so the subtraction procedure removed the contribution of attentional/cognitive factors, since these were assumed to be constant across the paired tasks. They did the same for assessment of frequency resolution.

A number of important findings emerged from their study. First, the derived measures of temporal and frequency resolution were independent of age over the range tested (6 to 11 years), whereas performance on base tasks (i.e., prior to the subtraction procedure) improved with age. This suggested that what was improving with age were attentional or cognitive skills, rather than auditory processing skill. Second, the derived measures of temporal and frequency resolution were usually not correlated with cognitive test scores (e.g., nonverbal IQ, digit span, nonword repetition), whereas the base measures were. As a further step in the analysis, Moore et al. (2010) compared the bottom 5% of performers on the auditory processing tasks with the remainder of the sample. The groups were usually not significantly different on the basis of derived temporal and frequency resolution measures but usually were significantly different on the base auditory processing scores, cognitive tasks, and most of the clinical presentation measures. Moore et al. (2010) concluded that CAPD (i.e., poor performance on base auditory processing tests, and poor listening and communication skills in their study) had more to do with "poor engagement with sounds, rather than impaired hearing" (p. e389). It was not lost on Moore et al. (2010) that CAPD is so often comorbid with independently diagnosed attentional disorders (e.g., Chermak et al., 1999).

Some readers might feel uncomfortable with Moore et al.'s (2010) conclusion. In the first place, it is an assumption on their part that the cognitive/attentional processes involved in the base auditory processing tests making up each pair to provide the derived measures are in fact the same. One can easily imagine that the instructions given to the participants were the same for the pair members, but this does not mean that the higher level perceptual/cognitive processes engaged by the tests were the same. This issue occurs in auditory temporal gap-detection studies (see below). The instructions given to participants performing within- and between-channel gap detection tasks can be formally identical, but the perceptual/cognitive processes engaged by the two tasks may be very different (Phillips, 2012; Phillips et al., 1997). Second, the subtraction procedure, employed to eliminate attentive/cognitive contributions common to the performance of the related tasks, may also subtract out specifically auditory processing contributions common to both tasks. A third reason for some concern with the Moore et al. (2010) study is that it is not clear whether any of the bottom 5% of participants would necessarily have met the clinical diagnostic criteria for CAPD, though some of us are optimistic that they may have.

A strength of the Moore et al. (2010) study lies in prompting the following question. Could it be that among the (nonneurological) population, there is a continuous distribution of auditory per-

ceptual skill—in the broadest sense of that term—and that the clinical criteria for CAPD provide a cutoff point at the lower end of that distribution for the purpose of identifying the listeners most in need of some kind of intervention? That is, are diagnostically positive and diagnostically negative individuals members of the same distribution of CAP prowess, but differing only in their location along that continuum, and not discrete groups or categories of individuals (as one may expect in the case of overt neurological disease)? Is the situation rather like that of reading skill, that is, that there is probably a continuous distribution of reading efficiency, with some people being expert readers, others poorer readers, and most in the middle? (Parenthetically, it is noteworthy that a comparison of expert and normal readers reveals that expert readers have the better, that is, lower, within-channel auditory temporal gap detection thresholds [Au & Lovegrove, 2001].)

One line of evidence on this question comes from recent studies of auditory gap detection. The gap detection task comes in at least two general forms. In the within-channel paradigm, the listener is presented with two streams of sound, one of which (the signal) contains a brief silent period (gap) at or near its temporal midpoint, while the other (the standard) does not. The important point is that the sounds that delimit or bound the gap are identical. This two-alternative forced-choice trial is embedded in an adaptive threshold-tracking routine. The task of the listener in each trial is to identify the signal, and the adaptive tracking routine automatically concludes when the shortest-duration gap that can be reliably detected has been ascertained. Because the peripheral and central neurons that encode the sound are the same ones for the sounds preceding and following the gap, the perceptual operation required to perform this task ultimately reduces to the detection of a discontinuity in the activity of the neural/perceptual channel activated by the stimulus (Phillips et al., 1997). Depending on the spectral bandwidth of the stimulus, within-channel gap thresholds can be as low as 1 to 3 ms (Eddins et al., 1992).

In the between-channel paradigm, the sounds that bound the silent period are different—for instance, in spectral content or ear of presentation, or both (Formby et al., 1998; Phillips et al., 1997; Taylor et al., 1999). Once again, only the signal contains a nonzero duration temporal gap, but now both the signal and the standard contain a discontinuity as the stimulus switches from the one that precedes the gap to the one that follows it. This means that the task of detecting the gap requires a relative timing of the offset of the leading sound, and the onset of the sound marking the end of the gap. There is no peripheral machinery capable of executing this relative timing (but see Forrest & Formby, 1996; Phillips, 2012), and so it must be performed centrally, and it may be based in part on shifts of attentional allocation between the neural/perceptual channels representing the leading and trailing markers of the signal (and standard) stimulus. The task of the listener is again simply to detect the signal in each two-alternative forced-choice trial, and this trial structure is embedded in an adaptive, threshold-tracking staircase. Between-channel gap thresholds vary with the perceptual similarity of the markers bounding the gap but are often as much as an order of magnitude longer than within-channel gap thresholds in the same listeners.

Let us now return to the issue at hand. Phillips et al. (2010) studied within- and between-channel gap detection performance, using a method-of-limits strategy, in three groups of age-matched children: normal controls, children referred for CAPD assessment and found to be diagnostically positive (i.e., two standard deviations below the mean on each of at least two of six standardized clinical tests), and children referred for CAPD assessment but found to be diagnostically negative (i.e., "failed" zero or one out of six standardized clinical tests). In practice, within-channel best (minimal) gap durations failed to distinguish the three participant groups (modal best gap durations were 3.5 ms for all groups). As expected, between-channel best (minimal) gap durations were much longer. The distribution of best gap durations for controls extended from about 24 to 80 ms, but that for the diagnostically positive children extended from 53 to 180 ms. These distributions were significantly different. The interesting point is that the referred but diagnostically negative children had a distribution of best duration gaps centered between those of the other two groups (24 to 120 ms).

There are a number of points to be made here. First, within each participant group, best (minimal) gap durations were continuously distributed. Second, the distributions of best gap durations for controls, CAPD–, and CAPD+ children were shifted with respect to each other (toward longer gap durations as the participant "category" became more worrisome). This raises the possibility that had there been sufficient data to test the hypothesis, CAPD+ children failing three, four, five, or all six of the standardized clinical tests would have had best gap durations shifted still further toward long values. That is, there may be a continuous distribution of CAP prowess within the diagnostically positive group that is an extension of that already seen in the diagnostically negative and control groups. This is only a speculation that needs to be tested empirically, but if confirmed, then it also would suggest the existence of a continuous distribution of CAP efficiency within which CAPD– and CAPD+ "categories" are the result of the imposition of arbitrary diagnostic criteria on what may in fact be a continuous and perhaps quite broad distribution of CAP prowess. This is not necessarily a negative thing: The practice of imposing such criteria identifies the children most in need of intervention, and this is helpful. It is somewhat similar to the case of audiometric hearing loss (the familiar mild, moderate, severe, and profound degrees) in middle/inner ear disease. Given equivalent dB "distances" between boundaries, there is as much variance within a category as there is between midpoints of adjacent categories. That is, the location of the category boundaries is arbitrary, but it is helpful in being a guide to the most appropriate type of intervention required. On the other hand, the imposition of arbitrary diagnostic criteria on a continuous distribution can create the impression of discrete listener categories when no such categories exist independently of those criteria.

A third point concerns the nature of the task that revealed the extended distributions of best gap duration scores in the various participant populations. It was the between-channel gap detection task. Of the two gap detection tasks, this is the one thought to have the greater attentional/cognitive overhead (after Phillips et al., 1997; Phillips, 2012). This finding is compatible with

Moore et al.'s (2010) hypothesis in the sense that the greater the attentional/cognitive overhead required of the task, the greater is the likelihood of a CAPD+ listener showing poor performance. It appeals to the distinction between deficits in the sensory processing stream to the perceptual processor, and the higher level operations executed on the strictly sensory data information stream. On the other hand, preattentive MMN responses (see above), specifically localized to the auditory cortex, are seen in response to both within- and between-channel gap detection stimuli of comparable *perceptual* salience, even though the listener is not required to discriminate the relevant standards and deviants while the MMN is recorded (Heinrich et al., 2004). Parenthetically, these data may challenge the hypothesis that between-channel gap detection necessarily relies heavily on attentional processes (Phillips et al., 1997).

An entirely different approach to this general question comes from consideration of the heterogeneity with which CAPD presents itself. We saw above that listeners diagnostically positive for CAPD may or may not have overt neurological disease. Among the former group, it is obvious that the details of the presentation will vary with the locus and nature of the neuropathology. There is no reason why a listener with agenesis of the corpus callosum should have a clinical presentation indistinguishable from a listener with auditory neuropathy or a localized brainstem tumor. And, indeed, the clinical presentations of such listeners are different (cf. Starr et al., 1996; Musiek et al., 1994). Certainly, from the neurological point of view, these would be treated as distinct diagnostic entities. The question that arises, then, is whether neurologically normal listeners with CAPD are also heterogeneous. In this case, we cannot appeal to neurology to distinguish subgroups of listeners with CAPD, but we can look for evidence of systematic, or even idiosyncratic, constellations of clinical presentation signs. If CAPD in nonneurological cases is indeed systematically heterogeneous, then the hypothesis that CAPD is purely a disorder of top-down (and specifically attentional) processing faces the challenge of explaining the existence of the subtypes. In this regard, while there may be disagreement about how best to specify subtypes of CAPD, there is little doubt about the heterogeneity of its presentation (Bellis & Ferre, 1999; Jutras et al., 2007).

It is clear, then, that even if we are currently unable to identify the sources of variance in CAP skill/efficiency, that variance exists. Identifying the sources of that variance is a task for the future. An important point, however, is that CAP performance can be trained, and that this training is paralleled by the development of preattentive auditory electrophysiological responses (see above). This effect of training is entirely consistent with the animal neuroscience literature. Training animals in a way that makes a particular stimulus parameter (or point along it) behaviorally relevant enhances that parameter's central representation, likely by means of a cholinergic (nucleus basalis) mediated plasticity of cortical connectivity such that the cortex "self-selects" behaviorally relevant ascending inputs for cortical elaboration (Suga et al., 1997; Weinberger, 1997; see above). It is highly likely that this particular form of plasticity is expressed as an increase in the neural synchrony of those inputs to the affected cortical neurons/networks (after Eggermont, 2007). It is perhaps this increase in synchrony that results

in the development of scalp-recorded responses in humans during auditory training (see discussion of MMN and related responses, above). By the same token, it is this malleability in central representation and behavioral performance that offers potential for auditory training interventions in CAPD.

Two Novel Forms of CAPD

One of the most striking forms of CAPD must be congenital amusia, and a particularly clear case was presented by Peretz et al. (2002). The case study described a middle-aged woman with above-average intellectual, memory, and language skills, no history of neurological disease, no evidence of auditory cortical atrophy or pathology, but a quite severe deficit in the ability to detect pitch changes—as is typically also described in group studies (Ayotte et al., 2002; Foxton et al., 2004). In the Peretz et al. (2002) case study, the listener was reliably able to detect a pitch change only if the pitch change was an upward one; this pattern held for both pure tones and piano notes. The listener's detection of upward changes of pitch was also impaired, though not as severely. This asymmetry in sensitivity to pitch changes strongly argues against the deficit being a high-level cognitive one (e.g., attention, working memory) because there is no obvious reason why such high-level cognitive operations should be coupled to the direction of pitch change (Peretz et al., 2002). Interestingly, the pitch change deficit did not extend to the perception of intonation contours (e.g., statements versus questions, based a downward versus upward pitch change in the terminal word of an utterance) in the Peretz et al. (2002) case, although it does affect discrimination of speech intonation in about 30% of amusics in group studies (Patel et al., 2008).

An entirely different line of work bearing on CAPD comes from Eggermont's laboratory. Norena et al. (2006) reared cats for about 20 weeks (beginning around day 75) in an "enhanced auditory environment" (EAE), in which the animals were passively exposed to brief tones, spanning 5 to 20 kHz, presented in random order and overlapping in time, at an overall average rate of 96 Hz and 80 dB sound pressure level (SPL) for 24 hrs/day. After the exposure, the cats almost all had normal ABR thresholds to tones from 3 to 32 kHz, indicating an auditory periphery with normal sensitivity. By comparison with control animals (raised in quiet), the cortical representation of frequencies across the EAE bandwidth was disrupted. There were fewer neurons in the cortical territory of the EAE bandwidth with normal, short-latency responses within their frequency response areas, and there was a previously undescribed population of neurons with long-latency responses tuned either to frequencies below 5 kHz or above 20 kHz. Within the EAE representation, evoked neural firing rates were lower than those seen in the same frequency representation in control animals, and lower than those seen in representations of frequencies outside the EAE bandwidth. The firing rate distribution across frequency was thus the reverse of that seen in control animals. Many of these findings were seen in a later study using the same bandwidth of EAE, but this time for 6 weeks, at 68 dB SPL (Pienkowski & Eggermont, 2009) and then providing up to 12 weeks of recovery (quiet environment after EAE). Once again, ABR thresholds in the experimental animals

were normal, indicating normal peripheral sensitivity. Tonotopic maps were disrupted in the representation of the EAE bandwidth, as was neural frequency tuning. Following recovery, there was some restoration of neural frequency tuning, but a disordered tonotopy persisted in animals given up to 12 weeks recovery time. Mean neuronal firing rates were normal in the EAE cats, but the mean amplitudes of local field potentials were nearly halved. This distinction is important, because it suggests the existence of a reduced "gain" exerted on the input to the cortex; this is because cochlear output and brainstem sensitivity as indexed by the ABR were normal. Within the EAE region, neural synchrony of spontaneous discharges was enhanced in EAE animals, but only for electrode separations of 0.25 and 0.35 mm. Outside the EAE region, neural synchrony was enhanced for electrode separations of up to almost 1.5 mm. These changes in synchrony showed no evidence of reverting to normal even in animals given 12 weeks of recovery from EAE. They are important findings. Recall that neurons routinely fire "in synchrony" when driven by a common stimulus; the enhanced synchrony seen in spontaneous discharges suggests a synaptic reorganization. Most recently, Pienkowski and Eggermont (2010) studied the cortex of cats reared with intermittent (12 hr/day) 68 dB EAE for only 6 weeks. There was no loss of peripheral sensitivity as indexed by tone-evoked ABRs, but EAE exposure again resulted in a loss of responsiveness in the EAE bandwidth representation, and disordered tonotopy. The perceptual correlates of the cortical reorganizations seen in the studies from Eggermont's laboratory are at present unknown, but it is difficult to imagine that the changes to neural frequency tuning, tonotopy, response timing, and neural synchrony are without behavioral consequence. For our understanding of CAPD, it is important to appreciate that these neurophysiological changes occurred in the absence of peripheral hearing loss.

For the cases of both congenital amusia and the effects of EAEs, no appeal to attentional factors, at least in any conventional understanding of that term, seems capable of explaining the data. Some might argue that the congenital nature of amusia speaks indirectly to a neurological disorder (albeit a relatively benign one) and that the Eggermont data are direct evidence of a neurological disturbance. We walk a fine line here. Ultimately, all behavior is mediated by the brain, so any differences in behavior, either between individuals (e.g., those with more or less developed a skill) or within an individual across experience (e.g., auditory training), must be accompanied by differences in brain organization or physiology. The fine line is therefore this: At what point do we demarcate normal variation from a clinical disorder? The presence or absence of overt neurological disease (tumor, stroke, neuropathy) makes the case easy. The effects of experience or epigenetics, that is, experiential effects on gene expression for neurological change, make that decision much more subtle.

Conclusions

Hearing and listening are active processes. What we have seen develop in this chapter is an acknowledgement that the central auditory nervous system is *adaptive* (see Weinberger, 2011 for a

more developed account of the cortex as an "auditory problem solver"). To be sure, the general layout and topographic connectivity of the CANS, and the general psychophysical processes they support, are probably determined genetically, but the details of the connectivity are subject to the listener's experience. Structural or functional plasticity in the connectivity details are likely mediated at least in part by the numerous feedback loops between the auditory cortex and the auditory brainstem nuclei. The descending connections are the medium through which the forebrain auditory system can regulate or fine-tune its own input. It is perhaps this mechanism that contributes to the "gain control" on cortical inputs effected by enhanced auditory environments (Pienkowski & Eggermont, 2009). From a teleological point of view, perhaps the content of the EAE is behaviorally irrelevant to the animal, and so inputs transmitting information about the EAE are downregulated. On the other hand, when behaviorally significant discriminations need to be learned, then it is through the medium of feedback loops that signals of interest are afforded expanded forebrain *neural* representations, and presumably greater *perceptual* elaboration than they would otherwise receive. The devil is indeed in the details, and the particular devil in this case is the adaptivity that enables the listener's CANS to optimize the salience of the neural representations of stimuli that are behaviorally significant.

At the outset, we suggested that a primary function of the CANS is to establish a "neurological picture" (sensory representation) of the acoustic world that is veridical and that can support a seamless, continuous, private perceptual experience. That remains true, but we acknowledge now that details of both the representation and its perceptual elaboration reflect the interplay of data-driven sensory processing and top-down modulation of the ascending inputs. At an arguably higher level of processing, we see further evidence of top-down processing in the disambiguation of acoustic signals by context (e.g., phonemic restoration), the formation of auditory perceptual objects through grouping mechanisms (e.g., auditory saltation), and a clear role of attentional processes in the perceptual filtering of the auditory scene (e.g., change deafness).

The fact that normal CANS function reflects an interplay of bottom-up and top-down processes has the consequence that, in principle, a CAPD could arise from deficits in either direction of information flow (or both; see also Chermak & Bellis, Chapter 20, this volume). Indeed, the effects of deficits in centripetal and/or centrifugal information flow are awkward to disentangle precisely because of their functional interplay. In the clinic, however, we are able to strip speech stimuli of semantic context (e.g., use of isolated words or nonwords) or use stimuli that are sufficiently analytical that the contributions of grouping processes or attentional filtering are at least minimized. Moreover, deficits in so-called supramodal (e.g., attentional) processes can be assessed independently (Chermak et al., 1999; see also Chermak, 2003; Petkov et al., 2004), and are thus somewhat isolable from data-driven processing. To help matters, it may be possible to develop combined behavioral/electrophysiological tasks that tap ascending and descending processing somewhat separably (Jerger & Reagor,

2012). Certainly, the use of MMN and N1–P2 responses as an index of what perceptual discriminations the listener is capable of is a huge advance. To date, the best data on this point have come from group studies, but one hopes that these methods can be refined for use with individual listeners.

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CHAPTER 23

FUTURE DIRECTIONS IN THE IDENTIFICATION AND DIAGNOSIS OF CENTRAL AUDITORY PROCESSING DISORDER

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The future directions to be taken in the identification and diagnosis of central auditory processing disorder (CAPD) are difficult to predict. Without a crystal ball, the projections offered in this chapter are somewhat speculative, although they are derived from our clinical experiences and evolve logically from the tremendous research advances in auditory neuroscience and related areas. The reader also is referred to the clinical practice guidelines in CAPD published recently by the American Academy of Audiology, which includes directions for future research in diagnosis and intervention, as well as regarding professional issues and education (American Academy of Audiology, 2010).

Early Advances in Central Auditory Assessment

Perhaps one of the best ways to predict the future is to study the past. In this regard, CAPD has a varied and interesting history. In introducing the concept of central auditory assessment, Bocca and his colleagues (1954) affirmed several pivotal principles that are likely to continue to guide central auditory testing in the future. One key principle guiding their work was their recognition of the limited value of pure-tone sensitivity measures in detecting and revealing compromise of the central auditory nervous

system (CANS). This principle led Bocca and his associates to develop auditory tests placing greater challenge on the CANS to therefore be more sensitive than pure tone thresholds to central auditory dysfunction. To increase the challenge, they developed low-redundancy speech tests (e.g., filtered speech). Although we now know that the utility of filtered speech tests for CANS evaluation may be compromised by the effects of peripheral hearing loss, Bocca's understanding of the need to challenge the CANS has been confirmed over the years. (See Tests Abandoned or of Limited Value below). Another pioneer active at essentially the same time as Bocca was Helmer Myklebust, a psychologist interested in childhood hearing loss. In his 1954 book, Myklebust described children with auditory difficulties who had no measurable pure tone hearing loss. He was one of the first to relate that auditory disorder should include not only peripheral but also central auditory dysfunction (see Jerger, 2009; Myklebust, 1954).

Shortly after Bocca and colleagues began developing their test strategies and Myklebust's work was acknowledged, another breakthough was achieved in 1961 when Doreen Kimura published her seminal article on dichotic listening. Based on a relatively large population of patients with temporal lobe damage, Kimura (1961) reported poorer performance for the ear contralateral to the involved cerebral hemisphere, findings that were consistent with Bocca's inferences regarding the structure of the CANS. Shortly after Kimura's dichotic studies, Katz (1962) introduced the Staggered Spondaic Word (SSW) test, also grounded in the anatomy and physiology of the CANS, which became a widely used test of central auditory function. Using a dichotic listening paradigm, Milner (1968) demonstrated the role of the corpus callosum in dichotic listening, an important principle that later was well solidified in the areas of neuroscience and audiology (Musiek et al., 1984). At about the same time, Pinheiro and Ptacek (1971) introduced the original paradigm for testing pattern perception.

In the mid-1970s, Willeford (1977) observed that children with learning disabilities may be at risk for central auditory dysfunction, an association that has been documented over the years (ASHA, 2005). Also evolving in the 1970s was the auditory brainstem response (ABR), which would become a major clinical and research tool. Sensitive to dyssynchrony of the brainstem, the ABR continues in use today to assess the integrity of this more caudal portion of the CANS (Musiek, Gollegly, Kibbe, & Verkest, 1988). Throughout the 1980s and 1990s, the sensitivity of other auditory evoked potentials to dysfunction of the CANS was demonstrated for purposes of research (e.g., mismatched negativity [MMN]), as well as research and clinical application (e.g., middle latency response [MLR], late-latency responses, N1–P2 response, and P300). (See Musiek & Lee, 1999 for a review and Chapter 6 of Volume 2 of the Handbook for a review of the history and pioneers in CAPD.)

Progress Demands Communication and Change

In order for research to continue to advance, it is important to take a retrospective look to examine where the field has been in order to identify unan-

swered questions, as well as the advances that have occurred. Based on that retrospective, decisions can be reached as to what kind of research efforts should be emphasized in the future. Unfortunately, it seems clear that in the area of CAPD, basic science has advanced at a much faster pace than clinical research and application. Advances in auditory neuroscience have been striking, while clinical application of these developments has lagged. Perhaps communication shortcomings between researchers and clinicians, as well as clinical resistance to change have contributed to the slower pace in the clinical domain.

Developments in central auditory processing are at least partially dependent on clinicians' ability to embrace change. It seems clear that many basic and clinic science advances have never been translated into clinical use (e.g., click lateralization, tests of localization, cross-channel gap detection, backward masking, interaural timing procedures). This is a curious and unfortunate circumstance, as it slows the pace of improvements in clinical services. However, the problem is not one-sided: Researchers must consider the practical needs of clinicians. Diagnostic tests must be developed that are clinically feasible and can be incorporated within the constraints and conditions of typical clinical settings, and thus appeal to clinicians, not just to other researchers. Failure to consider these issues leads to delays or, worse yet, rejection of promising new clinical tools. Basic researchers and clinical scientists interested in advancing new clinical tests and procedures must develop paradigms that are not only sensitive and specific, but also are easy to administer and score and are time efficient and use equipment that is commonly available in most audiology clinics. Moving a procedure from the lab to the clinic must proceed incrementally and with generous communication between the scientist and the clinician. In fact, it seems that there is a need for "clinical research" to be more clinical. That is, perhaps more research should actually be performed in the clinical setting using exact clinical protocols, and so forth. This would enhance the uptake of new procedures by assurance that they can survive in a real clinical situation (Dhar, 2011, Mayo Clinic Conference). Often, clinical research conducted in the laboratory does not realistically approximate what happens in the true clinical environment, slowing or worse yet stalling the transition from laboratory to clinic.

Although some procedures are quickly adapted for clinical use (e.g., dichotic listening), others require extended periods of time to integrate fully into the clinical test battery. In our own experience, we have noticed that the frequency pattern test is still viewed by some as a *new procedure,* even though it has existed for over 25 years and has been clinically used for almost that long (Chermak, Traynham, Seikel, & Musiek, 1998). Researchers can hasten clinicians' adoption of new tools by developing them for clinical (as opposed to laboratory) application and then explaining their use in clinical terms and forums. Clinicians must be at the ready to critically evaluate new tools, and if determined to be better than what currently is available, they must accept and embrace these new tools and secure the continuing education that might be necessary to fully implement the new procedure. Failure to incorporate efficient tools will not only slow down the pace of clinical improvements for our patients, but may also discourage creative and innovative approaches in the

basic and clinical science arenas. Change can be unsettling; however, as clinicians committed to quality patient care, we must be willing to put away older and perhaps more comfortable approaches in favor of better—more effective and efficient—approaches and tools.

The ability of the clinician to adapt to new procedures depends on their readiness. The clinician well grounded in the basic sciences will be better able to make insightful interpretations of new tests, design patient programs, and understand subtle signs of disorders to better help their patients. Basic scientists who have an interest in the clinical sciences will better help the clinician by providing "usable" innovations.

Clinicians' Knowledge and Interpretation of Test Outcomes

Test interpretation is crucial to improved diagnostic capability. Clinicians must be well versed in the strengths and limitations of the tests in their battery and the bases (e.g., anatomy, physiology and pathophysiology) of these strengths and limitations. Passing or failing a test or battery of tests may not be the only factor guiding diagnosis. In fact, it is clinically feasible that two different clinicians with different depths of knowledge could render two different diagnoses for the same patient presenting the same test results. This would likely mean that a misdiagnosis would occur, with significant consequences to the patient. Both the efficiency of the test and the knowledge brought to bear by the clinician *interpreting* the test are essential to accurate diagnosis. In order to advance our diagnostic precision, our knowledge of underlying mechanisms, both normal and pathologic, must be grounded firmly.

In order to improve CAPD diagnostics, clinicians must be attuned to patients' complaints and conceptualize those complaints within a broad context, which includes test results, symptoms, history, and family, workplace, educational, and recreational issues that may bear on the situation. The current central auditory test battery does not allow us to probe every type of auditory processing problem. Indeed, the number and types of auditory processes might actually exceed those currently identified (AAA, 2010; ASHA, 2005). Fortunately, patients' symptoms are invaluable indicators of processing deficits, particularly if the symptoms are interpreted correctly.

To optimally utilize information pertaining to the patient's symptomatology, the clinician must understand and apply knowledge of anatomy, physiology, and psychoacoustics. For example, for some patients with central auditory deficits secondary to a neurological insult, the clinician might be able to *predict* the patient's performance on certain central auditory tests based on the patient's symptoms and an accompanying magnetic resonance image (MRI). We are not suggesting that one can or should diagnose CAPD without confirming deficits using efficient central auditory tests; rather, we are emphasizing that by properly interpreting a thorough case history combined with careful testing using efficient tests and procedures, we can obtain the maximum amount of information and insight to best serve the patient. To maximize the information gleaned from the history and best interpret test results, however, clinicians must be knowledgeable. Improving diagnosis of CAPD is not

just a matter of developing better tests; improving diagnostic capability also is dependent on more insightful interpretation of efficient tests.

Auditory Psychophysics and Confounds of Behavioral Tests

Test Development: Language and Other Confounds

Behavioral tests of central auditory function have both advantages and disadvantages. Behavioral tests allow the patient (in most instances) to relate what/how they hear, which can be very enlightening for the clinician. Also, behavioral tests often approximate real listening situations, which enhance their relevancy and provide guidance for intervention. In addition, behavioral tests usually require less time to administer relative to electrophysiologic procedures and often are easier to score and interpret. However, because successful performance on behavioral tests, even those employing simple responses, requires the coordination of many systems (e.g., attention, memory, motor control, and sometimes language), there are liabilities associated with behavioral testing of central auditory function.

Interpreting performance on behavioral central auditory tests that use speech stimuli (i.e., language) can be difficult, especially with pediatric populations, nonnative speakers, and older adults following cerebral vascular accidents or affected by other conditions that compromise language and/or cognitive function. One must consider that performance on behavioral tests that employ speech stimuli might reflect language, memory, and attention factors as well as true auditory function. In children, this potential confound is underscored by the frequency with which children suspected of having CAPD also have language processing problems (see Sloan, 1992). Most would agree that language difficulties can manifest as auditory processing deficits and that CAPD can manifest as a language problem. These two entities also can coexist (AAA, 2010; ASHA, 2005; Sloan, 1992).

To minimize the potential confounding effects of language, audiologists should select behavioral central auditory tests that employ nonspeech stimuli (e.g., frequency and duration patterns, gap detection) or low-level language stimuli (e.g., dichotic digits). (Electrophysiologic procedures are also useful in this regard as long as they employ nonspeech stimuli; however, as noted in Chapter 22 of this volume, electrophysiological procedures do not tell us what listeners perceive, but rather the capacity of the CANS to represent auditory stimuli.) Expanding the variety of tests and procedures in the central auditory battery that use nonspeech stimuli (and are not highly dependent on attention and memory) and are focused on specific auditory processes (e.g., lateralization, cross-channel gap detection) will provide the audiologist with additional options to examine multiple central auditory processes and augment our ability to differentially diagnose the range of comorbidly presented disorders (e.g., CAPD, attention deficit-hyperactivity disorder, language impairment).

One such test that may soon emerge in the clinical arena involves interaural timing. In this procedure, clicks are presented to both ears with differing time

delays and intensity levels, resulting in defined changes in intracranial lateralization. Under experimental conditions this procedure has been shown to be highly sensitive to lesions of the CANS (Pratt et al., 1998). Moving this procedure from the lab to the clinic will require some procedural streamlining and attention to the issues discussed above.

Developing tests using nonspeech stimuli and do not therefore require native language proficiency will serve the needs of the growing number of countries around the world that have begun establishing programs for evaluation and management of CAPD. By developing tests that can be used universally, insightful comparisons can be made about potential cultural differences and their influences on auditory processing outcomes.

As noted above, in selecting and interpreting tests, audiologists must take into consideration the potential confounding effects of systems other than audition. The interdependencies among these systems suggest, however, that multidisciplinary assessment is essential to fully evaluate and differentially diagnose a range of *look-alike* conditions. For example, recognizing the potential confound imposed by language, as well as the frequency with which CAPD and language problems co-occur, it is essential that a full speech and language evaluation be completed on all children referred for CAPD testing. Having this information in hand at the time of audiologic testing should assist the audiologist in the selection and interpretation of tests. If multidisciplinary evaluations are to provide useful information, the data provided by our colleagues in other disciplines must meet the same high standards of sensitivity and specificity as audiologists require of central auditory tests.

The use of psychophysical functions can be of great help in differential diagnosis and in sorting out the underlying cause(s) of poor performance on a given test, which might be due to auditory dysfunction, lack of understanding (e.g., perhaps language-related), or cognitive factors such as inadequate attention. By keeping the task constant and easing the auditory demands (e.g., by increasing presentation level), performance should improve, indicating the subject understands and is following the task. If easing auditory demands does not lead to improved performance on an auditory task, then nonauditory factors should be entertained as the source of difficulty. Psychophysical functions allow us to use patients as their own controls (e.g., intrasubject comparisons, as in interaural differences—see below), another useful clinical approach that enhances the rigor and efficiency of the diagnostic test battery (Bellis, Billiet, & Ross, 2011; Cacace & McFarland, 1998, 2005).

The Role of Tests Using Speech Stimuli in the Central Auditory Test Battery

Ironically, some of our most efficient tests of central auditory function involve speech stimuli (e.g., dichotic tests) and are, therefore, linked to language processes (Musiek, 1983). Despite the potential for confounding effects, there is no question that tests using speech stimuli should remain in the central auditory test battery, given the primacy of speech as stimulus to the CANS and the likely coevolution of the speech and auditory systems. Speech testing has value ecologically. It is dominant in our everyday environment, it is critical to our well-

being, and without it individuals struggle to contribute to society. To not assess this key stimulus would be a shortcoming of the evaluation process. Moreover, speech signals provide access to different CANS processing mechanisms than do nonspeech stimuli. For example, increased response to the human voice compared with nonvocal sounds is seen in the left and right superior temporal cortex (Grossman et al., 2010). Because the degree of temporal processing required for accurate perception of spoken language is significantly greater than required for perception of nonspeech sounds, processing of speech signals may be more vulnerable to disruption by CANS dysfunction (e.g., Fitch et al., 1997; Griffiths et al., 1999; Shannon et al., 1995; Zatorre & Belin, 2001). In fact, central auditory processing deficits may only be revealed with speech tasks (e.g., Benavidez et al., 1999; Johnson et al., 2005). Speech stimuli garner greater attention when testing younger subjects than do nonspeech tasks, which are more abstract, thereby reducing a potential confound.

Awareness of possible contaminating factors of speech stimuli and resisting overinterpretation is key.

When properly interpreted, certain performance patterns on tests using speech stimuli can help the audiologist differentiate auditory system versus other system deficits. For example, central auditory dysfunction is commonly manifested by depressed performance in only one ear or asymmetric performance across ears, with one ear's performance much poorer than the other. When this pattern of result is observed, it is highly likely that the source is an auditory problem, as speech, language, and memory deficits would not lateralize (see Chermak & Musiek, 1997 for review). More research is needed to explore lateralization in procedures using speech stimuli, such as dichotic listening. Moreover, speech-evoked auditory potentials (see Auditory Evoked Potentials below) may soon offer additional clinical applications for differential diagnosis.

Development of New Tests of Central Auditory Processes

Ironically, our current central auditory test battery still does not contain a test of auditory discrimination, as noted in the first edition of the Handbook. There have been some developments in assessment of localization, however, since the first edition, as discussed below. Despite the fact that auditory discrimination is one of the most basic hearing functions, it is seldom clinically evaluated. So basic is auditory discrimination to auditory processing, that frequency, intensity, and duration discrimination should be assessed as part of both the peripheral and the central auditory test batteries. No doubt, auditory discrimination problems are likely to be present in many children and adults with CAPD; therefore, our failure to assess this fundamental auditory process limits our ability to gain a full understanding of the patient's difficulties.

There has been some groundwork accomplished on the development of a frequency discrimination procedure for clinical use—but with little uptake by the clinical community (Cranford, Rye, & Stream, 1982). Cranford et al. (1982) demonstrated that frequency discrimination is severely degraded, especially when there are temporal restrictions, in patients with damage of the CANS. Developing a clinically useful measure of audi-

tory discrimination is all the more important given the considerable evidence that auditory discrimination can be improved with training (Delhommeau et al., 2005).

The psychoacoustic tuning curve is another test procedure that involves frequency analysis and could be developed for clinical use in the future. This procedure, which has been commonly used in psychoacoustics research, provides an indication of the status of the frequency selectivity of the auditory system.

A clinically feasible test of sound localization has long been needed. Even informal tests of sound localization appear to be highly sensitive to dysfunction of the CANS (Sanchez-Longo & Forester, 1958). Therefore, recent efforts to standardize a clinical test of this process are of great interest (Cameron & Dillon, 2005). Because measures of sound localization are usually obtained in heavily sound-treated rooms or anechoic chambers, the clinical feasibility of localization in the clinical setting had been questioned. However, the recently developed LiSN-S (Listening in Spatialized Noise–Sentence test) holds promise to fill the void in clinically feasible localization procedures, although the LiSN-S is not a true localization procedure, but rather approximates these kinds of tasks by including spatial orientation as a component of the sentence understanding in noise identification task (Cameron & Dillon, 2007). This procedure appears to have clinical utility and should contribute to a more comprehensive test battery. In the first edition, we expressed great hope that virtual reality techniques would soon be applied to sound localization in the clinical setting (Besing & Koehnke, 1995); however, it appears that this technique is not being embraced clinically or to any great extent in the laboratory either.

[redacted]sts Abandoned or of Limited Value

Temporal processing is fundamental to audition and involves many subprocesses and abilities, only a few of which are tested clinically (e.g., temporal sequencing, temporal resolution or discrimination). Additional measures are needed to fully explore temporal processes, including temporal masking and temporal integration. In this context, it is interesting to note the fate of a test of temporal integration around which there was a flurry of activity in the late 1970s. The test was called brief tone audiometry (Wright, 1978). This technique, though it seemed useful to many, did not survive as a clinical procedure, despite some compelling reports on its use with central auditory disorders (Baru & Karaseva, 1972). Given this history, a test of temporal integration may not reemerge soon as a clinical audiologic test procedure.

Monaural low-redundancy speech tests (e.g., filtered speech, compressed speech, speech in noise) are among the oldest types of central auditory tests. Unfortunately, they have been shown to be highly confounded by hearing loss and language disorders. These confounds, combined with their relatively mediocre sensitivity and specificity, have led to a continued decline in their use, with continuing decline projected into the future.

[redacted]ltimodality Testing

Recent interest in cross-modality testing to assist in the differential diagnosis of CAPD has stimulated much debate. Proponents assert that CAPD must be

defined as an exclusively modality-specific perceptual disorder that can truly exist only if it can be demonstrated that the auditory system is the only modality involved (Cacace & McFarland, 2005). Others, including the authors of this chapter, define CAPD as a *primarily* modality-specific perceptual dysfunction that cannot be attributed to peripheral hearing loss or higher order, global cognitive, attention, or related disorders (AAA, 2010; ASHA, 2005; Musiek, Bellis, & Chermak, 2005).

In order to fully examine these competing conceptualizations of CAPD, other modalities must be assessed with the same rigor applied to the auditory modality to ascertain fully the status of these other modalities, as well as the status of supramodal systems (i.e., attention and memory) that influence processing across modalities (Musiek, Bellis, & Chermak, 2005). Limiting cross-modality measurement and comparisons at this time are a number of theoretical, procedural, and professional scope of practice issues. For example, the equivalence of multimodal tests that differ only in sensory stimulus has not yet been demonstrated. Moreover, if audiologists are not presently qualified to test other modalities and determine possible interactions with pansensory systems, broader based training for audiologists or increased dependence on other professionals to evaluate individuals suspected of CAPD will be required. Perhaps of greatest relevance are findings that cross-modality test comparisons do not contribute unique data that enhance differential diagnosis beyond those obtained through intratest comparisons of efficient central auditory tests (Bellis et al., 2011). Additional issues may emerge with more research and clinical trials.

Auditory Evoked Potentials

Topographic Mapping as a Clinical Instrument

In the first edition,it was mentioned that it was likely that auditory evoked potentials (AEPs) would serve an increasingly larger role in the diagnosis of CAPD. Testing with AEPs affords the clinician the advantage of little contamination from language, cognition, or other potential confounds. Also advantageous, AEPs permit testing of various levels of the CANS. A recent survey, however, indicated decreased clinical use of evoked potentials, reporting that 70% to 90% of audiologists report never using electrophysiology in their test battery (Emanuel, Ficca, & Korczak, 2011). At first glance this is puzzling; however, several reasons for the decline in clinical use of AEPs seem probable, including issues related to reimbursement, startup costs, prolonged learning curve, and the absence of normative data. These issues certainly could drive down the use of AEPs—especially in a private practice setting.

While AEPs provide precise temporal information about CANS activity, topographic mapping provides spatial as well as temporal detail. Topographic mapping is the process of viewing evoked-potential responses from multiple electrode arrays (often 64–128 electrodes) placed on the scalp. Not only can the generator sites across the scalp be analyzed, but also the time course of these potentials can be observed. Activity across the scalp is color coded, usually with lighter, brighter colors indicating high amplitude response and darker colors conveying lower amplitudes.

Topographic mapping will continue to serve as a valuable research tool to explore the neurophysiology underlying CAPD. The key question, however, is whether topographic mapping or some modification can become a *clinically* useful procedure for diagnosing CAPD. Clinical trials in regard to its diagnostic test efficiency must be conducted. If proven efficient for diagnosis of CAPD, the procedure would need to be streamlined so that it could become a clinical tool. Technologically, topographic mapping could, in all likelihood, be streamlined to make it a clinically feasible procedure; however, the issue then becomes whether the cost of a topographic procedure could be tolerated by third-party or even private payers.

Middle and Late Auditory Evoked Potentials

The MLR and the late (N1–P2) AEPs have been used successfully in the evaluation of CAPD (Musiek & Lee, 1999; Hall 2007 for review); however, these evoked potentials are still not being utilized as much as perhaps they should be (see above). These AEPs can likely become more diagnostically powerful with certain alterations. For example, by recording the AEPs in the presence of acoustic competition and/or using some form of abbreviated speech as stimulus rather than clicks and tonal stimuli—both of which would increase the practical relevance of these measures—MLR and N1–P2 could provide valuable indices and insights regarding CANS function, particularly through comparisons with results obtained without competition and for nonspeech stimuli. In fact, recent investigations using short speech segments with an ABR paradigm have reported impressive findings in individuals with learning problems (Russo, Nicol, Zecker, Hayes, & Kraus, 2005). (See Chapter 7.) Longer speech segments could be employed with the late AEPs—for which there is a good experimental track record in identifying CANS dysfunction using standard click and tonal stimuli (see Musiek & Lee, 1999 for review).

Another AEP that holds promise for the future is the Auditory Steady State Response (ASSR). The ASSR is triggered by the depth of frequency, intensity, or both frequency and intensity modulation. Comparison of the ASSR with behaviorally obtained measures of modulation (e.g., frequency difference limen defined as the smallest modulation depth discerned by the listener) might offer a powerful pair of physiologic and psychoacoustic measures to diagnose frequency discrimination problems and central auditory dysfunction. Some preliminary research already has been conducted on this technique (John et al., 2000). Also promising, ASSR threshold and behavioral threshold comparisons may help differentiate central versus peripheral involvement. In contrast to subjects with normal hearing or cochlear hearing loss where the ASSR–behavioral threshold correlation is good (Shinn, 2005), there is some evidence that subjects with CANS involvement demonstrate poor correlation between behavioral and ASSR thresholds.

P300 and Multimodality Testing

Given the issues raised above regarding behavioral measurement of multimodal performance, perhaps the most promising means to conduct multimodal measures would be via the P300. It is well known that P300s can be obtained using

auditory, visual, and tactile stimulation (Regan, 1989). Therefore, with sufficient normative data, the auditory, visual, and tactile P300s could be compared to help determine the modality specificity of a processing deficit. Though cross-modality P300s have been examined (Regan, 1989), this technique has not received clinical application.

Concurrent Recording of Electrophysiologic and Behavioral Tests

Several studies have reported on the use of concurrently recording AEPs while the patient performs central auditory behavioral tests to establish a diagnosis of CAPD (Atcherson, Gould, Mendel, & Ethington, 2009; Palmer & Musiek, in press; Jerger et al., 2002; Musiek, Baran, Shinn, & Jones, 2011, pp. 296–328). This approach can be useful in difficult cases and to help corroborate findings across the test battery. Behavioral tests and electrophysiological procedures serve different purposes and provide complementary information—one probes the listener's perceptions and the latter provides insights into the anatomy and physiology of the listener's CANS. In fact, these two approaches assess, at least to some degree, different neural substrates, thereby incorporating both in a clinical text battery, maximizing the opportunity to identify different regions and types of dysfunction.

The Role of Imaging Techniques

Functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) are the two main imaging techniques commonly used in auditory research. Both techniques reflect changes in metabolic activity for various regions of the brain, generally in response to some type of stimulation. In the future, both of these techniques will play an increasing role in CAPD diagnosis and that of communication disorders in general. Another method of MRI that is gaining in popularity is diffusion tensor imaging (DTI). DTI maps water molecules in brain tissue. Since water diffuses in the direction of fiber tracts, DTI measures white matter and connectivity (Schmithorst et al., 2011). This technique would appear to be especially useful for defining microstructure of myelin tissue, such as in the corpus callosum (Jerger, Martin, & McColl, 2004). Imaging procedures will reveal the areas of the brain that are activated during a particular central auditory test procedure. In so doing, imaging will advance our understanding of the physiological and anatomical correlates of test procedures and subject performance. In addition, DTI in particular may be able to identify the specific neurological substrate supporting difference behavioral measures, allowing clinicians to streamline the test battery to those procedures offering unique, rather than redundant information (Schmithorst et al., 2011). Imaging may also allow monitoring recovery or treatment progress. Combining behavioral tests with functional imaging should allow us to determine the neurophysiologic basis for changes observed in patient performance. Correlating functional imaging results with behavioral test results may also provide a basis for differentiating auditory, language, and attention deficits. Cost and equipment issues are likely to determine whether imaging emerges as a clinical tool.

Education and Training

If the area of CAPD is to advance, a major change must occur in the education and training of audiologists. The education and clinical training for most audiology students in the area of CAPD and its neuroscience foundations have been inadequate. In a survey, Chermak et al. (1998) noted that 80% of the respondents reported not to have taken even one course as a graduate student that was dedicated to CAPD. In that same survey, 20% of the respondents reported that they had never taken one graduate course in the structure and function of the CANS. Our recently completed follow-up survey revealed little change in graduate students' education, despite the replacement of master's programs with the more extensive AuD degree program (Chermak, Silva, Nye, Hasbrouck, & Musiek, 2007). Also troubling is the lack of diversity in the populations students evaluate for CAPD. Commonly, student clinical experience with CAPD is restricted to children with learning problems. Even within this limited population, experiences are meager, with the vast majority of students obtaining less than five clock hours in this category (Chermak et al., 1998), a finding replicated in our follow-up survey (Chermak et al., 2007). Patients with neurological involvement (e.g., strokes, closed head injuries, epilepsy, mass lesions) are seldom seen and yet it is this population that perhaps allows us to learn the most about the nature of CAPD.

Emanuel, Ficca, and Korczak (2011) found that less than 20% of audiologists responding to their survey used any type of electrophysiological measure as part of their CAPD assessment, and 32% of the responding audiologists did not offer treatment for CAPD due to lack of training. These findings are of particular concern, since half of the audiologists responding in 2011 held doctoral degrees, and several clinical practice documents and guidelines have noted the value of electrophysiological measures in the diagnosis of CAPD (AAA, 2010; Jerger & Musiek, 2000). Although nearly 100% of respondents indicated that audiologists should be the professionals to diagnose CAPD, these same respondents identified speech-language pathologists (74%) and educators (52%) as responsible for providing intervention (Emanuel et al., 2011). Although our field has evolved and expanded in numerous directions, including balance and tinnitus diagnosis and treatment, a primary focus has remained on the fitting of hearing aids and other advanced technology, with therapy being deemphasized, including in the area of CAPD. This is in stark contrast with scope of practice documents and guidelines that specifically state that the audiologist is responsible for the diagnosis and treatment/management of children and adults with CAPD (AAA, 2010; ASHA, 2005a).

It is ironic that such serious training issues are prevalent in one of the most demanding areas within audiology. Encouraging, however, is Emanuel et al.'s finding that a large majority(80%) of audiologists responding to their survey reported customizing test batteries to reflect the patient's age and case history, and customizing management recommendations (75%) based on the findings of diagnostic tests (Emanuel, 2011). Recognizing that many AuD programs include specific courses in CAPD and its underlying anatomy and physiology, we remain hopeful that re-administration of these surveys in 5 to 10 years will reveal the benefits of the transition of audiology to a doctoral level profession.

It is beyond the scope of this chapter to detail the courses and training experiences needed to properly prepare students to serve patients with CAPD, although a recent technical report offers some guidance in this regard (AAA, 2010). At a minimum, undergraduate students aspiring to the audiology profession should focus on biological and physical sciences, as well as courses in psychology focused on the physiological, experimental, and perceptual foundations. At the graduate level, the neurosciences should be pursued along with focused courses on anatomy and physiology of the CANS. Psychoacoustics and pediatric audiology courses also are crucial, in addition to a heavy dose of courses devoted to CAPD, including electrophysiology. It is interesting to note that some of the most used procedures in evaluating CAPD are also the oldest (Emanuel et al., 2011). This is understandable from one respect but also may indicate a rather pedestrian uptake in the education and knowledge of newer (and at times more involved) procedures.

In addition to more extensive course work and clinical experiences, it is probably as important that faculty and clinical supervisors instill in their students an appreciation of the essential role of science in professional practice. As elaborated above, a strong relationship between the scientist and clinician needs to be nurtured.

A specialty certification in the area of CAPD (as well as strong continuing education programs) may be needed to achieve the considerable enhancements and consistency in education and training that emphasize neuroscience and pathophysiology. A specialty certification could require certain types of clinical experiences and courses prior to delivering services in the area of CAPD. Alternatively, or in conjunction with specialty certification, one could make opportunities available to clinicians to work alongside *master* clinicians (e.g., fellowships) who have proven knowledge and experience in CAPD, either during or after university training. From a continuing education perspective, in 2012 four major international meetings (Hong Kong, Boston, Lyon, and Melbourne) were held on the topic of CAPD. These meeting were well attended and certainly provided considerable opportunities for clinicians and scientists to enhance their knowledge of CAPD. Until there is even greater emphasis on CAPD and related topics in the university training programs, continuing education efforts need to be maximized to keep professionals current.

[redacted]derserved Clinical [redacted]ulations With CAPD

One of the most exciting facets of future diagnostic work in the area of CAPD is the potential for expansion of the clinical population base. Currently, children with learning problems are the largest component of audiological referrals for central auditory testing. While audiologists must continue to serve this clinical population, there are other clinical populations that currently are underserved, to the detriment of both the patients and the clinician.

Traumatic Brain Injury

Many patients with head injury have a compromised CANS and when tested demonstrate central auditory deficits (Musiek et al., 2004). Unfortunately, most often these patients receive no audiological

evaluation of any sort. This is especially unfortunate given the emerging evidence that auditory training techniques can help these patients (Musiek et al., 2004). From an audiological viewpoint, this population is clearly underserved, and efforts should be made to expand services to this population in the future.

Surgery of the CANS

In the field of otology it is commonplace to conduct pre- and postsurgical audiological evaluations. For example, pre -and posthearing testing allows one to measure the effect of myringotomy and tubes for otitis media. Strikingly, however, auditory testing seldom precedes neurosurgical procedures that remove portions or all of the temporal lobe, despite risk to the CANS imposed by these major surgical procedures (Berlin, Lowe-Bell, Jannetta, & Kline, 1972). Inroads must be made here to better serve the audiological needs of these patients.

Older Adults

CAPD is a major component of presbycusis. The prevalence of CAPD among older adults has been rather well documented, ranging from 23% to 76% in community-based samples (Cooper & Gates, 1991; Golding, Carter, Mitchell, & Hood, 2004) to 70% in a clinical sample (Stach et al., 1990). Older listeners tend to require longer temporal cues and exhibit delayed N1–P2 latencies for speech stimuli (Gordon-Salant et al., 2008; Tremblay et al., 2004). In fact, age-related decline in temporal processing may commence much earlier, perhaps in the fourth decade of life (Kumar, 2011). The interdependence of the peripheral and central auditory systems has also been demonstrated. Even a modest degree of hearing loss can reduce the degree to which central auditory structures (e.g., bilateral superior temporal gyri, thalamus, brainstem) are activated (as seen in fMRI) when listening to complex sentences. Moreover, a linear relationship has been observed between hearing acuity and gray matter volume in the primary auditory cortex (Peelle et al., 2011). These findings clearly carry implications for interventions to improve communication in the elderly, who can be expected to present with both peripheral and central auditory deficits. (See Amplification below and Chapter 15 of Volume 2 of this Handbook.)

Stuttering

Renewed interest in investigating the CANS of individuals who stutter may be on the horizon. Historically, some postulated that those who stutter might experience CANS dysfunction. Implicating the possible linkage of stuttering and the CANS have been reports of differences in dichotic listening and temporal processing between those who stutter and controls (Cimorell-Strong, Gilbert, & Frick, 1983; Meyers, Hughes, & Schoney, 1989; Sommers, Brady, & Moore, 1975). More recently, delayed auditory feedback as well as frequency transposition feedback techniques have demonstrated clear effects on stuttering severity (Stuart & Kalinowski, 2004). Though most current auditory-stuttering research is directed toward auditory evoked potentials, behavioral techniques likely will continue to reveal interesting central auditory relationships in those with fluency disorders. Future studies should

employ extensive testing with efficient procedures, both behavioral and electrophysiological. In addition, stutterers representing various age groups should be studied.

Auditory Hallucinations

Although the primary population experiencing auditory hallucinations are those with psychiatric disorders (schizophrenia) (Johns et al., 2002), audiologists have been interested in auditory hallucinations recently, especially in Europe. Recent studies demonstrate that auditory hallucinations involve the CANS and can be precipitated by hearing loss (i.e., deprivation) and/or damage to the CANS (Berrios, 1990). Electrophysiological studies have documented different patterns of auditory processing in individuals with chronic schizophrenia (Domjan, Csifcsak, Drotos, Janka, & Szendl, 2012), and functional imaging studies have shown activation of auditory areas of the cortex during auditory hallucinations (Shergill et al., 2004). Given the frequency with which individuals with schizophrenia have auditory hallucinations, audiologists should be involved in multidisciplinary evaluations of this population. Even more compelling, it has been shown that those with schizophrenia also have decreased neural volume in the auditory cortex, likely secondary to some form of degeneration, as well as poor central auditory function involving Heschl's gyrus and the corpus callosum (i.e., interhemispheric transfer) (Henshall et al., 2012; Hubl et al., 2010). Audiologists may one day become involved in the evaluation and treatment of central auditory dysfunction related to this intriguing psychiatric disorder.

Dyslexia

Significant progress has been made toward understanding the biological basis of dyslexia. Human and animal studies have shown anatomical, behavioral, and electrophysiological auditory abnormalities in dyslexia (Amitay, Ahissar, & Nelken, 2002; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Poelmans, Luts, Vandermosten, Boets, Ghesquiere, & Wouters, 2012; Szalkowski & Fitch, 2012; Widmann, Schroger, Tervaniemi, Parkarinen, & Kujala, 2012; Zaidan & Baran, 2013). In humans, ectopic areas in and near the auditory areas of the brain and polymicrogyria often involving the auditory cortex have been reported (Boscariol, Guimaraes, Hage, Cendes, & Guerreiro, 2011; Galaburda et al., 1985), as well as dysfunction of the auditory thalamus (the medial geniculate body [Diaz, Hintz, Kiebel, & von Kriegstein, 2012]). Rodents with spontaneous and induced malformations of the developing cortex, like those anatomic malformations seen in humans with dyslexia, exhibit deficits in auditory processing similar to those performance deficits seen in humans with central auditory processing and learning deficits (Threlkeld et al., 2007).

Dyslexia is a polygenic developmental reading disorder (Giraud & Ramus, 2013). Szalkowski and Fitch (2012) identified several genes that lead to aberrant neuronal migration in the neocortex, aberrant migration that is similar to that observed in brain tissue taken postmortem from individuals with dyslexia. These same genes have been implicated in deficits in rapid auditory processing, spatial learning, and working memory in animal models. Central auditory processing deficits have been documented in humans with dyslexia and in genetically

engineered animal models of dyslexia (Boscariol et al., 2011; Szalkowski & Fitch, 2012). Clearly, the accumulating basic science evidence implicates the auditory system as perhaps the key system expressing dysfunction in this common learning problem. Additional audiological study of this disorder is needed.

Peripheral Hearing Loss and Amplification

Audiologists who see individuals requiring amplification may find useful the following two concepts related to CAPD: binaural interference and asymmetry of function. Binaural interference is reflected in poorer binaural function than the best monaural function (Jerger, Silman, Lew & Chmiel, 1993). This can result from considerable unilateral compromise at the peripheral or central level. The clinical uptake from binaural interference is that in these cases monaural amplification may be more effective than binaural amplification, contrary to the predominant approach whereby most patient populations benefit from the numerous advantages of binaural hearing.

The second concept is often, but not always, related to the first. Individuals with CAPD who are hearing aid candidates due to comorbid peripheral auditory involvement may demonstrate symmetrical hearing for tonal thresholds and speech recognition, but asymmetrical performance on behavioral and/or electrophysiological central auditory tests (Musiek & Baran, 1996). Such findings must be considered in the hearing aid evaluation process and certainly could be the basis for poor hearing aid adjustment after receiving amplification (Jerger, Silman, Lew, & Chmiel, 1993).

[redacted]reening and Early Identification

Better screening tools and earlier detection of CAPD are essential to minimize the functional consequences of CAPD. Certainly the earlier CAPD can be identified, the earlier intervention can be started, which is crucial to minimizing the impact of CAPD and successful intervention. Given the variability of young, normal children's performance on the currently available behavioral measures of central auditory function, it is difficult to identify those young children (below 7–8 years) in need of follow-up. Contributing to much of this variation is the long maturational course of the CANS (Thompson et al., 2003). Sensitive and specific screening procedures must be developed that can be readily administered and easily completed by young children. Developing such procedures is difficult while still maintaining the level of difficulty required to challenge the CANS. This indeed will be one of our great challenges, but one that must be met.

Several studies suggest that evoked potentials, perhaps paired with traditional behavioral paradigms assessing central auditory behaviors that have a short maturational course, may hold promise as tools for early identification. For example, infrequent stimuli with silent gaps were shown to modulate P2 and generate MMNs in normal 6-month-old infants (Trainor, Samuel, Desjardins, & Sonnadara, 2001). Also illustrating the potential to identify CANS dysfunction in younger children, Trehub, Schneider, and Henderson (1995) reported that normal 6-month-old infants detected gaps down to 12 ms using visual reinforcement audiometry.

Summary and Conclusions

The future of CAPD is one with many challenges, but also one with many opportunities. It is necessary that we continue to develop tests that are highly sensitive and specific to central auditory dysfunction. This may mean depending more on electrophysiological procedures, as well as behavioral tests that are resistant to confounds such as language and attention. Our clinical doctoral (AuD) programs must better education tomorrow's clinicians. Clinicians must embrace change and become better grounded in the neurosciences. Likewise, researchers hoping to advance clinical practices must understand clinical needs and constraints. Improvements in all of these areas will yield dividends for our patients as well as our discipline and profession. The challenges ahead will be exciting and advances will come. We must be steadfast, patient, and proceed with strong science and enthusiasm.

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GLOSSARY

Absorption. Property of a material or an object whereby sound energy is converted into heat by propagation in a medium or when sound strikes the boundary between two media. It is determined for a specified frequency or for a stated frequency band.

Accommodation. Making facilities and programs accessible to and usable by persons with disabilities through appropriate changes, including policy adjustments, task restructuring, adjusted schedules, equipment acquisition or alteration, training, or provision of qualified readers or interpreters, and other similar accommodations.

Acoustic access. Access through the auditory channel, either unaided or aided, to acoustic information.

Acoustic saliency. An acoustically salient phoneme (speech sound) or word is one that is obvious and prominent in an utterance. In a sentence context, acoustically nonsalient morphemes are shorter in duration and softer than louder phonemes in adjacent portions of the utterance.

Adaptive training. Training in which stimulus parameters vary on the basis of the participant's performance on preceding trials.

Afferent. Used to refer to neurons carrying information to the brain, such as those in the ascending auditory pathways.

Amplitude modulation (AM). Variation in the envelope of a sound over time.

Analog. Refers to a signal that varies continuously over time.

Assessment. Formal and informal procedures to collect data and gather evidence; delineation of functional areas of strength or weakness and/or determination of ability or capacity in associated areas.

Assistive listening system. A device that delivers sound to individuals with peripheral or central auditory deficits to mitigate listening problems (e.g., frequency modulated (FM) systems, personal amplifiers, infrared systems).

Association area. Areas of the cerebral cortex not believed to receive direct sensory inputs or send outputs to motor neurons, but to communicate with other cerebrocortical areas.

Attention. Gateway to conscious experience; maintains primacy of certain information in ongoing information processing.

- **Selective (focused) attention.** Ability to focus on relevant stimuli while ignoring simultaneously presented, but irrelevant stimuli (i.e., distractors).
- **Divided attention.** Ability to attend to multiple stimuli simultaneously.
- **Sustained attention (vigilance).** Ability to inhibit interference; requires sustained focus for a period of time while awaiting the occurrence of a target stimulus.

Attention deficit hyperactivity disorder (ADHD). Persistent pattern of inattention and/or hyperactivity-impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development; manifested in multiple settings; interferes with developmentally appropriate social, academic, or occupational functions; and has been present prior to age 12 years.

- **Combined type.** Attention deficit characterized by hyperactivity-impulsivity and inattention.
- **Predominantly inattentive type.** Presents primary symptoms of inattention.
- **Predominantly hyperactive-impulsive type.** Behavioral regulation disorder.

Attenuation. Reduction in magnitude of a physical quantity such as sound, either by electronic means (e.g., by an attenuator), or by a physical barrier, including various absorptive materials. It is usually measured in decibels.

Auditory brainstem response (ABR). A sequence of synchronous electrical activity in the auditory brainstem (i.e., auditory [eighth nerve] nerve, cochlear nuclei, superior olivary complex, trapezoid body, lateral lemniscus, inferior colliculus, and possibly the medial geniculate body) in response to an auditory or acoustic stimulus.

Auditory cortex. Area of the cerebral cortex that is the final destination of auditory inputs; located in the floor of the lateral sulcus in the superior temporal gyrus; see also Primary auditory area.

Auditory discrimination. Differentiating similar acoustic stimuli that differ in frequency, intensity, and/or temporal parameters.

Auditory evoked response/auditory evoked potential (AEP). Synchronous electrical activity (potentials) within the auditory nerve or auditory regions of the brain evoked by auditory or acoustic stimuli.

Auditory late latency response (ALLR). Synchronous electrical activity (N1–P2–N2 complex) occur-

ring in the range of approximately 60 to 250 msec arising from thalamocortical regions (i.e., superior temporal plane, lateral temporal lobe, and adjacent parietal regions).

Auditory middle latency response (AMLR). Synchronous electrical activity (potential) arising from the midbrain (i.e., Na component) and the superior temporal gyrus (i.e., Pa) within the auditory cortex.

Auditory neuropathy spectrum disorder (ANSD). A *peripheral* neural disorder, thought to arise from spiral ganglion cells, their processes, and/or the eighth (auditory) cranial nerve, characterized by normal otoacoustic emissions (OAEs), a recordable cochlear microphonic (CM), and an absent or grossly abnormal auditory brainstem response (ABR). The primary complaint of a person diagnosed with ANSD is difficulty understanding speech, especially in background noise.

Auditory training. Direct auditory skills remediation to maximize processing (use) of acoustic signals; set of acoustic conditions and/or repetitive tasks designed to activate auditory and related systems, change their neural base, and improve the ability to perceive auditory events.

Aural rehabilitation. 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 functioning.

Backward masking. The presence of one sound renders a previously presented sound less detectable.

Binaural fusion. The fusion (merging) of signals from the two ears into a single coherent sound image at the level of the brainstem.

Binaural interaction. Central auditory processing of intensity or time differences of acoustic stimuli presented diotically to the two ears.

Binaural interference. A phenomenon whereby the performance of the poorer ear interferes with that of the better ear resulting in poorer binaural performance than monaural performance of the better ear.

Binaural masking level difference. A measure of the advantage in signal detection that can result from the use of binaural cues; the difference in signal threshold between a situation in which the masker and signal have the same interaural time difference and interaural level difference, and a situation in which the interaural time and/or level differences for the masker and the signal differ.

Binaural summation. A summation of the acoustic energy that is presented to the two ears simultaneously, which affects thresholds and suprathreshold loudness.

Bottom-up processing. Information processing that is data driven; properties of the data are primary determinants of higher level representations and constructions.

Brain imaging. Procedures used to map the structure and metabolic and electrophysiological properties of the brain; includes computed tomography, magnetic resonance imaging, positron emission topography, regional cerebral blood flow, and brain electrical activity mapping.

Central auditory nervous system (CANS). The auditory brainstem, subcortical pathways, auditory cortex, and corpus callosum.

Central auditory processes. Auditory system mechanisms and processes that underlie the following abilities or skills: sound localization and lateralization; auditory discrimination; auditory pattern recognition; temporal aspects of audition including, temporal integration, temporal discrimination (e.g., temporal gap detection), temporal ordering, and temporal masking; auditory performance with competing acoustic signals (including dichotic listening); and auditory performance with degraded acoustic signals.

Central auditory processing disorder (CAPD). Difficulties in the perceptual processing of auditory information in the central nervous system, that cannot be *attributed to* higher order language, cognitive, or related supramodal confounds, and manifests as poor performance in one or more of the central auditory processes, with associated changes in the neurobiologic activity underlying those processes that give rise to the auditory evoked potentials.

Characteristic frequency. The pure tone frequency to which a given place on the basilar membrane, or a given neuron in the auditory system, is most sensitive at low stimulation levels.

Classroom audio distribution system (CADS). A system whose primary design goal is to electroacoustically distribute the audio portion of spoken communications and curricular content throughout the learning space or listening area.

Clear speech. Speech produced by a speaker who has been instructed to speak as clearly as possible, as if trying to communicate in a noisy background.

Clinical decision analysis (CDA). A quantitative, systematic approach to clinical decision making derived from signal detection theory.

Closure. The ability to subjectively complete and make whole an incomplete form. Listeners use language knowledge and inductive and deductive reasoning, as well as auditory and grammatic closure to derive the meaning of words and messages.

- **Auditory closure.** The ability to recognize a whole word despite the absence of certain elements.
- **Grammatic closure.** The ability to complete phrases or sentences despite missing words or morphemes (e.g., filling in the verb form *are* versus *is* to conjugate with the subject *they*).
- **Verbal auditory closure.** The ability to use spoken contextual information to facilitate speech recognition.

Cochlear microphonic (CM). An alternating current potential that follows the waveform of the stimulus and the vibrations of the basilar membrane. One of two potentials comprising the ECochG. See Electrocochleography.

Cognition. Activity of knowing, encompassing the acquisition, organization, and use of knowledge; automatic and unconscious processes that transform, reduce, elaborate, store, recover, and use sensory input; processes involved in knowing, including perceiving,

recognizing, conceiving, judging, sensing, and reasoning; primary phase in the development of knowledge.

Cognitive style. An individual's approach to processing information, problem solving, and cognitive tasks (e.g., bottom-up/top-down, impulsive/reflective, field dependent/field independent).

Commissure. A group of axons of neurons passing from one side of the brain, usually, to a similar structure on the opposite side of the brain.

Commissurotomy. The medical term for surgical sectioning of a brain commissure, usually the corpus callosum.

Comorbidity. Existence of two or more disorders, diseases, or pathologic processes in an individual that are not necessarily related.

Compensation. Rehabilitative approach directed toward reducing the negative impact of a disorder or disease not amenable to complete recovery through treatment.

Consonant-vowel (CV). Nonsense syllable comprised of a consonant followed by a vowel (e.g., ba, da, ga).

Corpus callosum. Principal commissure of the cerebral hemispheres.

Critical distance. Distance from a sound source at which direct sound level and reverberant sound level are equal.

Damping. Dissipation of energy with time or distance; loss of energy in a system resulting from friction (internal or external) or other resistance.

Deductive inferencing. Reasoning from the general to the specific.

Desktop sound field FM system. A self-contained system designed for personal or small group listening in which the FM signal transmitted over a wireless microphone is presented to the listener(s) via a speaker which is placed on the user's desk.

Depolarization. An increase in the electric potential of a hair cell or neuron from a negative resting potential.

Diagnosis. Identification and categorization of impairment/dysfunction; determination of presence and nature of a disorder.

Dichotic. Simultaneous presentation of two different acoustic events, one to each ear.

Difference limen. Just noticeable difference or smallest detectable change in a stimulus, usually pertaining to frequency, intensity, or duration; the difference in a quantity that a listener can just detect at some criterion level of performance.

Differential diagnosis. Distinguishing between two or more conditions presenting with similar symptoms or attributes.

Diffraction. Bending of sound waves around obstacles whose dimensions are smaller than the wavelength of the sound; the spreading out of waves beyond openings that are smaller than the wavelength of the sound. Diffraction involves a change in direction of a wave as it passes through a small opening or around a barrier in its path.

Diffusion. Process of spreading or dispersing radiated energy so that it is less direct or coherent. In acoustics, diffusion is caused by sound waves reflected from an uneven surface.

Distortion. Undesired change of a waveform resulting in the presence of some frequency components in

the output signal that are not present in the input signal.

Dynamic assessment. Approach to evaluation focused on the different ways by which an individual achieves a score rather than the score achieved; approach is characterized by guided learning to determine an individual's potential for change.

Effectiveness. Effects of treatment; how well a treatment works in real-world settings.

Effect size. Calculated measure used to determine the extent of practical significance for particular research results.

Efferent system. The portion of the auditory system, also called the descending system, that courses from the brain down to the cochlea following a similar pathway as the afferent system.

Efficacy. Effects of treatment; how well a treatment can work under ideal circumstances and adequate control; documenting treatment efficacy requires demonstrating that a particular treatment produces the desired outcomes or behavior change in an efficient manner (e.g., cost effective) as a result of the treatment.

Efficiency. A measure of a test's combined sensitivity and specificity; ability of a test to identify correctly those individuals who have the dysfunction/disorder and correctly identify those individuals who do not have the dysfunction.

Electroacoustic measures. Recordings of acoustic signals from within the ear canal that are generated spontaneously or in response to acoustic stimuli (e.g., otoacoustic emissions, acoustic reflexes).

Electrocochleography (ECochG). An auditory evoked response that arises from the cochlea and eight (auditory) cranial nerve within the first 2 to 3 msec. following an abrupt stimulus. See Cochlear microphonic.

Electrophysiologic measures. Recordings of electrical potentials that reflect synchronous activity generated by the central nervous system in response to a wide variety of acoustic events (e.g., auditory brainstem response, steady-state evoked potentials, auditory middle-latency response, frequency following response, cortical auditory event-related potentials [P1, N1, P2, P300]).

Endogenous. Refers to evoked potentials (e.g., P300) that are relatively invariant to changes in the eliciting physical stimulus, but are highly influenced by subject state and require an internal or mental activity (e.g., perceptual or cognitive process) to generate the potential.

Evaluation. Interpretation of assessment data, evidence, and related information.

Evidence-based practice. Explicit and judicious use of current best evidence in making decisions about the care of individual patients by integrating individual clinical expertise with the best available external clinical evidence from systematic research; a systematic method to evaluate and implement best practices for assessment and treatment in clinical fields.

Executive function. Component of metacognition; set of general control processes that coordinate knowledge (i.e., cognition) and metacognitive knowledge, transforming such knowledge into behavioral

strategies, which ensure that an individual's behavior is adaptive, consistent with some goal, and beneficial to the individual; self-directed actions of an individual that are used to self-regulate so as to accomplish self-control, goal-directed behavior, and maximize future outcomes.

Exogenous. Refers to evoked potentials that are highly dependent on acoustic features of the stimulus.

Extra-axial. Lesions of the brainstem that do not arise from within the brainstem, but from near structures that encroach upon the brainstem.

Forward masking. The presence of one sound renders a subsequent sound less detectable.

Free field. A sound environment in which there are no significant effects on sound propagation from boundaries and the medium (air) is homogeneous and motionless; under free-field conditions, the loss of energy with distance may be predicted by the inverse square law.

Gyrus (*pl.* gyri). Bulge on the surface of the cerebral cortex consisting of gray matter with an inner core of white matter.

Hearing assistive technology. See Assistive listening system.

Impedance. Quotient of a dynamic field quantity (e.g., sound pressure) by a kinematic field quantity (e.g., particle velocity), at a specified frequency; total opposition to energy flow expressed in ohms.

Incidence. Number of individuals who contract a disease during a particular period of time.

Individuals with Disabilities Education Act (IDEA)/Individuals with Disabilities Education Improvement Act (IDEIA). Federal education acts that guarantee special education and related services to children with disabilities.

Induction learning. Discovery learning; a three-step process through which a learner recognizes a pattern or relationship, explains the pattern or relationship, and hypothesizes the rule governing the pattern or relationship.

Inductive inferencing. Reasoning from the particular facts to a general conclusion.

Inferencing. Reaching a conclusion on the basis of facts or evidence.

Information processing. Assigning meaning to sensory input based on the extraction of cues or constraints through various processes or stages of cognition, including encoding, organizing, storing, retrieving, comparing, and generating or reconstructing information; these stages involve the interaction between sensory (e.g., auditory processes) and central processes (e.g., cognitive and linguistic processes) through feedback and feedforward loops.

Interaural timing. Refers to a behavioral task requiring the subject to determine the order of two acoustic events presented to each ear separately at slightly different times.

Intervention. Comprehensive, therapeutic treatment and management of a disorder.

Intra-axial. Refers to lesions of the brainstem that evolve from the brainstem tissue itself, as opposed to extra-axial lesions that arise from nonbrainstem tissue. Extra-axial lesions often are in contact with the brainstem.

Inverse square law. Principle whereby under free field conditions, sound intensity varies inversely with the

square of the distance from the source; sound intensity I (in W/m^2) measured at distance r (in m) from the source producing the power P (in W) is described as $I = P/(4\pi r^2)$. Thus, if distance is doubled, sound intensity decreases by a factor of four. When expressed in decibels, level decreases by 6 dB for each doubling of the distance from the source to the point of measurement.

Isolation point. A real-time word recognition processing event, which occurs at the gate when the listener initially identifies the target word.

Latency. The time between occurrence of a physiologic event, usually a spike or evoked potential, and a stimulus.

Lateralization. Process of determining the location of a sound inside the head (i.e., intracranial) within the plane between the two ears.

Learning disabilities. A heterogeneous group of disorders, presumed to be due to central nervous system dysfunction, manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing, reasoning, or mathematical abilities.

Learning style. An individual's characteristic cognitive, affective, modality, and physiological behaviors and preferences employed in perceiving, interacting with, and respond to the learning environment.

Lexical access. A spoken language processing event in which a percept comes in contact with various features of stored lexical representations.

Lexical activation. Some change in status of a subset of word candidates contained in the mental lexicon.

Linguistic-contextual information. Anything that influences the a priori probability of an uncoming utterance or the post hoc, retroactive recognition of an ongoing utterance.

Localization. Process of determining the location of a sound in the environment.

Management. Procedures (e.g., compensatory strategies, environmental modifications) targeted toward reducing the effects of a disorder and minimizing the impact of the deficits that are resistant to remediation.

Masking. Process by which the threshold of one sound is raised by the presence of another (masking) sound; presence of one sound renders a subsequent sound less detectable.

Memory. Capacity to encode, process, and retrieve events, knowledge, feelings, and decisions of the past.

- **Short-term memory.** Brief storage of limited capacity with minimal processing requirements.
- **Working memory.** Temporary storage of information used during reasoning and planning; involves both storage and executive processing and manipulation of information.
- **Long-term memory.** Declarative or explicit memory and procedural or implicit memory; long-term storage of unlimited capacity; involves both storage and processing of information.
- **Declarative or explicit memory.** Conscious awareness or recollection of previously acquired information, retrieved on demand.
- **Procedural or implicit memory.** Use of previous experience or

knowledge, in the absence of conscious awareness or recollection, to support learning and guide performance.

Mesencephalic. Referring to the midbrain, just rostral to the pons.

Meta-analysis. Synthesis of treatment efficacy literature (randomized controlled trials) on a given topic using mathematical procedures to integrate results from multiple studies.

Metacognition. Awareness and appropriate use of knowledge; awareness of the task and strategy variables that affect performance and the use of that knowledge to plan, monitor, and regulate performance, including attention, learning, and the use of language; second phase (following cognition) in the development of knowledge which is active and involves conscious control over knowledge.

Metalinguistics. Aspects of language competence that extend beyond unconscious usage for comprehension and production; involves ability to think about language in its abstract form-- to reflect on aspects of language apart from its content, analyze it, and make judgments about it; metalinguistic knowledge underlies performance on a number of tasks, including phonological awareness (e.g., segmentation, rhyming), organization and storage of words (e.g., multiple meaning words), and figurative language (e.g., metaphor, idiom, humor); may be considered a subset of metacognition since using language is one of the goals of metacognitive processes.

Metamemory. Knowledge and awareness of one's own memory systems and strategies.

Minimum audible angle. The smallest detectable angular separation between two sound sources relative to the head.

Mismatch negativity (MMN) response. A negative wave (electrical response), generated from a broad region extending from the frontal lobes to the auditory regions of the temporal lobes, to an unattended, rare (or deviant) auditory stimulus resulting from the subtraction of the waveform to the standard (attended) stimulus from the waveform to the deviant stimulus (i.e., "difference wave").

Mnemonics. Artificial or contrived memory aids for organizing information (e.g., acronyms, rhymes, verbal mediators, visual imagery, drawing).

Myogenic. A response that is generated by muscle contractions.

Neural synchrony. Pattern of neural activity in which large populations of neurons fire simultaneously; this type of neural activity generates the electric activity giving rise to auditory evoked potentials. Neural synchrony facilitates transmission of activity across central synapses and becomes more important downstream the auditory pathway.

Neuroaudiology. Study of the auditory nervous system as it relates to hearing.

Neurobiology. Encompasses neuroanatomy, physiology, neurochemistry, and neuropharmacology.

Neuropharmacology. Effects of drugs on neuronal tissue.

Neurotransmitter. Chemical agent released by vesicles of a nerve cell that permits synaptic transmission between neurons, between sensory cells and neurons, and between neurons and muscle cells.

No Child Left Behind Act (NCLB). A federally mandated statute enacted in 2002 designed to improve student achievement in the public schools.

Otoacoustic emissions. Subaudible sounds generated by the outer hair cells in the cochlea either spontaneously or evoked by sound stimulation.

P300. A cognitive/sensory response to auditory (or other sensory modality stimulation) reflecting attention and sensory processing to a stimulus thought to arise from neural generators in the medial temporal lobes, temporal-parietal region, and prefrontal cortex.

Pansensory. Referring to higher level mechanisms that are common to and that support processing across all modalities.

Perceptual training. Regimens in which basic perceptual attributes (e.g., sound frequency or duration) are trained through repeated exposure to a task (typically discrimination or identification).

Personal FM system. A system consisting of a wireless microphone transmitter used by the speaker and a receiver used by the listener which is coupled to the listener's ears via headphones, ear buds, or through personal hearing aids (using direct audio input, induction neckloop, or Bluetooth).

Phase. Proportion of a period through which the waveform of a sound has advanced relative to a given time.

Phase-locking. Tendency of an auditory neuron to fire at a particular time (or phase) during each cycle of vibration on the basilar membrane; more generally, as seen in central neurons, the firing at roughly the same phase of the stimulus frequency cycle, but generally not for every cycle (this happens only for frequencies well below 1 kHz).

Pharmacology. Sources, chemistry, actions, and uses of drugs.

Phonemic analysis. Separating words or syllables into a sequence of phonemes.

Phonemic synthesis. Blending of discrete phonemes into the correctly sequenced, coarticulated sound patterns.

Phonological awareness. Explicit awareness of the sound structure of language, including the recognition that words are composed of syllables and phonemes.

Plasticity. Reorganization of the cortex by experience, often reflected in behavioral change (i.e., learning); alteration of neurons to conform better to immediate environmental influences, often associated with a change in behavior; changes in the properties of individual neurons or neuronal assemblies following specific use, pattern of stimulation, injury or during development; neural reorganization may be possible to some extent across the life span, as well as following injury (compensatory plasticity), and in response to learning.

Posterior probabilities. The probability that a patient actually has a disease given a positive test result, the probability that a patient does not have a disease given a negative result, and the probability of being incorrect given a test result (i.e., the patient does not have disease despite a positive test result or the patient has the disease despite a negative test result). Posterior probabilities reflect the probability of an outcome

given the known prevalence of the disorder in the population.

Precedence effect. Refers to the dominance of information from the leading sound (as opposed to delayed or reflected versions of that sound) for the purpose of sound localization; the effect occurs for stimulus time delays varying from fractions of a millisecond to the upper limit for auditory fusion, after which separate sounds are perceived.

Presbycusis. Age-related hearing loss; the gradual, progressive loss of hearing that occurs as people age.

Prevalence. Total number of cases of a specific disease or disorder existing in a given population at a certain time.

Prevention. Procedures targeted toward reducing the likelihood that impairment will develop.

Primary auditory area (or cortex). The main auditory area of the brain, typically considered to be Heschl's gyrus.

Problem solving. Generating a variety of potentially effective responses to a situation and recognizing and implementing the most effective response.

Prosody. Suprasegmental aspects of spoken language; the dynamic melody, timing, rhythm, and amplitude fluctuations of fluent speech.

Psychoacoustics. The study of the relation between sound (i.e., physical parameters) and perception (i.e., psychological correlates) using behavioral measurement techniques.

Real-time speech. The transitory, ephemeral nature of an ongoing speech signal; when speech is presented in a real-time manner, listeners must quickly recognize phonemes, syllables, and words based on preceding linguistic-contextual cues and ongoing acoustic-phonetic information.

Reasoning. Evaluation of arguments, drawing of inferences and conclusions, and generation and testing of hypotheses.

Receiver operating characteristic (ROC) curve. A plot of the effects of sensitivity (d′) and a subject's response criterion on the probability of hits (i.e., subject's correct detection of a signal) and false alarms (i.e., subject's detection of a signal when it is not present). A plot used in clinical decision analysis (CDA) to examine how sensitivity and specificity change as a function of different test cutoff scores.

Reciprocal teaching. Alternating roles between the client and clinician, allowing the client to assume the role of teacher as well as learner.

Reflection. Acoustical phenomenon that occurs whenever sound strikes a surface; reflected sound is the portion of the sound energy striking the surface that bounces off the surface.

Reliability. The consistency, dependability, reproducibility, or stability of a measure.

Remediation (or treatment). Procedures targeted toward resolving an impairment.

Reverberation. Persistence or prolongation of sound in an enclosed space, resulting from multiple reflections of sound waves off hard surfaces after the source of the sound has ceased. Reverberation time (RT_{60}) refers to the time required for a steady-state sound

to decay 60 dB from its initial peak amplitude offset.

Schema. Structured cluster of concepts and expectations; an abstract and generic knowledge structure stored in memory that preserves the relations among constituent concepts and generalized knowledge about a text, event, message, situation, or object.

- **Formal schema.** Linguistic form that organizes, integrates, and predicts relationships across propositions (e.g., additives [*and, furthermore*], adversative [*although, nevertheless, however*], causal [*because, therefore, accordingly*], disjunctive [*but, instead, on the contrary*], and temporal connectives [*before, after, subsequently*], as well as patterns of parallelism and correlative pairs [*not only/but also*; *neither/nor*]).
- **Content or contextual schema.** Provides a generalized interpretation of the content of experience; organizes facts and establishes a framework that imposes certain structures on events, precepts, situations, and objects and facilitates interpretation.

Screening. Procedures used to identify individuals who are at risk for an impairment.

Segmentation. Parsing spoken language into its constituent and successive segments; parsing sentences, words, or syllables into their constituent phonetic units; the manner in which listeners demarcate the ongoing spoken utterance into units of lexical access.

Self-regulation. Encompasses metacognitive knowledge and skills, as well as affective/emotional, motivational, and behavioral monitoring and self-control processes.

Semantic network. Construct representing a mental system of nodes and links connecting lexical units; vocabulary building in such a network involves adding new nodes and links, as well as changing activation values of the links between nodes (e.g., building synonymy by strengthening the relationships between nodes).

Sensitivity. The ability of a test to yield positive findings when the person tested truly has the dysfunction/disorder; ability of a test to identify correctly those individuals who have the dysfunction/disorder.

Signal-to-noise ratio. Relationship between the sound levels of the signal and the noise at the listener's ear, commonly reported as the difference in decibels between the intensity of the signal and the intensity of the background noise (e.g., if the speech signal is measured at 70 dB and the noise is 64 dB, the signal-to-noise ratio is +6 dB).

Sound field. The area and/or pattern of air pressure disturbance caused by the compression and rarefaction of energy in the audio frequency range.

Specificity. Ability of a test to identify correctly those individuals who do not have the dysfunction/disorder.

Spectrum level. Level of sound contained in a 1-Hz-wide band; a measure of spectral density.

Speech intelligibility. Percentage of words, sentences or phonemes correctly received out of those transmitted; an important measure of the effectiveness or adequacy of a communication system or of the ability of people to communicate in noisy environments.

Spoken language processing. An interactive system of peripheral and central functions used to recog-nize and understand real-world transitory utterances as meaningful speech.

Standing wave. Phenomenon resulting from the interference of sound waves of the same frequency and kind traveling in opposite directions; characterized by the absence of propagation and the existence of nodes and antinodes that are fixed in space.

Sulcus. Infoldings on the cerebral surface separating gyri.

Synapse. Junction where information is transmitted between two neurons.

Synaptic transmission. Passage of an electrical impulse across a synapse through transduction to a chemical neurotransmitter presynaptically and transduction back to an electrical signal postsynaptically.

Systems theory. Study of systems as an entity rather than a conglomeration of parts; provides a conceptual framework for understanding the organization, interaction, and dynam-icity of elements comprising systems.

Temporal integration. Refers to the relationship between stimulus duration and intensity within a time frame of less than one-half second; integration of energy sampled within a time frame of approximately 200 milliseconds; sensitivity improves as signal duration increases up to approximately 200 to 300 milliseconds, after which thresholds remain essentially constant; also known as temporal summation.

Temporal masking. Masking that occurs when the signal and the masker do not overlap in time; also known as nonsimultaneous masking.

Temporal ordering. See Temporal sequencing.

Temporal processing. Auditory mechanisms and processes responsible for temporal patterning (e.g., phase-locking, synchronization) of neural discharges and the following behavioral phenomena: temporal resolution (i.e., detection of changes in durations of auditory stimuli and time intervals between auditory stimuli over time), temporal ordering (i.e., detection of sequence of sounds over time), temporal integration (i.e., summation of power over durations less than 200 milliseconds), and temporal masking (i.e., obscuring of probe by pre- or poststimulatory presentation of masker).

Temporal resolution. Refers to the shortest time period over which the ear can discriminate two signals; also known as temporal discrimination.

Temporal sequencing. The ability to discern the correct order of rapid acoustic events as they occur over time.

Tonotopic. Organization of auditory neurons in a particular structure according to their responsiveness to specific frequencies; a system of sound frequency representation in which the frequency determines the place (e.g., in a neural array) of activation.

Top-down processing. Information processing that is knowledge or concept driven such that higher level constraints guide data processing, leading to data interpretation consistent with these constraints.

TORCH+S complex. A group of perinatal medical problems often linked to hearing loss. T = toxoplasmosis; O = other (e.g., associated

ophthalmologic disease); R = rubella; C = cytomegalovirus; H = herpes; S = syphilis.

Total acceptance point. A late event in the real-time word recognition process when a listener recognizes the target word with a high level of confidence.

Treatment (remediation). Procedures targeted toward resolving an impairment.

Treatment outcomes. General term to denote change on measurements from pre to post intervention.

Tuning curve. A graph depicting the response of a neuron at a given percentage (e.g., 10%) above spontaneous activity, plotted as a function of stimulus intensity and frequency. The lowest sound level to which the neuron responds is represented by the tip of the tuning curve (i.e., characteristic frequency).

Two-alternative forced choice method (2AFC). A psychophysical method in which the participant is asked to decide which of two successive intervals contains a signal.

Validity. The degree to which a test measures what it is intended to measure.

Wernicke's area. The receptive auditory-language associational area of the cortex that may include part of the planum temporale and the postero-superior temporal gyrus.

Word predictability. Amount of fill-in-the blank meaningfulness in a preceding spoken context. In predictability-high (PH) sentences, preceding semantic-contextual information is presented in the form of clue words; no such clue words are available in predictability-low (PL) sentences.

Word recognition. A spoken language processing event marking the conclusion of the word selection phase; also refers to a listener's ability to perceive and correctly identify a set of words usually presented at suprathreshold hearing level.

Working memory. Holding information in mind while simultaneously manipulating or transforming that information.

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