CURRENT TOPIC

Aetiology and clinical presentations of auditory processing disorders—a review

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Abstract
Auditory processing disorders may have detrimental consequences on a child's life, if undiagnosed and untreated. We review causes of auditory processing disorders in order to raise clinical awareness. Auditory processing disorders may present against a background of neurological disease or developmental disorders, as well as in isolation. Clinicians need to be aware of potential causes and implications of auditory processing disorders.
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Hearing is a complex process that orchestrates transduction of the acoustic stimulus into neural impulses by the ears, transmission of the neural impulses by the auditory nerves to the brain, and perceptual registration and cognitive elaboration of the acoustic signal by the brain as well as conscious perception of the sound. Hearing impairment(s) arising from pathology of the brain may have detrimental consequences on a child's life if untreated; however, diagnostic and management strategies for these "central" hearing impairments in childhood are rarely implemented. These auditory deficits have been collectively termed "auditory processing disorders", in order to incorporate in the term the interaction between peripheral and central pathways.[1]

A rough prevalence estimate for auditory processing disorders (APD) in childhood is 7%.[2] Despite the frequency of the problem, a systematic approach to the diagnosis and rehabilitation of APD in children has only started emerging over the past 30 years, as a result of developments in basic sciences; emphasis has shifted from identification of the lesion that causes the disorder to identification of the impaired individual's difficulties and their appropriate remediation.[3]

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Anatomy of the central auditory nervous system

The central auditory nervous system (CANS) extends from the cochlear nucleus in the brain stem to the auditory cortex. The superior olivary complex, lateral lemniscus and inferior colliculus, medial geniculate body, and reticular formation are important relay stations. The cortical and subcortical auditory areas mainly consist of Heschl's gyrus, the planum temporale (extending from the posterior aspect of Heschl's gyrus to the end of the Sylvian fissure), and the Sylvian fissure with the insula.[4] The cerebral hemispheres are connected by the corpus callosum. The CANS is characterised by an intrinsic "redundancy"—that is, an extensive interaction of its structures that is responsible for the resistance of the system to exhibit deficits on standard auditory testing in the presence of a lesion.[4] In children, myelination and maturation continue until 10–12 years of age.[5] The young brain has an inherent ability for plasticity: the forebrain sensory representations may change in response to altered receptors, sensory environment, or use and learning.[6]

Clinical presentation

Children with auditory processing disorders appear to be uncertain about what they hear, and may have difficulties listening in background noise, following oral instructions, and understanding rapid or degraded speech in the presence of normal peripheral hearing.[1] Symptoms may become apparent in the early school years or at a later academic stage of the child's life, due to changes in the acoustic environment or to increased academic demands. In rare cases, these symptoms may be the first manifestation of a neurological disorder.[7] As a consequence of

Table 1 Deficits and resulting symptoms and behaviours which characterise APDs

Auditory deficits in:	*Resulting symptoms and behaviours*
Sound localisation	Poor performance in confusing environments
Auditory pattern recognition	Difficulties following oral instructions
Auditory discrimination	Language, reading, and spelling disorders
Temporal processing	
Processing degraded auditory signals	Difficulties with rapid/degraded speech and with auditory closure
Processing the auditory signal when embedded in competing acoustic signals	Difficulties in background noise
	Inattention
	Distractibility
	Academic difficulties

the primary auditory difficulties, children with APDs may have secondary characteristics of language, reading and spelling disorders, as well as inattention and distractibility (table 1)[2]; this profile requires careful consideration and diagnostic evaluation for differentiation from other common childhood developmental disorders.

Auditory processing disorders may result from disruption of processes specific to audition, as well as from more global deficits (for example, memory or attention deficits) that have a negative impact on the processing of auditory information.[8] Irrespective of the causal mechanism, an APD may manifest as a deficit in sound localisation, discrimination, pattern recognition, temporal processing, and performance deficits when the auditory signal is degraded or embedded in competing acoustic signals (table 1). These deficits have electrophysiological as well as behavioural correlates.[8]

Diagnosis

Central auditory testing clinically evaluates the integrity of the CANS and provides a "bottom up" sensory cognitive approach to learning and behavioural problems as well as an index to neuropathological constellations. Central auditory nervous system problems may be isolated or associated with more pervasive processes and conditions which closely interact with other sensorimotor modalities as well as "top down" cognitive functions. The diagnosis of APD thus requires a multidisciplinary approach with careful consideration of cognitive, memory, and linguistic parameters. Diagnosis relies on synthesis of information from history (medical, educational, developmental), behavioural and electrophysiological tests, as well as ancillary procedures such as neuroimaging, speech and language assessment, and psychological/cognitive assessment, after careful consideration of confounding factors.[1]

The testing battery (table 2) includes:

- Baseline audiometric assessment to exclude a peripheral hearing loss
- Behavioural central auditory tests. These tests may tap into more than one auditory process, and fall into three main categories[9]:
  - monaural low redundancy: speech stimuli that are either degraded (e.g. in terms of frequency content), or embedded in competing signals (e.g. in noise or multispeaker babble) are presented to one ear, and the child is requested to identify the speech stimulus
  - dichotic/binaural interaction tests: stimuli are presented to both ears, and the task requires the child to attend to one ear only or to both at the same time
  - temporal tests, e.g. sequencing tasks
- Electrophysiological tests. These may include auditory brain stem evoked responses and middle latency response, which are key measures for auditory structures in the brain stem and in subcortical to cortical levels respectively; and late potentials, which may or may not be affected by attention, such as the P300 or Mismatch Negativity (MMN).[10]

Table 2 Testing battery for APDs

Baseline audiometric tests	Pure tone audiogram Tympanogram Otoacoustic emissions
Behavioural central auditory tests	*Monaural low redundancy tests* e.g. filtered words, auditory figure-ground test *Dichotic tests/binaural interaction tests* e.g. dichotic digits, competing sentences *Temporal tests* e.g. frequency pattern test, temporal gap detection
Electrophysiological tests	Auditory brain stem evoked responses Middle latency response P300 Mismatch negativity

Classification

In terms of pathophysiological mechanisms, APD may be classified as occurring in the presence of[2]: neurological conditions; delayed central nervous system maturation; or other developmental disorders.

NEUROLOGICAL CONDITIONS ASSOCIATED WITH APD

Few cases of APD in children have an underlying neurological deficit. However, an APD may occasionally be the only or the presenting manifestation of a neurological disorder, highlighting the necessity for a high clinical index of suspicion and the value of neurological and developmental examination.

Tumours of the CANS

The concept of (central) APDs may be traced back to Bocca's audiological findings in adults with brain tumours that affect the auditory areas.[9] Children with CANS tumours have similar ear deficits to adults,[11] notwithstanding the young brain's capacity for plasticity.[6] In the presence of severe neurological symptomatology, auditory difficulties may not be perceived as a major symptom, even in the presence of grossly abnormal central auditory test results.[7] Conversely, APD may be the first and only manifestation of a space occupying lesion,[7] and the auditory deficits may be mistaken for a learning disability. Appropriate surgical and rehabilitational intervention may lead to improvements in behavioural and electrophysiological findings,[7] highlighting the young brain's potential for plasticity and the need for aggressive rehabilitation of the young patient after brain surgery.

Prematurity and low birth weight

Preterm infants with low birth weight may suffer from APD which significantly improves with time; however, by the age of 14 years some of these children will continue manifesting subtle auditory deficits, such as poor auditory memory span, in a significantly greater proportion than the normal birth weight population.[12]

Extrinsic damage to the brain

Bacterial meningitis is implicated as a cause of auditory processing disorder, but the supporting evidence is inconclusive.[13] Single case reports also indicate that *herpes simplex encephalitis* can be associated with central deafness in children—that is, central auditory system dysfunction that results in practically no useable hearing.[7]

Lyme disease, a tick borne infection caused by the spirochete *Borrelia burgdorferi*, may have long term sequelae of auditory processing difficulties[14] which may persist following treatment.

APD may be also be caused by *head trauma*.[15] Children who sustain closed head injury may suffer from atrophy of the posterior corpus callosum, resulting in auditory hemispheric disconnection.[15]

Low level *heavy metal exposure* in children may affect sites in the CANS. Blood mercury levels may correlate with auditory brain stem response (ABR) delayed latencies,[16] as well as with poorer central auditory processing abilities.[17] Similarly, prenatal exposure to *cigarette smoke*,[18] *alcohol*,[19] or postnatal *anoxia*[20] may also be implicated in higher prevalence of APD.

Cerebrovascular disorders

The auditory deficit in stroke in childhood can be quite dramatic, with no behavioural response to sound despite the presence of normal otoacoustic emissions and ABR, as in the case of a 3 year old child with *Moyamoya disease*.[21]

Metabolic disorders

Cortical deafness with auditory agnosia may be a presenting feature of adrenoleucodystrophy; this symptom may temporarily respond to treatment.[22] There are no systematic studies of APD in the presence of inborn errors of metabolism, although several of these conditions are known to affect central auditory structures with abnormal auditory evoked response potentials (see, for example, Kaga and colleagues[23]). In view of new treatment possibilities, and of the brain's capacity for plasticity, such studies are urgently required.

Epilepsy

Central auditory impairment has been reported in association with bihemispheric *seizure disorder*. Following successful surgery to control the epilepsy,[24] children may show improvement in measures of central auditory function, but results are variable.

Landau–Kleffner syndrome is characterised by acquired aphasia and epileptic seizures, with onset in childhood.[25] The major feature of the disease is the inability to understand spoken language; this has in turn been interpreted as reflecting an impairment of auditory phonological discrimination,[26] a generalised auditory agnosia rather than a phonological decoding deficit,[27] or a phonological deficit underlined by insensibility to loudness and a defect in temporal resolution.[28] The length of electrical status epilepticus in sleep has a strong negative correlation with receptive as well as expressive language scores, highlighting the need for timely medical or surgical intervention.[29]

DELAYED MATURATION OF THE CENTRAL AUDITORY PATHWAY

The human auditory system is fully developed at birth; however, *myelination* continues for several years in the higher auditory pathways, as reflected in ABR and middle/late auditory potentials indices, which reach adult values around 2 years of age and by 10–12 years of age respectively,[10] as well as in the improved behavioural performance with age in several behavioural central auditory tests.[9] *Auditory deprivation* may have deleterious effects on the organisation of the auditory pathway; thus maturation of some aspects of central auditory function may be limited by the onset and duration of the *period of deafness* prior to cochlear implantation.[30] Similarly, auditory deprivation may underlie delayed maturation of the central auditory pathway in children who have a history of *glue ear*, and who show significantly poorer performance in behavioural as well as prolonged ABR wave latencies[31] than normal controls.

DEVELOPMENTAL ABNORMALITIES

Attention deficit hyperactivity disorder

The diagnosis of attention deficit hyperactivity disorder (ADHD) is made on the basis of reported symptoms of inattention, impulsivity, and hyperactivity that are developmentally inappropriate and which are observed in at least two different settings. In contrast, APD is diagnosed on the basis of history, audiometric behavioural and electrophysiological test findings, and ancillary procedures (see table 3). Shortcomings arising from diagnostic methodologies and overlapping symptomatology of the two conditions may account for the debate as to whether APD and ADHD are a single[32] or two distinct but co-morbid developmental disorders.[33] Clinicians can identify a reasonably exclusive set of diagnostic behavioural characteristics for ADHD and APD.[33] However, consistency does not ensure validity of the diagnosis, and APD and the predominantly inattentive subtype of attentional deficit disorder may yet be a single developmental disorder.[33] Neurophysiological studies have found smaller MMN amplitudes to auditory stimuli in children with ADHD,[34] and this may underlie the presence of an auditory processing deficit; however, this is not a specific finding. There is a clear need to identify electrophysiological indices that would permit a confident diagnosis of ADHD and/or APD in order to choose appropriate modes of treatment.

Table 3 Behavioural signs of APD and ADHD, as ranked by clinicians in terms of frequency (high to low) for each disorder

	ADHD	*APD*
1	Inattentive**	Difficulty in hearing noise
2	Distracted*	Difficulty following oral instructions
3	Hyperactive	Poor listening skills
4	Fidgety or restless	Academic difficulties
5	Hasty or impulsive	Poor auditory association skills
6	Interrupts or intrudes	Distracted*
7		Inattentive**

Adapted from Chermak and colleagues.[33]
Asterisks represent commonly observed behaviours in ADHD and APD; note the difference in ranking.

Dyslexia

There is still debate as to whether dyslexia is a specifically linguistic disorder[35] or whether the underlining phonological deficit is caused by an auditory temporal processing deficit.[36 37]

Characteristic structural abnormalities of auditory areas in the brain have been reported in dyslexics.[37–39] Behavioural studies have indicated that dyslexics suffer from temporal processing deficits which are differentially related to lexical and non-lexical reading strategies.[40]

There is abounding empirical support that an auditory processing deficit may underlie some forms of dyslexia; however, it needs to be clarified to what degree this temporal deficit affects other modalities such as vision, and whether other potential factors might contribute to dyslexia. We believe that dyslexics with listening behaviours strongly suggestive of an auditory deficit should be referred for detailed audiological evaluation and appropriate audiological rehabilitation.

Language impairment

The issue of an auditory temporal processing deficit as opposed to a purely linguistic deficit being causal to *specific language impairment* (SLI) (developmental dysphasia) remains controversial. Specific language impairment refers to language impairment that cannot be explained on the basis of neurological, cognitive, motor, or sensory deficits. However, this definition may be inappropriate, as from the early 1970s, there is evidence to support the causal link of an auditory processing deficit to specific language impairment.[41] Neuropathological studies in developmental dysphasia have identified structural abnormalities of auditory areas of the brain.[42] Subsequent studies led to the hypothesis that the temporal processing deficit may also affect other sensory modalities, but the auditory processing deficit seems to be of more crucial importance for the language impairment.[43] The strongest argument in favour of the auditory basis for SLI comes from remedial studies which found significantly greater improvement in auditory and language processing in children with SLI who received training with acoustically modified speech than in the control group who had been trained with natural speech.[44]

Learning disability

In some cases, learning disabled children may have central auditory deficits similar to those observed in adult patients with surgical section of the corpus callosum, indicating that the auditory deficits may be due to disruption of interhemispheric processing of auditory information, possibly due to delayed myelination.[5] These children may also have longer latencies of the middle latency response[45] as well as diminished MMN to stimuli of specific rapid speech changes than normal children.[46] Learning disabled children are a heterogeneous group; however, identification of underlying specific auditory deficits may indicate what remedial action is appropriate.

Management

Management of APD (see table 4) consists of the following[47]:

- Signal enhancement strategies which aim to improve the signal to noise ratio, for example by minimising background noise or by using frequency modulated systems in the classroom.
- Auditory training which makes use of the brain's plasticity and can be formal (by means of sophisticated equipment and strictly controlled stimuli) or informal. Formal auditory training may include: computerised commercial programs such as FastForWord (Scientific Learning Corporation, 1997; http://www.scilearn.com/) and Earobics (Cognitive Concepts, Inc., 1997; http://www.earobics.com/), which alter speech acoustics and adaptively speed up neural processing; or training in the audiology clinic with modified central auditory tasks. Informal strategies can be applied at home or at school and include tasks such as vowel/consonant training, simple games such as "Simon", etc.
- Linguistic and cognitive strategies which aim to increase use of compensatory strategies.

APD management is not without controversy. As critics point out,[48] these interventions are based on certain assumptions, including a bottom up (sensory to cognitive) model of the brain's processing of incoming speech signals, the assertion that auditory processing defects cause language impairments, and the acceptance that targeting the auditory defect by a training programme will lead to improved language. However, these assertions are still under debate. Despite this continuing debate on the exact relation of audition and language, there is a growing body of evidence that APD management is beneficial.[44] While further research is needed to clarify why and how this management actually works, it is important to identify and to address these auditory processing deficits by appropriate specific strategies.

Conclusion

Auditory processing disorders may be a feature of both neurological and developmental disorders. However, whereas APD appears to be

Table 4 Management strategies forAPDs

Management strategies	*Examples*
Signal enhancement strategies	Minimise background noise Minimise reverbation levels Assistive listening devices: – Personal FM systems – Classroom FM systems
Auditory training	Formal: – Earobics – Fast ForWord – Test driven auditory training in clinic Informal: – Vowel/consonant training – Auditory directive tasks – "Simon" game
Linguistic strategies	Vocabulary building
Cognitive strategies	RIDER: Read, make an Image, Describe image, Evaluate for completeness, Repeat for next sentence

well documented in relation to certain syndromes, in other disorders the quality of existing evidence is inconclusive and the relation of APD to the coexisting disorder is poorly understood. Further research into the interface between APD and neurological and developmental disorders is needed. Clear insight into the nature of the auditory processing deficit may have implications for appropriate management, in agreement with the trend to provide multimodal intervention for these disorders. Moreover, a detailed understanding of the structural and functional substrate of auditory processing disorders will enable phenotypic evaluation specifically for the purposes of genetic research.

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