

Review

Perspectives on the Pure-Tone Audiogram

DOI: 10.3766/jaaa.16061

Frank E. Musiek*
 Jennifer Shinn†
 Gail D. Chermak‡
 Doris-Eva Bamious§

Abstract

Background: The pure-tone audiogram, though fundamental to audiology, presents limitations, especially in the case of central auditory involvement. Advances in auditory neuroscience underscore the considerably larger role of the central auditory nervous system (CANS) in hearing and related disorders. Given the availability of behavioral audiological tests and electrophysiological procedures that can provide better insights as to the function of the various components of the auditory system, this perspective piece reviews the limitations of the pure-tone audiogram and notes some of the advantages of other tests and procedures used in tandem with the pure-tone threshold measurement.

Purpose: To review and synthesize the literature regarding the utility and limitations of the pure-tone audiogram in determining dysfunction of peripheral sensory and neural systems, as well as the CANS, and to identify other tests and procedures that can supplement pure-tone thresholds and provide enhanced diagnostic insight, especially regarding problems of the central auditory system.

Research Design: A systematic review and synthesis of the literature.

Data Collection and Analysis: The authors independently searched and reviewed literature (journal articles, book chapters) pertaining to the limitations of the pure-tone audiogram.

Results: The pure-tone audiogram provides information as to hearing sensitivity across a selected frequency range. Normal or near-normal pure-tone thresholds sometimes are observed despite cochlear damage. There are a surprising number of patients with acoustic neuromas who have essentially normal pure-tone thresholds. In cases of central deafness, depressed pure-tone thresholds may not accurately reflect the status of the peripheral auditory system. Listening difficulties are seen in the presence of normal pure-tone thresholds. Suprathreshold procedures and a variety of other tests can provide information regarding other and often more central functions of the auditory system.

Conclusions: The audiogram is a primary tool for determining type, degree, and configuration of hearing loss; however, it provides the clinician with information regarding only hearing sensitivity, and no information about central auditory processing or the auditory processing of real-world signals (i.e., speech, music). The pure-tone audiogram offers limited insight into functional hearing and should be viewed only as a test of hearing sensitivity. Given the limitations of the pure-tone audiogram, a brief overview is provided of available behavioral tests and electrophysiological procedures that are sensitive to the function and integrity of the central auditory system, which provide better diagnostic and rehabilitative information to the clinician and patient.

Key Words: audiogram, auditory evoked potentials, central auditory processing disorder, diagnostic techniques

*The University of Arizona, Tucson, AZ; †The University of Kentucky, Lexington, KY; ‡Washington State University Health Sciences Spokane, Spokane, WA; §University College London Hospitals, London, UK

Corresponding author: Frank E. Musiek, The University of Arizona, Tucson, AZ 85719; E-mail: fmusiek@email.arizona.edu

This paper was presented orally in part at the American Academy of Audiology in Anaheim, CA, April 3–6, 2013.

Abbreviations: ABR = auditory brainstem response; AEP = auditory evoked potential; AMLR = auditory middle latency response; AR = acoustic reflex; CANS = central auditory nervous system; CAPD = central auditory processing disorder; EP = evoked potential; GIN = Gaps-in-Noise Test; IHC = inner hair cells; MLD = masking level difference; OAD = obscure auditory dysfunction; OAE = otoacoustic emission; SIN = Speech-in-Noise Test; VA = Veterans Administration

INTRODUCTION

In the early 1980s, author Frank E. Musiek of this article discussed in formal and informal venues the concept of “beyond the pure-tone audiogram.” Though the details of these discussions have faded from memory over the years, these conversations led to a formal publication in 1986 entitled, “Hearing beyond the eighth nerve” (Musiek, 1986). This editorial highlighted the shortcoming of relying on only the pure-tone audiogram to evaluate hearing status. This was especially true in light of patients who had central auditory deficits. Clinical populations with compromise of the central auditory system often reported symptoms of hearing difficulties; however, their symptoms were not reflected in what was then the standard test of hearing—pure-tone audiometry—and its graphic representation, the audiogram.

Well before 1980, a number of reports demonstrated rather marked auditory deficits in people with central nervous system disorders; however, these deficits were manifested only on tests more complex than pure-tone audiometry. Research by Bocca and his Italian colleagues, as well as by Jerger, Kimura, and Katz in the mid-to-late 1950s and early 1960s, drew attention to the need for evaluating patients with central auditory lesions with audiological tests “sensitized” to central auditory dysfunction (Bocca et al, 1954; Jerger, 1960; Kimura, 1961; Katz, 1962). These early researchers ushered in the development of new tests and demonstrated the futility of using pure-tone thresholds to assess auditory compromise in patients with neuroauditory disorders.

Over the next several decades, considerable research amassed in the area of central auditory dysfunction, and mostly by inference, the limitations of pure-tone audiometry (Chermak and Musiek, 1997; Baran and Musiek, 1999; Musiek and Chermak, 2007). Several reports made clear that changes in the CANS can also occur in individuals with hearing loss. Several studies showed that both young and older adults with bilateral symmetric sensorineural hearing loss who received a monaural hearing aid fitting experienced a progressive decrement in their word recognition scores in their unaided ear, while word recognition scores in their aided ear remained unchanged in the absence of any change in their pure-tone audiograms. Individuals aided binaurally did not experience any decline in word recognition scores (Silman et al, 1984; Gelfand et al, 1987; Hurley, 1998). These findings suggest strong central effects of auditory deprivation in the unaided ear of

individuals fitted monaurally, that is, undetectable by the pure-tone audiogram. These studies corroborated patients’ complaints of decreased hearing with no apparent change in pure-tone thresholds.

The accumulating evidence of the pure-tone audiogram’s limitations has penetrated all aspects of audiology. In addition, advances in auditory neuroscience have begun to emphasize the considerably larger role of the central auditory nervous system (CANS) in hearing and related disorders. As a result of changing views on synaptic degeneration, tinnitus, homeostatic plasticity, brain reorganization, and the effects of noise on the auditory system, attention has turned from almost exclusive focus on the auditory periphery to include more central mechanisms (Schwaber et al, 1993; Morest et al, 1998; Lockwood et al, 2002; Eggermont, 2012).

This change in thinking has become apparent at major audiological conferences over the past two or three years. Featured presentations that focused on the topic of “beyond the pure-tone audiogram” were well attended (Shinn and Musiek, 2013; Musiek, 2015). More presentations than ever before have included comments referencing pure-tone limitations (Shinn and Musiek, 2013; Edwards, 2015). Audiologists now recognize the importance of assessing the central auditory system to fully evaluate auditory or hearing function. The purpose of this article is to elaborate this point, its rationale, and its implications. While recognizing the important role of the pure-tone audiogram in clinical audiology, we review data demonstrating the shortcomings of pure-tone threshold testing in the broader context of assessing overall hearing status. The audiogram’s appropriate use will be discussed, including the role of threshold versus suprathreshold measures. Next, several unique studies are highlighted to demonstrate that audiograms do not always reflect damage to the cochlea—a finding seemingly counterintuitive. This is followed by a section devoted to reports of various degrees of auditory symptoms and difficulties presented by individuals with normal pure-tone audiograms, reflecting an important disassociation between the pure-tone audiogram and patient complaints. Next, we discuss the finding, although seen in only a minority of cases, of normal pure-tone thresholds in those with auditory nerve involvement. We continue with a review of the initial impetus for testing beyond the audiogram—central auditory involvement—and conclude with a brief overview of available tests and procedures that assess the central auditory system, tests which in some instances also may provide insight into the function of the auditory periphery.

VALUE OF THE AUDIOGRAM

There is no denying that pure-tone audiometry has become the hallmark measure in the evaluation of patients with hearing disorders. It serves as a critical tool in the diagnosis of many audiological and otologic disorders. It is important in providing information regarding type, degree, and configuration of hearing loss. Additionally, it has vital diagnostic value in assisting physicians in making surgical decisions. The audiogram is an old tool, however, and has advanced relatively little over the years. As noted by Feldmann (1970), audiometric-related events date back to 377 BC, when Hippocrates reported clinical findings of hearing loss. Richardson coined the word “audiometer” in 1879, with Hartmann creating the first “auditory chart” in 1885 (Vogel et al, 2007). The first audiometers were manufactured in the 1930s, and while substantial technological advancements have occurred since (e.g., computer-based platforms), the fundamental technical and diagnostic principles underlying audiometry have remained essentially unchanged over the last 50+ years.

As audiologists, we are charged with evaluating, diagnosing, and treating an array of disorders of the auditory and vestibular systems. To do so requires that we understand the intricacies of the anatomy and physiology of these systems. While most audiologists are relatively well versed with respect to peripheral anatomy and physiology, fewer have the same depth of knowledge of the CANS. As a result, many audiologists are less comfortable with neuroaudiologic assessment and rehabilitation. As Chermak and colleagues documented in 2007 (Chermak et al, 2007), too few audiologists evaluate central auditory function, a situation which has improved somewhat in the last decade, although based on the inquiries the authors and their colleagues receive on a regular basis, locating an audiologist with this expertise can be challenging, especially in certain parts of the authors’ respective home countries, the United States and Great Britain.

The audiogram is excellent at determining degree and configuration of hearing loss, and in conjunction with other basic audiological tests (e.g., immittance and speech audiometry) helps determine the type of hearing loss. However, the audiogram provides the clinician with information regarding only hearing sensitivity, and no information about central auditory processing (despite the fact that pure-tone signals do reach the primary auditory cortex). Nor does the pure-tone audiogram provide any information regarding the auditory processing of real-world signals (i.e., speech, music). The CANS is an intricate neural communication network that plays a vital role in hearing. In fact, patients with CANS compromise (e.g., auditory agnosia) can present marked deficits in auditory pro-

cessing despite normal audiograms and, usually, relatively normal-hearing sensitivity (Kazui et al, 1990). (Also see discussion of central deafness below.) As our profession advances, so should our view and approach to diagnostic assessment and rehabilitation of the total auditory system, not simply the periphery.

A prime example of our professional transition away from the dependency on the audiogram is the evolution of candidacy criteria for cochlear implant recipients. During the early years of cochlear implants, only profoundly hearing-impaired patients were considered candidates. In more recent years, candidacy guidelines have become significantly less stringent, allowing patients with lesser degrees of hearing impairment to satisfy eligibility requirements. Perhaps even more important, however, is that functional performance in noise (e.g., using tests such as the Hearing-in-Noise Test and AzBio Test (Nilsson et al, 1994; Spahr et al, 2012) is given significantly more weight than pure-tone thresholds in determining eligibility for a cochlear implant.

The pure-tone audiogram is considered to offer limited insight into functional hearing and is therefore not considered to provide the most ecologically valid information regarding auditory function in real-world settings. There are numerous examples in the literature demonstrating that patients who present with CANS involvement may, and often do, yield normal auditory thresholds. As early as 1942, Karlin reported that the audiogram was a poor predictor of performance in complex listening environments (Karlin, 1942). An example of the failure of the audiogram to reveal evidence of higher auditory dysfunction also was observed by Bocca and colleagues in the 1950s (Bocca et al, 1954; 1955). Bocca’s work confirmed that patients with lesions of the CANS often present with normal audiograms, despite significant deficits with respect to processing degraded speech. This would have been undetected if the audiogram were the only test used to evaluate the auditory system.

Perhaps some of the most compelling examples of the limitations of the audiogram are seen in patients who present with central deafness (Musiek and Lee, 1998; Musiek, Baran, et al, 2007). Musiek, Baran, et al (2007) described a patient who presented with bilateral cerebrovascular accidents. The pure-tone audiogram revealed a severe to profound hearing loss bilaterally; however, further examination using otoacoustic emissions (OAEs), acoustic reflexes (ARs), and the auditory brainstem response (ABR) revealed normal function, bilaterally. Of note, however, was the finding that the middle and late auditory potentials were severely compromised. This case underscores the need to examine the entire auditory system, using a variety of behavioral tests and electrophysiological procedures, and the importance of a thorough understanding of the anatomical structures involved in the measured responses. Had the entire auditory

system not been thoroughly evaluated, this patient might have been inappropriately fit with amplification or even a cochlear implant, only to experience limited benefit at best. Even more serious than not having benefited the patient, inappropriately providing a cochlear implant might well have unnecessarily and inadvertently obliterated a normal-functioning cochlea.

More recently, the Veterans Administration (VA) has examined auditory deficits in veterans returning from recent military operations (i.e., Operation Iraqi Freedom/Operation Enduring Freedom/Operation New Dawn). Specifically, the VA has examined veterans with blast-related injuries (Lew et al, 2007; Gallun et al, 2012; Oleksiak et al, 2012; Saunders and Echt, 2012; Saunders et al, 2015). Many of these veterans have expressed concerns regarding their hearing, particularly in noisy situations or less than ideal listening environments; however, they present with normal peripheral hearing sensitivity. These findings led Gallun and colleagues (2012) to compare veterans with and without blast-related injuries. Those that were blast exposed demonstrated significant deficits in temporal resolution, binaural integration, and speech recognition in noise compared to the control group. In addition, they demonstrated differences in objective electrophysiological measures. While the ABR did not reveal significant differences between groups, the blast-exposed group demonstrated both latency (delayed) and amplitude (reduced) differences in the right ear on late auditory evoked potentials (AEPs; P300) relative to the control group. Saunders et al (2015) examined 99 blast-exposed veterans through self-report and behavioral measures. The most common deficit was speech-in-noise difficulties (>75%). More than half of the veterans presented deficits on three to six of the ten functional measures that were administered (e.g., Hearing-in-Noise Test, North American Listening in Spatialized Noise-Sentence Test, Staggered Spondaic Words). Their confirmed blast injuries likely contributed to their central deficits, further demonstrating why one cannot rely solely on the audiogram to comprehensively assess the auditory system. Further demonstrating the limitations of the pure-tone audiogram in revealing the underlying status of the auditory system, Saunders and Echt (2012) found that more than half of VA audiologists (53%) reported encountering two to three veterans per month reporting auditory complaints, despite normal audiograms, with nearly all VA audiologists (93%) reporting at least one veteran per month with that profile.

In fact, self-reported hearing difficulties among adults with normal audiograms are not confined to veterans. Shinn and colleagues recently reported on patients seen in a general audiology/otolaryngology clinic. Of those patients who presented with concerns regarding hearing despite having normal audiograms, 13% specifically reported auditory processing deficits (as reflected by their challenges hearing in noise and in difficult listening environments), and 48% were concerned

about hearing loss (Shinn et al, 2016). Those with concerns regarding “hearing loss” were more than likely experiencing some degree of abnormal auditory perception that requires further evaluation for identification and treatment planning.

Additional evidence of the limitations of the audiogram arises from studies of patients with tinnitus and those exposed to noise. A number of studies have shown that patients with tinnitus present with subtle audiological deficits not observed on the audiogram (Schiette and McAlpine, 2011; Epp et al, 2012; Ishak et al, 2013). Moreover, subtle auditory deficits of both the peripheral and central mechanisms due to noise exposure have been observed on OAEs (Boger et al, 2012), sound localization (Menezes et al, 2014), and temporal processing (Kumar et al, 2007), despite the presence of normal-hearing sensitivity (as reflected on the audiogram).

NORMAL PURE-TONE THRESHOLDS AND COCHLEAR DAMAGE

Conventional thinking posits that the pure-tone audiogram reflects the functional status of the cochlea. This certainly is supported, at least partially, by studies showing elevated pure-tone thresholds in cochleas with damage to hair cells (Schuknecht, 1974). However, over the years, a few reports have demonstrated that this correspondence may not be absolute. For example, 50 years ago, a classic histological study of the cochlea questioned the degree to which histological damage to the hair cells correlated with pure-tone thresholds, thereby casting some doubt on the physiological validity of pure-tone thresholds (Bredberg et al, 1965). Hunter-Duvar and Elliott (1972) reported normal thresholds in a squirrel monkey with considerable hair cell loss. Ward and Duvall (1971) reported on two animals with normal pure-tone thresholds despite marked hair cell damage at the basal and middle turns of the cochlea. Clark and Bohne (1986), examining damage from low-frequency, high-intensity sound exposure to the apical section of the cochlea of chinchillas, concluded that up to 50% of the hair cells could be missing without significantly affecting the animal’s low-frequency hearing sensitivity. Though these authors reported better correlations between pure-tone thresholds and hair cell integrity at the basal turn of the cochlea, they interpreted their overall results as reflecting the insensitivity of the pure-tone audiogram to cochlear histology. Following additional analyses, Clark and Bohne concluded that “pure-tone thresholds are not adequate to determine functional status of the ear” (p. 78). More recently, Lobarinas et al (2013) made a similar observation following carboplatin-damaged inner hair cells (IHCs) in chinchillas (N = 9). They reported only minimal shift in pure-tone thresholds, with most frequencies remaining within normal range, even when inner hair cell

damage approached 80% of the total number of IHCs. Lobarinas et al concluded that there was poor correlation between loss in IHCs and pure-tone thresholds. As is now known, IHCs are not, for the most part, sensitive to low intensity, but rather are activated at high intensities (Kujawa and Liberman, 2009). Notwithstanding this distinction, Lobarinas et al's findings further demonstrate the limitations of pure-tone threshold procedures. A number of studies preceding those mentioned here also commented on the presence of normal or near-normal pure-tone thresholds despite cochlear damage. These studies primarily examined auditory neurons absent secondary to hair cell damage. Because these investigations focused on auditory nerve fibers, they are reviewed in the "Normal Pure-Tone Thresholds and Auditory Nerve Compromise" section on the auditory nerve.

While audiologists often assume that abnormal pure-tone thresholds reflect damage to the cochlea—usually the hair cells—this may not be accurate in all cases. Indeed, in the absence of histological data, such as in a clinical situation, we are left to infer the status of the hair cells and cochlear dysfunction based on pure-tone thresholds. However, this may lead to incorrect inferences. Clearly, there is a relationship between pure-tone thresholds and hair cell damage; however, in many instances of hair cell depletion, pure-tone thresholds do not accurately reflect the degree of depletion, and in fact, may not reflect hair cell depletion at all, as seen by essentially normal-hearing sensitivity. The reports of basic scientists suggest the need for assessment tools beyond pure-tone audiometry to monitor hair cell damage and the status of the cochlea (Clark and Bohne, 1986).

NORMAL PURE-TONE THRESHOLDS AND AUDITORY NERVE COMPROMISE

Considerable evidence confirms that damage to the auditory nerve results in loss of hearing sensitivity and elevated pure-tone thresholds, as seen in cases of vestibular schwannomas (i.e., acoustic tumors). In fact, it has been reported that $\sim 3/4$ of patients with acoustic tumors present unilateral, high-frequency sensorineural hearing loss, with various other audiometric configurations (including normal thresholds) constituting the remaining 25% (Johnson, 1977). Indeed, there are a surprising number of patients with acoustic neuromas who have essentially normal pure-tone thresholds. One of the first reports that pure-tone thresholds might not consistently reveal dysfunction of the auditory nerve was published by Crowe et al (1934), who studied human temporal bones from individuals with documented normal pure-tone thresholds. Crowe et al reported considerable auditory nerve atrophy (up to 50%) at the basal turn, which was considered to be secondary to hair

cell damage in this region. Schuknecht and Weollner (1953) reported normal detection thresholds in one of three cats who had $\sim 80\%$ of their spiral ganglion cells destroyed. In a follow-up study on cats, Schuknecht and Weollner (1953) reported that 50% of spiral ganglion cells could be damaged without a shift in normal pure-tone thresholds. Subsequently, other animal studies have reported normal pure-tone thresholds despite extensive damage to spiral ganglion cells (Elliott, 1962).

In a review of several extensive studies of humans with confirmed acoustic neuromas, 72 (or 5%) of a total of 1,685 patients presented normal pure-tone thresholds for the involved side (Beck et al, 1986; Dornhoffer et al, 1994; Kanzaki et al, 1997; Magdziarz et al, 2000; Mackle et al, 2007). This figure of 5% is consistent with estimates by others (Musiek et al, 1986). In these studies, the criteria for normal pure-tones thresholds varied from lax (normal thresholds for frequencies 500–2000 Hz; <25 dB HL) to strict (250–8000 Hz; <20 dB HL and no more than 10-dB asymmetry). Of note was one study that reported a 5% rate of normal pure-tone hearing thresholds; however, of this group of 21 patients with acoustic tumors and normal thresholds, 13 reported subjective hearing loss (Beck et al, 1986), a finding generally consistent with the report by Musiek et al (1986). Musiek and colleagues (1986) reported hearing loss or hearing difficulties in 12 of 16 patients with cerebellopontine angle tumors and essentially normal pure-tone thresholds. It could be argued, however, that since most acoustic tumors arise from the vestibular nerves that, at least early on, the audiogram would be relatively unaffected. Therefore, one might not anticipate pure-tone deficits in at least some of the 5% of acoustic neuroma patients who present normal audiograms. This has led some to recommend the use of other measures beyond the audiogram when evaluating suspected acoustic neuromas (Musiek et al, 1986; Valente et al, 1995).

Kujawa and Liberman (2009) reported interesting findings in mice relative to auditory nerve involvement and pure-tone thresholds after noise exposure. The animals' pure-tone thresholds resolved to normal following temporary threshold shift; however, the animals incurred damage to afferent auditory nerve terminals and subsequent degeneration of the auditory nerve. Kujawa and Liberman noted that pure-tone audiograms did not reflect the overall and progressive damage to hearing in these animals. They also demonstrated that the ABR revealed auditory nerve dysfunction and was, therefore, a more accurate index of the animal's true hearing status.

These animal and human reports reveal that most auditory nerve compromise is reflected by pure-tone shift; however, this correspondence is not consistent, despite the widespread assumption that it is. Normal pure-tone thresholds can be seen despite damage to

or pathology of the auditory nerve. Clearly, clinicians investigating acoustic neuromas should include measures in addition to pure-tone thresholds to more accurately evaluate hearing in such patients (Musiek et al, 1986; Valente et al, 1995).

AUDITORY SYMPTOMS OF INDIVIDUALS WITH NORMAL AUDIOGRAMS

Since the seminal observations of Bocca et al (1954), we have known that patients with brain pathology may complain of hearing difficulties despite the presence of normal-hearing thresholds and normal speech recognition in quiet. As part of a psychoacoustic pattern discrimination test validation study, Blaettner et al (1989) administered a hearing questionnaire to patients with unilateral cerebrovascular lesions of the telencephalic auditory structures to assess potential auditory perceptual problems in everyday hearing situations. They found that about half (49%) their patients reported auditory perceptual problems, particularly in situations with simultaneous speakers; however, the vast majority (79%) of those patients who gave abnormal results on the psychoacoustic test reported auditory perceptual problems. Of interest, the latter patients often stated that they did not have any hearing complaints before the questionnaire being administered. This is consistent with our own anecdotal observations that neurological patients with self-reported difficulties in several hearing activities (e.g., localization, understanding speech in noise, perception of music) may not attribute these difficulties to hearing, since their ability to hear soft sounds may be unimpaired. Blaettner et al (1989) proposed that the reduced ability of patients with central auditory lesions to follow or select a speaker in a noisy environment is similar to the disability experienced by individuals with a peripheral (cochlear) hearing loss, and this central auditory disability should be taken into account when evaluating the communication abilities of patients with brain lesions. Bamiou et al (2012) administered the modified Inventory for Auditory Disability questionnaire (Kramer et al, 1995) to 21 nonaphasic adult patients with stroke of the auditory brain regions and relatively preserved audiometric thresholds and 23 normal age- and hearing-matched controls. Stroke patients' scores for sound recognition and localization were significantly worse than scores for normal controls. Moreover, the questionnaire scores correlated significantly with the results of central auditory processing test scores, but not with hearing thresholds. Bamiou et al (2012) proposed that the questionnaire could help identify stroke patients who need further audiological assessment for central auditory processing disorder (CAPD).

Listening difficulties in the presence of normal pure-tone thresholds are not confined to those with neurological-type lesions or presentations. In nonneurological cases,

~4% of working-age adults (Hind et al, 2011) and up to 10% of all adults (Kumar et al, 2007) who present to audiology clinics with complaints of significant listening difficulties have normal pure-tone thresholds. A proportion of these patients demonstrate abnormal performance on complex psychoacoustic tests, and when their auditory nerve function is intact, their presentation is attributed to CAPD. Saunders and Haggard (1992) conducted a study to characterize patients (whom they classified as having obscure auditory dysfunction [OAD], as discussed in the "Limitations of the Audiogram with Other Populations" section) with difficulties in hearing speech in noise, and determine the etiology of their symptoms. The study protocol included hearing tests (e.g., pure-tone audiometry, frequency resolution, Speech-in-Noise Test (SIN), a sentence completion task, and a lip-reading test), as well as psychological assessments (i.e., a personality questionnaire and hearing difficulties questionnaire). They showed that these patients differed significantly from the controls in three domains: (a) psychological domain (i.e., greater anxiety on personality test), (b) psychoacoustic domain (i.e., impaired frequency resolution, SIN threshold), and (c) cognitive/linguistic domain (i.e., lower scores on the focused attention condition of a dichotic listening test). They also reported that while patients with OAD and normal pure-tone hearing thresholds usually were discharged from audiology departments, the findings of a normal audiogram did not satisfy these patients, who subsequently sought a second opinion and further assessments. Their research led to the development of a package of performance tests, questionnaires, and protocols for counseling (Saunders et al, 1992). Higson et al (1994), in a further study of 59 new OAD patients, replicated the findings of the previous study by Saunders and Haggard (1992) and proposed that such patients had a consistent profile of poorer speech reception threshold (SIN) and considered themselves handicapped by their symptoms.

Neijenhuis et al (2003) assessed hearing disability in 24 otherwise neurologically normal adults with a suspected CAPD (i.e., patients who complained of hearing difficulties on the Amsterdam Inventory [Kramer et al, 1995] despite the presence of a normal audiogram and speech recognition in quiet audiometry). Sixty-eight percent of these adults gave abnormal results (scores below the 90th percentile of the normal control group) on the central auditory test battery. As a group, the participants with a suspected CAPD reported significantly more complaints regarding hearing abilities than normal controls for all five factors assessed by the Amsterdam Inventory, with speech in noise and sound localization as the most frequently reported difficulties. Bamiou et al (2015) assessed hearing symptoms in adult patients with CAPD (N = 39) versus controls (N = 19) as revealed on the Speech Spatial Qualities hearing scale (Gatehouse and Noble, 2004), the (Modified) Amsterdam Inventory

for Auditory Disability (Kramer et al, 1995), and the Hyperacusis questionnaire (Khalifa et al, 2002). Speech in noise emerged as the key issue for these patients.

PURE-TONE THRESHOLDS AND COMPROMISE OF THE CANS

The limited sensitivity of pure-tone thresholds to cochlear and eighth-nerve damage certainly reduces their utility in clinical evaluations of hearing function. The disassociation between patient reports of hearing difficulties with CANS involvement and normal pure-tone thresholds has become a reasonably well-known and accepted finding. This lack of correspondence was revealed in early studies by Bocca and his colleagues (Bocca et al, 1954). A principle assertion of these germinal studies was that pure-tone thresholds were of little value in measuring auditory deficit in patients with central auditory compromise. This understanding led these early investigators to embark on developing new test procedures that were more sensitive to detecting central auditory dysfunction. This understanding continues to guide the development of central auditory tests to better reveal with more objective measures the source(s) of the auditory symptoms of the patient with central auditory involvement (Musiek et al, 1994).

Though Bocca and colleagues' early studies began to raise awareness of the shortcomings of pure-tone thresholds in revealing central auditory dysfunction, the results of other early reports were perhaps even more striking. These reports profiled individuals who had undergone hemispherectomies and resections of major portions of the temporal lobe with essentially no effects on the pure-tone audiogram (Goldstein et al, 1956; Berlin et al, 1972). The Goldstein et al (1956) report was especially compelling in that pure-tone thresholds for four patients with infantile hemiplegia did not significantly change from preoperative levels after hemispherectomy. While we now know that brain plasticity could be operating in these patients, this was an important early report calling attention to the insensitivity of pure-tone thresholds to a major insult to the central nervous system and the auditory cortex. It should be mentioned that one of these patients was tested several years later with degraded speech stimuli and showed a clear deficit in the ear contralateral to the hemispherectomy, demonstrating the potential of more sensitized stimuli and tests to more accurately reflect the status of the CANS.

In the 1960s and 1970s, additional reports emerged corroborating the extremely limited value of pure-tone thresholds in measuring central auditory integrity. At the same time, sensitized tests such as monaural low-redundancy speech and dichotic listening began to demonstrate their worth in evaluating those with central auditory disorder (Jerger, 1960; Kimura, 1961; Lynn

and Gilroy, 1972; Jerger and Jerger, 1975; Speaks et al, 1975). Such reports helped to persuade some audiologists, regardless of whether they worked in the area of central auditory disorders, that pure-tone thresholds simply do not reflect deficits of the CANS and that different test approaches and strategies are required for thorough and accurate evaluation of these patients.

Over the last 30 yr, additional tests of the CANS have been developed. In a large study of consecutive patients with a wide variety of brainstem lesions and after excluding patients with cerebellopontine angle lesions or acoustic neuromas as well as patients with hearing loss attributable to other causes ($N = 309$), only 26% presented pure-tone thresholds poorer than 30 dB ISO (Luxon, 1980). This figure represents a "high" estimate given the study's skewed population, as they were presented to a tertiary care Neurology Neurosurgery specialist hospital. Furthermore, diagnosis of brainstem pathology was based on clinical neurological and otological grounds in all patients, and diagnosis was confirmed at operation or postmortem examination in 35%—that is, a sample with a high prevalence of advanced or life-threatening brainstem pathology—to merit operation. Conversely, a series of studies has documented the rather ubiquitous finding of the sensitivity of a variety of sensitized central auditory tests to compromise of the brainstem auditory pathway in the absence of any consistent influence on the pure-tone audiogram. For example, masking level differences (MLDs) have been shown to be abnormal in brainstem lesions despite normal audiometric thresholds (Olsen and Noffsinger, 1976; Lynn et al, 1981; Noffsinger et al, 1985). Similar findings were observed using interaural timing tasks in individuals with brainstem involvement (Furst et al, 2000; Aharonson and Furst, 2001; McCullagh and Bamiou, 2014). Studies using low-redundancy speech tests on patients with brainstem involvement showed no poorer than moderate hearing loss, with many of these individuals presenting normal or near-normal pure-tone sensitivity (Jerger and Jerger, 1974; Karlsson and Rosenhall, 1995). Dichotic listening studies also have demonstrated good sensitivity to brainstem disorders despite normal or near-normal pure-tone findings (Musiek, 1983).

Testing beyond the pure-tone audiogram may include electrophysiological procedures as well as the behaviorally sensitized tests. The ABR is a powerful measure of auditory brainstem pathway integrity, which, as discussed earlier, is seldom reflected in pure-tone thresholds (Luxon, 1980; Musiek and Geurkink, 1982; Musiek, Shinn, et al, 2007). Patients with multiple sclerosis demonstrate the insensitivity of the audiogram to brainstem integrity. Although it has been well documented that ABRs are commonly abnormal in patients with multiple sclerosis due to involvement of the auditory brainstem pathways (Paludetti et al, 1985; Musiek et al, 1989), their

pure-tone auditory thresholds are rarely elevated (Doty et al, 2012). The ABR also is highly sensitive and specific to various brainstem disorders, whether they are vascular, mass, or degenerative in type, yet pure-tone thresholds provide essentially no diagnostic insight to the patients' auditory problems (Musiek and Geurkink, 1982; Noffsinger et al, 1982; Musiek et al, 1988; Musiek, Shinn, et al, 2007). If the pure-tone thresholds were used exclusively in these cases of brainstem involvement, the overwhelming majority of patients' auditory difficulties would remain undocumented. In fact, normal pure-tone thresholds at times could provide a false sense of security to the examiner, who might be inclined to dismiss the patient's symptoms when in actuality the auditory system may be compromised.

Earlier in this section, it was mentioned that initial reports of normal pure-tone thresholds in cases of hemispherectomy were critical in gaining insight and providing momentum in the evolution of our understanding of the need to move beyond the pure-tone audiogram. Perhaps the best illustration of the shortcomings of pure-tone thresholds is reports showing the absence of pure-tone threshold changes from lesions of auditory areas of the cortex or cerebrum (Musiek et al, 1994; Hurley and Hurley, 2014). In many studies examining auditory cortex involvement and use of central auditory tests, patients typically were required to have normal-hearing sensitivity to qualify for the study. These patients' central auditory test performance was clearly abnormal due to their documented lesions of the auditory cortex. Major strokes and related vascular problems, head injury, mass lesions, and degenerative disorders of the auditory cortex have been shown to yield abnormal results on a myriad of behavioral test procedures, including temporal processes (Musiek et al, 1990; 2005; 2012; Baran and Musiek, 1999; Shinn, 2014), dichotic listening (Musiek, 1983; Musiek et al, 2012; Weihing and Atcherson, 2014), low-redundancy speech (Lynn and Gilroy, 1972; 1977; Krishnamurti, 2015), and binaural interaction procedures (McCullagh and Bamiou, 2014).

In addition to the ABR, other electrophysiological procedures (e.g., middle latency and late AEPs) tell a similar story. These evoked potentials (EPs) can be sensitive to cortical damage to the auditory areas and in most cases are accompanied by a normal audiogram (Knight et al, 1980; Musiek et al, 1994; 1999; Musiek and Lee, 1999). Though middle and late EPs are not, in most cases, routine clinical procedures, in the right situations they may be helpful. However, understanding the limitations of these EPs should be exercised in their application.

LIMITATIONS OF THE AUDIOGRAM WITH OTHER POPULATIONS

Over the years, there has been a series of reports of individuals with normal pure-tone thresholds

with presumed damage to the CANS (based on symptoms such as difficulty understanding speech in background noise) or possibly damage to the cochlea. These individuals presented performance deficits on more complex auditory tests. For example, elderly participants presented a decrease in speech recognition in noise over time in the absence of any change in the pure-tone audiogram (Dubno et al, 1984). Similarly, there have been reports of changes in temporal processing ability with increasing age with essentially no change in pure-tone thresholds (He et al, 2008; Hopkins and Moore, 2011). Age-related reductions in the brainstem frequency following response synchrony and amplitude, as well as prolonged latencies, have been observed in middle-aged individuals, even in the absence of significant hearing loss (Clinard and Tremblay, 2013). Based on recordings of speech-evoked ABRs in quiet and in noise in older adults with normal-hearing sensitivity, Anderson et al (2011) reported that those older adults with poor speech recognition in noise performance demonstrated greater disruption of response morphology in noise, reflecting a reduction in neural synchrony.

Another population commonly seen by audiologists is children with normal-hearing sensitivity with CAPD and language-related learning problems in the absence of frank underlying neuropathology (Bishop, 2000). A number of studies suggest that at least some of these children present with some underlying benign, diffuse, or focal neuroanatomic/neuromorphological abnormalities (e.g., ectopias [normal but misplaced cells], polymicrogyria [abnormally small gyri]) or maturational delay due to a slower course of myelination or auditory deprivation (Galaburda et al, 1985; Boscarriol et al, 2009; Boscarriol, Garcia, Guimarães, et al, 2010; Boscarriol, Guimarães, Hage, et al, 2010; Boscarriol et al, 2011; Chermak and Musiek, 2011; Boscarriol et al, 2015). This pediatric population has characteristically been defined by normal pure-tone thresholds, the presence of auditory symptoms, and related learning difficulties. They also show deficits on central auditory tests. These children can be helped by various intervention techniques, as the recent literature has shown (Musiek et al, 2014; Weihing and Musiek, 2014). Again, if only pure-tone audiograms were obtained from these children, there might have been no documentable reason to pursue intervention. Unfortunately, this failure may occur more often than we might wish to acknowledge.

Based on the animal work of Kujawa and Liberman (2009) and Pienkowski and Eggermont (2012), it appears that noise exposure can result in damage beyond the cochlea without affecting the pure-tone audiogram. Alvord (1983) reported that individuals with a history of noise exposure and normal audiograms still demonstrated poorer speech recognition in noise scores than did a control group not exposed to noise. Despite a methodological concern (i.e., the noise group had slightly

more loss at 4 kHz than the control group), Alvord's study contributed to the early seeds of thought concerning the inability of the pure-tone audiogram to reflect damage to the auditory system, whether that damage was peripheral or central. A striking study published in 2012 by Kumar et al compared a group of noise-exposed workers with normal pure-tone audiograms to a matched control group without a history of noise exposure. Though the audiograms were essentially the same across groups, the noise-exposed group performed more poorly on (temporal) tests of modulation detection and duration pattern perception (generally considered a measure of central auditory function). Kumar et al's results could be interpreted to suggest that noise damaged the central auditory system but not the cochlea, or that the noise damaged both peripheral and central systems, but the audiogram did not reflect the peripheral auditory compromise.

Recently published data reveals how exposure to moderate levels of noise over long durations can precipitate tonotopic disorganization in the cortex of cats (Pienkowski and Eggermont, 2012). This cortical compromise evolves without affecting detection thresholds. These findings again demonstrate the failure of audiometric threshold measures to provide any insight as to what appears to be devastating dysfunction of the cortex. While recognizing the limitations of generalizing from animal data to humans, these data do suggest that a patient with a history of noise exposure could experience degraded frequency processing that could affect overall hearing function, which would not be heralded by any pure-tone threshold changes. Again, clinicians should adopt a cautious approach in rendering a diagnosis of "normal hearing" based solely on the pure-tone audiogram.

WHEN THE PURE-TONE AUDIOGRAM BEARS LITTLE IF ANY RELEVANCE TO PERIPHERAL INTEGRITY

We have discussed the limitations of the pure-tone audiogram to reveal damage to the cochlea (as well as the CANS). Central deafness provides a fascinating condition in which the pure-tone audiogram may in fact reflect massive dysfunction of the CANS without any relevance to peripheral integrity. Indeed, pure-tone thresholds may be absent in complete central deafness or show severe decrements in central deafness; however, the source of these deficits in sensitivity is central in origin: they do not originate in the peripheral auditory system (Musiek et al, 1994; Musiek, Baran, et al, 2007). In fact, the term "central deafness" is preferred to "central hearing loss," since the term "hearing loss" implies a peripheral origin. Typically, OAEs are present, which suggests normal cochlear function (i.e., outer hair cell function) or at most a mild hearing loss, as well as normal

ABRs, which confirm the integrity of the inner ear and cochlear nerve as well as the auditory pathways within the low brainstem. Speech recognition typically is severely depressed—often more severely depressed than predicted based on pure-tone thresholds (Kaga et al, 2004). EPs that assess electrophysiological responses that originate in the cortex or the radiations to the auditory cortex reveal abnormalities in central deafness and complete central deafness (Musiek et al, 2004). Typically, patients can speak, read, and write. Auditory deficits (e.g., pure-tone thresholds, patient's assessment of "hearing") typically are poorer in the ear contralateral to the hemisphere with the most damage (Heffner and Heffner, 1986; Musiek et al, 1994). Inconsistent responses to sound may result from attention deficits, or improper triggering of attention by ascending auditory tracts (Musiek et al, 1994).

CENTRAL AUDITORY TESTS AND PROCEDURES

Given the limitations of the pure-tone audiogram, we provide a brief overview of available clinical tests, behavioral tests, and electrophysiological procedures that are sensitive to the function and integrity of the central auditory system and are useful for clinical evaluation and diagnosis of CAPD. Tests reviewed include those of dichotic listening, binaural interaction (e.g., MLDs), temporal processing (e.g., temporal resolution and temporal patterning), and monaural low-redundancy measures (e.g., speech in noise or competition, filtered speech, time-compressed speech). Current and new paradigms involving measurement of AEPs also are mentioned. Finally, we note the potential of software to develop behavioral tests to assess auditory functions for which there are no or few commercially available options. The reader is referred to Weising et al (2015) and Musiek, Chermak, et al (2011) for discussion of the sensitivity and specificity of the tests reviewed below. The reader is referred to Musiek and Chermak (2014) and AAA (2010) for detailed reviews of the tests and procedures outlined below.

Though primarily considered a test of cochlear function, OAEs, whether transient or distortion product, can assist in the diagnosis of central auditory involvement (Musiek and Chermak, 2015). OAEs usually are absent or abnormal when cochlear hearing loss exceeds 30 dB HL for transient OAEs and ~40 dB HL for distortion product OAEs. Though it is well known that auditory nerve function is not necessary for generation of OAEs; eighth nerve disorders such as acoustic tumors can disrupt OAEs.

The AR and other audiological tests (e.g., air- and bone-conduction thresholds, tympanometry, and reflex decay) can help differentiate middle ear, cochlear, and eighth nerve problems; however, these tests are not

able to differentiate auditory nerve from low brainstem involvement. The AR is mediated in the lower pons, and, therefore, reflects only a small portion of the CANS. The AR, however, can provide insight as to central auditory dysfunction in the caudal brainstem (Musiek and Chermak, 2015). AR abnormalities have been observed in only a relatively small portion of children with CAPD. This observation suggests that the central dysfunction underlying their CAPD was rostral to the brainstem (Hall and Johnston, 2007). Abnormal AR findings, however, have been reported in a number of different central auditory disorders with established brain pathology, such as tumors of the brainstem (Jerger and Jerger, 1977), multiple sclerosis (Jerger et al, 1986), head injury (Hall et al, 1982), and more recently in lead poisoning (Counter et al, 2011).

Dichotic listening (i.e., listening to different acoustic events presented to each ear simultaneously) is among the most widely used behavioral tests of the CANS and central auditory processing. The most commonly used clinical tests employing the dichotic paradigm are the Staggered Spondaic Word test (Katz, 1962), Dichotic Digits (Musiek, 1983), the competing words and competing sentences subtests of the SCAN-3:C (tests for auditory processing disorders for children) (Keith, 2009b) and of the SCAN-3:A (tests for auditory processing disorders in adolescents and adults) (Keith, 2009a), and Dichotic Words (DW; Meyers et al, 2002). In patients with well-localized lesions, the ear contralateral to the involved hemisphere often exhibits poor performance relative to norms (interparticipant) or to the other ear (intraparticipant, interaural comparison) (Kimura, 1961; Musiek, 1983; Musiek and Weihing, 2011). If the corpus callosum is involved, a left-ear deficit is seen consistently (Musiek and Weihing, 2011). A left-ear deficit also is seen in young children (<12 yr) whose corpus callosa have not yet attained their full complement of myelin, as well as in the elderly and in individuals with diseases affecting myelin (e.g., multiple sclerosis) (Musiek et al, 1984; Musiek and Pinheiro, 1985; Musiek and Weihing, 2011). However, there are well-established top-down cognitive effects on such tests that reflect the interaction of auditory processing with auditory cognition (e.g., Jerger and Martin, 2006; Martin et al, 2007; Gates et al, 2010; Davis et al, 2012).

Binaural interaction tasks require the listener to combine complementary inputs distributed between the ears, synthesizing intensity, time, or spectral differences of otherwise identical stimuli presented simultaneously or sequentially (e.g., localization and lateralization tasks) to the two ears. Localization refers to the identification of the location of a sound source in a sound field. Lateralization is a similar process in which the individual determines the location in the head (intracranial) of a sound image presented through headphones. The effects of

brain lesions on sound localization have been demonstrated extensively in animals and humans. Damage to the CANS (as well as peripheral hearing loss) reduces the ability to accurately localize a sound source (Bamiou, 2007). No clinical measure of localization has been adopted clinically. However, a binaural interaction task, MLD, is used clinically and reveals an individual's ability to detect a target signal (usually low-frequency tones or speech) in noise when the binaural phase relationships between the signal and noise are altered. Noffsinger et al (1972) demonstrated a decrease in the MLD relative to norms in a large population of patients with multiple sclerosis, which primarily affects the central auditory pathway. Lynn et al (1981) reported that patients with low pontine lesions presented smaller MLDs than controls or those with lesions of the upper brainstem or cortex, demonstrating the differential sensitivity of MLDs to various anatomical regions of the CANS. The Listening in Spatialized Noise–Sentences (Cameron and Dillon, 2007) test offers another approach to examine binaural interaction and correlates well with the MLD (Cameron et al, 2006). An added benefit of this test is that it provides a derived measure for voice and spatial cue, thus minimizing, if not entirely eliminating, linguistic influence on test performance.

Auditory temporal processing refers to the perception of sound or the alteration of sound within a restricted or defined time domain (Musiek et al, 2005). Temporal processing is comprised of (a) temporal ordering or sequencing, (b) temporal resolution or discrimination, (c) temporal integration or summation, and (d) temporal masking. Clinical measures of temporal ordering and temporal resolution are available; however, at this time, there are no clinically feasible measures of temporal integration or temporal masking. The most widely used clinical tests of temporal ordering are the Frequency Pattern Test and the Duration Pattern Test (Emanuel et al, 2011). Individuals with cerebral lesions demonstrate temporal ordering deficits (Belmont and Handler, 1971; Karasseva, 1972; Swisher and Hirsh, 1972; DeRenzi et al, 1977). Patients with compromise of the auditory areas of either hemisphere or of the interhemispheric pathways present difficulties on the Frequency Pattern Test and Duration Pattern Test (Musiek et al, 1980; 1990; Pinheiro and Musiek, 1985; Musiek and Pinheiro, 1987; Musiek et al, 1994). These tests impose some degree of cognitive load in view of the complex response mode required; however, more recently developed tests for pitch and time sequence analysis hold promise for the assessment of these important facets of auditory processing (Grube et al, 2012; 2013).

Temporal resolution or discrimination refers to the shortest duration of time in which an individual can discriminate between two auditory signals (Gelfand, 1998). One of the most common measures of temporal resolution using behavioral methods is gap detection,

which requires participants to indicate whenever they hear a “silent” interval embedded in an ongoing sound or noise burst. Two measures of gap detection are commonly used clinically: the Gaps-in-Noise ([GIN]; Musiek et al, 2005) and the Random Gap Detection Tests (Keith, 2002). Findings in these two tests are, however, dissociated in clinical populations, and this has been attributed to a higher load on attention for the Random Gap Detection Test task compared to the GIN (Iliadou et al, 2014). The GIN appears to be more sensitive to cortical compromise than to brainstem involvement (Musiek et al, 2005). Several new clinically viable gap detection procedures have been introduced recently (Griffiths et al, 2001; Lister et al, 2006). One such procedure, the Adaptive Test of Temporal Resolution, uses a computerized, adaptive psychophysical methodology to evaluate temporal resolution (Lister et al, 2006).

Monaural low-redundancy speech tests are among the oldest tests used to assess the CANS (Bocca et al, 1954; 1955). These tests are administered monaurally using stimuli that have been degraded electroacoustically or digitally in the frequency/spectral, temporal, or intensity domain (i.e., low-pass filtered speech tests, SIN (or speech-in-message competition), and time-compressed speech tests). In addition to some degree of sensitivity to CANS lesions (i.e., moderately sensitive to brainstem and cortical dysfunction), they also provide insights regarding an individual’s functional deficits (i.e., auditory closure and listening in noise), as well as one of the most common complaints of individuals seen for CAPD evaluation (i.e., understanding speech in background noise), and therefore offer practical information for intervention (Bellis and Ferre, 1999; Bellis, 2003; Musiek and Baran, 2004). 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, 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. Peripheral hearing loss, as well as CANS dysfunction, reduces the intrinsic redundancy of the auditory system. Peripheral hearing loss reduces the ability of the auditory system to resolve spectral detail; therefore, monaural low-redundancy tests are subject to the potential confound of peripheral hearing loss (Chermak and 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 and Musiek, 1999).

Given the relative insensitivity of the pure-tone audiogram to the function and integrity of the central au-

ditary system, the authors recommend that audiologists consider including a screen for central auditory function in their basic audiological evaluation. A screening measure is indicated when the patient’s auditory complaints are inconsistent with the degree of deficit as represented by the pure-tone audiogram or when the patient’s history raises the possibility of higher order auditory involvement. Unfortunately, research has lagged on the development of screening tests; however, dichotic digits and gap detection may be used for screening (as well as diagnostic purposes) since they pose minimal language and cognitive load and they are sensitive to dysfunction across the CANS (Hurley and Musiek, 1997; Musiek et al, 2005; Musiek, Chermak, et al, 2011). Obviously, other tests might be appropriately used as screeners; however, only tests with established sensitivity to CANS involvement that can be administered in a relatively short period of time should be considered.

AEPs

Electrophysiologic potentials can be useful in the evaluation of the CANS. These potentials provide a more objective measure of the CANS, and their mechanisms of generation can be more directly tied to their underlying anatomy and physiology (i.e., generator mechanisms) than is possible with behavioral tests. However, as noted above, one must be prudent in their use and interpretation. A brief discussion follows of the ABR, auditory middle latency responses (AMLRs), the cortical auditory evoked response (P1–N1–P2), and the cognitive P300 response.

The ABR arises from the auditory brainstem nuclei and pathways. The ABR consists of five to seven peaks, with waves I and II recognized as the scalp-recorded eighth nerve compound action potential. Waves III–V are indicative of activation of nuclei of the cochlear nucleus, superior olivary complex, lateral lemniscus, and the neural tracts connecting them. Wave III is associated with lower brainstem nuclei whereas waves IV–VII, and especially wave V, are associated with upper brainstem nuclei. The absence or abnormality of ABR peaks beyond waves I and II is indicative of brainstem lesions (see Musiek et al, 2007 for review).

The AMLR is generated by the auditory thalamus (medial geniculate body), the fiber tract (auditory radiation) to the cortex from thalamus, and the primary auditory cortex on Heschl’s gyrus. Lesions of the primary auditory cortex may reduce or eliminate the AMLR. Cortical lesions of the temporal lobe result in abnormality or absence of the P1–N1–P2 complex (Knight et al, 1980; Bahls et al, 1988; Musiek and Baran, 2004; Musiek and Chermak, 2007; Cavinato et al, 2012). Studies also have shown P300 measures to be affected by various disorders of the central nervous system. It is important, however, that these middle and late AEPs be

employed with the utmost caution by highly experienced individuals who understand the limitations of these procedures as neurodiagnostic measures.

Recording AEPs in response to stimuli used in behavioral tests of central auditory function as well as recording AEPs while participants actually perform a central auditory test are promising hybrid approaches emerging in the diagnosis of CAPD. While the measurement of hearing thresholds using AEPs is common in clinics and laboratories worldwide, the recording of AEPs in response to more complex stimuli and/or while an individual concurrently performs a behavioral central auditory test has emerged recently as a potentially powerful research and clinical tool. Jerger et al (2002) demonstrated the potential of this procedure using gap detection and dichotic listening tasks with twins, one of whom was diagnosed with CAPD. Palmer and Musiek (2013) reported that amplitude of the N1–P2 late potential changed in response to changes in the duration of gaps presented in broadband noise, and that the N1–P2 potential closely approximated behavioral gap detection thresholds in normal participants. Other behavioral procedures also have been inserted into various AEP paradigms. For example, dichotic stimuli have been employed using mismatch negativity to determine hemispheric advantages in dichotic listening (Yasin, 2007). Mismatch negativity also has been used to measure auditory discrimination, yielding impressive correlations with results obtained behaviorally (Pakarinen et al, 2007). The CANS of individuals who successfully perform the behavioral task and produce repeatable AEPs for a given auditory task must present high auditory integrity, reflecting both the integrity of the neural substrate underlying the behavioral task as well as the neural substrate of the pathways responsible for generating the AEP (Musiek, Chermak, and Cone-Wesson, in press).

Software to Develop Clinical Tests

As noted above, there are auditory functions for which we have limited tools or no commercially available measurement tools with documented sensitivity and specificity (e.g., sound localization, temporal integration, temporal masking). Clinicians with sufficient computer skills might use MatLab or Adobe Audition (formerly Adobe Cool Edit Pro) to develop measures of auditory skills. At a minimum, the clinician must obtain norms for these novel measures, and ultimately establish the measure's sensitivity and specificity if the measure is to be used for clinical diagnosis (see Weihing and Atcherson, 2014, for discussion of clinical decision analysis).

SUMMARY

The pure-tone audiogram has served and will continue to serve an important role in audiological

evaluation. However, audiologists recognize the importance of assessing the central auditory system to fully evaluate auditory or hearing function. The purpose of this article is to elaborate this point, its rationale, and its implications. While recognizing the important role of the pure-tone audiogram in clinical audiology, we reviewed data demonstrating the shortcomings of pure-tone threshold testing in the broader context of assessing overall hearing status. We emphasized that the audiogram provides insight as to the sensitivity of the auditory system to simple stimuli, but that is of little value in providing information about other aspects of auditory function, such as suprathreshold processes, discrimination, temporal processes, binaural processes, and other complex acoustical processes. These limitations of the pure-tone audiogram are particularly evident in individuals with central auditory disorders where commonly observed “normal findings” on a pure-tone audiogram can be misleading if such findings are assumed to reflect the status of the entire auditory system, including central auditory structures and processes. Even in cases of cochlear and auditory nerve involvement, normal pure-tone thresholds are surprisingly common, further revealing the shortcomings of this routine audiometric measure. Several unique studies were highlighted to demonstrate that audiograms do not always reflect damage to the cochlea—a finding seemingly counterintuitive. A section was devoted to reports of various degrees of auditory symptoms and difficulties presented by individuals with normal pure-tone audiograms, reflecting an important disassociation between the pure-tone audiogram and patient complaints. Although seen in only a minority of cases, we discussed the finding of normal pure-tone thresholds in those with auditory nerve involvement. We reviewed the initial impetus for testing beyond the audiogram—central auditory involvement—and concluded this perspective piece with a brief overview of available tests and procedures that assess the central auditory system, which in some instances also may provide insight into the function of the auditory periphery, as well as suggestions for screening central auditory function.

Acknowledgments. The authors acknowledge the help of University of Arizona audiology doctoral students Alyssa Everett and Nicole Denny for their work in reviewing and preparing the manuscript. They also thank the Royal Arch Research Assistance for indirect support of this work.

REFERENCES

- Aharonson V, Furst M. (2001) A model for sound lateralization. *J Acoust Soc Am* 109(6):2840–2851.
- Alvord LS. (1983) Cochlear dysfunction in “normal-hearing” patients with history of noise exposure. *Ear Hear* 4(5):247–250.

- American Academy of Audiology (AAA). (2010) Clinical practice guidelines for the diagnosis, treatment, and management of children and adults with central auditory processing disorder. <http://www.audiology.org/publications-resources/document-library/central-auditory-processing-disorder>. Accessed April 2016.
- Anderson S, Parbery-Clark A, Yi HG, Kraus N. (2011) A neural basis of speech-in-noise perception in older adults. *Ear Hear* 32(6):750–757.
- Bahls FH, Chatrian GE, Mesher RA, Sumi SM, Ruff RL. (1988) A case of persistent cortical deafness: clinical, neurophysiologic, and neuropathologic observations. *Neurology* 38(9):1490–1493.
- Bamiou DE. (2007) Measures of binaural interaction. In: Musiek FE, Chermak GD, eds. *Handbook of Central Auditory Processing Disorder: Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing, 257–286.
- Bamiou DE, Iliadou VV, Zanchetta S, Spyridakou C. (2015) What can we learn about auditory processing from adult hearing questionnaires? *J Am Acad Audiol* 26(10):824–837.
- Bamiou DE, Werring D, Cox K, Stevens J, Musiek FE, Brown MM, Luxon LM. (2012) Patient-reported auditory functions after stroke of the central auditory pathway. *Stroke* 43(5):1285–1289.
- Baran J, Musiek FE. (1999) Behavioral assessment of the central auditory system. In: Musiek FE, Rintelmann WF, eds. *Contemporary Perspectives on Hearing Assessment*. Boston, MA: Allyn & Bacon, 375–415.
- Beck HJ, Beatty CW, Harner SG, Ilstrup DM. (1986) Acoustic neuromas with normal pure tone hearing levels. *Otolaryngol Head Neck Surg* 94(1):96–103.
- Bellis TJ. (2003) Auditory processing disorders: it's not just kids who have them. *Hear J* 56(5):10–19.
- Bellis TJ, Ferre JM. (1999) Multidimensional approach to the differential diagnosis of central auditory processing disorders in children. *J Am Acad Audiol* 10(6):319–328.
- Belmont I, Handler A. (1971) Delayed information processing and judgment of temporal order following cerebral damage. *J Nerv Ment Dis* 152(5):353–361.
- Berlin CI, Lowe-Bell SS, Jannetta PJ, Kline DG. (1972) Central auditory deficits after temporal lobectomy. *Arch Otolaryngol* 96(1):4–10.
- Bishop DVM. (2000) How does the brain learn language? Insights from the study of children with and without language impairment. *Dev Med Child Neurol* 42(2):133–142.
- Blaettner U, Scherg M, von Cramon D. (1989) Diagnosis of unilateral telencephalic hearing disorders. Evaluation of a simple psychoacoustic pattern discrimination test. *Brain* 112(Pt 1):177–195.
- Bocca E, Calearo C, Cassinari V. (1954) A new method for testing hearing in temporal lobe tumours; preliminary report. *Acta Otolaryngol* 44(3):219–221.
- Bocca E, Calearo C, Cassinari V, Migliavacca F. (1955) Testing “cortical” hearing in temporal lobe tumours. *Acta Otolaryngol* 45(4):289–304.
- Boger ME, Sampaio AL, Oliveira CA. (2012) Otoacoustic emissions in normal-hearing workers exposed to different noise doses. *Int Tinnitus J* 17(1):74–79.
- Boscariol M, Casali RL, Amaral MI, Lunardi LL, Matas CG, Collela-Santos MF, Guerreiro MM. (2015) Language and central temporal auditory processing in childhood epilepsies. *Epilepsy Behav* 53:180–183.
- Boscariol M, Garcia VL, Guimarães CA, Hage SR, Montenegro MA, Cendes F, Guerreiro MM. (2009) Auditory processing disorders in twins with perisylvian polymicrogyria. *Arq Neuropsiquiatr* 67(2B):499–501.
- Boscariol M, Garcia VL, Guimarães CA, Montenegro MA, Hage SR, Cendes F, Guerreiro MM. (2010) Auditory processing disorder in perisylvian syndrome. *Brain Dev* 32(4):299–304.
- Boscariol M, Guimarães CA, Hage SR, Cendes F, Guerreiro MM. (2010) Temporal auditory processing: correlation with developmental dyslexia and cortical malformation. *Pro Fono* 22(4):537–542.
- Boscariol M, Guimarães CA, Hage SR, Garcia VL, Schmutzler KM, Cendes F, Guerreiro MM. (2011) Auditory processing disorder in patients with language-learning impairment and correlation with malformation of cortical development. *Brain Dev* 33(10):824–831.
- Bredberg G, Engstrom H, Ades HW. (1965) Cellular pattern and nerve supply of the human organ of Corti; a preliminary report. *Arch Otolaryngol* 82(5):462–469.
- Cameron S, Dillon H. (2007) Development of the listening in spatialized noise-sentences test (LISN-S). *Ear Hear* 28(2):196–211.
- Cameron S, Dillon H, Newall P. (2006) Development and evaluation of the listening in spatialized noise test. *Ear Hear* 27(1):30–42.
- Cavinato M, Rigon J, Volpato C, Semenza C, Piccione F. (2012) Preservation of auditory P300-like potentials in cortical deafness. *PLoS One* 7(1):e29909.
- Chermak GD, Musiek FE. (1997) *Central Auditory Processing Disorders: New Perspectives*. San Diego, CA: Singular Publishing Group.
- Chermak GD, Musiek FE. (2011) Neurological substrate of central auditory processing deficits in children. *Curr Pediatr Rev* 7(3):241–251.
- Chermak GD, Silva ME, Nye J, Hasbrouck J, Musiek FE. (2007) An update on professional education and clinical practices in central auditory processing. *J Am Acad Audiol* 18(5):428–452, quiz 455.
- Clark WW, Bohne BA. (1986) *Cochlear Damage: Audiometric Correlates. Sensorineural Hearing Loss*. Iowa City, IA: University of Iowa, 59–82.
- Clinard CG, Tremblay KL. (2013) Aging degrades the neural encoding of simple and complex sounds in the human brainstem. *J Am Acad Audiol* 24(7):590–599, quiz 643–644.
- Counter SA, Buchanan LH, Ortega F, van der Velde J, Borg E. (2011) Assessment of auditory brainstem function in lead-exposed children using stapedius muscle reflexes. *J Neurol Sci* 306(1–2):29–37.
- Crowe SJ, Guild SR, Polvogt LM. (1934) Observations on the pathology of high-tone deafness. *J Nerv Ment Dis* 80(4):480.
- Davis T, Martin J, Jerger J, Greenwald R, Mehta J. (2012) Auditory-cognitive interactions underlying interaural asymmetry in an adult listener: a case study. *Int J Audiol* 51(2):124–134.
- De Renzi E, Faglioni P, Previdi P. (1977) Spatial memory and hemispheric locus of lesion. *Cortex* 13(4):424–433.

- Dornhoffer JL, Helms J, Hoehmann DH. (1994) Presentation and diagnosis of small acoustic tumors. *Otolaryngol Head Neck Surg* 111(3 Pt 1):232–235.
- Doty RL, Tourbier I, Davis S, Rotz J, Cuzzocreo JL, Treem J, Shephard N, Pham DL. (2012) Pure-tone auditory thresholds are not chronically elevated in multiple sclerosis. *Behav Neurosci* 126(2):314–324.
- Dubno JR, Dirks DD, Morgan DE. (1984) Effects of age and mild hearing loss on speech recognition in noise. *J Acoust Soc Am* 76(1):87–96.
- Edwards B. (2015) Emerging Technologies in amplification, Translational research I. Presented at the American Auditory Society annual conference, Scottsdale, AZ, March 5–7, 2015.
- Eggermont J. (2012) *The Neuroscience of Tinnitus*. Oxford, UK: Oxford University Press.
- Elliott LL. (1962) Backward masking: monotonic and dichotic conditions. *J Acoust Soc Am* 34(8):1108–1115.
- Emanuel DC, Ficca KN, Korczak P. (2011) Survey of the diagnosis and management of auditory processing disorder. *Am J Audiol* 20(1):48–60.
- Epp B, Hots J, Verhey JL, Schaette R. (2012) Increased intensity discrimination thresholds in tinnitus subjects with a normal audiogram. *J Acoust Soc Am* 132(3):EL196–EL201.
- Feldmann H. (1970) *A History of Audiology; a Comprehensive Report and Bibliography from the Earliest Beginnings to the Present*. Chicago, IL: The Beltone Institute for Hearing Research.
- Furst M, Aharonson V, Levine RA, Fullerton BC, Tadmor R, Pratt H, Polyakov A, Korczyn AD. (2000) Sound lateralization and interaural discrimination. Effects of brainstem infarcts and multiple sclerosis lesions. *Hear Res* 143(1–2):29–42.
- Galaburda AM, Sherman GF, Rosen GD, Aboitiz F, Geschwind N. (1985) Developmental dyslexia: four consecutive patients with cortical anomalies. *Ann Neurol* 18(2):222–233.
- Gallun FJ, Diedesch AC, Kubli LR, Walden TC, Folmer RL, Lewis MS, McDermott DJ, Fausti SA, Leek MR. (2012) Performance on tests of central auditory processing by individuals exposed to high-intensity blasts. *J Rehabil Res Dev* 49(7):1005–1025.
- Gatehouse S, Noble W. (2004) The speech, spatial and qualities of hearing scale (SSQ). *Int J Audiol* 43(2):85–99.
- Gates GA, Gibbons LE, McCurry SM, Crane PK, Feeney MP, Larson EB. (2010) Executive dysfunction and presbycusis in older persons with and without memory loss and dementia. *Cogn Behav Neurol* 23(4):218–223.
- Gelfand S. (1998) *Hearing: An Introduction to Psychological and Physiological Acoustics*. New York, NY: CRC Press.
- Gelfand SA, Silman S, Ross L. (1987) Long-term effects of monaural, binaural and no amplification in subjects with bilateral hearing loss. *Scand Audiol* 16(4):201–207.
- Goldstein R, Goodman AC, King RB. (1956) Hearing and speech in infantile hemiplegia before and after left hemispherectomy. *Neurology* 6(12):869–875.
- Griffiths TD, Dean JL, Woods W, Rees A, Green GGR. (2001) The Newcastle Auditory Battery (NAB). A temporal and spatial test battery for use on adult naïve subjects. *Hear Res* 154(1–2):165–169.
- Grube M, Cooper FE, Griffiths TD. (2013) Auditory temporal-regularity processing correlates with language and literacy skill in early adulthood. *Cogn Neurosci* 4(3–4):225–230.
- Grube M, Kumar S, Cooper FE, Turton S, Griffiths TD. (2012) Auditory sequence analysis and phonological skill. *P Roy Soc Lond B Bios* 279(1746):4496–4504.
- Hall JW, 3rd, Huang-fu M, Gennarelli TA. (1982) Auditory function in acute severe head injury. *Laryngoscope* 92(8 Pt 1):883–890.
- Hall JW, 3rd, Johnston KN. (2007) Electroacoustic and electrophysiologic auditory measures in the assessment of (central) auditory processing disorder. In: Musiek FE, Chermak GD, eds. *Handbook of (Central) Auditory Processing Disorders: Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing, 287–316.
- He NJ, Mills JH, Ahlstrom JB, Dubno JR. (2008) Age-related differences in the temporal modulation transfer function with pure-tone carriers. *J Acoust Soc Am* 124(6):3841–3849.
- Heffner HE, Heffner RS. (1986) Effect of unilateral and bilateral auditory cortex lesions on the discrimination of vocalizations by Japanese macaques. *J Neurophysiol* 56(3):683–701.
- Higson JM, Haggard MP, Field DL. (1994) Validation of parameters for assessing Obscure Auditory Dysfunction—robustness of determinants of OAD status across samples and test methods. *Br J Audiol* 28(1):27–39.
- Hind SE, Haines-Bazrafshan R, Benton CL, Brassington W, Towle B, Moore DR. (2011) Prevalence of clinical referrals having hearing thresholds within normal limits. *Int J Audiol* 50(10):708–716.
- Hopkins K, Moore BC. (2011) The effects of age and cochlear hearing loss on temporal fine structure sensitivity, frequency selectivity, and speech reception in noise. *J Acoust Soc Am* 130(1):334–349.
- Hunter-Duvar IM, Elliott DN. (1972) Effects of intense auditory stimulation: hearing losses and inner ear changes in the squirrel monkey. *J Acoust Soc Am* 52(4):1181–1192.
- Hurley RM. (1998) Is the unaided ear effect independent of auditory aging? *J Am Acad Audiol* 9(1):20–24.
- Hurley RM, Hurley AE. (2014) Psychoacoustic considerations and implications for the diagnosis of central auditory processing disorder. In: Musiek FE, Chermak GD, eds. *Handbook of Central Auditory Processing Disorder: Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing.
- Hurley RM, Musiek FE. (1997) Effectiveness of three central auditory processing (CAP) tests in identifying cerebral lesions. *J Am Acad Audiol* 8(4):257–262.
- Iliadou VV, Bamiou DE, Chermak GD, Nimatoudis I. (2014) Comparison of two tests of auditory temporal resolution in children with central auditory processing disorder, adults with psychosis, and adult professional musicians. *Int J Audiol* 53(8):507–513.
- Ishak WS, Zhao F, Rajenderkumar D, Arif M. (2013) Measurement of subtle auditory deficit in tinnitus patients with normal audiometric thresholds using evoked otoacoustic emissions and threshold equalizing noise tests. *Int Tinnitus J* 18(1):35–44.
- Jerger JF. (1960) Observations on auditory behavior in lesions of the central auditory pathways. *AMA Arch Otolaryngol* 71(5):797–806.

- Jerger J, Jerger S. (1974) Auditory findings in brain stem disorders. *Arch Otolaryngol* 99(5):342–350.
- Jerger S, Jerger J. (1975) Extra- and intra-axial brain stem auditory disorders. *Audiology* 14(2):93–117.
- Jerger S, Jerger J. (1977) Diagnostic value of crossed vs uncrossed acoustic reflexes: eighth nerve and brain stem disorders. *Arch Otolaryngol* 103(8):445–453.
- Jerger J, Martin J. (2006) Dichotic listening tests in the assessment of auditory processing disorders. *Audiol Med* 4(1):25–34.
- Jerger J, Oliver TA, Rivera V, Stach BA. (1986) Abnormalities of the acoustic reflex in multiple sclerosis. *Am J Otolaryngol* 7(3):163–176.
- Jerger J, Thibodeau L, Martin J, Mehta J, Tillman G, Greenwald R, Britt L, Scott J, Overson G. (2002) Behavioral and electrophysiologic evidence of auditory processing disorder: a twin study. *J Am Acad Audiol* 13(8):438–460.
- Johnson EW. (1977) Auditory test results in 500 cases of acoustic neuroma. *Arch Otolaryngol* 103(3):152–158.
- Kaga K, Nakamura M, Takayama Y, Momose H. (2004) A case of cortical deafness and anarthria. *Acta Otolaryngol* 124(2):202–205.
- Kanzaki J, Ogawa K, Inoue Y, Shiobara R. (1997) Hearing preservation surgery in acoustic neuroma patients with normal hearing. *Skull Base Surg* 7(3):109–113.
- Karasseva TA. (1972) The role of the temporal lobe in human auditory perception. *Neuropsychologia* 10(2):227–231.
- Karlin JE. (1942) A factorial study of auditory function. *Psychometrika* 7(4):251–279.
- Karlsson AK, Rosenhall U. (1995) Clinical application of distorted speech audiometry. *Scand Audiol* 24(3):155–160.
- Katz J. (1962) The use of staggered spondaic words for assessing the integrity of the central auditory nervous-system. *J Aud Res* 2:327–337.
- Kazui S, Naritomi H, Sawada T, Inoue N, Okuda J. (1990) Subcortical auditory agnosia. *Brain Lang* 38(4):476–487.
- Keith RW. (2002) *Random Gap Detection Test*. St. Louis, MO: Auditec.
- Keith RW. (2009a) *SCAN-3:A Tests for Auditory Processing Disorders for Adolescents and Adults (SCAN-3:A)*. San Antonio, TX: Pearson.
- Keith RW. (2009b) *SCAN-3:C Tests for Auditory Processing Disorders for Children (SCAN-3:C)*. San Antonio, TX: Pearson.
- Khalifa S, Dubal S, Veuillet E, Perez-Diaz F, Jouvent R, Collet L. (2002) Psychometric normalization of a hyperacusis questionnaire. *ORL J Otorhinolaryngol Relat Spec* 64(6):436–442.
- Kimura D. (1961) Some effects of temporal-lobe damage on auditory perception. *Can J Psychol* 15(3):156–165.
- Knight RT, Hillyard SA, Woods DL, Neville HJ. (1980) The effects of frontal and temporal-parietal lesions on the auditory evoked potential in man. *Electroencephalogr Clin Neurophysiol* 50(1–2):112–124.
- Kramer SE, Kapteyn TS, Festen JM, Tobi H. (1995) Factors in subjective hearing disability. *Audiology* 34(6):311–320.
- Krishnamurti S. (2015) Application of neural network modeling to identify auditory processing disorders in school-age children. *Adv Artif Neural Syst* 2015:1–13.
- Kujawa SG, Liberman MC. (2009) Adding insult to injury: cochlear nerve degeneration after “temporary” noise-induced hearing loss. *J Neurosci* 29(45):14077–14085.
- Kumar UA, Ameenudin S, Sangamanatha AV. (2012) Temporal and speech processing skills in normal hearing individuals exposed to occupational noise. *Noise Health* 14(58):100–105.
- Kumar G, Amen F, Roy D. (2007) Normal hearing tests: is a further appointment really necessary? *J R Soc Med* 100(2):66.
- Lew HL, Jerger JF, Guillory SB, Henry JA. (2007) Auditory dysfunction in traumatic brain injury. *J Rehabil Res Dev* 44(7):921–928.
- Lister JJ, Roberts RA, Shackelford J, Rogers CL. (2006) An adaptive clinical test of temporal resolution. *Am J Audiol* 15(2):133–140.
- Lobarinas E, Salvi R, Ding D. (2013) Insensitivity of the audiogram to carboplatin induced inner hair cell loss in chinchillas. *Hear Res* 302:113–120.
- Lockwood AH, Salvi RJ, Burkard RF. (2002) Tinnitus. *N Engl J Med* 347(12):904–910.
- Luxon LM. (1980) Hearing loss in brainstem disorders. *J Neurol Neurosurg Psychiatry* 43(6):510–515.
- Lynn GE, Gilroy J. (1972) Neuro-audiological abnormalities in patients with temporal lobe tumors. *J Neurol Sci* 17(2):167–184.
- Lynn GE, Gilroy J. (1977) Evaluation of central auditory dysfunction on patients with neurological disorders. In: Keith RW, ed. *Central Auditory Dysfunction*. New York, NY: Grune and Stratton, 177–221.
- Lynn GE, Gilroy J, Taylor PC, Leiser RP. (1981) Binaural masking-level differences in neurological disorders. *Arch Otolaryngol* 107(6):357–362.
- Mackle T, Rawluk D, Walsh RM. (2007) Atypical clinical presentations of vestibular schwannomas. *Otol Neurotol* 28(4):526–528.
- Magdziarz DD, Wiet RJ, Dinces EA, Adamiec LC. (2000) Normal audiologic presentations in patients with acoustic neuroma: an evaluation using strict audiologic parameters. *Otolaryngol Head Neck Surg* 122(2):157–162.
- Martin J, Jerger J, Mehta J. (2007) Divided-attention and directed-attention listening modes in children with dichotic deficits: an event-related potential study. *J Am Acad Audiol* 18(1):34–53.
- McCullagh J, Bamiou DE. (2014) . (.)Measures of binaural interaction. In: Musiek FE, Chermak GD, eds. *Handbook of Central Auditory Processing Disorder: Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing.
- Menezes Pde L, Andrade KC, Carnauba AT, Cabral FB, Leal Mde C, Pereira LD. (2014) Sound localization and occupational noise. *Clinics* 69(2):083–086.
- Meyers JE, Roberts RJ, Bayless JD, Volkert K, Evitts PE. (2002) Dichotic listening: expanded norms and clinical application. *Arch Clin Neuropsychol* 17(1):79–90.
- Morest DK, Kim J, Potashner SJ, Bohne BA. (1998) Long-term degeneration in the cochlear nerve and cochlear nucleus of the adult

chinchilla following acoustic overstimulation. *Microsc Res Tech* 41(3):205–216.

Musiek FE. (1983) Assessment of central auditory dysfunction: the dichotic digit test revisited. *Ear Hear* 4(2):79–83.

Musiek FE. (1986) Hearing beyond the eighth nerve. *Am J Otol* 7(5): 313–314.

Musiek FE. (2015) Adult diagnostics with an emphasis on electrophysiology. Presented at Student Academy of Audiology Conference (SAA), San Antonio, TX, March 25, 2015.

Musiek FE, Baran JA. (2004) Audiological correlates to a rupture of a pontine arteriovenous malformation. *J Am Acad Audiol* 15(2): 161–171.

Musiek FE, Baran JA, Pinheiro ML. (1990) Duration pattern recognition in normal subjects and patients with cerebral and cochlear lesions. *Audiology* 29(6):304–313.

Musiek FE, Baran JA, Pinheiro ML. (1994) *Introduction to Case Studies in Neuroaudiology. Neuroaudiology–Case Studies*. San Diego, CA: Singular Publishing Group.

Musiek FE, Baran JA, Shinn JB, Guenette L, Zaidan E, Weihing J. (2007) Central deafness: an audiological case study. *Int J Audiol* 46(8):433–441.

Musiek FE, Baran J, Shinn J, Jones R. (2012) *Disorders of the Auditory System*. San Diego, CA: Plural Publishing.

Musiek FE, Charette L, Kelly T, Lee W, Musiek E. (1999) Hit and false-positive rates for the middle latency response in patients with central nervous system involvement. *J Am Acad Audiol* 10(3): 124–132.

Musiek FE, Charette L, Morse D, Baran JA. (2004) Central deafness associated with a midbrain lesion. *J Am Acad Audiol* 15(2): 133–151.

Musiek FE, Chermak GD. (2007) Auditory neuroscience and (central) auditory processing disorder: an overview. In: Musiek FE, Chermak GD, eds. *Handbook of (Central) Auditory Processing Disorder: Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing, 3–12.

Musiek FE, Chermak GD, eds. (2014) *Handbook of Central Auditory Disorder: Auditory Neuroscience and Diagnosis* Vol. 1. 2nd ed. San Diego, CA: Plural Publishing.

Musiek FE, Chermak GD. (2015) Psychophysical and behavioral peripheral and central auditory tests. In: Celesia GG, Hickok G, eds. *Handbook of Clinical Neurology: The Human Auditory System: Fundamental Organization and Clinical Disorders*. Amsterdam, The Netherlands: Elsevier, 313–332.

Musiek FE, Chermak GD, Cone-Wesson BK. (in press) Central deafness. In: Oliver, D, ed. *Neurobiology of Hearing*. New York, NY: John Wiley & Sons.

Musiek FE, Chermak GD, Weihing J. (2014) Auditory training. In: Chermak GD, Musiek FE, eds. *Handbook of Central Auditory Processing Disorder: Comprehensive Intervention*. San Diego, CA: Plural Publishing.

Musiek FE, Chermak GD, Weihing J, Zappulla M, Nagle S. (2011) Diagnostic accuracy of established central auditory processing test batteries in patients with documented brain lesions. *J Am Acad Audiol* 22(6):342–358.

Musiek FE, Geurkink NA. (1982) Auditory brain stem response and central auditory test findings for patients with brain stem lesions: a preliminary report. *Laryngoscope* 92(8 Pt 1):891–900.

Musiek FE, Gollegly KM, Kibbe KS, Reeves AG. (1989) Electrophysiologic and behavioral auditory findings in multiple sclerosis. *Am J Otol* 10(5):343–350.

Musiek FE, Gollegly KM, Kibbe KS, Verkest SB. (1988) Current concepts on the use of ABR and auditory psychophysical tests in the evaluation of brain stem lesions. *Am J Otol* 9(Suppl):25–35.

Musiek FE, Kibbe-Michal K, Geurkink NA, Josey AF, Glasscock M, 3rd. (1986) ABR results in patients with posterior fossa tumors and normal pure-tone hearing. *Otolaryngol Head Neck Surg* 94(5):568–573.

Musiek FE, Kibbe K, Rackliffe L, Weider DJ. (1984) The auditory brain stem response I-V amplitude ratio in normal, cochlear, and retrocochlear ears. *Ear Hear* 5(1):52–55.

Musiek FE, Lee WW. (1998) Neuroanatomical correlates to central deafness. *Scand Audiol Suppl* 49(4):18–25.

Musiek FE, Lee W. (1999) Auditory middle and late potentials. In: Musiek FE, Rintelmann W, eds. *Contemporary Perspectives on Hearing Assessment*. Boston, MA: Allyn & Bacon, 243–272.

Musiek FE, Pinheiro ML. (1985) Dichotic speech test tests in the direction of central auditory dysfunction. In: Pinheiro ML, ed. *Assessment of Central Auditory Dysfunction: Foundations and Clinical Correlates*. Baltimore, MD: Williams & Wilkins, 201–217.

Musiek FE, Pinheiro ML. (1987) Frequency patterns in cochlear, brainstem, and cerebral lesions. *Audiology* 26(2):79–88.

Musiek FE, Pinheiro ML, Wilson DH. (1980) Auditory pattern perception in ‘split brain’ patients. *Arch Otolaryngol* 106(10): 610–612.

Musiek FE, Shinn JB, Jirsa RE. (2007) The auditory brainstem response in auditory nerve and brainstem dysfunction. In: Burkard R, Eggermont J, Don M, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application*. Baltimore, MD: Lippincott Williams & Wilkins, 291–312.

Musiek FE, Shinn JB, Jirsa R, Bamiou DE, Baran JA, Zaida E. (2005) GIN (Gaps-In-Noise) test performance in subjects with confirmed central auditory nervous system involvement. *Ear Hear* 26(6):608–618.

Musiek FE, Weihing J. (2011) Perspectives on dichotic listening and the corpus callosum. *Brain Cogn* 76(2):225–232.

Neijenhuis K, Snik A, van den Broek P, Neijenhuis K. (2003) Auditory processing disorders in adults and children: evaluation of a test battery. *Int J Audiol* 42(7):391–400.

Nilsson M, Soli SD, Sullivan JA. (1994) Development of the Hearing in Noise Test for the measurement of speech reception thresholds in quiet and in noise. *J Acoust Soc Am* 95(2):1085–1099.

Noffsinger D, Martinez CD, Schaefer AB. (1982) Auditory brainstem responses and masking level differences from persons with brainstem lesion. *Scand Audiol Suppl* 15:81–93.

Noffsinger D, Martinez CD, Schaefer AB. (1985) Puretone techniques in evaluation of central auditory function. In: Katz J, ed. *Handbook of Clinical Audiology*. Baltimore, MD: Williams and Wilkins, 337–354.

- Noffsinger D, Olsen WO, Carhart R, Hart CW, Sahgal V. (1972) Auditory and vestibular aberrations in multiple sclerosis. *Acta Otolaryngol Suppl* 303:1–63.
- Oleksiak M, Smith BM, St Andre JR, Caughlan CM, Steiner M. (2012) Audiological issues and hearing loss among veterans with mild traumatic brain injury. *J Rehabil Res Dev* 49(7):995–1004.
- Olsen WO, Noffsinger D. (1976) Masking level differences for cochlear and brain stem lesions. *Ann Otol Rhinol Laryngol* 85(6 Pt. 1):820–825.
- Pakarinen S, Takegata R, Rinne T, Huutilainen M, Näätänen R. (2012) Measurement of extensive auditory discrimination profiles using the mismatch negativity (MMN) of the auditory event-related potential (ERP). *Clin Neurophysiol* 118(1):177–185.
- Palmer SB, Musiek FE. (2013) N1-p2 recordings to gaps in broadband noise. *J Am Acad Audiol* 24(1):37–45.
- Paludetti G, Ottaviani F, Gallai V, Tassoni A, Maurizi M. (1985) Auditory brainstem responses (ABR) in multiple sclerosis. *Scand Audiol* 14(1):27–34.
- Pienkowski M, Eggermont JJ. (2012) Reversible long-term changes in auditory processing in mature auditory cortex in the absence of hearing loss induced by passive, moderate-level sound exposure. *Ear Hear* 33(3):305–314.
- Pinheiro ML, Musiek FE. (1985) Sequencing and temporal ordering in the auditory system. In: Pinheiro ML, ed. *Assessment of Central Auditory Dysfunction; Foundations and Clinical Correlates*. Baltimore, MD: Williams and Wilkins, 201–217.
- Saunders GH, Echt KV. (2012) Blast exposure and dual sensory impairment: an evidence review and integrated rehabilitation approach. *J Rehabil Res Dev* 49(7):1043–1058.
- Saunders GH, Field DL, Haggard MP. (1992) A clinical test battery for obscure auditory dysfunction (OAD): development, selection and use of tests. *Br J Audiol* 26(1):33–42.
- Saunders GH, Frederick MT, Arnold M, Silverman S, Chisolm TH, Myers P. (2015) Auditory difficulties in blast-exposed veterans with clinically normal hearing. *J Rehabil Res Dev* 52(3):343–360.
- Saunders GH, Haggard MP. (1992) The clinical assessment of “Obscure Auditory Dysfunction” (OAD) 2. Case control analysis of determining factors. *Ear Hear* 13(4):241–254.
- Schaette R, McAlpine D. (2011) Tinnitus with a normal audiogram: physiological evidence for hidden hearing loss and computational model. *J Neurosci* 31(38):13452–13457.
- Schuknecht HF. (1974) *Pathology of the Ear*. Cambridge, MA: Harvard University Press.
- Schuknecht HF, Woellner RC. (1953) Hearing losses following partial section of the cochlear nerve. *Laryngoscope* 63(6):441–465.
- Schwaber MK, Garraghty PE, Kaas JH. (1993) Neuroplasticity of the adult primate auditory cortex following cochlear hearing loss. *Am J Otol* 14(3):252–258.
- Shinn J. (2014) Temporal processing tests. In: Musiek FE, Chermak GD, eds. *Handbook of Central Auditory Processing Disorder: Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing.
- Shinn J, Long A, Rayle C, Bush M. (2016) Primary auditory symptoms in patients with normal peripheral hearing sensitivity: redefining hearing loss. *Hear Balance Commun* 14(1):44–49.
- Shinn J, Musiek FE. (2013) Beyond the audiogram Part I & II, Tier I, featured session. Presented at the American Academy of Audiology Meeting, Anaheim, CA, April 3–6, 2013.
- Silman S, Gelfand SA, Silverman CA. (1984) Late-onset auditory deprivation: effects of monaural versus binaural hearing aids. *J Acoust Soc Am* 76(5):1357–1362.
- Spahr AJ, Dorman MF, Litvak LM, Van Wie S, Gifford RH, Loizou PC, Loiselle LM, Oakes T, Cook S. (2012) Development and validation of the AzBio sentence lists. *Ear Hear* 33(1):112–117.
- Speaks C, Gray T, Miller J. (1975) Central auditory deficits and temporal-lobe lesions. *J Speech Hear Disord* 40(2):192–205.
- Swisher L, Hirsh IJ. (1972) Brain damage and the ordering of two temporally successive stimuli. *Neuropsychologia* 10(2):137–152.
- Valente M, Peterein J, Goebel J, Neely JG. (1995) Four cases of acoustic neuromas with normal hearing. *J Am Acad Audiol* 6(3):203–210.
- Vogel DA, McCarthy PA, Bratt GW, Brewer C. (2007) The clinical audiogram: its history and current use. *Commun Disord Rev* 1(2):81–94.
- Ward WD, Duvall AJ, 3rd. (1971) Behavioral and ultrastructural correlates of acoustic trauma. *Ann Otol Rhinol Laryngol* 80(6):881–896.
- Weihing J, Atcherson S. (2014) Primer on clinical decision analysis. In: Musiek FE, Chermak GD, eds. *Handbook of Central Auditory Processing Disorder: Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing.
- Weihing J, Guenette L, Chermak G, Brown M, Ceruti J, Fitzgerald K, Geissler K, Gonzalez J, Brenneman L, Musiek F. (2015) Characteristics of pediatric performance on a test battery commonly used in the diagnosis of central auditory processing disorder. *J Am Acad Audiol* 26(7):652–669.
- Weihing J, Musiek F. (2014) The influence of aging on interaural asymmetries in middle latency response amplitude. *J Am Acad Audiol* 25(4):324–334.
- Yasin I. (2007) Hemispheric differences in processing dichotic meaningful and non-meaningful words. *Neuropsychologia* 45(12):2718–2729.