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Auditory processing disorder (APD)

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A recent submission to *Ear and Hearing* on early diagnosis of APD¹ ended up on my desk after three expert reviewers and another member of our Editorial Board provided a highly detailed critique of the paper. What most caught my attention was that 2 of 3 reviewers questioned the premise of whether an earlier diagnosis of APD will ultimately prove useful since the status of APD is so controversial (Reviewer 1) or difficult to verify (Reviewer 2). I know of no other area of audiology or neuroscience where such a long-standing intellectual, theoretical and practical impasse exists. In contrast to the ‘consensus on CAPD’ (ASHA 2005; AAA 2010) cited by the authors of the above submission, it has been increasingly argued recently that most if not all cases of childhood ‘APD’ are either better characterized by more commonly recognized learning disorders, especially language disorder (de Wit et al. 2018), or by specific difficulties, for example hearing speech in noise (DeBonis 2015) or spatial hearing (Cameron et al. 2014), that are firmly grounded in psychoacoustics. So the bigger question I began to ask is whether and how *Ear and Hearing* and other academic journals should be handling work submitted on ‘APD’. After consultation with and encouragement from our Editorial Board, I thus prepared this article that is endorsed by the whole Board and that will become journal editorial policy, effective immediately for new submissions. Briefly, the new policy is that “articles that either implicitly or explicitly assume APD is a single diagnostic characteristic of the auditory system likely will not be considered for publication”.

History, ancient and modern

It isn’t clear when the term APD was first used. Robert Keith, recently retired from the University of Cincinnati, has been in the field longer than most and he first heard it in 1973, used by Jack Willeford at an ASHA meeting. Long before that, Myklebust (1954) had observed that some children with apparently normal hearing sensitivity “cannot listen ... they cannot direct their attention selectively to an unexpected sound”. James Jerger has a very readable chapter on the history of APD in a book that was influential on my thinking, ‘*Controversies in Central Auditory Processing Disorder*’ (Cacace & McFarland 2009). Numerous international audiology societies have published their own guidelines on APD, but I would like to cite one part of the British Society of Audiology guidelines (BSA 2011; 2018; Moore et al. 2013) that may help define and limit what it is I object to. The BSA proposed three types of APD: Secondary APD, Acquired APD and Developmental APD. Secondary APD was so-called because of its association with a known genetic cause or

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¹The terms APD and Central (C)APD are used interchangeably in the literature

peripheral hearing impairment. Acquired APD is associated with a known neurological event such as trauma, stroke or infection. But it is Developmental APD, normal audiometry with no other known etiology and presumably present from birth, to which this article is primarily directed. I do not think it is controversial that people with hearing loss or identified brain lesions may have (additional) hearing deficits originating in the central auditory system. However, the notion that children with listening difficulties² have a disorder of central auditory function that may be diagnosed with some arbitrary combination of the commonly used clinical tests for APD (Emanuel et al. 2011) is unjustified in the view of many scientists and clinicians, including the Editorial Board of this Journal. Such a diagnosis could even be harmful if it prevents children receiving evidence-based and soundly reasoned diagnosis and treatment.

Theory

Several scholarly papers have questioned the construct of APD. For example, Cacace and McFarland (2009) have long argued that, to be a useful construct, APD should produce poor performance that is relatively specific to tasks involving auditory stimuli. They have demonstrated that aspects of Acquired APD may satisfy this criterion, but that is not the case for Developmental APD. Based on criteria firmly established in medical science, Vermiglio (2014) contrasted the case for ‘APD’ with that of ‘hearing speech in noise’ as ‘clinical entities’ (disease categories), concluding that whereas hearing speech in noise *does* meet the criteria, APD *does not*, despite the recent inclusion of APD in the International Classification of Diseases (ICD-10). DeBonis (2017) has recently presented an evaluation of the American Academy of Audiology recommendations on APD (AAA 2010) in terms of their effectiveness as credible and useful clinical practice guidelines. He found that they did not meet many of those key requirements and therefore failed to provide meaningful guidance for clinicians.

One key theoretical issue advocated by proponents of APD is that the major current tests derive legitimacy from their sensitivity and specificity. However, there is no recognized ‘gold standard’ for APD (Dillon et al. 2012; Vermiglio 2016). Comparison with such a gold standard would be the normal route for determining clinical sensitivity and specificity. Instead, the argument for sensitivity and specificity (AAA 2010; pp.15–16) appears to be based on the following logic. Adults with brain lesions known to involve the central auditory system often perform poorly on the most commonly used behavioral tests for APD. Some children *without* suspected or confirmed brain lesions also perform poorly on those same tests. Since the tests provide good sensitivity and specificity for central auditory lesions in adults, so the argument goes, a similar pattern of test results in children confirms a biological entity, APD, in those children, with assumed similar test sensitivity and specificity. The primary flaw in this argument is a simple logical one, that if A(lesions) is associated with B(poor test results), and B is associated with C(some children), then A must be associated with C. Although we might accept that impaired ‘auditory processing’ is

²‘Listening difficulties’ is used here and in the literature to summarize any self-, caregiver- or professional-reported problems of hearing or listening, despite normal audiometry. It does not imply any mechanism.

associated with brain lesions in adults, there are many factors other than brain lesions that produce impaired auditory perception in both children and adults, for example, inattention.

Another erroneous belief that appears to underpin much of the foundation of APD is that impaired auditory performance in someone with a 'normal' pure tone audiogram must be due to disordered processing in the central auditory system. Although this may be the case in some instances, there are at least two other well-recognized sources of such disordered processing. One is the cochlea, where we know that impaired hair cell function and resulting changes in cochlear compression influences spectral and temporal tuning somewhat independently of pure tone sensitivity (Oxenham & Bacon 2003). The second is so-called 'top-down' influences on hearing, for example attention, memory, emotion and learning as elaborated below.

A final issue concerns the words auditory, processing, and disorder. As argued above, APD does not satisfy the criteria of a 'disorder' and, below, the problems it attempts to describe are not confined to the central 'auditory' system. Neuroscientists and psychoacousticians have also expressed strong reservations about the word 'processing' (e.g. Brian Moore, Novartis Foundation, London, 2009). It has been used to imply central neural function, but major 'processing', including transduction of sound, also occurs in the ear. It is thus my contention that 'processing' is everything – and therefore nothing – as a description of auditory function and pathology.

Evidence

Based on my own experience of reading and reviewing hundreds of submissions and publications on APD over a 15 year period of research in this area, I offer the following observations. Some papers are based on original experimental data and have been peer-reviewed, but publications on APD often state a series of opinions or clinical anecdotes that lack rigorous review and have no solid foundation in theory or evidence. Many of these publications are lavishly referenced, but the references are often to inappropriate, obscure or unreviewed sources. Among those that have been reviewed, the premise for the study usually assumes, and review sometimes insists on adherence to the ASHA and AAA position paper guidelines that identify a child with APD based on performance 2 or 3 standard deviations below the norm for one or more commonly used clinical tests (DeBonis 2017; Emanuel et al. 2011). For the most part, these norms do not actually exist and, where they do, they may only be gleaned from sources unavailable for verification. There are some exceptions (e.g. SCAN-3 battery; Keith 2009), but performance on the tests used is, without exception, dependent on a complex set of skills of which 'processing' within the classic central auditory system (auditory nerve to auditory cortex) is just one. Rather than carefully describing the data and pointing to the myriad interpretational issues and limitations of the study, publications in this area typically advocate their results as supporting a clinical diagnosis for children whose symptoms lack any clear connection with the tests they have been given, or any well-evidenced form of intervention, other than simple advice on listening strategies or other acoustic enhancements. A paper by Wilson and Arnott (2013) examined performance on commonly used APD tests of 150 children with normal peripheral hearing using nine sets of diagnostic criteria drawn from published sources. Based on

published guidelines, they found rates of potential APD ranging from 7% to 93% of the study children. The authors supported “calls to abandon the use of (C)APD as a global label”.

Clinical prevalence, presentation, and evaluation

There is no doubt that many children have listening difficulties and that many of those who present at audiology or other pediatric clinics have ‘normal’ audiograms. In a retrospective record review covering a 5 year period at Cincinnati Children’s Hospital, we identified 1,113 children who had normal audiograms and had undergone a “central auditory processing evaluation” (Moore et al. 2018). These children almost all had other complications, most typically speech/language or attention impairments, and a variety of other well-recognized learning disorders (e.g. behavioral/emotional, cognitive delay, dyslexia). Evaluation of all children included the SCAN battery, but just 14 children would be classified “Disordered” according to the SCAN manual (Keith 2009). Eleven of those 14 had a diagnosis of either or both speech/language or attention disorders, two had academic problems, and just one child lacked an associated difficulty or an alternative explanation of their listening difficulties.

After a two-year search in an ongoing, prospective study, we have found two out of nearly a hundred extensively examined children who may have a specific, auditory-based problem. All these children had normal pure-tone audiograms, but caregiver reported listening difficulties, assessed using the ‘ECLiPS’ questionnaire (Barry et al. 2015). Several of those who have come closest to the elusive ‘pure APD’ had sub-clinical or minimal hearing loss (e.g. high frequency pure tone thresholds of 15–25 dB HL). No physiological, central auditory system processing abnormalities have yet been observed. Testing has included click-evoked brainstem responses, frequency following responses, and 3-T magnetic resonance imaging of auditory cortex in an attempt to differentiate these children with listening difficulties from their typically developing, age-matched peers. Faced with this evidence, or lack of evidence, it cannot be said that APD does not exist. However, using the most specific and sensitive tests of central function in children selected by concerns about their ability to listen, we don’t see any clear APD. By contrast, there is no shortage among the children with listening difficulties in this sample who have poor speech-in-noise hearing, correlated with slight to moderate impairment of cognitive skills (using the NIH Cognition Toolbox; Weintraub et al., 2013).

Action

Let’s be bold! For too long mainstream auditory science has simply turned a blind eye to APD. In the meantime, thousands of children are being inappropriately or inadequately advised about the nature and management of their listening difficulties. On the clinical side, only about half the larger audiology services in US children’s hospitals offer an APD evaluation, or at least they did three years ago when a small survey of 18 services was performed (L.L. Hunter, personal communication). At Cincinnati Children’s Hospital, the number of children being tested for and diagnosed with APD has been on a steep downward spiral over the last decade (Moore et al. 2018). Why do so many services with some of the best records for audiological research and training not have an active APD clinic? I cannot

provide evidence on this, but personal enquiry about APD in those services usually results in rolling eyes and a polite suggestion we move the conversation along to things that are defined and measurable. It may be impossible to separate fully the sensory and cognitive components of hearing. And the separation becomes more difficult as stimulus and task complexity become increasingly realistic. To make a verbal or other report of what an individual hears it is necessary for a sound or its neural representation to pass through the various elements of the middle ear, cochlea, brainstem, midbrain, thalamus, auditory cortex, the many multimodal parts of the cortex with their heavy, bidirectional interconnectivity and, finally, the motor (action) centers and their effector systems that all play a critical role in even the most simple auditory-verbal tasks. How then, as recommended in APD guidelines, does an audiologist working in a busy clinic use their “professional skills” to ensure that a child with listening and other behavior issues is attentive, motivated and intellectually gifted enough for those multitudinous, multimodal systems not to influence the child’s perception of a threshold-level, complex auditory task?

Audiologists will continue to see children with listening difficulties but with normal audiograms. There is active discussion and some implementation of alternate clinical models (BSA 2018; Cameron et al. 2015). These typically begin with a well-validated questionnaire, to establish or confirm the nature of the reported difficulties, and a thorough audiological exam. They also incorporate speech-in-noise testing, following rigorous standardized procedures. They may include one or more standardized tests of attention, memory or language to assist management advice or onward referral. Optimally, there should be interdisciplinary consultation, but this is often unfeasible. The testing may be arranged hierarchically, to enable successive examination of specific hypotheses concerning the nature of the child’s problems. A scan of the contents of *Ear and Hearing* over the last couple of years suggests an assessment regimen will soon be feasible that is age appropriate, evidence led, realistic, psychoacoustically valid, standardized, efficient, intervention focused, and internationally agreed. These characteristics will be the key drivers to lead clinical management in a new and better direction. Crucially, further sensitized objective measures to detect mechanisms underlying listening difficulties in newborns and infants may lead to game-changing prevention and intervention.

I have no firm suggestion of an overarching name for the outcome of such an alternate assessment, a label for what we are assessing³. The term ‘listening difficulties’ has been gaining traction and could serve as a diagnostic billing category. However, as above, it does not imply any specific mechanism and, if used, it should remain an umbrella term until specific mechanisms are identified.

What role should journals play?

During preparation of this article, several colleagues, including members of the *Ear and Hearing* Editorial Board questioned a total censure of ‘APD’ I had initially proposed. Following much discussion, the resulting, revised policy is as follows: Papers that contribute

³Harvey Dillon (personal communication) has suggested retaining APD as an “umbrella term”, and Wayne Wilson (2018) has recently argued that APD could be viewed as a “spectrum disorder”

scientific evidence to the development of perceptual processes have always been and will continue to be welcome, but articles that either implicitly or explicitly assume APD is a single diagnostic characteristic of the auditory system likely will not be considered for publication.

More broadly, is it the role of a scholarly journal to place even a partial ban on a controversial subject? I think the answer is in the word ‘scholarly’. Definitions include ‘learning’ and a ‘fund of knowledge ... drawing on the ancients’. In science, unless we learn from reason, and respect our legacy of experiments, painstakingly documented, reviewed and curated, we are nothing.

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