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All Audiologists Encounter Patients with Auditory Processing Disorders (APD). Show us an audiologist who has never evaluated a patient with APD, and we'll show you an audiologist who relies on the audiogram as a complete hearing test. Over-reliance on the audiogram is nothing new. More than 50 years ago, Dr. Helmer R. Myklebust, in highlighting the importance of assessing the central auditory system, noted that "the diagnostician of auditory problems in children has traditionally emphasized peripheral damage" (Myklebust, 1954, p. 54). However, with advances in auditory neuroscience there is growing awareness that "we hear with our brain, not with our ears." The practical implication of this statement is clear... hearing assessment is not complete until speech perception is evaluated under difficult, yet commonly encountered, listening conditions. In addition, evaluation of the auditory periphery is not enough. If the act of "hearing" extends from the ears to the brain, the clinician charged with evaluating hearing should evaluate the entire system, as well.

In recent years, several groups of audiologists have re-defined APD. Reporting the findings of a panel of experts at a conference held in Dallas in 2000, Jerger & Musiek (2000) stated that APD "is broadly defined as a deficit in the processing of information that is specific to the auditory modality." A few years later, a task force organized by the American Speech-Language-Hearing Association (ASHA), focusing mostly on hearing processes within the brain, broadly defined (central) auditory processing as "the efficiency and effectiveness by which the central nervous system utilizes auditory information" (Technical Report of ASHA Working Group on (Central) Auditory Processing Disorders, ASHA 2005).

Examples of auditory processes typically cited by these and other groups of experts include: auditory discrimination, auditory pattern recognition, temporal aspects of audition, temporal ordering, auditory performance in competing acoustic signals, and auditory performance with degraded acoustic signals. Experts also emphasize that a variety of listener-related, and non-auditory, variables may influence auditory processing and its clinical measurement, among them cognitive factors (e.g., intelligence level, memory, and processing speed), attention, motivation, motor skills, and linguistic abilities. For a thorough review of non-auditory factors in hearing, the reader is referred to a recent special issue of the *Journal of the American Academy of Audiology* (volume 18, number 7, July/August 2007) devoted entirely to cognition in audiology.

Conspicuously absent from these definitions of auditory processing are constraints regarding specific patient populations, e.g., only children. In addition, the definition of

APD suggested by Jerger & Musiek (2000) clearly indicates that auditory processing disorders may have anatomic origins anywhere within the auditory system, from the cochlea to the cortex. However, to differentiate APD from deficits attributed solely to the peripheral auditory system, and to decrease the confusion that ensued from the use of both labels CAPD and APD, ASHA (2005) used the term *(Central) Auditory Processing Disorders ((C)APD)*. The use of this term was intended to emphasize that APD is fundamentally a deficit in the central auditory pathways, but that it can occur secondary to peripheral pathology or other auditory disorders as a result of neuroplastic changes. Thus, the two terms can be used interchangeably.

The critical point is that, if auditory processing disorders can occur secondary to auditory dysfunction anywhere within the auditory system, and may be found in both children and adults, then any audiologist evaluating any patient should remain always vigilant for the possibility of deficits in auditory processing of peripheral and central origin.

APD IS FOR REAL

Beginning especially in the 1990s, basic neuroscience research has generated progressively more and more evidence in support of the nature and anatomic origins of APD. Hundreds of published papers on neuro-diagnostic techniques, such as functional magnetic resonance imaging (fMRI) and cortical auditory evoked responses have contributed to the description of the neuro-anatomic and neuro-physiologic underpinnings of APD. Audiologists can document the rather voluminous auditory neuroscience literature by conducting via the Internet a Medline search (www.nlm.nih.gov) with key words such "auditory," "auditory processing," "fMRI" (for functional magnetic resonance imaging), "auditory evoked responses," and many others. Support for the legitimacy of APD is also evident by the allocation of resources of professional organizations for task forces charged with the development of technical statements (ASHA, 2005) and clinical guidelines for assessment and management of APD (e.g., the American Academy of Audiology Task Force on APD).

RISK FACTORS FOR APD IN PEDIATRIC AND ADULT PATIENT POPULATIONS

A common question that arises is that of which patients are at risk for APD and should be considered for APD assessment. Risk factors for APD in children are highly varied. There is general agreement that children with poor academic performance are at risk for APD, particularly if other educational and sensory explanations have been ruled out. Certain disorders that often co-exist with APD, such as specific

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language impairment (SLI), learning disabilities, reading impairment, and attention deficit hyperactivity disorder (ADHD), increase the likelihood of a deficit in auditory processing, especially when appropriate management of the disorder fails to produce adequate improvement in academic performance. Children are also may be also at risk for APD if they have a history of neonatal factors associated with neurological dysfunction (e.g., asphyxia), later insults to the central nervous system (e.g., head injury), clinical signs and symptoms of neurological dysfunction (e.g., seizure disorders), and chronic otitis media.

APD also is regularly encountered quite often clinically in adult patient populations by audiologists who include screening or diagnostic measures in their test batteries. The increased likelihood of deficits in auditory processing in aging adults has long been appreciated (e.g., Bellis & Wilber, 2001; Jerger, 1973). At the very least, additional audiologic assessment with sensitive measures of auditory processing are indicated when patients express concerns about their hearing despite normal hearing sensitivity, when the patient's hearing complaints are greater than those expected for the pure-tone audiogram, when benefit from amplification is less than anticipated, and when clinical observations and/or case history suggest the possibility of central nervous system disease or dysfunction (e.g., dementia, other cognitive deficits, or head injury). In short, any patient with hearing or listening complaints should be considered at risk for APD until proven otherwise. Understandably, modern-day audiologists who are under growing pressure to see more patients in less time, and with diminishing reimbursement, are tempted to reject such advice as not clinically feasible. However, as audiologists, we alone among educational and health care professionals are responsible for the assessment of hearing, and for management of hearing problems. We haven't evaluated hearing until we've described auditory performance at the highest levels of the nervous system, and we can't expect patient satisfaction or good management outcomes if we haven't identified and addressed all of the patient's auditory complaints.

ASSESSMENT OF APD IN THE AVERAGE TYPICAL AUDIOLOGY CLINIC

The optimal audiologic test battery consists of clinically feasible and proven procedures with sensitivity to peripheral and central auditory dysfunction. With adherence to the definitions of APD cited above (ASHA, 2005; Jerger & Musiek, 2000), the audiologist should attempt to measure auditory processing function in the cochlea and also within neural structures and centers from the 8th cranial nerve through the auditory cortex. Practically speaking, however, the audiologic test battery should be streamlined to minimize the time required to complete the assessment while maximizing the

information obtained from the assessment. One overall principle in the application of a test battery can help to achieve these apparently conflicting goals. Each procedure selected for the initial test battery administered to a new patient must add value to the audiologic diagnosis and management of the patient.

How can sensitive measures of auditory processing be included in the test battery with the time constraints typical of clinical audiology today? Two general test strategies help to resolve this clinical dilemma. One is to eliminate procedures that contribute little or nothing to the diagnosis of auditory dysfunction for a given patient. For example, time-consuming measurement of bone-conduction hearing thresholds contributes nothing to the diagnosis of a patient with no history of middle-ear disorder, and no clinical evidence of middle ear dysfunction on prior test procedures, e.g., normal tympanograms, acoustic reflexes present, and normal OAEs in the low to mid-frequency region. Measurement of aural immittance and OAEs can be performed in far less time than bone-conduction pure-tone hearing thresholds. Support personnel can even carry out these objective electro-acoustic tests. Moreover, by starting the test process with immittance and OAE measures, the likelihood of detecting middle-ear and cochlear auditory dysfunction is considerably higher, and the chances of invalid findings due to malingering and other non-audiologic factors (e.g., attention, cognitive decline, motivation) lower, than for traditional bone-conduction pure-tone measures. The minutes saved with this strategy can be invested in procedures with documented sensitivity to more complex and higher level auditory processing, such as a dichotic procedure and/or a test of auditory performance in background noise. However, it should be remembered that beginning with these types of procedures may be contraindicated for some populations (e.g., children with aversions to placing items in the ear, etc.). Therefore, the best strategy is always an individualized testing approach guided by the patient's presenting complaints.

Published descriptions of test batteries for APD assessment in children are readily available (e.g., ASHA, 2005; Bellis, 2003; Hall, 2000; Hall, 2007; Hall & Mueller, 1997; Hall, 2000; Hall, 2007; Bellis, 2005; ASHA, 2005; Musiek & Chermak, 2007). Assessment of APD in adult patients, particularly those with sensory hearing loss, requires the use of procedures that are least influenced by peripheral auditory dysfunction. Examples include the Hearing in Noise Test (HINT) and synthetic sentence identification test with ipsilateral competing message (SSI-ICM) for the assessment of auditory performance in background noise, the Gaps-in-Noise (GIN) test for temporal auditory processing, the Dichotic Digits and Staggered Spondaic Word (SSW) tests for binaural integration, and Frequency or Duration Pattern Tests for temporal patterning

and detection of high-level deficits in auditory processing. With careful selection of the procedures to be included in the test battery, based on the patient's history, hearing and listening complaints, and by choosing other procedures based on the findings of the initial audiologic tests, it is possible to evaluate simple and complex auditory processing abilities within a reasonable, clinically acceptable time period (e.g., less than 45 minutes).

INTERVENTION OPTIONS FOR APD IN PEDIATRIC AND ADULT PATIENT POPULATIONS


Identification and diagnosis of APD is, of course, a fruitless exercise if the information does not contribute to management and, ultimately, improved communicative outcome. Decisions on which strategies are required for effective management of APD are largely dependent on an accurate and complete assessment and diagnosis. A detailed discussion of strategies and techniques used in the management of APD in children is far beyond the scope of this paper. As noted above in reference to publications describing diagnostic test procedures for APD assessment in children, plenty of handy resources on the topic are easily accessible to the clinical audiologist. It is reasonable to question how diagnostic information on auditory processing contributes to management decisions for adults with normal hearing sensitivity or with a sensorineural hearing loss.

In adult patient populations, the pattern of deficits in auditory processing emerging from the diagnostic assessment will influence three different, yet related, components of management: 1) counseling and patient/family information, 2) selection of hearing aids and assistive devices, and 3) other forms of audiologic rehabilitation.

APD test findings most assuredly will affect patient and

family counseling. Take, for example, an elderly woman brought to the clinic for a hearing assessment by a caring son or daughter who is very concerned about the mother's hearing difficulty, particularly in noisy settings. The audiogram shows a very mild high-frequency sensory hearing loss, actually not much considering the patient's age, and fair word recognition scores. Lacking further diagnostic test findings, the audiologist would be tempted to simply to reassure the patient and her family that her hearing is really quite good, and a hearing aid is not necessary. However, what if speech perception in background noise were assessed and showed a marked deficit in auditory performance? Further, what if central auditory tests indicate deficits in binaural integration, temporal patterning, interhemispheric transfer of auditory information, or other dysfunction? The audiologist would take a very different approach with patient and family counseling and instruction and the audiologist would offer additional forms of intervention (e.g., FM technology, additional environmental modifications, and or perhaps a computer-based auditory training program). Findings of concomitant central auditory dysfunction in adults with documented sensory hearing loss prompt the audiologist to look into features of hearing aids that would not otherwise be considered or even warranted. Finally, some patterns of findings for APD measures for adult patients (e.g., markedly asymmetric and/or abnormal performance) may suggest the need for medical referral and diagnostic follow-up for possible neurological disorders. Many other examples could be cited to illustrate the impact of comprehensive diagnostic assessment of APD in adult patients on management strategies and overall outcome.

CONCLUSIONS

The possibility of APD should always be entertained in the audiologic assessment of children and adults. We encourage audiologists to include in clinical protocols indicators for assessment of auditory processing beyond the simple audiogram and measures of speech threshold and recognition, and in the audiologic test battery sensitive procedures for the identification and diagnosis of auditory processing deficits that will influence real-world communicative function. 

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