

## Research Report

# Auditory processing disorder in relation to developmental disorders of language, communication and attention: a review and critique

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### Abstract

*Background:* Auditory Processing Disorder (APD) does not feature in mainstream diagnostic classifications such as the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV), but is frequently diagnosed in the United States, Australia and New Zealand, and is becoming more frequently diagnosed in the United Kingdom.

*Aims:* To familiarize readers with current controversies surrounding APD, with an emphasis on how APD might be conceptualized in relation to language and reading problems, attentional problems and autistic spectrum disorders.

*Methods & Procedures:* Different conceptual and diagnostic approaches adopted by audiologists and psychologists can lead to a confusing picture whereby the child who is regarded as having a specific learning disability by one group of experts may be given an APD diagnosis by another. While this could be indicative of co-morbidity, there are concerns that different professional groups are using different labels for the same symptoms.

*Conclusions & Implications:* APD, as currently diagnosed, is not a coherent category, but that rather than abandoning the construct, we need to develop improved methods for assessment and diagnosis, with a focus on interdisciplinary evaluation.

*Keywords:* auditory processing, children, learning difficulties, language impairment, attention deficit hyperactivity disorder (ADHD), autistic spectrum disorders (ASD).

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**What this paper adds***What is already known*

In a recent survey of British audiologists and speech and language therapists, most respondents admitted that they do not have clear knowledge of Auditory Processing Disorder (APD) or diagnostic procedures for APD. This is a confusing area, and one in which conceptualization of APD and best practice for assessment and treatment is developing.

*What this paper adds*

There is currently no evidence for APD as a coherent category. However, as auditory problems are associated with a range of conditions, rather than abandoning APD improved methods for assessment and diagnosis are required. Systems for APD sub-typing and prescribing treatment have been proposed, but these should be approached with caution. In order to avoid misdiagnosing language difficulties as auditory problems, APD may be best diagnosed using non-speech stimuli in the context of multidisciplinary assessment by both audiologists and speech and language therapists.

**Introduction**

Auditory Processing Disorder (APD) is widely diagnosed in the United States and Australasia (Cameron and Dillon 2005a, Emanuel 2002), and is receiving more attention in the United Kingdom (Hind 2006). Initially a diagnostic entity proposed by the audiological community, APD has become widely diagnosed in children with learning disabilities. Presumed to have a causal basis in subtle abnormalities in the central auditory nervous system (CANS), the primary symptom of APD is difficulty identifying or discriminating sounds despite having normal peripheral hearing. Poor ability to understand speech in noise is the most common manifestation. Despite it being routinely identified, there is a lack of agreement on how to diagnose APD, what APD is, the relationship between language and reading and APD, and even if APD actually exists at all.

This review aims to familiarize readers with the concept of APD as applied to children to explain some of the controversies surrounding it, and consider what the implications are for assessment and intervention. We shall start by summarizing what is known about central auditory pathways and their function, followed by an account of APD definition and diagnosis as conceptualized by audiologists and auditory scientists. We then move on to outline difficulties with this construct, before discussing APD in relation to common developmental disorders: attention deficit hyperactivity disorder (ADHD), autistic spectrum disorder (ASD), specific language impairment (SLI), and dyslexia. Finally, we consider future directions for clinical practice and research in APD.

**Central auditory processing**

The structure and function of the peripheral auditory system, which includes the outer, middle and inner ear, are well established (Evans 1992). Central auditory processing (CAP) is much less well understood. 'Central auditory system' refers to

structures beyond the cochlea and up to the non-primary auditory cortex. There are three subdivisions: brainstem, thalamus, and cortex (Boatman 2006).

Knowledge of CAP is based on lesion, neuroimaging, and electrocortical mapping studies in animals and humans. Phillips states that central auditory processing is:

an umbrella term for all of the operations executed on peripheral auditory inputs, and which are required for the successful and timely generation of auditory percepts, their resolution, differentiation, and identification. (Phillips 2002: 255)

Specific aspects of processing are thought to have different physiological bases.

Phillips (1995, 2002) describes a number of general principles in relation to CAP which have bearing on the concept of APD. First, the whole of the central auditory system is organized according to frequency, or is 'tonotopically' organized. Second, there are patterns of convergence and divergence within the auditory pathway and a well-developed descending auditory pathway. The existence of complex patterns of connectivity mean that it is difficult to assign a specific role in auditory analysis to any particular structure. Third, sensory representations within the CANS take a number of forms. These include tonotopic organization in the case of sound frequency and distribution of cells 'tuned' to specific response rates in the case of coding of transient temporal events. A further point is that structural organization of sensory pathways including cortical maps may vary considerably between individuals, and is subject to some degree of plasticity even in adulthood. In sum, the CANS is complex in structure, plastic in adaptability, individual in organization and diverse in function. CANS pathology need not respect functional neurological boundaries.

### **Definition and diagnosis**

Early work on 'central auditory processing disorder' was concerned with adults, some with acquired lesions of auditory pathways, who reported persistent difficulties with sound perception despite normal peripheral hearing (Hinchcliffe 1992, Kimura 1961). Subsequently, the diagnosis was extended to cover cases of children with no known pathology who had normal peripheral hearing but persistent listening difficulties (Jerger 1998). However, this extension to the developmental context has raised numerous problems, leading the American Speech–Language–Hearing Association (ASHA) to set up expert panels to define the condition. The latest report (ASHA 2005) builds upon an earlier document (ASHA 1996) and defines auditory processing, as involving the following skills:

- Sound localization and lateralization.
- Auditory discrimination.
- Auditory pattern recognition.
- Temporal aspects of audition, including temporal integration, temporal ordering and temporal masking.
- Auditory performance in competing acoustic signals.
- Auditory performance with degraded acoustic signals.

APD is defined as involving a deficit in one or more of the above. However, this definition continues to engender debate among experts in APD.

*Is APD a valid syndrome?*

In response to the 1996 document, critics pointed out that the definition is no more than a list of the kinds of things reported in the literature that people with APD have difficulty with (Chermak 2001). It is not clear whether this list defines a coherent syndrome, although one might expect some overlap between the skills listed, as they depend upon many of the same more basic auditory skills, such as frequency or intensity discrimination.

*Should APD be defined as a 'pure' disorder?*

In the 1996 ASHA report on APD, central auditory processing was also seen as involving the deployment of non-dedicated global mechanisms of attention and memory. Therefore, according to the 1996 report, for some persons APD could result from a general dysfunction that would affect performance across modalities, such as an attention deficit. However, such an over-inclusive definition could lead to the diagnosis simply becoming a synonym for other recognized conditions such as ADHD. Some authors have insisted that modality specificity — deficits in auditory processing only — need be demonstrated for a satisfactory definition of APD (Cacace and McFarland 2005). Others have countered that this position may not be neurophysiologically tenable; multimodality is a basic feature of neural coding and manipulation and that there are few if any areas of the brain that are responsible for processing in any one modality (ASHA 2005, Bellis and Ferre 1999). The latest ASHA report recognizes both points of view, recommending that APD should be recognized in individuals when the sensory processing deficit is *most pronounced* in the auditory domain, and that in some people, it may be possible to demonstrate modality specificity.

This solution does not seem ideal either. Rosen (2005) agreed that it is not reasonable to label poor auditory performance as APD *if it results from* a supramodal cause like impaired attention, but he argued there is no reason why something that causes a deficit in auditory processing might not also cause a deficit in processing of other modalities. Rosen gives the example of a demyelinating disease like multiple sclerosis in which a variety of cognitive and perceptual processes would be affected by the same disease, though the person could still usefully be described as having APD.

The APD literature might be confused because it does not recognize that there are different purposes for defining and diagnosing a disorder. If one wants to understand causal mechanisms (in a research context), one needs to focus on 'pure' cases so that results are not confounded by the presence of other problems, and one can discover how far different deficits are dissociable. However, in a clinical setting this approach may be unrealistic as most cases will also have abnormalities of language, literacy, attention or social behaviour (for example, Cameron and Dillon 2005b). Indeed, if there were no associated difficulties, it is unlikely that a child would merit referral. There is theoretical usefulness in demonstrating that APD *can* occur independently of problems in other modalities, however this is likely to be over-restrictive if one then concludes that APD *only* occurs in this pure form. This debate gets at the nub of difficulties with the construct of APD, namely, the uncertainty about whether an observed auditory deficit is a primary *cause* of a child's problems, or a secondary *consequence*. Suppose we have a child who performs poorly on a test of central auditory function and also has attentional difficulties. Logically, there are several

possible reasons for this conjunction of deficits. (1) The auditory deficit could be the primary cause leading to the attentional deficit: i.e., the child has difficulty working out what people are saying and so learns to 'switch off'. (2) Conversely, a primary attention deficit could affect auditory processing. Thus, poor performance on auditory tasks could arise because of fluctuating attention to auditory stimuli. (3) A further possibility is that the auditory deficit and attention deficit are co-occurring disorders both caused by the same aetiological process, but not causally linked. If we restricted diagnosis of APD to children without attention deficit, we could exclude option 2. However, it would be a mistake to make such a restriction, unless we were sure that option 1 was not feasible. We cannot escape this logical impasse unless we have ways of establishing direction of causality. This is a point to which we shall return when considering future directions.

*Should APD be diagnosed on the basis of non-speech auditory processing?*

In the ASHA definition APD is presumed to affect perception of both speech and non-speech signals, and, as we shall see, tests used to diagnose APD often use speech stimuli. This might seem reasonable insofar as any auditory impairment is likely to affect both speech and non-speech sounds. The degree to which the perception of a particular sound was affected would depend both on what specific auditory processes were impaired as well as the complexity and acoustic makeup of the sound in question (Griffiths *et al.* 1999, Price *et al.* 2005, Tallal 2004). However, there is also evidence that speech perception is a special case; speech is processed differently to other sound (Mody *et al.* 1997). This would mean that it would be possible to have a speech-specific perceptual deficit. It then becomes a moot point as to whether this should be regarded as a form of APD, or rather a case of linguistic (phonetic) impairment. Griffiths (2002) defines CAP as the generation of auditory percepts (or 'sound objects') *before* these sound objects acquire meaning (or undergo semantic processing), and so potentially could include cases where there was, for instance, a deficit in discriminating phonemes in non-words. However, this runs again into problems of identifying direction of causation: if a child has language difficulties and poor speech discrimination, we may be unable to tell whether the discrimination problem is the cause or consequence of language impairment.

The British Society of Audiology (BSA) (2005) adopts a more restrictive definition that side-steps this difficulty by defining APD as something that affects non-speech sounds:

A central auditory processing disorder is a hearing disorder resulting from impaired brain function; characterized by poor recognition, discrimination, separation, grouping, localization, or ordering of non-speech sounds. (bottom of page)

This low level auditory perceptual impairment would be assumed to affect both speech and non-speech sounds, but to demonstrate this unambiguously one would need to use non-speech sounds. According to this definition, if an auditory deficit was seen only in speech processing or phonological categorization it would not be recognized as APD.

In sum, APD is defined as a disorder affecting auditory processing, which may co-occur with other cognitive and perceptual impairments. The difficulty is to disentangle them. There is a concern, for instance, that the same child may receive a

diagnosis of 'SLI' if seen by a speech and language therapist or 'APD' if seen by an audiologist. We will briefly turn to consider how differences in neuropsychological conceptualization of auditory processing contribute to this state of affairs, before addressing assessment issues.

### **Models of auditory processing**

One reason for mutual miscommunication among professionals who see children with suspected APD may be because they adopt different theoretical frameworks. As Friel-Patti (1999) noted, audiologists generally tend to subscribe to a pathway model, where although there is some feedback from higher to lower levels of processing, auditory processing proceeds sequentially in the auditory nervous system (Ehret and Romand 1997). Specific tests are administered that focus on a particular level of processing, similar to the process of lesion localization in neuropsychology. Much of the research in this area has come from adults with cerebral lesions affecting auditory pathways, but the model is presumed to be applicable to children with APD of developmental origin as well. A different type of model, known as a network model, is described by Medwetsky (2002) in the context of specifying processes involved in perception of spoken language. This incorporates feed-forward and backward components, and takes into account impact of higher level processes such as language and world knowledge, pattern recognition, synthesized auditory memory and allocation of processing resources by a central executive. In addition to auditory processes, this model incorporates aspects of information-processing that will be familiar to psychologists and speech and language therapists.

Although some (Cacace and McFarland 2005) would disagree with the inclusion of factors that are not strictly 'central auditory', such 'higher-order' domain-general cognitive resources, others argue that one cannot study the central auditory system in isolation, but must take into account the possible impact on auditory processing these factors have. For example, Bellis and Ferre (1999) and Chermak and Musiek (1997) note that clinical presentation and expectations about the impact of APD would be different in an adult with acquired APD versus a child with developmental APD but with similar constraints on AP. The adult is likely to be an expert language user, a fluent sight reader, have a mature level of world knowledge and have a wealth of top-down resources that can be mobilized to support auditory processing. Adults may also be able to develop compensatory strategies to cope with their APD. The child is still in the process of acquiring skill in language and world knowledge as well as learning the associations between sounds and letter names involved in beginning to read. While the effect of APD might be exactly the same at some levels of auditory processing, the outcome is likely to be very different depending on the effect of other, not strictly central auditory factors.

The interactive nature of the network model highlights that there is a danger of concluding that a child has an APD when the primary problem is poor language or weak short-term memory. Those who adopt a network model for APD argue that the primary deficit must be one of auditory processing (that is, reflecting dysfunction originating in the pathway from cochlea to auditory cortex), but it is a process that can be impacted upon by a range of top-down factors, such as language level and memory. However, although this distinction is easy to draw in principle, in practice it can be difficult to sort out which is which. The difficulty arises whenever an attempt is made to take a model from adult neuropsychology and apply it in a developmental context (Karmiloff-Smith

1998); one is unlikely to find pure deficits analogous to those seen after focal lesions because impairment at one level can affect development of other systems.

### Assessment of APD

A 'best practice' assessment framework is described in the latest ASHA report on APD (ASHA 2005). This recommends diagnosis by a team, minimally an audiologist working with a speech and language therapist. Peripheral hearing should be thoroughly investigated using hearing thresholds, immittance measures and otoacoustic emissions. The report does not suggest a particular AP test battery, although it does describe categories of AP tests — auditory discrimination tests, temporal processing and patterning tests, dichotic speech tests, artificially degraded speech, binaural interaction tests, and electrophysiological measures (Table 1). A battery with a selection of different types of tests is recommended in order to address all aspects of auditory processing. Note, however, that as the number of tests increases, the chances of a child doing poorly on at least test purely by chance also increases and this might be incorrectly interpreted as a specific deficit. The report recommends an individual approach to testing that allows for factors such as age, language level, cultural background, visual acuity, memory, and motor skill. The exact selection of tests depends upon an individual patient's presentation, although exactly how this is done is not described. The 2005 ASHA report states that diagnosis of APD might be made on the basis of comparisons

**Table 1. Descriptions of common Auditory Processing Disorder (APD) tests**

Description (categories adapted from American Speech–Language–Hearing Association (ASHA) 2005)	Example
Temporal resolution: ability to discriminate different durations of auditory stimuli or detect silent gaps between stimuli	Random Gap Detection Test (Keith 2000a) <sup>ns</sup>
Temporal ordering: perception and processing of the order of two or more auditory stimuli over time	Pitch Patterns Sequence Test (Pinheiro 1977) <sup>ns</sup>
Perception of artificially degraded speech: speech may be time compressed, filtered, interrupted or competing with background noise	Filtered Words and Auditory Figure Ground subtests from the SCAN (Keith 2000b) <sup>s</sup>
Dichotic listening: two auditory stimuli are presented simultaneously, one to each ear; the listener is asked to attend to and report one or both stimuli	Competing Words and Competing Sentences subtests from the SCAN (Keith 2000b) <sup>s</sup> ; Staggered Spondiac Word Test (Katz 1962) <sup>s</sup>
Binaural interaction, localization and lateralization: processing involving signals from both ears, dependent on inter-aural time and intensity differences	The Listening in Spatialised Noise test (Cameron <i>et al.</i> 2006) <sup>s</sup>
Electrophysiological measures: recording of electrical brain responses to auditory stimuli <sup>a</sup> ; the timing and shape of components of the recorded signal are thought to represent sequential stages of processing by different components of the auditory central nervous system	Auditory brainstem response; middle latency response; late evoked response

Notes: <sup>ns</sup>, Non-speech stimuli; <sup>s</sup>, Speech stimuli.

<sup>a</sup> Speech or non-speech stimuli can be used to elicit responses. See 'electrophysiological studies' below.

with normative data, in which case performance below two standard deviations is a typical (albeit arbitrary) cut-off, or on a specific pattern of deficit on the basis of intra or inter-test performance. The report concludes its comment on assessment by saying that test selection and results can be mapped onto APD sub-profiles that can serve as a guide for treatment and management strategies, but acknowledges that these have not been validated by research.

In terms of a general approach to APD assessment, Jerger (1998) and Bamiou *et al.* (2006) recommend assessment including information from several different areas: clinical observation of the child in various listening environments, behavioural testing of different aspects of hearing, speech and language assessment, and electrophysiological testing. Various researchers have also made suggestions about a specific test battery for APD (Table 2). All include at least one dichotic task though no battery contains a test from each of ASHA's AP test categories. Some batteries include more than one test in each category. Only one battery includes electrophysiological measures and binaural interaction/localization tasks are also commonly left out. Though it omits

**Table 2. Suggested Auditory Processing Disorder (APD) test batteries**

Test category	Test	Test battery <sup>a</sup>				
		1	2	3	4	5
Temporal resolution	Temporal gap detection	√√				
	Tallal tests <sup>b</sup>		√			
Temporal ordering	Backward masking				√√	
	Duration Pattern Sequence test	√√			√√	
Perception of artificially degraded speech	Frequency Patterns test	√√			√√	√√
	Tests of temporal patterning			√√		
	Words in noise				√√	
	Sentences in noise				√√	
Dichotic listening	Monaural low redundancy speech			√√		
	Compressed speech		√			
	Filtered speech				√√	
	Dichotic digits	√√	√√	√√	√√	√√
	Competing sentences		√√			√√
Binaural interaction, localization and lateralization	Staggered Spondiac Word test		√			√√
	Binaural interaction tasks			√√		
Electrophysiological measures	Binaural fusion				√√	
	Auditory brainstem response	√√				
	Middle latency response	√√	√			
Other	Cortical event-related potentials (ERPs)	√				
	Auditory versus visual continuous performance	√				
	Questionnaire				√√	

Notes: <sup>a</sup>1, Jerger and Musiek (2000); 2, Musiek and Chermak (1994); 3, Bellis and Ferre (1999); 4, Neijenhuis *et al.* (2001); 5, Musiek *et al.* (1982).

<sup>b</sup>Also known as the Repetition Test. For a description, see Tallal and Piercy (1973).

√√, Main test; √, supplementary test.



two categories (localization/binaural interaction and degraded speech), Jerger and Musiek's (2000) battery seems the most comprehensive test battery. This battery was the result of a consensus meeting by 14 eminent auditory scientists and clinicians with interest in APD, although it has subsequently been roundly criticized by group of equally eminent scientists and clinicians (Katz *et al.* 2002).

A survey of common assessment practice by audiologists in the United States (Emanuel 2002) found that typical practice consisted of basic audiometric evaluation (otoscopy, tympanometry and pure tone audiometry) followed by auditory processing battery; dichotic speech tests, artificially degraded speech and questionnaires. Most took advice from other professionals, most often speech and language therapists (SLTs) and educational psychologists. A minority included temporal processing tests (pitch pattern sequence test, for sequencing of acoustic events and gap detection, a test of temporal resolving power) and auditory brainstem responses (ABR) (for evaluation of vestibulo-cochlear nerve and brain stem responses). Very few used cortical evoked potentials. The most commonly used tests were the commercially available SCAN<sup>1</sup> tests (Keith 1986, 2000b) and Auditory Continuous Performance Test<sup>2</sup> (ACPT) (Keith 1994a).

In the UK, a survey of SLPs and audiologists who diagnose APD found that most diagnosed APD on the basis of self report with the use of questionnaires (the CHAPS, (Smoski *et al.* 1998), or a locally developed one) and on the basis of a single commercially available test (most often the SCAN or SCAN-C), with a minority using a mixture of different tests including electrophysiological and language and cognitive assessments (Hind 2006).

None of the reported assessment batteries in either the United States or the UK corresponded with that recommended by Jerger and Musiek's (2000) consensus statement on APD testing.

### **Problems with current AP assessments**

Clinicians and researchers must be wary in their selection of appropriate tests for APD; many of them suffer from a number of serious problems.

#### *Psychometric characteristics*

There are numerous APD tests available from individuals or marketed commercially, but many have no normative or reliability data. Test reliability in particular is problematic for APD tests, as young children's psychoacoustic test performance is notoriously variable (Werner 1992). Without adequate psychometric data, ASHA recommendations to take an individual approach to testing, taking into account factors such as age or language level, are unworkable, as there is no empirical basis for making such judgements.

Some tests such as Keith's SCAN tests for children and adults (Keith 1994b, 2000b) do have high quality norms, and this is one factor that has ensured their popularity. In the case of the SCAN-C, standardization involved 650 children from a sample that was representative of the US population in terms of sex, geographical location, race and socio-economic status. Re-testing of 145 children was also carried out. However, there can be substantial accent effects on performance, making the norms inappropriate for children who do not speak American English (Dawes and Bishop 2007, Marriage *et al.* 2001).

*Test validity*

Even if a test is reliable and well-standardized, there remains concern about validity. Procedure-related skills (such as language, memory, attention, IQ) can have a significant impact on performance and this needs to be taken into account. Many APD tests use linguistic stimuli, and many demand a spoken response. This means that supposed AP tests can be influenced by language ability (Marriage *et al.* 2001, Moore 2006, Rosen 2005), as acknowledged in the latest ASHA (2005) report. One AP task, competing sentences, requires the child to repeat spoken sentences, a task that is similar to one that has been shown to be a sensitive marker of language impairment (Conti-Ramsden *et al.* 2001). It is sometimes assumed that one can avoid the influence of a child's linguistic knowledge by using meaningless non-words, but prior linguistic knowledge can affect performance on such a test (Thorn and Gathercole 1999). For children with language or reading difficulties, even spatial relational concepts that refer to auditory perceptual properties such as 'high' or 'low' can be problematic. No task can ever be completely free of impact from verbal abilities, when covert labelling of stimuli might be used to facilitate performance, such as 'high' or 'low' or 'beep' and 'boop' (Bishop 1997). Furthermore, design of psychophysical tasks has a critical impact on performance (Sutcliffe and Bishop 2005, Wightman *et al.* 1989), and this is especially so for clinical groups (Bishop *et al.* 1999).

For many tests, exactly what they *are* measuring is unclear, and some have suggested they draw on overlapping sets of auditory skills and that it may not be possible to expect a one to one relation between auditory tests and auditory processes (Schow *et al.* 2000). Some researchers have attempted to clarify what dimensions of auditory processing some popular assessments relate to. Schow and Chermak (1999) administered a battery of common AP tests that were expected to address four key skills: dichotic and temporal processing, auditory closure and auditory foreground–background differentiation. The authors reported a large amount of variance that was unexplained by a four factor model, and suggested that other factor/s might explain that variance.

If one assumes that the different APD subtests are measuring different skills, then it would be expected that APD tests would not correlate well with each other, as indeed was found by Schow and Chermak. However, McFarland and Cacace (2002) also point out an alternative interpretation that the different AP subtests are measuring something entirely different from AP. The four groupings of AP assessments therefore need to be validated by comparison with other indices of these separate functions.

Overall, there are numerous concerns about the validity of APD tests, and whether they are really assessing the integrity of the CANS, or whether performance is due more to non-auditory factors.

**APD sub-types**

Some researchers have suggested that the APD population may be divided into clinically useful sub-categories. Two such systems, the Buffalo model and the Bellis–Ferre model are described below.

*Buffalo model*

The Buffalo model (Katz 1992) derives from clinical testing done by Katz and colleagues at the University at Buffalo, and is based on patterns of performance on

the Staggered Spondaic Word (SSW) test (Katz 1962), a phonemic synthesis test and a speech-in-noise task. In the SSW, overlapping two syllable (spondaic) words are presented via headphones and the task is to report the spondaic word directed to a particular ear. The pattern of errors is thought to relate to specific neurological abnormalities. The phonemic synthesis task involves blending separate phonemes into familiar monosyllabic words, such as /n-o-z/ → nose.

Katz (1992) described four main categories of APD:

- ‘Decoding’, diagnosed by a poor ‘right competing’ score on the SSW, in which there is thought to be breakdown at the level of phonemic processing, possibly because of poorly specified phonological representations.
- ‘Tolerance-fading memory’, characterized by poor speech-in-noise performance and poor phonemic synthesis and generally poor short-term memory.
- ‘Integration memory’, characterized by a poor ‘left-competing’ score on the SSW. Symptoms in this case are suggested to be caused by difficulties in integrating all types of information, including auditory and visual information.
- ‘Organizational’. This group is characterized by word and sound reversals on the SSW and phonemic synthesis test and is thought to involve problems maintaining sequences and organizing information. General disorganization in daily life, poor handwriting and reversals in spelling and reading are thought to be typical.

Katz *et al.* (1992) tested ninety-four 6–12-year-old children referred for auditory testing with the battery of three tests and found he could classify 91% of children with these categories as follows: Decoding 50%, Tolerance-fading memory 20%, Integration memory 17%, and Organization 4%.

#### *Bellis–Ferre (1999) model*

As with Katz’s Buffalo model, this multidimensional model is based on the authors’ clinical impressions, and a series of prototypical case studies are offered to illustrate each category in the model. Categories are based on patterns of findings on a battery of different combinations of auditory processing tests including Dichotic Listening (the Dichotic Digits test, Dichotic Rhyme, Competing sentences or the Staggered Spondaic Word test), Monaural low-redundancy speech (Low-pass filtered speech), Temporal Patterning (Frequency Patterns test) and Binaural interaction (Consonant–vowel–consonant binaural fusion). Several alternative tests are suggested for each area. There are three main categories:

- ‘Auditory decoding deficit’ is characterized by bilaterally depressed performance on dichotic speech and poor monaural low-redundancy speech recognition and generally poor auditory closure skills. Difficulties in challenging listening environments and poor reading and spelling are typical. This category is similar to the ‘Decoding’ category of the Buffalo model.
- ‘Integration deficit’ is characterized by left ear suppression in dichotic speech tasks, difficulty with linguistic labelling in temporal patterning tests with good performance on monaural low redundancy speech (see Table 1 for descriptions of common APD tests). Difficulties across modalities are expected, in fact with any task that requires inter-hemispheric communication.

Bi-manual and bi-pedal motor skills are poorly coordinated, and providing extra cues, for example visual, to support auditory tasks worsens performance. In this case, difficulties are supposed to be due to a deficit in information transfer across the corpus callosum. This category is similar to the 'Integration' category of the Buffalo model.

- 'Prosodic Deficit'. This category represents difficulty with acoustic contours and a left ear deficit on dichotic tasks. People in this category typically have poor pragmatics, generally poor sequencing skills, flat expressive prosody and difficulties with perceiving prosody and with judging speaker's intent. The Buffalo model does not have any obvious parallel to this group.

#### *APD subtypes: evaluation*

A major concern is that both classification systems are derived from linguistically based tests, on which scores could be readily affected by poor language skills. The emphasis on deficits in phonological processing in defining APD in the Buffalo system entails that most children with phonological processing difficulties (that is, most cases of dyslexia and SLI) would be diagnosed as having APD. *None* of the categories in either system met the BSA's criterion for APD (described above), as diagnosis is made on the basis of impaired speech processing or phonological categorization. There has not yet been any independent experimental validation of the proposed categories or validity of the proposed treatments for either system of classification.

#### **APD in relation to other diagnostic categories**

##### *Attention deficit hyperactivity disorder (ADHD)*

In addition to the auditory problems, children with APD are often described as having behavioural problems such as inattentiveness, distractibility and poor organization (ASHA 1996, BSA 2005), symptoms that are reminiscent of ADHD (American Psychiatric Association (APA) 2000). Riccio *et al.* (1994) found that in 30 children diagnosed with APD, 50% would also fit a diagnosis of ADHD based on formal evaluation. This raises major questions about direction of causation: attention affects children's performance on psychophysical assessment (Wightman and Allen 1992), and listening skills probably also impact upon some assessments of attention. Are APD and ADHD distinct and separate entities that can occur co-morbidly or does one cause the other? Or is APD just an alternative label for ADHD symptoms seen from the perspective of an audiologist? It has been suggested that ADHD is a disorder that impacts on a range of perceptual processes, including auditory ones (Sagvolden *et al.* 2005), thus APD may be considered just one aspect of ADHD. A second possibility is that auditory processing deficits cause some cases of ADHD (Chermak *et al.* 1999).

There is not much support for the second view, although one can imagine that a child who is unable to hear well may become inattentive. However, in the case of ADHD, there are a range of neurophysiological findings including measures of attention and executive functions, list learning and information processing speed that are inconsistent with a purely auditory perceptual basis for the observed behaviour problems (Woods *et al.* 2002). Studies attempting to differentiate APD and ADHD

have taken a number of different approaches, including comparisons of behavioural observations, psychometric and electrophysiological testing. Such comparisons are complicated by the fact that there are no objective diagnostic tests for either APD or ADHD, with the latter diagnosed on the basis of parent and/or teacher report.

Chermak *et al.* (2002) suggested that ADHD and APD might be distinguished on the basis of behavioural presentation. They compared audiologists' and paediatricians' rankings of 58 behaviours associated with APD and the predominantly inattentive type of ADHD and found that none of the four behaviours that were ranked highest were in common for both disorders. They concluded that there were an exclusive set of behaviours that characterized APD and ADHD. However, McFarland and Cacace (2003) objected that this analysis was misleading, and re-analysed Chermak *et al.*'s data focusing on the whole range of reported symptoms for both conditions. They found that overall rankings of behavioural features were highly correlated for APD and ADHD. In other words, there was a high degree of overlap in these professionals' descriptions of behaviour of children with APD and ADHD.

In an earlier paper, Chermak *et al.* (1999) suggested that children with ADHD and those with APD have different sorts of attentional difficulties. They suggested that the attentional deficit in ADHD is characterized by a deficit in sustained and selective attention, while that of APD characterized by focused and divided attention, for example on a dichotic listening task. Chermak *et al.* proposed that these different attentional problems would be reflected in patterns of performance on auditory continuous performance tests, which could then be used to differentiate the two conditions. In auditory continuous performance tests, children have to respond appropriately to infrequent auditory stimuli, a task designed to generally tax one's attention. Children with APD would be expected to miss more targets (errors of omission), while those with ADHD would respond impulsively (errors of commission).

However, the conceptual basis of this argument does not seem to be reasonable. According to DSM-IV criteria for ADHD (APA 2000), while both inattentiveness and impulsivity are features of ADHD, impulsivity is not a *necessary* feature of ADHD. An impulsive pattern of responding would not provide a very specific distinction between ADHD and APD. Others have suggested that while auditory continuous performance tests are highly sensitive to ADHD, they are not very specific (Oyler *et al.* 1998, Riccio and Reynolds 2001). Riccio *et al.* (1996) directly examined the validity of Keith's (1994a) Auditory Continuous Performance Test to differentially diagnose children with and without coexisting ADHD, and found that although there was a tendency for the ADHD group to find the task more difficult, there were not significant differences on any index of performance between groups.

Early studies (Gascon *et al.* 1986) had suggested that behavioural tests for APD were actually measuring symptoms of ADHD. Later studies suggested that APD and ADHD are distinct disorders that may co-occur and that tests for APD were actually measuring something distinct from attention. Riccio *et al.* (1996) compared patterns of performance on the Staggered Spondiac Word Test (SSW) (Katz 1962) by children with APD to that of children with ADHD. They found different patterns of results for the two groups and a low correlation between SSW test results and ADHD behaviours. Similar findings have been obtained by Breier *et al.* (2003), who used experimental tests designed to tax auditory temporal processing with children who had either reading disability, or ADHD, both diagnoses or neither. The main effect

of ADHD was non-significant on both speech and non-speech tasks, indicating that attentional deficit does not necessarily disrupt performance on psychoacoustic tasks.

A particularly strong test of the impact of ADHD symptoms on auditory processing is to compare the same children when on or off medication to control ADHD symptoms. Tillery *et al.* (2000) investigated the impact of ADHD stimulant medication on performance of tests of attention and auditory processing by children with both ADHD and APD. They found that while medication significantly improved performance on a test of attention, it had no effect on performance on tests of auditory processing. However, different results were found by Sutcliffe *et al.* (2006), who also compared performance on auditory psychophysical tests by children with ADHD while on and off medication. They found that attentional state affected performance on one task (frequency discrimination), but not the other (detection of 20 Hz frequency modulation), despite both tasks using the same task procedure. It seems that even among very similar tests, some may be more susceptible to the effects of attention than others. It is encouraging that some APD tests do seem relatively resistant to attentional variation. It would be a worthwhile exercise to discover which tests are more resilient and why this might be.

Another approach to disentangling auditory deficit from more general attentional difficulties is to include a visual test of attention. The prediction would be that a child with ADHD would have attentional problems regardless of modality, whereas APD should show up only on auditory tests. Riccio *et al.* (2005) adopted this approach and found no correlation between a visual test of attention and auditory processing measures (SCAN-C and SSW) among children with APD, ADHD or both. They concluded that APD is not necessarily associated with attention deficit, and it is possible to separate the two using behavioural measures.

Electrophysiological methods could provide a way of accurately differentiating APD and ADHD. Some abnormalities in auditory event-related potential (ERP) response have been found in children with ADHD (Oades *et al.* 1996), but attempts to differentiate APD children with and without ADHD using electrophysical measures have not been successful to date (Ptok *et al.* 2004).

In sum, though studies of behaviour of children with APD and ADHD report a large degree of overlap of reported symptoms, there are some behaviours more often associated with one disorder than the other (Chermak *et al.* 2002, Ptok *et al.* 2006, Riccio *et al.* 1994). Although the clinician needs to be alert to the possibility that poor attention may affect performance on tests of auditory processing, it does not seem reasonable to argue that APD is just another way of describing ADHD. Rather, it seems as though APD and ADHD are frequently co-morbid while being distinct entities. This is a common finding in neurodevelopmental disorders, possibly because the same aetiological factors can affect more than one developing system (Bishop 2006).

#### *Autistic Spectrum Disorders (ASD)*

Although perceptual abnormalities are not part of the diagnostic criteria for autistic disorder, they are a commonly mentioned feature. For instance, children with autism have been described as being indifferent to sound, ignoring such salient stimuli as someone speaking their own name. At the same time, children with autism may be hypersensitive to some sounds, being able to hear soft sounds that others find undetectable, or exhibiting extreme aversive reactions to sounds that others find

innocuous (Frith 2003). Rosenhall *et al.* (1999) reported 18% of 199 children with autism had hyperacusis, as opposed to none in an age-matched control group. Alcantara *et al.* (2004) found that people with high functioning ASD complained of difficulties listening in noise and had poorer speech-in-noise performance compared with age- and intelligence-matched controls.

Such reports, coupled with increasing recognition of autism, or milder forms of autistic spectrum disorder (ASD) in children of normal intelligence (Baird *et al.* 2006), raise the question as to whether the diagnosis of APD might be being applied to children with ASD who have abnormal auditory perception. We have found that ASD is over-represented among children referred for APD testing at a large APD specialist clinic in London, where 9% had a formal diagnosis of autism or ASD (Dawes *et al.* 2008). In our behavioural testing of children who received a diagnosis of APD, preliminary analysis suggested a high rate of autistic features though ASD had not been formally diagnosed. We are not aware of any published studies that have employed conventional autism diagnostic instruments with children referred for APD, but there is a literature on auditory perception in ASD that is of relevance.

There are findings of both enhanced and impaired auditory skills in people with autism, though there is uncertainty about how these might relate to the condition as a whole. An explanation that has been advanced for the mixed auditory processing profile in autism is that there is enhanced or spared local processing (or processing of detail, for example single note changes in melody) with impaired global processing (or processing of the whole, for example changes in melody contour) (Frith and Happe 1994, Mottron *et al.* 2006). However, research findings do not always seem to fit well with this explanation. The reverse of what would be predicted is sometimes found, for example, enhancement of perception of musical affect (Heaton *et al.* 1999) (global) versus impairments in pitch discrimination (Tecchio *et al.* 2003) (local).

Two recent ERP studies suggest that the observed impairment is due to a speech-specific, post-sensory impairment related to attentional orienting (Ceponiene *et al.* 2003, Whitehouse and Bishop 2008).

Overall, the pattern of results suggests that any auditory deficits seen in autism are heavily modulated by the meaning of the stimuli, and thus due to top-down influences on auditory processing, rather than caused by a primary problem in detecting or discriminating auditory features. Based on observed associations between ASD and abnormal auditory behaviour, two recommendations follow. First, that further research is carried out into the nature and significance of auditory processing problems in ASD and second, that children who receive a diagnosis of APD be screened for communication difficulties associated with ASD. Given the evidence of top-down influences on auditory processing in autism, we judge it as unlikely that listening problems in children with ASD would be helped by the kinds of environmental modification that have been recommended for APD (Bamiou *et al.* 2006).

#### *Specific language impairment (SLI) and developmental dyslexia*

The most research — and the most debate — has been on the relationship between APD and disorders of language and literacy. There is a high prevalence of language and reading difficulties among children diagnosed with APD (Chermak and Musiek 1997, Katz 1992). There is also a long history of findings of auditory processing

difficulties in children and adults with dyslexia and SLI (Tallal 2004, Witton *et al.* 1998), with some researchers proposing that auditory (or more general pan-sensory) processing difficulties underlie language and reading difficulties (Stein 2001).

While there is strong evidence that a phonological deficit underlies reading problems (Snowling 2001), a theory first posited by Tallal and Piercy (1973) suggests that in some children, a temporal auditory deficit may be the cause of these phonological problems. For example, if a young child were to have difficulty in resolving rapid temporal changes in sound at the level of phonemes of speech, this might result in poorly defined phonetic categories with consequences for phonemic awareness and literacy acquisition. In severe cases, this could also lead to problems in learning vocabulary and syntax (that is, language problems). Researchers in hearing and APD have suggested further behavioural consequences of auditory processing problems; difficulties with following amplitude or pitch changes at a level of seconds rather than tens of milliseconds (as for phoneme-level perception) might lead to difficulties with perception of prosody, correct interpretation of meaning and appreciation of music (Bellis and Ferre 1999, Griffiths *et al.* 1999). Depending on developmental history and environmental interactions, different levels of auditory processing impairment might manifest as SLI, dyslexia, difficulties listening in noise, or amusia (for amusia, cf Ayotte *et al.* 2002). Although initially promising as an explanatory hypothesis for reading and language problems, numerous independent groups have failed to replicate Tallal's findings of auditory temporal processing deficits in dyslexia and SLI (McArthur and Bishop 2001). However, Tallal claimed that an auditory processing problem may explain language and literacy problems in *some* children (Tallal *et al.* 1991). An explanation offered for a failure to replicate findings is that there is inherent heterogeneity within the SLI/dyslexia population, and that a subgroup of children has perceptual deficits that underlie their language or reading difficulties.

An additional and more serious problem is posed by failures to replicate Tallal's original finding of strong correlations between auditory temporal processing and measures of phonological processing, such as non-word reading (cf Bretherton and Holmes 2003). A number of reviews of the temporal auditory hypothesis have concluded that this theory can not account for most reading or language problems. Several reviews (Bailey and Snowling 2002, McArthur and Bishop 2001, Ramus 2003, 2004, Rosen 1999, 2003) identified major difficulties with the auditory hypothesis. First, only a minority of children with dyslexia have auditory perceptual deficits and there is typically overlap in performance between affected and control groups; many affected individuals show no auditory deficit, whereas some controls score badly. Second, for those that do have deficits, these are not restricted to brief or rapid stimuli as the auditory theory hypothesizes. Third, perceptual difficulties are unrelated to phonological problems, although phonological problems are convincingly associated with reading problems. Studdert-Kennedy and Mody also found poor readers were not able to discriminate speech sounds, but were able to discriminate acoustically matched non-speech sounds (Mody *et al.* 1997, Studdert-Kennedy and Mody 1995). They suggested that the phonological deficits in poor readers relates to a speech-based, phonetic problem rather than a general problem with auditory processing. In reference to language impairment, Bishop *et al.* (1999) compared performance on a range of auditory tasks by children with language impairment and matched controls. There was no significant difference between groups on any auditory task. They concluded that auditory deficits are neither necessary nor sufficient cause of language impairment.



Despite controversy about the association and causality, many studies have found a significant increase in auditory processing problems among children with language and reading problems (for an overview, see Tallal 2004) and this needs to be explained. One possibility is that some people with SLI/dyslexia do have auditory processing abnormalities, but while these auditory abnormalities might have the same aetiology as the language/reading difficulties (for example, impairment of neurological development), they are not themselves causally related to the language/reading difficulties (cf. Ramus 2004). A second possibility is that a perceptual deficit is a risk factor that increases the likelihood that a language or reading disorder will be expressed in those with genetic predisposition (Bishop 2006). A third possibility is that more general, non-sensory difficulties may underlie poor performance on psychophysical tasks (for example, Roach *et al.* 2004). The variability of performance may reflect differing co-morbid mixtures of SLI/dyslexia and other more general deficits, such as memory and attentional problems.

A further possibility is that the pattern of association between auditory deficits and phonological problems might change with age (Tallal 2004). An early auditory deficit may affect how phonological representations are set up, leaving a lasting phonological deficit even when auditory deficits may change or resolve. It is difficult to test this proposition without longitudinal data; however, there is some evidence of association between early auditory discrimination and later language development (Benasich *et al.* 2006).

Some feel that APD can be totally discounted as a cause of SLI because of the existence of (1) children with auditory deficits and no SLI and (2) children with SLI and no auditory deficits, but as we have seen above, this may be a premature conclusion. SLI and dyslexia are complex, multifactorial disorders; while there is a strong heritable contribution, they may only be manifest in conjunction with other intrinsic or extrinsic factors. A difficulty with auditory processing might constitute such a factor. APD may be a risk factor for language and literacy problems, with some forms of APD being more significant than others, depending on which aspect of processing is impaired.

### **Future directions**

It is clear that there remains considerable tension between audiologists who diagnose APD, and see it as a common cause of children's specific learning difficulties, and experts in language and literacy problems, many of whom would agree with Kamhi and Beasley's (1985) designation of APD as 'a twentieth-century unicorn' (extending now into the 21st century!). We need to consider how this state of affairs may be rectified. The development of well-standardized, age-appropriate, valid and reliable measures of non-verbal auditory processing is a tractable task that urgently needs to be addressed by the audiological community (Moore 2006). In addition, we recommend that children referred for evaluation of APD should be assessed by a speech and language therapist in order to establish the extent of associated communication problems. However, as we have stressed in this review, there is a deeper problem that cannot be resolved so easily, and that concerns causal relationships between co-existing impairments in children. Even if we have a psychometrically strong measure of auditory processing that reveals a deficit in a child with a language impairment, it can be difficult to know whether this is causally linked to the disorder. We suggest three lines of research that could help us escape from this impasse: electrophysiological studies, aetiological research, and intervention studies.

*Electrophysiological studies*

Electrophysiological measures provide a non-invasive way of observing the temporal course of brain responses to auditory stimuli. Most of the responses that occur within the first 300 ms or so post-stimulus onset are termed 'obligatory', which means that they can be elicited without active attention by the child. There is therefore considerable interest in using these measures to complement and validate behavioural tests of APD because they can avoid the problems inherent in interpreting behavioural tests that may depend on the child's attention and motivation.

Some recent research suggests that a number of late auditory ERP indices might be useful in identifying APD (Liasis *et al.* 2002) and monitoring changes in auditory processing following intervention (Baran *et al.* 2006). However, variability of response between normal individuals and within typical development poses a serious challenge. Furthermore the mismatch negativity, which was initially hailed as an electrophysiological measure of auditory discrimination, has not lived up to its promise as a clinical tool because of low reliability at the individual level (Bishop 2007).

In recent work, Kraus and colleagues have examined the possible contribution of brainstem level encoding of auditory stimuli to learning problems. The brainstem response to sound (auditory brainstem response ABR) is an electrophysiological response that can be reliably recorded at the individual subject level. The ABR represents a faithful neural 'snapshot' of the stimulus, in that onset, fundamental frequency and harmonics are all represented in the ABR (Banai *et al.* 2005, Kraus and Nicol 2005). Kraus suggested that there are particular, dissociable 'source' and 'filter' related components of the ABR that are especially important for perception of linguistic content of a speech signal. Source related components refer to regularly spaced, later occurring peaks in the ABR that correspond to the fundamental frequency of the stimulus and relate to prosodic information in speech. Filter components correspond primarily to the initial and final peaks in the ABR and to the onset and offset of the stimulus. Filter components correspond to phonemic content.

Filter components are reported to be vulnerable to disruption in some children, especially in the presence of background noise (Kraus and Nicol 2005). The ABR response subserves cortical processing of speech signals, which is in turn associated with performance on standardized measures of educational achievement (Cunningham *et al.* 2000).

Banai *et al.* (2005) examined the relation between brainstem responses to synthetic speech stimuli and literacy skills in normal and learning disabled children who had received a diagnosis of a learning disability by independent clinicians. All children had otherwise normal hearing and intelligence (standard score greater than 85). They found that about 40% of the learning disabled group had an abnormal ABR, and that this was associated with more severe learning problems (poorer reading and listening comprehension).

Wible *et al.* (2005) also looked at ABR and cortical responses to /da/. They found a group of children with learning problems who had abnormal ABR and abnormal cortical processing. These children also demonstrated a weaker correlation between ABR and cortical response compared with controls, and this correlation was more susceptible to disruption by noise. One might predict that children diagnosed with APD might show a similar pattern. However, in Wible *et al.*'s study, this pattern was evident in only approximately one-quarter of the learning disabled sample, with the

rest similar to controls. It was concluded that only a minority of children with learning disabilities have auditory-based language learning problems. One piece of evidence that supports a causal role for impaired brainstem encoding in speech perception deficits is the study by King *et al.* (2002). As for the work described above, King *et al.* discovered abnormal cortical processing of synthetic speech syllables in noise for learning-disabled children with ABR abnormalities, not for learning disabled children with no ABR deficit. They then trained a small number of children with the Earobics computerized training package (Cognitive Concepts, Inc. 1997). They found that for children with abnormal ABR, post-training there was increased resilience of cortical responses in the presence of noise and improved speech sound discrimination. The Earobics programme involves training in phonological awareness, auditory processing and language skills. Some of the training involves the use of acoustically enhanced signals designed to facilitate auditory training, and the assumption was that it was this auditory training that resulted in the observed changes. Definite conclusions are difficult to draw on this basis of this work alone, apart from that the ABR can be modified by experience.

We should be wary of concluding that, because we see a response in the brain, we have uncovered the 'cause' of a child's difficulties. It is always possible that the development of auditory pathways is influenced by higher centres and/or a coincident rather than causal deficit. For instance, an alternative explanation of the findings of Wible *et al.* is that abnormal ABRs to speech in noise are simply a marker of atypical brain development, and not causally linked to the learning difficulties (cf. Bishop 2006). Nevertheless, although they do not solve the problem of detecting direction of causation, electrophysiological methods do provide an important complementary approach to the study of higher auditory functions, and we anticipate that this line of work will burgeon over the next decade.

### *Aetiology*

There is evidence for strong genetic influence on the aetiology of ADHD, ASD, SLI and dyslexia (Bishop and Rutter 2009). Genetically informative designs, such as twin studies, can help determine whether two co-occurring deficits have common origins. Using this approach, Bishop *et al.* (1999) were able to show that, although auditory discrimination problems were more common in children with language impairments than in a control group, auditory and language problems had different origins, with the auditory problems appearing to be entirely environmentally determined. This approach has barely been applied to the study of APD and other disorders, but merits attention because of its potential to disentangle causal pathways (for more discussion, see Bishop 2006).

### *Intervention*

Strong evidence for causality is provided if one can show that reducing one deficit improves another. For instance, the demonstration by Sutcliffe *et al.* (2006) that performance on an auditory task was dramatically improved in children with ADHD by administration of stimulant medication provided evidence that it was the attentional difficulties (that were ameliorated by the intervention) which had caused the high auditory thresholds.

Particularly compelling evidence of an auditory basis to children's language or literacy problems would be provided if one could show that training non-verbal auditory discrimination improved verbal skills. However, despite early promise (Tallal *et al.* 1996), this has not been convincingly demonstrated. Although training can improve discrimination of non-verbal auditory stimuli, there appears to be no generalization to language or literacy skills (for example, Agnew *et al.* 2004, Berwanger and Von Suchodoletz 2007). Even where discrimination of acoustically modified speech is combined with intensive computerized language training, there is little evidence of efficacy in improving language skills when properly controlled trials are conducted (Cohen *et al.* 2005, Pokorni *et al.* 2004, Rouse and Krueger 2004). This area of research is still at an early stage and further studies, ideally combining neurophysiological with behavioural measures, are needed (cf Santos *et al.* 2007). However, in our current state of knowledge, the consensus is that one is more likely to improve reading or language by training those skills directly, rather than by attempting to improve non-verbal auditory processing.

Bamiou *et al.* (2006) reviewed contemporary approaches to intervention with APD, noting the dearth of studies on efficacy. It is noteworthy that rather than attempting to ameliorate the auditory problem directly, several approaches aim to lessen the impact of the impairment, by environmental modification (for example, seating the child at the front of the class) or signal enhancement (having the teacher using a directional microphone). There would be considerable interest in evaluating the impact of such intervention on language and literacy as well as auditory functioning.

### Conclusion

We argue that there is both clinical and theoretical support for the category of Auditory Processing Disorder (APD): it is plausible that in some children higher auditory processing is specifically impaired compared with their detection of sound. Furthermore, there are children who complain of disproportionate difficulties in hearing in difficult listening conditions, and their difficulties cannot be explained away as attentional or linguistic. Nevertheless, it would appear that in current practice, APD is over diagnosed, with some practitioners using criteria that will include virtually all children with language and literacy problems. One reason why APD has proven to be such a problematic category to diagnose and assess may be because it sits on the crossroads of different disciplines. Children with APD have a mixture of auditory and learning difficulties, with language and literacy often implicated. A current concern is that the same child who is treated as a case of specific language impairment (SLI) by a speech and language therapist may be diagnosed with APD by an audiologist, causing confusion to both professionals and parents. Audiologists have traditionally adopted a 'pathway' model, in which the task is to identify the stage of processing that is affected, and may be unaware of the impact that verbal ability can have on some of their tests. On the other hand, speech and language therapists may be unaware that auditory deficits play a role in causing language problems if they assume that an audiogram stating 'normal hearing' means the auditory system is intact. We regard it as crucial that these different professional groups work together in carrying out assessment, treatment and management of children and undertaking cross-disciplinary research. A particular need is for reliable and well-standardized measures that avoid the confound of language level, such as are

currently being developed in a large-scale study in the United Kingdom (Moore 2006). Speech-based tests can also be valuable, provided one can demonstrate that the deficits found are not the consequence of poor language level (Cameron and Dillon 2005a). In more specialized clinical settings, the electrophysiological methods pioneered by Kraus and colleagues hold out promise for providing converging evidence of auditory processing disorders.

It may be that auditory processing problems are one of a number of deficits commonly found in developmental disorders. Future research may help clarify the significance of auditory processing difficulties and how they factor in the causation and manifestation of developmental disorders and learning disabilities. Rather than considering APD as a separate disorder, it may be more helpful to clinicians and researchers as well as the children and families concerned to consider auditory processing problems as one of several dimensions of impairment associated with a range of developmental conditions, rather than being a categorical disorder in its own right. The risk with the latter is that this may divert management and treatment efforts away from other useful areas. Productive areas for research involve (1) development of reliable and valid clinical measures of auditory function capable of differentiating auditory sensory from language, attention, memory and cognitive functions; (2) defining a disorder of auditory processing and clarifying its impact on development of language and literacy as well as functional consequences on listening and behavioural competence in challenging auditory environments such as classrooms, in the home and among peers; (3) the incidence of these auditory problems; and (4) effectiveness of various interventions, including which features of the intervention are most effective and which children are likely to benefit.

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### Notes

1. The SCAN tests are individually administered in audiometric or quiet room conditions. Stimuli are recorded on a CD and presented over headphones. Patients are scored on the accuracy with which they repeat monotonically presented single-word stimuli that have been acoustically degraded or presented in the presence of multi-talker babble, as well as dichotically presented single words and sentences. Accuracy scores are compared with age-based performance norms.
2. The ACPT is designed to measure a child's ability to attend and respond selectively to a specific linguistic cue and to maintain attention for an extended period of time. Tape-recorded stimuli are presented at a rate of one word per second. Children must listen for and respond by a thumb raise to the infrequent target word 'dog' presented randomly among other single syllable words. Comparison against criterion scores yields either a pass or a fail.

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