

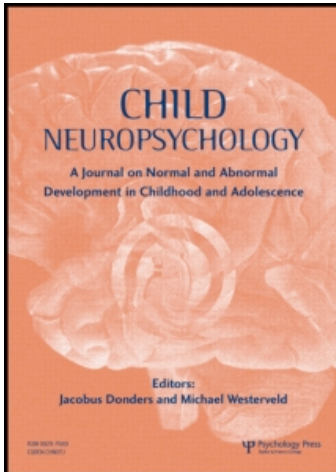
This article was downloaded by: [Bailey, Teresa]

On: 8 November 2010

Access details: Access Details: [subscription number 927410944]

Publisher Psychology Press

Informa Ltd Registered in England and Wales Registered Number: 1072954 Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



Child Neuropsychology

Publication details, including instructions for authors and subscription information:

<http://www.informaworld.com/smpp/title~content=t713657840>

Auditory Pathways and Processes: Implications for Neuropsychological Assessment and Diagnosis of Children and Adolescents

Teresa Bailey^a

^a Private Practice, Los Altos, California, USA

First published on: 29 September 2010

To cite this Article Bailey, Teresa(2010) 'Auditory Pathways and Processes: Implications for Neuropsychological Assessment and Diagnosis of Children and Adolescents', *Child Neuropsychology*, 16: 6, 521 – 548, First published on: 29 September 2010 (iFirst)

To link to this Article: DOI: 10.1080/09297041003783310

URL: <http://dx.doi.org/10.1080/09297041003783310>

PLEASE SCROLL DOWN FOR ARTICLE

Full terms and conditions of use: <http://www.informaworld.com/terms-and-conditions-of-access.pdf>

This article may be used for research, teaching and private study purposes. Any substantial or systematic reproduction, re-distribution, re-selling, loan or sub-licensing, systematic supply or distribution in any form to anyone is expressly forbidden.

The publisher does not give any warranty express or implied or make any representation that the contents will be complete or accurate or up to date. The accuracy of any instructions, formulae and drug doses should be independently verified with primary sources. The publisher shall not be liable for any loss, actions, claims, proceedings, demand or costs or damages whatsoever or howsoever caused arising directly or indirectly in connection with or arising out of the use of this material.

AUDITORY PATHWAYS AND PROCESSES: IMPLICATIONS FOR NEUROPSYCHOLOGICAL ASSESSMENT AND DIAGNOSIS OF CHILDREN AND ADOLESCENTS

Teresa Bailey

Private Practice, Los Altos, California, USA

Neuroscience research on auditory processing pathways and their behavioral and electrophysiological correlates has taken place largely outside the field of clinical neuropsychology. Deviations and disruptions in auditory pathways in children and adolescents result in a well-documented range of developmental and learning impairments frequently referred for neuropsychological evaluation. This review is an introduction to research from the last decade. It describes auditory cortical and subcortical pathways and processes and relates recent research to specific conditions and questions neuropsychologists commonly encounter. Auditory processing disorders' comorbidity with ADHD and language-based disorders and research addressing the challenges of assessment and differential diagnosis are discussed.

Keywords: *Auditory processing; Auditory pathways; Auditory subcortical pathways; Attention deficit disorder; Auditory processing disorder; (Central) auditory processing; (Central) auditory processing disorder; Learning disability.*

INTRODUCTION

Auditory processing refers to the electrical transformation and transmission of the auditory signal after it leaves the mechanical processes of the outer, middle, and inner ear (Musiek & Baran, 2007). The auditory signal is transmitted by the auditory nerve, a branch of the eighth cranial nerve, via the ascending auditory pathways, to termini in the auditory cortex and the cerebellum. The afferent pathways are still not completely understood. As a result of intensive research in both humans and animals in the last 20 years, the pathways and their functions are more completely mapped. The complexity of the transformation of auditory signals into what we recognize as sound and meaningful communication is beginning to be understood in ways that can be used more accurately to diagnose and treat developmental and acquired disorders in children and adolescents. A PubMed search in early December 2009 found 1,680 articles for the search term “auditory processing” and 522 articles when “auditory processing” was limited by the term “children.”

The author is grateful for valuable constructive criticism provided by James W. Hall III, PhD, of an early draft of this paper. Any inadvertent errors in the area of audiology and evoked potentials are the responsibility of the author.

Address correspondence to Teresa Bailey, P.O. Box 4058, Los Altos, CA 94024, USA. E-mail: tbaileyphd@me.com

In a technical report the American Speech and Hearing Association (ASHA, 2005) identified seven central auditory processing mechanisms: (a) sound localization, (b) lateralization, (c) discrimination, (d) pattern recognition, (e) temporal aspects of audition, including temporal integration, temporal discrimination (e.g., temporal gap detection), temporal ordering, and temporal masking, (f) auditory performance in competing acoustic signals (including dichotic listening), and (g) auditory performance with degraded acoustic signals (e.g., speech in noise).

An auditory processing disorder (APD) is present when an individual has significant difficulty in one or more of these processing mechanisms as demonstrated by abnormal performance on one or more tests of central auditory processes (ASHA, 2005; Chermak 2007). The impairment is not due to a disorder of attention, cognition, or language, although these may be comorbid. When they are comorbid, the auditory processing deficit or dysfunction is not caused by the other disorder(s). In light of this definition, an APD is not a discrete diagnostic entity but a term of convenience that alerts the clinician to the presence of one or more processing dysfunctions in one of the auditory processing mechanisms.

A substantial research literature supports the construct of auditory processing as physiological in nature. These physiological processes can be measured by valid and reliable electrophysiological, imaging, and behavioral assessment techniques (Musiek & Chermak, 2007). The basic dichotomous sorting of normal versus abnormal performance for adults is reasonably well established. Databases reflecting developmental norms for children's auditory development and performance on tests continue to expand.

Audiologists consistently assert the need for a multidisciplinary team to be involved in the diagnosis of APD due to multiple possible manifestations of behavioral, social, and academic problems associated with APD (ASHA, 2005; Musiek & Chermak, 2007). Neuropsychology as a clinical discipline that routinely evaluates all the impacted cognitive and behavioral domains is, however, absent from most APD research.

The diagnosis of an APD is made after it has been established by audiological evaluation that a patient has normal peripheral hearing (Musiek & Chermak, 2007). It has long been acknowledged that in children there are conditions short of deafness that can affect the reception of clear auditory signals, such as chronic otitis media, damage to the hair cells, and cochlear disorders. If no peripheral damage is detectable on pure tone audiometry and tympanography and in the absence of a lesion, it has been assumed that failure to track, to comprehend, and to respond appropriately to incoming auditory information is likely due to poor motivation, attention deficit disorder, receptive language disorder, and/or an expressive language disorder (Keller, 1992).

Studies of auditory processing began when Mykelbust (1954) and Bocca, Calearo, Cassinari, and Migliavacca (1955) published their research on the need for evaluation of auditory processing in children with communication disorders. Since that time most clinical research on central auditory processes has been carried out by audiologists and otolaryngologists.

Many pediatric behavioral tests of central auditory function were originally based on adult site of lesion studies. These tests were later re-normed to take into account the developmental stages and maturational performance changes in children (Kaplan, Gladstone, & Katz, 1984; Keith 2009a, 2009b). As the field has grown, psychometrically valid and reliable norms have been developed for both older and recent tests, and electrophysiological measurements have become available. Electrophysiological measures demonstrate good correlations with behavioral measures for distinguishing APD as a separate diagnostic entity (McArthur, Atkinson, & Ellis, 2009).

This review will address the following aspects of auditory processing: the afferent central auditory system, development and maturation, associated medical conditions, electrophysiological measures, behavioral assessment, differential diagnosis, and interventions.

AFFERENT AUDITORY SYSTEM

The central auditory system begins when the peripheral mechanical process is transformed into electrical impulses and conveyed to the brain via the auditory nerve, a branch of the eighth cranial nerve. From there the signal travels both ipsi- and contralaterally through a series of relay stations until it reaches the primary, or core, auditory cortex. It then travels ipsilaterally through the core and belt regions of the auditory cortex, and contralaterally via the corpus callosum to the opposite auditory cortex for further processing.

Other pathways that run through classical pathway structures, called the nonclassical pathways, bypass the thalamo-cortical portion of the classical pathway, and proceed to the auditory cortex by different pathways. Another pathway, known as the cortico-thalamic-cerebellar loop (Pastor, Vidaurre, Fernandez-Seara, Villanueva, & Friston, 2008; Sens & de Almeida, 2007), appears to have a role in auditory attention and the integration of behavioral responses to auditory and visual inputs (Pastor et al., 2008). The basal ganglia have been identified as having a role in processing auditory signal aspects of speech (Kotz, Schwartze, & Schmidt-Kassow, 2009), particularly timing.

Efferent pathways, thought to be part of a feedback loop that involves spatial orientation, help the listener move toward or away from the perceived stimuli. They contribute to basic awareness and attention (Parvizi & Damasio, 2001).

Before a cortical interpretation of the meaning of the incoming signal can occur and a behavioral response is generated, all sounds, both speech and nonspeech, are analyzed at subcortical levels for characteristics of frequency (pitch), duration, intensity (loudness), and interaural timing differences. This process begins in the peripheral auditory system, where frequency begins to be analyzed in the hair cells of organ of Corti. The hair cells are arranged to respond tonotopically (according to pitch, i.e., vibratory frequency). The range of frequencies that the hair cells respond to is fairly narrow. The hairs can be damaged by exposure to loud sounds. Damage to small groups of cells can result in selective pitch perception impairments for specific frequencies.

Most of the information in this section is a brief summary of information presented in Musiek and Baran (2007) and Musiek and Chermak (2007). Readers should consult these works for more precise information and detailed diagrams.

The afferent peripheral-central auditory junction is the auditory nerve. It carries four types of information: frequency (pitch), intensity (loudness), duration, and interaural timing information. The first relay station is the cochlear nucleus. The three constituent nuclei of the cochlear nucleus receive this information and relay it to different parts of the next steps in the pathway (see Figure 1 and Table 1).

All parts of the central pathway from this level through the cortical processing areas are arranged tonotopically, i.e., each pitch frequency is processed in an orderly array of cells arranged according to a specific pitch. In addition to the tonotopic arrangement, sound intensity and timing functions are analyzed by each relay station.

The *cochlear nucleus*, located in the pons, is the first connection between the auditory nerve and the central pathways. In addition to the three characteristics of pitch, duration, and intensity, analyzed here and at every other central location, areas within the

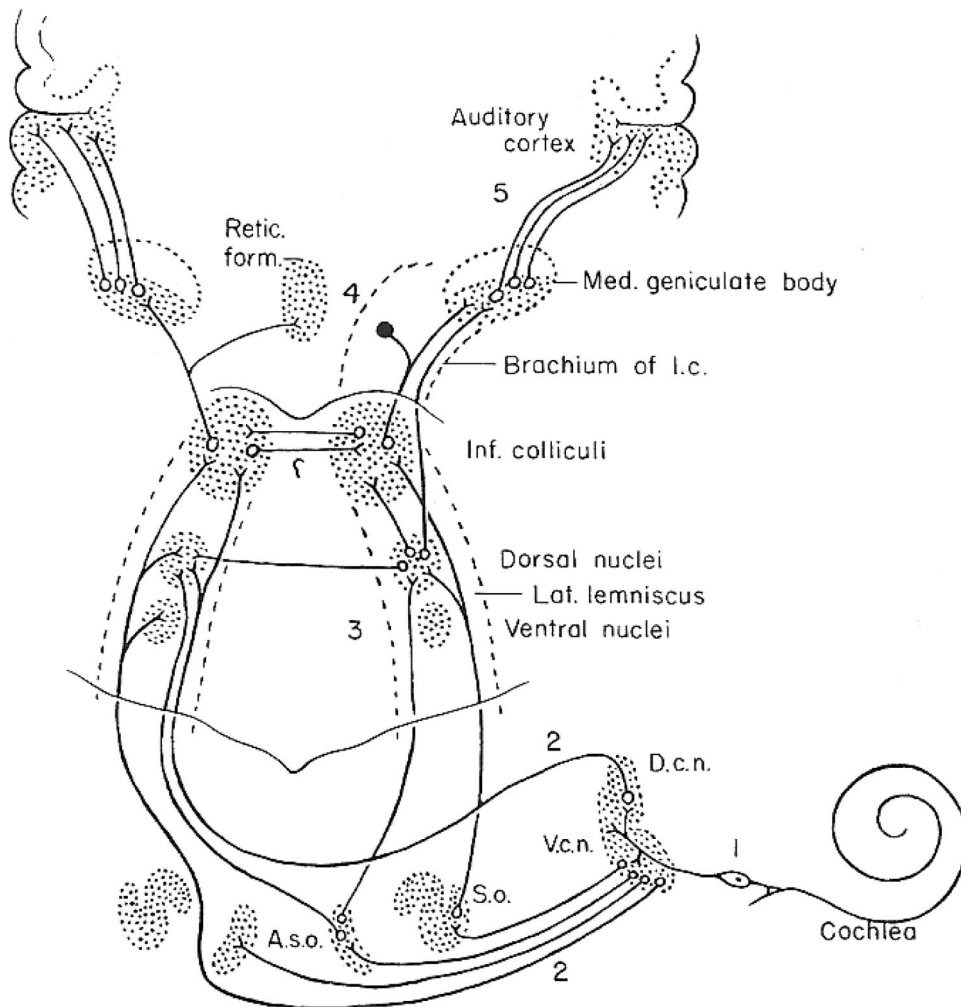


Figure 1 Afferent auditory pathways. Reprinted with permission from Gacek (1972).

cochlear nucleus respond selectively to speech-specific frequencies and assist in sound localization.

The *superior olivary complex* is located deeper in the pons than the cochlear nucleus. Spatial localization of the sound source is more accurately calculated by comparing the arrival times of pitches and intensity of the individual monaural inputs, on a frequency-by-frequency basis. This is the first level at which there is bilateral representation of the monaural data. The process by which this occurs is termed binaural fusion and integration. If the central pathway is compromised at this level, there can be noticeable difficulty localizing sounds. This has implications for tracking speech when a speaker is not stationary, as often happens when a speaker moves around a room while conveying information to the group as a whole.

This level of auditory processing can be affected by the presence of background noise. When ambient background noise and sound-reflective surfaces combine, listening becomes more difficult, and processing the information may be compromised (Shield & Dockrell, 2004).

Table 1 Afferent Auditory Pathways.

Structure	Processing Function	Evoked Response	Behavioral Process Test		Compromised Behavior
			Anatomic correlations inferred from lesion studies and auditory evoked responses	Anatomic correlations inferred from lesion studies and auditory evoked responses	
1. Auditory Nerve (AN) Part of Cranial Nerve VIII	1. Frequency (pitch), Intensity (loudness), Duration, Timing. Frequency preserves tonotopic organization from the cochlea	1. Waves I & II of Auditory Brainstem Response (ABR) Wave II often absent, even in normal population	1. Audiometry	1. Sensorineural hearing loss	Anatomic correlations inferred from lesion studies and auditory evoked responses
2. Cochlear Nucleus (CN) Consists primarily of three nuclei located in the pons.	2. Frequency, Intensity, Temporal Coding, Sound Localization	2. Wave III ABR	2. Acoustic Startle Reflex (a non-quantitative behavioral test for hearing in infants).	2. Poor hearing in noisy environment	
3. Superior Olivary Complex (SOC) Consists of several small diffuse nuclei. The caudal pons has a dense auditory region.	3. Frequency, Intensity, Timing, Spatial localization of sounds by comparing the arrival of interaural pitch and intensity of stimuli on a frequency-by-frequency basis, from binaural inputs. Fusion and integration of bilateral inputs.	3. Wave IV ABR	3. Auditory Figure-Ground, Speech-in-Noise Masking Level Differences (MLD) Interaural Time Differences, Interaural Intensity (IID) Differences (ITD) IID & ITD not used clinically	3. Difficulties in localization. Difficulties hearing speech in noise (Cocktail Party Effect)	
4. Ventral and dorsal nuclei of the Lateral Lemniscus (LL) tract Located in upper pons.	4. Frequency, Intensity, Timing Localization function based on sensitivity to Interaural Timing Differences (ITD) and Interaural Intensity Differences (IID)	4. Wave V ABR	4. IID, ITD	4. Not yet documented in humans	
5. Inferior Colliculus (IC) Located in the dorsal midbrain, the IC is the primary auditory structure of the midbrain.	5. Frequency, Intensity, Timing Localization of static & moving source. This is the first location with sound duration-sensitive neurons.	5. Wave Vn ABR Auditory Steady State Response (ASSR)	5. Gap Detection IID, ITD	5. Marked decrease in ability to localize and track sound; Problems hearing in noise; With some lesions, central deafness.	
6. Medial Geniculate Body (MGB) Located in dorsal and caudal Thalamus.	6. Frequency, Intensity, Timing Localization and Lateralization	6. Waves VI & VII of ABR, (not always present) Middle-Latency Response (MLR) especially Ventral and Medial GB	6. IID, ITD	6. Localization and lateralization of sound; Abnormal morphology seen in some dyslexic brains.	

(Continued)

Table 1 (Continued)

Structure	Processing Function	Evoked Response	Behavioral Process Test	Compromised Behavior
7. Insula	7. Frequency, Intensity, Timing Localization Phonological processing Nonverbal auditory processing Visual-auditory integration	7. Middle Latency Responses (MLR) Po, Na, Pa, Nb, Pb (P50)	7. Dichotic rhyme	Anatomic correlations inferred from lesion studies and auditory evoked responses 7. Some fMRI studies show decreased activation in the insula in dyslexic children; Phonological difficulties Central Deafness with bilateral lesion.
8. Auditory Cortex	8. Frequency, Intensity, Timing Localization (can distinguish timing differences as short as 1–2 milliseconds) Speech Perception Auditory figure-ground discrimination	8. Auditory Late Response (ALR) P1 (same as MLR Pb or P50), N1 (N100), P2, N2, P3 (P300)	8. Gap Detection Auditory figure-ground Pattern Recognition; provides diagnostic clarity among normal, cochlear, and cerebral deficits. May show bilateral deficits even if due to unilateral damage Abnormal Ear Advantage	8. Poor Frequency discrimination Auditory Agnosia
9. Corpus Callosum (CC)	9. Interhemispheric transfer of signals; mostly in sulcus area	9. Some tests of electrical impulse transmission speed; only used in research at this time.	9. Dichotic tests Pitch pattern perception in commissurotomy patients; Agenesis of CC patients within normal limits because of bilateral distribution of speech.	9. Commissurotomy patients show disconnection syndrome for language; Agnesis of CC patients usually perform within normal limits.
10. Cerebellum Not considered part of the classical afferent or efferent systems	10. Appears to be involved with both excitatory and inhibitory aspects of auditory timing, motor response to hearing; shifting and coordinating attention and actions among sensory modalities.	10. No specific tests at present	10. No specific tests at present	10. Unclear which behaviors are specific to the auditory aspects of the cerebellum.

11. Basal Ganglia Not considered part of the classical afferent or efferent systems	11. Excitatory & Inhibitory; Timing/beat processing of changes in duration, pitch, intensity; Detecting temporal cues in speech; Sequencing functions in syntax; Shifting attention/shifting set.	11. P300, P600	11. No specific tests at present	11. Unclear which behaviors are specific to the auditory aspects of the basal ganglia. Parkinson's patients: decrease in auditory attention, syntax processing increased pauses in speech, difficulties interpreting prosody (pitch, duration, intensity) in others' speech. When dysfunctional, likely places additional burden on cerebellar-thalamic-pre-supplementary motor area pathway.
--	---	----------------	----------------------------------	--

Just as there is an optic chiasm, there is an *auditory chiasm*. The auditory chiasm forms at the lateral superior olive. Another group of fibers in the superior olivary complex form the *lateral lemniscus*. This fiber tract contains several distinct nuclei within the larger tract. In addition, some lower fibers terminate within the lateral lemniscus. The function of the lateral lemniscus is not well understood.

The *inferior colliculus* is located in the dorsal midbrain. Contralateral spatial representation from the afferent fibers is preserved and possibly enhanced. This is the first level where there are dedicated duration-sensitive neurons. These neurons assist in sound localization. Disruptions at this level result in localization difficulties, problems hearing in noisy conditions and, with certain specific lesions, can result in central deafness.

Most fibers project to the ventral nucleus of the *medial geniculate body*, located in the dorsal and caudal thalamus. The inputs from the component nuclei of the inferior colliculus project to specific components of the thalamus. Temporal and frequency information regarding localization and lateralization of sound is more finely analyzed. Disruptions at this level lead to difficulties in the localization and lateralization of sound.

Fibers then project to the *insula* subcortex. At this level sound begins to be separated into speech and nonspeech sounds. The insula plays a poorly understood role in auditory signal and phonological processing. It is unclear the extent to which fibers connect with the arcuate fasciculus and are involved in auditory signal and language processing. This is the first level at which both visual and auditory information begins to be integrated outside the cerebellum (see Pastor et al., 2008). If a lesion is unilateral, difficulties in auditory processing will occur; if bilateral, central deafness can result.

From the insula, fibers project to the *auditory cortex*. In the past this area was described as primary and secondary. Current terminology uses the image of a core with at least two concentric areas around the core, the belt and the parabelt. The core is in the area of Heschel's gyrus, and the belts extend into neighboring sulci. At this level the tonotopic arrangement continues. Intensity is processed both in an inhibitory and excitatory fashion. Localization is analyzed according to interaural intensity and timing differences. Timing differences as short as 1 millisecond can be distinguished. Speech perception is recognized at the posterior temporal plane. Signal recognition in noise is differentiated in a figure-ground manner. The *corpus callosum* is involved in the interhemispheric transfer of information.

The auditory cortex is undergoing a rapid reevaluation of its structure and function. As imaging techniques give increasingly detailed static and functional information, researchers are beginning to identify regions where the brain separates and interprets the difference between speech and nonspeech sounds. Evidence points to this occurring beyond the traditionally defined auditory cortex. Different areas have specific functions for interpreting vowel sounds, distinguishing among vowel sounds, and the pitch at which the vowel is spoken. These functions are handled differently in the left and right hemispheres (Uppenkamp, Johnsrude, Norris, Marslen-Wilson, & Patterson, 2006).

The role of the *cerebellum* in auditory functions is not well understood. An emerging literature documents how auditory information is processed and relayed among the cerebellum, the classical and nonclassical afferent and efferent pathways. Sens and Almeida (2007) performed a systematic literature search on the auditory aspects of the cerebellum. Among their findings are support for the cerebellum's role in auditory timing and monitoring functions, auditory attention, especially selective auditory and complex sensory attention and focus; executing cortical commands that require coordination and timing; and inhibition based on incoming sensory information. The cerebellum is involved in

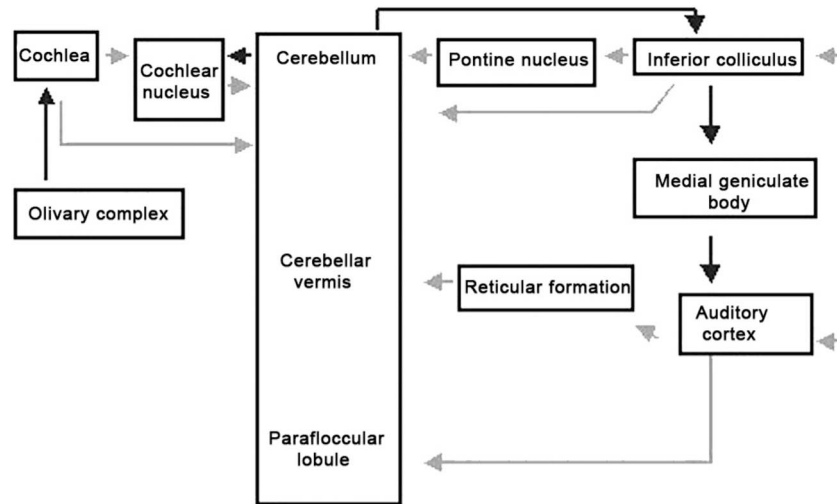


Figure 2 Cerebellar auditory pathways. Reprinted from Sens and de Almeida (2007).

voluntary control of cognitive processes, including shifting between visual and auditory stimuli.

Pastor et al. (2008) mapped an auditory cortico-cerebellar-thalamic loop (Figure 2). It appears to involve sensory integration functions. They hypothesize that this network is involved in selective auditory attention and possibly with motor output in conjunction with auditory signal processing.

The basal ganglia have recently been identified as having a role in auditory signal processing for language. Kotz et al. (2009) use the language deficits observed in Parkinson's disease patients to identify the processing of auditory beats and pauses. These beats can be the duration, pitch, and/or intensity aspects of speech-specific auditory signals.

DEVELOPMENT AND MATURATION OF CENTRAL AUDITORY FUNCTIONS

Fetuses have long been observed to respond motorically to external sounds that reach them in the womb. Newborn infants with normal hearing function respond to the sounds in their environments. Basic auditory functions are routinely screened by evoked potentials in both preterm and term infants (Hall, 2007). While normative data have been gathered to distinguish normal from abnormal electrophysiological responses, the full extent of the auditory maturational curve for children and adolescents has yet to be determined.

Auditory processes do not develop linearly (Musiek & Baran, 2007). Some are well developed at birth, while others continue to mature into late adolescence. In the first 6 months of life, babies gradually become better at distinguishing among high frequencies as the brainstem and middle ear continue to mature. By age 6 months babies can discriminate among higher frequencies as well as adults, while the ability to distinguish among low frequencies takes several more years to mature. Preschool children have difficulty separating sound foreground from background. By age 6 they have generally reached adult ability levels on formal testing; although they are still prone to becoming easily distracted by competing sounds. Between the ages of 7 and 10, children become better able to ignore

irrelevant sounds and to maintain focus in an environment with competing signals. Not until after age 12 are children able to discriminate as finely as adults among consonant categories. As a result, they rely more on multiple sources of cues for this information than do adults (Werner, 2007). Children's ability to identify consonant sounds accurately varies with the quality of the listening environment. This ability does not reach adult levels until about age 14 in reverberation-only and noise-only conditions. When confronted with noise-plus-reverberation conditions, however, adult level accuracy is not achieved until the late teen years (Johnson, 2000).

MEDICAL CONDITIONS KNOWN TO AFFECT CENTRAL AUDITORY PROCESSING

An expanding literature explores auditory processing in specific medical, developmental, and acquired conditions. Following are several pediatric conditions that are documented to involve central auditory processes. They appear in this text roughly in the order in which they tend to occur in developmental and diagnostic processes.

Perinatal Cerebellar Injuries

Limperopoulos, Robertson, Sullivan, Bassan, and du Plessis (2009) review the incidence of cerebellar injuries in preterm and term infants. They found that cerebellar injuries are not as rare as previously thought. Risks include preterm birth, increased intracranial pressure due to use of ECMO (extra corporeal membrane oxygenation) and other forms of assisted ventilation, instrument-assisted birth, and prolonged labor with excessive cranial molding. In term infants with cerebellar injuries, problems may occur with expressive language, socialization, and motor skills. Many of these problems have an auditory processing component. Some children with perinatal cerebellar injuries have developmental sequelae that eventually meet diagnostic criteria for autism spectrum disorders.

Hyperbilirubinemia

The cochlear nucleus is particularly vulnerable to damage from high bilirubin levels that can occur in the postnatal period. While closely followed infants are almost always treated appropriately, premature and low birth weight infants remain at risk for developing toxic levels that could damage the cochlear nucleus and basal ganglia. Emerging evidence suggests that damage from bilirubin occurs at what were previously considered safe levels (Nunez-Batalla, Carro-Fernandez, Antuna-Leon, & Gonzalez-Trelles, 2008; Shapiro & Nakamura, 2001).

Nunez-Batalla et al. (2008) investigated commonly occurring moderate hyperbilirubinemia that presents in the first week of extra-uterine life in up to 60% of full-term newborns, and as many as 80% of preterm infants. Both preterm and term infants and those who experience perinatal hypoxia are at increased risk for long-term toxic effects from this transient metabolic disorder.

Shapiro and Nakamura (2001) presented evidence that auditory processing problems occur in children without other signs of kernicterus. They cite studies with a range of documented abnormalities including auditory aphasia, auditory imperception, and word deafness. They argue that both the level of bilirubin and length of the hyperbilirubinemic state contribute to the pattern and degree of impairment observed. A range of often subtle

problems caused by bilirubin is now recognized and designated as Bilirubin-Induced Neurological Dysfunction (BIND).

Both groups of researchers argued that, although many children with elevated bilirubin and abnormal auditory brainstem response (ABR) findings usually resolve spontaneously within the subsequent year, these children remain at risk for developing auditory processing and learning disabilities. Shapiro and Nakamura urge more aggressive screening and follow-up with early interventions to prevent a substantial group of children from developing learning disorders, receptive language problems, and problems listening in noisy environments.

The findings of these two studies are important for neuropsychology because they demonstrate that there are identifiable impairments that are subclinical only in the sense that the classic neurotoxic syndrome rarely develops. They demonstrate that known sites of auditory pathway impairment can be identified, measured and can have late auditory processing and language effects. This has long-term clinical, developmental, educational, and social consequences in a subset of easily identifiable and trackable patients.

Neuropsychology can contribute to the growing knowledge base about children who have constellations of auditory attention, auditory memory, visual-motor integration, and some postural problems that do not fit any known disease process but that are encountered repeatedly in neuropsychological assessment. These children are often referred due to failure to meet educational expectations despite passing hearing tests, having intelligence within normal limits, and no hyperactivity. It is unknown how many attention deficit/hyperactivity disorder (ADHD) inattentive type patients actually may have an auditory processing problem that could be attributed to moderately high, until recently considered subclinical, bilirubin levels in infancy. When children with these complex presentations come for assessment, neuropsychologists often perform extensive records reviews dating back to gestation, delivery, and the postnatal period. Many health history questionnaires include questions about infantile jaundice and the use of bilirubin lights to treat transient hyperbilirubinemia. When the condition has not been severe or resolved quickly, no further note is usually taken of this phenomenon. In light of recent research, a history of treatment for hyperbilirubinemia, often noted by parents reporting that a child spent some time under fluorescent or "bili" lights, should raise the clinician's level of suspicion that auditory processing difficulties may need to become a focus of further clinical evaluation.

Otitis Media (OM) and Otitis Media with Effusion (OME)

Middle ear infections in infancy and toddlerhood are hypothesized to contribute to difficulty in the development of both receptive and expressive speech, as this is the period in which prelinguistic and linguistic skills are developing rapidly. Middle ear infections prevent auditory signals from being processed accurately and efficiently in the peripheral system for conduction to the central auditory processing system, by subcortical and intra-cortical pathways. It has been observed that with adequate treatment of ear infections, children with delayed receptive and expressive language usually catch up to their age peers. A subset of affected and treated children, however, do not progress as expected, and an even larger subset continues to demonstrate subtle language impairments and/or poor sustained auditory attention. Since ear infections are one of the most common childhood ailments, the effect of ear infection on auditory processing warrants more study than it has received to date.

Maruthy and Mannarukrishnaiah (2008) investigated the influence of otitis media in the first 12 months of life on brainstem and cortical auditory processing. Unlike most OM

studies, this study carefully controlled for socioeconomic status, age of infection, and age of follow-up assessment. Significant differences were found between patient and control groups only for 3-year-old children. By 4 years of age, the differences between groups had disappeared. The authors hypothesized that the early negative effects on brain stem function were compensated for by cortical structures becoming more easily excitable, i.e., via cortical gain, than they otherwise would have been without the decrease in signal input due to the ear infection. In a prospective study of 55 Dutch children from birth and 24 months through age 7, Zumach, Gerrits, Chenault, and Anteunis (2009) found that OM predicted a difference in performance in tests of speech in noise.

Gravel et al. (2006) prospectively followed 132 children in infancy and early childhood. The children underwent extensive testing at age 8. They found that OME with temporary hearing loss between the ages of 7 and 39 months resulted in abnormal performance on extended high-frequency hearing and brainstem measures at 8 years. They did not find any problems with binaural processing or speech in noise. Asbjornsen et al. (2005) found that children with a history of OME had impaired auditory attention skills compared to children with no known history of OME.

None of these studies found severe impairments in children who received adequate treatment for OM and OME. The range and inconsistency of findings across studies suggest that the extent and degree of subtle impairments are not established. Generalization from any of these findings to all children with a history of OM or OME is not yet warranted.

Craniosynostosis and Arnold-Chiari Malformations

Church, Parent-Jenkins, Rozelle, Eldis, and Kazzi (2007) examined evoked potentials in craniosynostosis patients. These patients may have disruptions of the eighth cranial nerve and auditory pathways in both the lower and upper brainstems. Abnormal auditory brainstem response (ABR) waveform patterns may be used to anticipate both the presence and severity of auditory processing disorders and their sequelae. These patients had asymmetric findings in the evoked potential results, due to displacement caused by craniofacial malformations. The authors observed similar problems in some patients with Chiari malformations.

The Arnold-Chiari malformations have a significant neurological and neuropsychological literature. Henriques-Filho and Pratesi (2006) studied Arnold-Chiari malformation types I (mostly adults) and II (mostly children). They used electrophysiological measures to demonstrate that 71% of the patients had abnormal auditory evoked responses (AER). They found that AERs were able to detect and localize the presence of functional abnormalities that cannot be examined with conventional structural measures such as MRI. The children showed significantly delayed maturation curves in their evoked responses. They did not reach normal values until 8 to 9 years of age, instead of the expected 4 years. Henriques-Filho and Pratesi argued that documentation of the presence and severity of the abnormal responses can lead to early interventions that will prevent additional functional deterioration.

Lesion Studies

Plaza, Rigoard, Chevrie-Muller, Cohen, and Picard (2001) presented a case of auditory processing dissociated from phonological processing. Their patient was a normally

developing boy who experienced a frontal trauma at age 23 months, with no apparent sequelae. Five months later the patient began to exhibit signs of Landau-Kleffner syndrome. At age 8.5 he underwent extensive neuropsychological and audiological evaluations. They observed that despite good phonological development for reading and writing, he was unable to respond adequately to spoken instructions or to learn orally presented information. He performed well when the same material was presented to him visually, in written form.

On tests of central auditory function, findings were significant for a complete left extinction for words and a nearly complete left extinction for digits on a dichotic listening test. Auditory evoked potentials were within normal limits. This suggested that the subthalamic auditory pathways were intact. The authors concluded that it is possible to have intact peripheral and subcortical processes, and even initial cortical processes, in the presence of abnormal processing of tones and speech sounds. Plaza et al. (2001) hypothesized that the compensatory use of visual processing to connect with linguistic material involved the phonological loop of the left inferior parietal and left inferior frontal cortices with input from primary visual areas, while bypassing the primary auditory cortex.

Hattiangadi et al. (2005) presented a thorough study of a child with nonpenetrating traumatic brain injury with identifiable areas of insult that resulted in auditory agnosia. He, too, was unable to respond to oral commands but responded easily to written instructions.

These studies demonstrate that disruption of the auditory pathways can have a variety of effects, depending on the location of the disruption. In both cases audiological and auditory processing evaluations provided critical information not otherwise available from standard neuropsychological and imaging studies. Some of the symptoms and neuropsychological test performances in these lesioned patients are similar to those in children without known lesions. While not equating the developmental disorders with discrete lesions, presentations with unexpected difficulties in following oral directions and conversations in the presence of normal hearing, spoken language, and reading, are seen in children without head trauma who are referred for evaluations. Both of these studies emphasize the need for traditional neuropsychological as well as auditory processing evaluation in order to obtain sufficient data to make appropriate treatment and accommodation recommendations.

ELECTROPHYSIOLOGICAL MEASURES

Both audiologists and neuropsychologists continue to grapple with the problem of making diagnoses based solely on verbal or motor responses to the perception and interpretation of speech and nonspeech auditory stimuli. Variable or impaired attention versus imperception, poor motivation, impaired oral and manual motor abilities, and receptive and expressive language disorders are all common confounds when testing auditory functioning with measures that require a behavioral response. A detailed discussion of electrophysiological measures is beyond the scope of this review. Hall (2007) is an authoritative resource for auditory evoked responses (AER) within the larger field of event-related potentials (ERP). Most of the information in this section is summarized from Hall. Readers are referred to his work for additional information.

An advantage of assessing the integrity of the central auditory system with evoked responses is that AERs do not depend on either directed attention or a volitional response to stimuli in order to elicit valid data on the state of the auditory system from the brainstem through the cortex. Some measures are already in routine clinical use, such as the auditory brainstem response (ABR) for screening individuals as young as infants, and P300 for

assessment of auditory and visual attention, response to treatments, and diagnosis of a range of neuropsychiatric disorders. Other measures, such as mismatch negativity (MMN), are currently used more in research studies than clinical practice. MMN shows potential for clinical use and studies involving this measure continue to increase (Bishop, 2007; McArthur et al., 2009; Sussman & Steinschneider, 2009).

Auditory-evoked potentials are elicited by presenting stimuli either monaurally or binaurally. The type and frequency of stimulus varies depending on which aspect of audition and which neural response is under investigation. As the signal travels from the ear along the auditory pathways, sensors on the scalp transmit the electrical impulses of the signal to a computer for processing. The traces and their characteristic shapes are analyzed and compared to normative databases. The amount of time elapsed from the signal presentation, measured in milliseconds, correlates with neuroanatomical locations along the auditory pathway.

Electrophysiological test terminology is inconsistent. Some measures such as ABR identify the location of the signal to be analyzed. Others, such as the middle latency (MLR) and auditory late (ALR) responses refer to the relative time window of the response. Still others, such as P300 or N1 refer to a specific time window. Measures with the designation positive (P) or negative (N) refer to the slope of a particular peak or trough that is the focus of analysis. See Table 1 for the anatomical correlates of the electrophysiological measures. Figure 3 shows the names, time windows, and amplitudes of the primary central auditory evoked responses, plotted on a logarithmic time scale.

The auditory brainstem response (ABR) is the first group of central evoked responses. There are seven characteristic waves; although not all are routinely generated in younger children. The waves are generated by the subthalamic portions of the central auditory system. The ABR waves are generated in the first 5 to 7 milliseconds after the onset of a stimulus. Each wave is designated with a Roman numeral. In addition to examining the shape and amplitude of the waveforms, the time latency between waves I and V is assessed to determine the presence or absence of dysfunction in the auditory nerve and

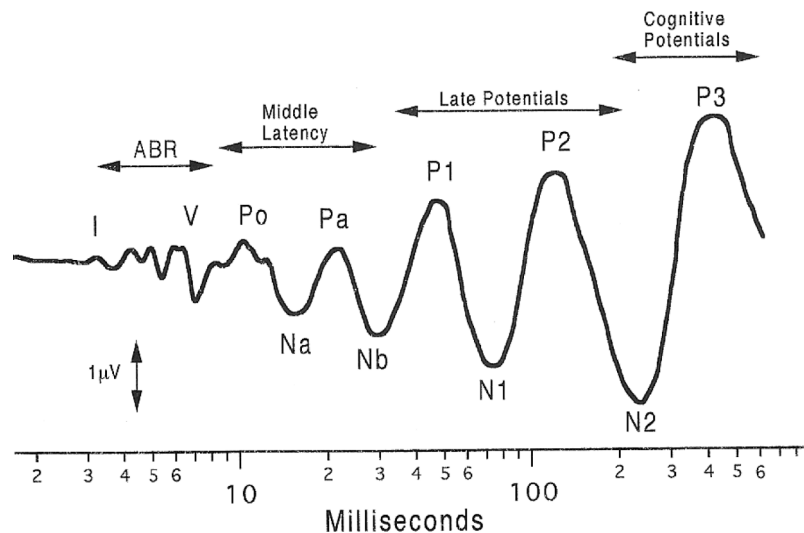


Figure 3 Auditory evoked potentials. © 1998 Wolters-Kluwer. Reprinted with permission from Sinniger and Abdala (1998).

auditory brainstem. ABR is not affected by sleep state or most medications. Portions of the wave are present as early as 25 weeks gestation. This measure is routinely used as a component of the evaluation of the integrity of infant auditory systems. Hall (2007) lists 38 medical conditions that have been reported in the literature for which ABR studies in the pediatric population exist. These range from the common, for example, the use of ototoxic medications and fetal alcohol syndrome, to rare syndromes with auditory processing components.

The middle latency response (MLR) is generated by the thalamus and parts of the auditory cortex. The MLR occurs between 10 and 60 milliseconds after stimulus onset. In order for it to be evoked reliably in children under the age of 10, modifications must be made to the stimulus presentation. Sleep and medication also must be carefully assessed. Among the clinical uses of the MLR is the diagnosis of an auditory processing disorder above the level of the brainstem. The final wave of the MLR is also a component of the first wave of the auditory late response (ALR). When analyzed as part of the MLR, the final wave is designated Pb or P50, as it occurs at approximately 50 milliseconds poststimulus onset. When analyzed as part of the ALR, the same peak is referred to as P1.

The auditory late response (ALR) occurs within the broad range of 50–500-plus millisecond time frame. It is easily affected by both sleep state and medications, especially sedatives. It is used to assess the integrity of the cortical auditory system, to diagnose auditory processing disorders, to evaluate auditory and visual attention to novel stimuli, and to evaluate other neuropsychiatric conditions. The P300 and P600 responses have been investigated for the basal ganglia's role in the basal ganglia-cortical loop for attention and temporal chunking and sequencing aspects of auditory processing (Kotz et al., 2009).

Specific late measures that relate to pediatric neuropsychology include mismatched negativity (MMN) and P300. MMN is an involuntary cortical response that occurs between 100–300 milliseconds after a stimulus onset. The name refers to the difference, or mismatch, of the negative troughs of two waveforms. The mismatch is calculated as the difference between the curve that results from presentation of a frequent stimulus compared to the curve for the presentation of an infrequent stimulus. The resulting mismatch curve provides information not only about characteristics surrounding the detection of a repeated stimulus but also whether the patient is able to register the occurrence of a different, infrequently presented stimulus. Because patients habituate easily to the frequent stimulus, MMN is not a good measure of sustained attention. It is useful, however, in assessing the ability to detect changes in signals, and the degree of cortical activation when stimuli change. Application of MMN to auditory processing, dyslexia, and specific language impairment is discussed below in the section on Differential Diagnosis. Figure 4 gives an example of how electrophysiological information for N1, P2, and MMN are displayed for analysis of the brain's response to habituated and novel stimuli.

The P300 response represents a response latency of approximately 300 milliseconds. The wave is also called P3 because it is the third positive wave in the late-response sequence. It is used to assess higher level auditory processing and visual and auditory attention to infrequent, novel stimuli. It is also used to assess the efficacy of treatment for a variety of neuropsychiatric disorders, including ADHD. There are two forms of P300 assessment. One, called the passive P3 or P3a, does not require focused attention. The other, P3b, is affected by an individual's ability to direct attention to the stimuli.

Most electrophysiological auditory measures are specific to audiology and otolaryngology. They require referral to specialists trained to conduct and interpret these measures. Some of the late responses, such as P300 and MMN, have wider applications in clinical

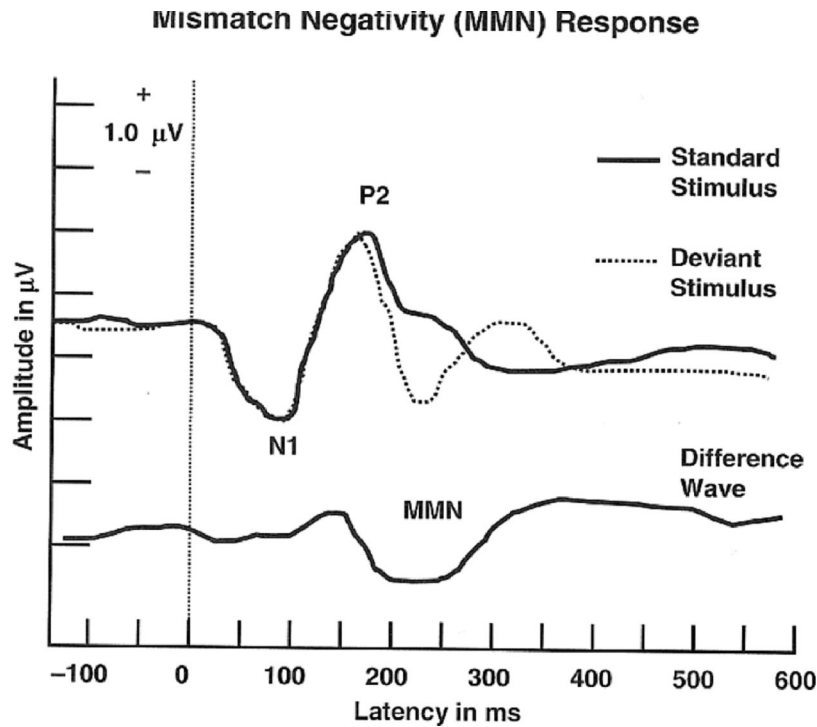


Figure 4 Mismatch Negativity (MMN) Response. Reprinted from Hall (2007).

and experimental psychology, cognitive neuroscience, neurology, and psychiatry. With proper training, assessment and interpretation of aspects of the late cortical responses are within the scope of practice of contemporary neuropsychology for assessing auditory and visual attention, medication titration, and diagnosing aspects of cognitive-behavioral impairment.

BEHAVIORAL ASSESSMENT

Behavioral tests of auditory processing were originally developed to correlate with known central lesion sites. These tests were later re-normed to take children's developmental trajectories into account. Behavioral tests present potentially confounding challenges due to the patient's need to attend closely to auditory stimuli that may be difficult to process, and the requirement to respond with language or motor output. These tests may have complex stimuli and instructions that place an additional cognitive burden on some patients. As a result, it can be difficult for the clinician to know whether a patient is responding poorly due to a problem understanding the oral instructions that may itself be a manifestation of an auditory processing disorder, or if some other cognitive process is involved. At the same time, behavioral tests are the most accessible screening tools available to neuropsychologists who need to understand the role audition may play in a patient's listening difficulties. Auditory processing is not currently routinely assessed in a systematic way by any organized group of healthcare providers, including audiologists. Research continues to accumulate; however, that demonstrates how auditory processing

functions impact a significant number of developmental, learning, and social behaviors, as well as acquired physiological impairments. Neuropsychology is in a unique position to bring this information together to achieve more accurate diagnoses as part of the routine assessment of attention and a wide variety of language-based functions.

In a longitudinal study of normally developing children between the ages of 6 and 12, Stollman, van Velzen, Simkens, Snik, and van den Broek (2004) distinguished three broad factors in auditory processing: (a) pattern recognition, (b) speech in noise, and (c) auditory closure and binaural fusion. Two additional factors that indicate the integrity and maturity of the auditory system are (d) temporal auditory processing, including gap detection and (e) sound localization (Musiek & Chermak, 2007). ASHA (2005) identified seven areas of central auditory functioning: localization, lateralization, discrimination, pattern recognition, temporal aspects, performance in competing acoustic signals, and performance with degraded signals. All seven functions are not routinely screened by audiologists. Gap detection has only recently begun to be assessed routinely in clinical settings (Keith, 2009a, 2009b). Sound localization is not yet in routine clinical use; although clinical feasibility studies are underway (Cameron et al., 2009).

A behavioral auditory screening battery, designed to be used by both audiologists and nonaudiologists is available for two age groups. The SCAN-3-C is for children aged 5 years to 12 years 11 months and the SCAN-3-A is for adolescents and adults aged 13 years to 50 years 11 months (Keith, 2009a, 2009b). Using headphones and a compact disc player, the initial screening tests three areas of auditory processing in 10–15 minutes: temporal auditory processing (Gap Detection), signal-to-noise or speech detection in the presence of background noise (Auditory Figure-Ground), and general indication of maturation of the central auditory system through interhemispheric transfer of information with dichotic listening (Competing Words). If any one of these screening tests is failed, more extensive testing with the core diagnostic battery is indicated. The core diagnostic battery consists of additional dichotic tests and a test of auditory closure in degraded conditions (Filtered Words). Supplementary tests include listening to rapid speech (Time Compressed Sentences) and an additional speech-in-noise test with a stronger signal-to-noise ratio at +12 decibels for both age groups. These additional tests can add 20–30 minutes to an evaluation. The SCAN-3 does not assess pitch or pitch-pattern perception, localization, or some important aspects of sound discrimination.

Interpretation of findings is addressed at three levels in the manual. These levels are at the individual test level, the composite index level, and left-right performance differences. Since performance on the Gap Detection subtest is not normally distributed, this test uses a cutoff score to determine normal and abnormal performance. A typically developing child in the age ranges covered by the SCAN-3 batteries should be able to distinguish the presence of a gap as short as 20 milliseconds (Keith, 2009a, 2009b). All other tests produce scaled scores.

Tests are interpreted individually for the underlying process they assess. All of the subtests, except Gap Detection, which uses a performance cutoff score, and Filtered Words, showed significant differences when comparisons were made between a group diagnosed with impaired central auditory function and a group of matched controls. Tables showing sensitivity and specificity for the subtests at each scaled score level are provided in the test manuals.

Given the significant differences achieved for the other scaled-score subtests, it is unclear why Filtered Words was retained in this revision of the battery. Further, if the Filtered Words test provides additional informational value about processing in degraded

conditions, it is unclear why this information is not included in the manual. The reasons for placing this subtest in the core diagnostic index instead of in the supplementary test group are unclear.

Another question about the construction of the SCAN batteries is the role of the Auditory Processing Composite (APC) for the SCAN battery. If, according to Chermak (2007) and ASHA (2005), an impairment in any of the seven auditory functions is sufficient for the diagnosis of an auditory processing disorder and if, due to the construction of composite index scores, significant differences can be obscured in the averaging process, it is unclear what diagnostic role the APC plays. There is no evidence in the audiology literature that a diagnosis of an APD is made based on averaged scores from different functional processes about which no correlational or factor data are presented.

Description of left-right ear score differences and the developmental role and trajectory for establishing a dominant ear are given very little attention in the manual, even though these scores are calculated. No additional references are provided in the manual. A discussion of lateralized ear advantage is beyond the scope of this review. Lateralized ear performance data are important, however, in understanding the level of maturity and the integrity of the monaural, and interhemispheric transfer of, the auditory signal.

This brief auditory processing battery provides useful information about the state of central auditory processes unavailable through other neuropsychological test procedures. This additional information can assist a neuropsychologist by providing information about (a) whether auditory processing difficulties contribute to attention deficits; (b) the ability to listen under suboptimal (degraded) conditions that often exist in classrooms, in the community, and even in the home; (c) an individual's ability to process rapid speech; and (d) an individual's ability to direct auditory attention under dichotic conditions. Based on this information, the neuropsychologist can generate hypotheses about the maturity of the system in general and integrity of interhemispheric transfer of auditory information. If problems are found on any of the subtests of the SCAN battery, referral to an audiologist with expertise in APDs is recommended. Even for patients who achieve scores grossly within normal limits, children performing in the low average and borderline levels will receive documentation of difficulties in this functional domain. Based on the individual pattern and level of performance, interventions and accommodations can be implemented.

Two auditory mechanisms not assessed by the SCAN battery are pitch perception and pitch-pattern recognition. Auditory pattern recognition is the fourth of seven central auditory processing capacities listed by ASHA (2005). One commonly used neuropsychological test of sustained attention, the Test of Variables of Attention (TOVA; Greenberg, 2007), tests auditory attention by means of a pitch discrimination task. The patient responds or inhibits response based on perceived relative pitch (i.e., whether the pitch is high or low relative to the other pitch in the test). If pitch perception and the ability to distinguish between a pattern of high versus low pitches are not tested prior to administration of this procedure, faulty diagnoses and recommendations may result.

The problems of pitch discrimination and pitch-pattern recognition are not limited to one neuropsychological test. A range of neuropsychological observations involve subjective judgment about an individual's ability to imitate and/or generate pitch-dependent aspects of speech, including prosody and verbal emotional expressions congruous with language content, and the ability to sing melodies to common songs such as "Happy Birthday." Some children, particularly those on the autism spectrum and those who have the so-called nonverbal learning disability pattern of symptoms, may speak in a flattened tone pattern and/or cannot correctly "read" the emotional tone from the prosody of another speaker.

The problem is compounded for children who speak tonal languages that depend on subtle relative pitch differences and patterns to specific word meanings (Zatorre & Gandour, 2008). These common manifestations of tonal and temporal patterns of auditory processing can be associated with developmental delay or impairment of central auditory processes for which children are referred for neuropsychological and speech and language evaluations.

DIFFERENTIAL DIAGNOSIS OF APD

The diagnosis of an auditory processing disorder continues to evolve (ASHA, 2005). Chermak (2007) makes clear that a diagnosis of an APD requires poor performance on at least one test that has demonstrated specificity and sensitivity to central auditory function. The SCAN batteries (Keith, 2009a, 2009b) report the specificity and sensitivity information for the component subtests of the battery. Even though there is considerable comorbidity among APDs, attention deficits, and language-based disorders, an APD itself is not the result of another disorder. The behavioral overlap of comorbid diagnoses is due to the widely distributed networks with which central auditory pathways are linked, in bottom-up, top-down, and reciprocal loop processes.

Attention Deficits

Auditory processing is an integral part of attention in persons without severe hearing impairment. In addition to the classical auditory pathways, the nonclassical pathways involve structures related to wakefulness, awareness, and attention, such as the reticular formation. These pathways closely parallel and integrate with other sensory pathways in the brainstem (Parvisi & Damasio, 2001) and the cerebellum (Sens & Almeida, 2007). The problem of differential diagnosis of an auditory processing disorder from an attention deficit disorder, especially in children, is longstanding. Keller (1992) observed a diagnostic bias according to which a professional first evaluated an individual. Given the same set of symptoms, particularly if a child were not severely hyperactive, audiologists would be more likely to diagnose an auditory processing disorder, while a psychologist would be more likely to diagnose an attention deficit. To begin to address this problem, Tillery, Katz, and Keller (2000) conducted a double-blind placebo controlled counterbalanced study. They demonstrated that while there is behavioral overlap between ADHD (Attention Deficit Hyperactivity Disorder) and APD, and while effective therapy with Ritalin can result in improved sustained attention on an auditory continuous performance test, Ritalin had no significant effect on the performance on three measures of central auditory functioning in patients who had improved scores on an auditory continuous performance test. The auditory tests that did not improve in medicated individuals were Staggered Spondaic Word (a dichotic interhemispheric measure), Phonemic Synthesis, and Speech-in-Noise.

Chermak (2007) addressed the differential diagnosis of an APD and ADHD based on commonly used behavioral observations and symptom checklists. There is no empirical diagnostic test that can identify ADHD. An ADHD diagnosis made based on age of symptom onset and behavioral symptoms listed in the *Diagnostic and Statistical Manual of Mental Disorders IV, Text Revision* (American Psychiatric Association, 2000). Thus, patient history is the main criterion for an ADHD differential diagnosis. Chermak notes that children diagnosed with ADHD often have histories of chronic otitis media, and that children with APD often have comorbid diagnoses of one or more learning disabilities and/or specific language impairment.

Chermak, Somers, & Seibel (1998) examined the interface between classical concepts of top-down processes in ADHD (i.e., a failure of executive function needed to sustain attention to auditory stimuli) and bottom-up processing of auditory inputs in APDs and asked whether the failure to attend were a result of poor executive control or were secondary to poor processing of auditory stimuli. They proposed a bidirectional interaction for auditory attention and comprehension. They argued that difficulties with attention and listening are separate but closely related behavioral manifestations of underlying dysfunctions. Understanding these relationships will require further neuroanatomical investigations of each process and how it relates to the others.

In an analysis of the commonly assumed behavioral overlap between ADHD and APD symptoms, Chermak, Hall, and Musiek (1999) found that only 2 of 11 behaviors appear in two different behavioral checklists used by pediatricians and audiologists to screen for attention and listening problems. APD was characterized as a selective auditory attention deficit with problems in language processing and accompanying academic problems, whereas ADHD was characterized more as a motor regulation problem with impulsive, interrupting speech. Chermak (2007) found that in a rank ordering of symptoms, ADHD inattentive type was characterized primarily as inattentive with accompanying academic problems and distractibility. Children diagnosed with an APD tended to ask for things to be repeated and had poor listening skills, problems following oral directions and difficulty hearing in ambient background noise. Academic difficulties and distractibility entered the list after the other symptoms. From a neuropsychological evaluation perspective, the APD group showed poor sustained auditory attention, poor auditory memory, and difficulty discriminating speech. None of these behaviors was present in the ADHD inattentive type checklist.

Riccio, Cohen, Garrison, and Smith (2005) addressed some of these concerns in a neuropsychological study of auditory processing measures and their correlation with neuropsychological measures of attention, memory, and behavior. They found no significant overlap between auditory processing measures and a sustained visual attention measure. They argued that if visual attention is found to be within normal limits but behavioral auditory attention is poor, APD may be a better diagnostic path to pursue. A shift between auditory and visual sensory modalities is measured by, if not yet part of the standard interpretation of, the IVA-Plus (Integrated Variables of Attention-Plus; Sanford & Turner, 1989, 2005) continuous performance test. This test requires frequent shifting between verbal auditory and visual stimuli. Both classical and nonclassical systems likely contribute to this complex set of attention demands (Kotz, 2009; Sens & Almeida, 2007). The verbal auditory stimuli are available in several languages. In contrast, the TOVA (Greenberg, 2007) measures visual and auditory attention in separate tests. The auditory version of the TOVA bypasses language but requires intact pitch discrimination and has additional cognitive loads for the spatial conceptual overlay of high and low pitch and the need to remember the referential pitches in the context of high and low pitch. The test, at present, does not require audiological confirmation of pitch discrimination or pitch-pattern sequencing prior to administration of the test.

While pharmacological treatment of ADHD that results in sustained attention and decreased impulsivity may aid in classroom performance, it does not address decrements in auditory processing (Tillery et al., 2000). The findings from Tillery et al., that stimulant medication does not improve abnormal auditory processing in children diagnosed with APD, whose performance on continuous performance tests does improve with this class of medications, may account for a proportion of patients who fail to respond adequately to medication prescribed for attention problems. As argued by Riccio et al. (2005), they may have an undiagnosed comorbid APD.

Sussman and Steinschneider (2009) used MMN and the ALR measure P3b to study active and passive auditory attention in children and adults. Their results show clear maturational factors in auditory attention and discrimination of sound differences. Their conclusions led them to argue that a more complex interaction exists among developmental attentional capacity, auditory processing, and language development than has been proposed on the basis of evoked potential studies to date. They further proposed that problems in attention may affect and reinforce problems in children with developmental language disorders and/or an APD.

Auditory Processing in Noisy Environments

Auditory processing in the presence of noise presents a special problem of differential diagnosis. If an individual has a normal tympanogram and normal auditory perception thresholds, common sense suggests that the individual is likely to be able to attend to and process speech in the presence of mild-to-moderate background noise. Failure to process speech in these common conditions may result in the assumption that the individual is either not well motivated to listen or may have an attention deficit. Research continues to accumulate in support of speech processing in the presence of background noise as an important component of central auditory processing (Musiek & Baran, 2007; Musiek & Chermak 2007; Ziegler, Pech-Georgel, George, & Lorenzi, 2009). Elementary school classrooms have been found to have an average background noise level of 72 decibels. This level of noise can reduce speech intelligibility more than 50% in persons with normal hearing and no auditory processing difficulties (Jamieson, Kranjc, Yu, & Hodgetts, 2004). Picard and Bradley (2001) reviewed the role of ambient noise levels in classrooms. They considered age-based needs and abilities as well as the special needs of children with problems processing sound in the presence of background noise. It is beyond the scope of this review to address the role of acoustics in classrooms, including the roles of construction, design, furnishing choices, and additional background sounds from increased use of noise-producing technologies as contributors to ambient noise levels and surface sound reflectivity. This is an area of cross-disciplinary research that bears integration into the assessment of the environments in which school children may spend a quarter or more of their waking hours (Jamieson et al., 2004; Johnson, 2000; Picard & Bradley, 2001; Shield & Dockrell, 2004; Zumach et al., 2009).

Ziegler et al. (2009) addressed a portion of the complex differential diagnostic challenge of the relationships among attention, auditory processing, and language-based disorders in relationship to processing speech in noisy environments. They found speech in noise perception abilities predicted 28%–44% of unique variance for phonological decoding and 18%–37% for reading. Their experiment involved dyslexic and normal readers who were matched for both chronological age, and reading age matched with younger age-appropriate readers. Among the conclusions generated by the study is that whatever other deficits underlie dyslexia, they are significantly exacerbated if a dyslexic reader has a comorbid processing deficit for speech perception in noise.

Language-Based Learning Disabilities

The role of auditory processing in speech and reading disorders gained prominence with the work of Tallal and Piercy (1973a, 1973b) who developed the rapid auditory temporal processing theory (RATP) based on their work with children with specific language

impairment (SLI) who had normal audiograms. Tallal (1980) extended this work to include children with dyslexia. Based on this work the Fast ForWord intervention was developed (Merzenich et al., 1996). Fast ForWord is discussed below under Interventions.

McArthur et al. (2009) examined the RATP theory as part of an extensive review of theories of auditory processing in language and reading impairments. They designed their multitiered research program to avoid many of the methodological difficulties that have tended to limit interpretation and generalization of much prior auditory processing research. The children in the McArthur et al. study underwent diagnostic evaluations for APD, SLI, and Specific Reading Disability (SRD) and were then matched with normal controls. McArthur et al. found that both diagnostic groups have members with atypical passive N1-P2 waves when presented tonal and consonant-vowel stimuli, whether the stimuli were presented slowly or rapidly. They interpreted their findings to mean that there is neither a speech-specific nor rate-specific deficit. Instead, they observed a generalized sound processing deficit in both diagnostic groups. Based on their own research and their review of other published studies, they estimate that this generalized deficit affects 33%–50% of children diagnosed with SLI or SRD.

They subsequently compared the children in the diagnostic groups who had atypical N1-P2 waveforms to children in the diagnostic groups who had typical waveforms. They found that the combined SLI and SRD typical waveform groups did not differ in spoken language skills. When the entire SLI group was examined in isolation, however, those with atypical waveforms performed worse on both repeating and reading non-words. The SLI group with normal waveforms performed poorly on one or the other non-word tests, but not both. McArthur et al. (2009) concluded by proposing the hypothesis that the observed N1-P2 auditory processing impairments represent a causal risk factor for both language and reading disorders. They outlined future experiments to link this documented physiological deficit to specific anatomical regions through functional imaging studies.

MMN has shown clinical usefulness in measurement of auditory discrimination in infants through adults. Bishop (2007) reviewed several international prospective studies of children with familial histories of dyslexia. Even when differences in design, presentation of stimuli, and language characteristics were considered, differences between children at risk for dyslexia were found compared to control groups in the lateralization of responses to stimuli. Most researchers found decreased left hemisphere activation in diagnostic groups compared to control groups.

In a wide-ranging review of the theoretical and experimental literature on the processing of speech, prespeech, and nonspeech sounds, Zatorre and Gandour (2008) reviewed the two leading theories of speech processing. The first proposes a speech domain-specific module governed by higher cortical processes. The second understands auditory processing of speech as a collection of generic neural processes that work together to process different acoustic components of speech. They cited evidence in support of each theory along with situations that cannot be answered by adherence to one position or the other. They propose that a combination of both positions is supported by recent experiments, functional imaging, and electrophysiological studies. McArthur et al. (2009) answer some but not all of the questions posed by Zatorre and Gandour (2008).

Zatorre and Gandour (2008) presented additional evidence collected on the role of tone and duration in processing both speech and nonspeech sounds across tonal and nontonal languages, including Mandarin Chinese, Thai, English, and German. These components,

temporal and pattern processing, are part of the ASHA (2005) list of core central auditory processes but have received little neuro-audiological research interest to date.

Zatorre and Gandour's (2008) work moves the field beyond current receptive-expressive language concerns to address auditory processing's role in problems of failure to perceive and express prosody in spoken language. The processing of prosody and semantic meaning leads to additional questions about difficulties interpreting and expressing emotional meaning through language that are often encountered in autism spectrum disorders and the so-called nonverbal learning disability syndrome. Gage, Siegel, Callen, and Roberts (2003) used magnetoencephalography (MEG) to investigate the M100, which is the same as the N100, auditory latency in children with autism. They found that compared to normal controls, children with autism displayed a significantly reduced range of tonal frequency modulation in the right hemisphere. This is the region where speech tone and prosody are interpreted.

INTERVENTIONS

Interventions for auditory processing disorders fall into two broad categories: therapy and accommodation. Neither type of intervention has sufficient documentation to meet an evidence-based standard of care for addressing auditory processing disorders. While some therapies show promise, they are still considered experimental (ASHA, 2004). Others that have peer-reviewed papers describing their efficacy in small experimental samples are not in widespread clinical use (Chermak & Musiek, 2007). Two widely available interventions, auditory integration therapies (AIT) such as Berard AIT (Berard, 1993) and temporal processing therapies such as Fast ForWord (Merzenich et al., 1996), have seen widespread use in clinical practice, but neither has met evidence standards (ASHA, 2004; Gillam et al., 2008). While some show promise, the current state of intervention lags far behind research on neuroanatomy and assessment of auditory functions (Chermak & Musiek, 2007).

Merzenich et al. (1996) developed Fast ForWord, a computer-based intervention. Fast ForWord is designed to treat deficits in rapid auditory processing by slowing down and then gradually increasing the speed of presentation of phonemes so that they can be more easily processed by an individual who is presumed to have a temporal (time-based) auditory processing disorder. While the use of Fast ForWord has demonstrated some improvements in auditory processing for some children with speech, language, and reading disorders, it has not proved to be superior to other interventions for improving reading fluency and comprehension (Gillam et al., 2008; Moore, Halliday, & Amitay, 2009). This does not mean, however, that impairments in the processing of rapidly flowing phonemes are irrelevant to the development of language and reading skills. The RATP theory appears to describe some, but not all, children with specific language impairment and dyslexia. Up to 50% of children who used Fast ForWord did not demonstrate the predicted temporal processing problems (McArthur & Bishop, 2004), but a significant percentage of children do. The extent to which these findings are confounded by problems with attention, motivation, fatigue, and other cognitive deficits is only now beginning to be explored systematically (Bishop, 2007; McArthur et al., 2009). More focused screening for temporal processing dysfunction may lead to the recommendation of this intervention for children who are more likely to benefit from it.

Wibel, Nicol, and Kraus (2005) found that some conditions, such as listening in noise, can be improved with training, but that different aspects of speech onset and tone

need further investigation. Zatorre and Gandour (2008) report studies showing that it is possible to train listeners to perceive a broader range of sounds in a relatively short time. Studies have not identified one particularly effective intervention compared to other interventions. This may be in part because the taxonomy of auditory processing disorders is not yet sufficiently differentiated to be able to assign individuals to specific interventions to address specific problems.

Given the lack of clinically available evidence-based interventions, most persons diagnosed with an APD are given recommendations for modifying the environment through accommodations. Another role the clinician can play is educating those who interact with patients with APD about the specific needs of individuals who have dysfunctions in one or more of auditory processing areas. Accommodation recommendations have strong face validity, but lack research to support their validity and efficacy. Even accommodations that would appear to be an easy focus of research, such as the use of local amplification systems in classrooms to improve signal-to-noise ratios, have not resulted in research-based implementation strategies. This is in part due to a lack of system standardization and the complex acoustic interactions in each specific listening environment. In situations where the use of personal amplification devices by non-hearing-impaired students are employed, contradictory findings appear to be based on the widely varying characteristics of the amplification systems in use (Johnson, 2000).

Education about the need for accommodations has strong face validity. An example is asking parents, teachers, and others to speak slowly, clearly and not become frustrated if the listener asks for clarifications more frequently than expected. Studies are lacking about the extent to which such awareness of the condition, whether modifications are implemented, and, if so, the extent to which they provide measurable benefit to the person with an APD.

CONCLUSIONS

Auditory processing is a complex process that involves the transformation of the mechanical processes of the outer and middle ear into meaningful sound, including speech, and the transmission of an electrical signal through a series of relay stations in the central auditory pathways. Research from the last 50 years has shown increasingly specific roles played by each stage of the process and the integration of the various component processes in both classical and nonclassical pathways and loops. Medical research continues to document various mechanical, metabolic, infectious, and pharmacological changes that affect the efficiency of auditory processing and therefore the development and maturation of auditory attention, sensory integration via feedback loops with the cerebellum, and higher order language-based processes including reading and receptive and expressive language.

Auditory processing and associated dysfunctions are not one diagnostic entity. The concept of auditory processing disorder as a unitary diagnosis should be revised to reflect current knowledge about the distinct components and subcomponents of auditory processing and their relationship to neuroanatomic networks. Seven areas of auditory processing are currently recognized (ASHA, 2005): (a) sound localization, (b) lateralization, (c) discrimination, (d) pattern recognition, (e) temporal aspects of audition, (f) performance in competing acoustic signals (dichotic listening), and (g) performance in degraded acoustic conditions (e.g., speech in noise). Each of these areas can be measured, and most have clinical evaluation tools in use. The others have evaluation tools under development for clinical applications. Understanding of these seven processes continues to be refined so

that each process and its behavioral manifestations can be expected to continue to be more clearly delineated for future study. Recent research is moving beyond top-down versus bottom-up arguments as more refined assessment tools allow better data acquisition and integration with developmental and acquired conditions in children and adolescents. Among the most recent findings are a demonstration of how impairments in processing speech in the presence of noise contributes to significant difficulties decoding of both spoken and written language (Ziegler et al., 2009), and that more generalized patterns of auditory dysfunction are found in up to half of children diagnosed with a specific language impairment or reading disability (McArthur et al., 2009).

Neuropsychology has a role to play in both clinical and research aspects of auditory processing. Neuropsychology is the only profession that routinely evaluates all affected cognitive domains and is involved in the differential diagnosis of ADHD, auditory attention, auditory memory, sequencing skills, listening skills, and higher order language processes, including reading. A screening battery, SCAN-3 (Keith 2009a, 2009b) based in auditory science, is available for nonaudiologists to screen children and adolescents in the areas of temporal processing, listening in degraded conditions, and listening with competing acoustic signals. Routine screening of auditory processes enables neuropsychologists to serve their patients by adding more precision to the differential diagnostic and referral processes, and by educating those who interact with affected children and adolescents about the role of audition in learning and social interactions.

Much work remains to be done before the full range of brain-behavior interactions are understood for central auditory processes. Neuropsychology's clinical and research experience with a broad range of assessment batteries and differential diagnosis of a wide variety of child and adolescent developmental and acquired conditions can assist in streamlining the research process as well as assist in ascertaining which interventions work best for which aspects of auditory processing dysfunctions.

This review has provided an introductory overview of the central auditory pathways and processes, normal developmental trajectories, and sources of developmental and acquired dysfunctions. It has provided an introduction to a broad research base on how central auditory processes are measured and relate to neuropsychological functions of attention and language. This review is by no means exhaustive. It is meant to provide the basis for further discussion and inquiry in a field that has not heretofore been the systematic focus of research or clinical training in child and adolescent neuropsychology.

Suggested reading for becoming acquainted with the foundational material in this article includes: Musiek and Baran (2007) for auditory pathways and functions; Musiek and Chermak (2007) for neuroscience and diagnosis of auditory processes; Hall (2007) for electrophysiological measures; Chermak and Musiek (2007) for interventions for auditory processing disorders; and Lalwani and Grundfast (1998) for medical background of auditory development and dysfunctions.

Original manuscript received September 12, 2009

Revised manuscript accepted March 16, 2010

First published online September 29, 2010

REFERENCES

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: American Psychiatric Association.

- American Speech and Hearing Association (ASHA). (2004). Working group on auditory integration therapies. *Central auditory processing: Current status of research and implications for research and clinical practice*. Technical Report.
- American Speech and Hearing Association (ASHA). (2005). Working group on auditory processing disorders. *(Central) auditory processing disorders*. Technical report.
- Asbjornsen, A., Obrzut, J., Boliek, C., Myking, E., Holmefjord, A., Reisaeter, S., et al. (2005). Impaired auditory attention skills following middle-ear infections. *Child Neuropsychology*, *11*(2), 121–133.
- Berard, G. (1993). *Hearing equals behavior*. New Canaan, CT: Keats Publishing.
- Bishop, D. V. M. (2007). Using mismatch negativity to study central auditory processing in developmental language and literacy impairments: Where are we, and where should we be going? *Psychological Bulletin*, *133*(4), 651–672.
- Bocca, E., Calearo, C., Cassinari, V., & Migliavacca, F. (1955). Testing “cortical” hearing in temporal lobe tumors. *Acta Otolaryngologica*, *32*, 289–304.
- Cameron, S., Brown, D., Keith, R., Martin, J., Watson, C., & Dillon, H. (2009). Development of the North American listening in spatialized noise-sentences test (NA LiSN-S): Sentence equivalence, normative data, and test-retest reliability studies. *Journal of the American Academy of Audiology*, *20*(2), 128–146.
- Chermak, G. D. (2007). Differential diagnosis of (central) auditory processing disorder and attention deficit hyperactivity disorder. In F. E. Musiek & G. D. Chermak (Eds.), *Handbook of (central) auditory processing disorder: Auditory Neuroscience and Diagnosis* (Vol. 1; pp. 365–394). San Diego, CA: Plural Publishing.
- Chermak, G. D., Hall, J. W., & Musiek, F. E. (1999). Differential diagnosis and management of central auditory processing disorder and attention deficit hyperactivity disorder. *Journal of the American Academy of Audiology*, *10*, 289–303.
- Chermak, G. D., & Musiek, F. E. (Eds.). (2007). *Handbook of (central) auditory processing disorder: Comprehensive intervention* (Vol. 2). San Diego, CA: Plural Publishing.
- Chermak, G. D., Somers, E., & Seibel, J. A. (1998). Behavioral signs of central auditory processing disorder and attention deficit hyperactivity disorder. *Journal of the American Academy of Audiology*, *9*, 78–84.
- Church, M. W., Parent-Jenkins, L., Rozzelle, A. A., Eldis, F. E., & Kazzi, S. N. J. (2007). Auditory brainstem response abnormalities and hearing loss in children with craniosynostosis. *Pediatrics*, *119*, e1351–e1360.
- Gacek, R. R. (1972). Neuroanatomy of the auditory system. In J. V. Tobias (Ed.), *Foundations of modern auditory theory* (Vol 2, pp. 239–263). New York: Academic Press.
- Gage, N. M., Siebel, B., Callen, M., & Roberts, T. P. (2003). Cortical sound processing in children with autism disorder: An MEG investigation. *Neuroreport*, *14*(16), 2047–2051.
- Gillam, R. B., Loeb, D. F., Hoffman, L. M., Bohman, T., Champlin, C. A., Thibodeau, L., et al. (2008). The efficacy of Fast ForWord Language intervention in school-age children with language impairment: A randomized controlled trial. *Journal of Speech, Language, and Hearing Research*, *51*, 97–119.
- Gravel, J. S., Roberts, J. E., Roush, J., Grose, J., Besing, J., Burchinal, M., et al. (2006). Early otitis media with effusion, hearing loss, and auditory processes at school age. *Ear and Hearing*, *27*(4), 353–368.
- Greenberg, L. M. (2007). *The Test of Variables of Attention* [Computer software]. Los Alamitos, CA: The TOVA Company.
- Hall, J. W., III. (2007). *New handbook of auditory evoked responses*. New York: Pearson Education, Inc.
- Hattiangadi, N., Pillion, J. P., Slomine, B., Christensen, J., Trovato, M. K., & Speedie, L. J. (2005). Characteristics of auditory agnosia in a child with severe traumatic brain injury: A case report. *Brain and Language*, *92*, 12–25.
- Henriques-Filho, P. S., & Pratesi, R. (2006). Abnormalities in auditory evoked potentials of 75 patients with Arnold-Chiari malformations types I and II. *Arquivos de Neuro-psiquiatria*, *64*(3-A), 619–623.

- Jamieson, D. G., Kranjc, G., Yu, K., & Hodgetts, W. E. (2004). Speech intelligibility of young school-aged children in the presence of real-life classroom noise. *Journal of the American Academy of Audiology, 15*(7), 508–517.
- Johnson, C. E. (2000). Children's phoneme identification in reverberation and noise. *Journal of Speech, Language, and Hearing Research, 43*, 144–157.
- Kaplan, H., Gladstone, V. S., & Katz, J. (1984). *Site of lesion testing, Audiometric interpretation* (Vol. 2). Baltimore, MD: University Park Press.
- Keith, R. W. (2009a). *SCAN-3 for adolescents and adults: Tests for auditory processing disorders*. San Antonio, TX: Pearson.
- Keith, R. W. (2009b). *SCAN-3 for children: Tests for auditory processing disorders*. San Antonio, TX: Pearson.
- Keller, W. D. (1992). Auditory processing disorder or attention-deficit disorder? In J. Katz, N. Stecker, & D. Henderson (Eds.), *Central auditory processing: A transdisciplinary view* (pp. 107–114). St. Louis, MO: Mosby Year Book.
- Kotz, S. A., Schwartz, M., & Schmidt-Kassow, M. (2009). Non-motor basal ganglia functions: A review and proposal for a model of sensory predictability in auditory language perception. *Cortex, 45*(8), 982–990.
- Lalwani, A. K., & Grundfast, K. M. (Eds.). (1998). *Pediatric otology and neurotology*. Philadelphia, PA: Lippincott-Raven.
- Limperopoulous, C., Robertson, R. L., Sullivan, N. R., Bassan, H., & du Plessis, A. J. (2009). Cerebellar injury in term infants: Clinical characteristics, magnetic resonance imaging findings, and outcome. *Pediatric Neurology, 41*(1), 1–8.
- Maruthy, S., & Mannarukrishnaiah, J. (2008). Effect of early onset otitis media on brainstem and cortical auditory processing [Electronic version]. *Behavioral and Brain Functions, 4*, 17. doi:10.1186/1744-9081-4-17 Retrieved May 9, 2009 from <http://www.behavioralandbrainfunctions.com/content/4/1/17>
- McArthur, G., Atkinson, C., & Ellis, D. (2009). Atypical brain responses to sounds in children with specific language and reading impairments. *Developmental Science, 12*(5), 768–783.
- McArthur, G. M., & Bishop, D. V. M. (2004). Which people with specific language impairment have auditory processing deficits? *Cognitive Neuropsychology, 21*, 79–94.
- Merzenich, M. M., Jenkins, W. M., Johnson, P., Scheiner, C., Miller, S. L., & Tallal, P. (1996). Temporal processing deficits of language-learning impaired children ameliorated by training. *Science, 271*, 77–81.
- Moore, D. R., Halliday, L. F., & Amitay, S. (2009). Use of auditory learning to manage listening problems in children. *Philosophical Transactions of the Royal Society B (Biological Sciences), 364*, 409–420.
- Musiek, F. E., & Baran, J. A. (2007). *The auditory system: Anatomy, physiology and clinical correlates*. Boston, MA: Pearson Education, Inc.
- Musiek, F. E., & Chermak, G. D. (Eds.). (2007). *Handbook of (Central) Auditory Processing Disorder: Vol. 1. Auditory Neuroscience and Diagnosis*. San Diego, CA: Plural Publishing.
- Myklebust, H. (1954). *Auditory disorders in children*. New York: Grune & Stratton.
- Nunez-Batalla, F., Carro-Fernandez, P., Antuna-Leon, M. E., & Gonzalez-Trelles, T. (2008). Incidence of hypoacusia secondary to hyperbilirubinaemia in a universal neonatal auditory screening programme based on otoacoustic emissions and evoked auditory potentials. *Acta Otorrinolaringologica Espanola, 59*(3), 108–113.
- Parvisi, J., & Damasio, A. (2001). Consciousness and the brainstem. *Cognition, 79*, 135–139.
- Pastor, M. A., Vidaurre, C., Fernandez-Seara, M. A., Villanueva, A., & Friston, K. J. (2008). Frequency-specific coupling in the cortico-cerebellar auditory system. *Journal of Neurophysiology, 100*(4), 1699–1705.
- Picard, M., & Bradley, J. S. (2001). Revisiting speech interference in classrooms. *Audiology, 40*, 221–244.

- Plaza, M., Rigoard, M. T., Chevrie-Muller, C., Cohen, H., & Picard, A. (2001). Short-term memory impairment and unilateral dichotic listening extinction in a child with Landau-Kleffner syndrome: Auditory or phonological disorder? *Brain and Cognition*, *46*(1–2), 235–240.
- Riccio, C. A., Cohen, M. J., Garrison, T., & Smith, B. (2005). Auditory processing measures: Correlation with neuropsychological measures of attention, memory, and behavior. *Child Neuropsychology*, *11*, 363–372.
- Sanford, J. A., & Turner, A. (1989). Integrated Variables of Attention [Computer software]. Richmond, VA: Braintrain Inc.
- Sanford, J. A., & Turner, A. (2005). Integrated Variables of Attention [Computer software]. Richmond, VA: Braintrain Inc.
- Sens, P. M., & de Almeida, C. I. R. (2007). Participation of the cerebellum in auditory processing. *Revista Brasileira Otorrinolaringologia*, *73*(3), 266–270.
- Shapiro, S. M., & Nakamura, H. (2001). Bilirubin and the auditory system. *Journal of Perinatology*, *21*, S52–S55.
- Shield, B., & Dockrell, J. E. (2004). External and internal noise surveys of London primary schools. *Journal of the Acoustical Society of America*, *115*, 730–738.
- Sininger, Y. S., & Abdala, C. (1998). Physiologic assessment of hearing. In A. K. Lawani & K. M. Grundfast (Eds.), *Pediatric otology and neurotology* (pp. 127–154). Philadelphia, PA: Lippincott-Raven Publishers.
- Stollman, M. H. P., van Velzen, E. C. W., Simkens, H. M. F., Snik, A. F. M., & van den Broek, P. (2004). Development of auditory processing in 6–12-year-old children: A longitudinal study. *International Journal of Audiology*, *43*, 34–44.
- Sussman, E., & Steinscheinder, M. (2009). Attention effects on auditory scene analysis in children. *Neuropsychologia*, *47*, 771–785.
- Tallal, P. (1980). Auditory temporal perception, phonics, and reading disabilities in children. *Brain and Language*, *9*, 182–198.
- Tallal, P., & Piercy, M. (1973a). Defects of non-verbal auditory perception in children with developmental dysphasia. *Nature*, *241*, 468–469.
- Tallal, P., & Piercy, M. (1973b). Developmental aphasia: Impaired rate of non-verbal processing as a function of sensory modality. *Neuropsychology*, *11*, 389–398.
- Tillery, K. L., Katz, J., & Keller, W. D. (2000). Effects of Methylphenidate (Ritalin) on auditory performance in children with attention and auditory processing disorders. *Journal of Speech, Language, and Hearing Research*, *43*, 293–301.
- Uppenkamp, S., Johnsrude, I. S., Norris, D., Marslen-Wilson, W., & Patterson, R. D. (2006). Locating the initial stages of speech-sound processing in human temporal cortex. *Neuroimage*, *31*(3), 1284–1296.
- Werner, L. A. (2007). Issues in human auditory development. *Journal Communications Disorders*, *40*(4), 275–283.
- Wible, B., Nicol, T., & Kraus, N. (2005). Correlation between brainstem and cortical auditory processes in normal and language-impaired children. *Brain*, *128*(2), 417–423.
- Zatorre, R. J., & Gandour, J. T. (2008). Neural specializations for speech and pitch: Moving beyond the dichotomies. *Philosophical Transactions of the Royal Society B Biological Sciences*, *363*(1493), 1087–1104.
- Ziegler, J., Pech-Georgel, C., George, F., & Lorenzi, C. (2009). Speech-perception-in-noise deficits in dyslexia. *Developmental Science*, *12*(5), 732–745.
- Zumach, A., Gerrits, E., Chenault, M. N., & Anteunis, L. J. (2009). Otitis media and speech-in-noise recognition in school-aged children. *Audiology and Neurootology*, *14*(2), 121–129.