

Short title: Profile and aetiology of children diagnosed with Auditory Processing Disorder (APD)

Word count: 4800 (including references & tables)

Key words: Auditory processing disorder, aetiology, otitis media, obstetric optimality

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Abbreviations: ASD; Autistic spectrum disorder, CHAPS; Children’s Auditory Performance Scale, DPT; Duration Patterns Test, GOSH; Great Ormond Street Hospital, GIN; Gaps in Noise, OES; Obstetric Enquiry Schedule, PPT; Pitch Patterns Test, RGDT; Random Gap Detection Test

Abstract

Objective: Auditory processing disorder (APD) is characterised by listening difficulties despite a normal audiogram. APD is becoming ever more widely diagnosed in children, though there is controversy over definition, diagnosis and aetiology. This study sought to describe presenting features and investigate aetiological factors for children diagnosed with APD compared to those for whom APD was excluded.

Methods: Medical notes for children referred to an specialist hospital-based APD clinic were reviewed in relation to presenting features and potential aetiological factors.

Results: 32 children diagnosed with APD and 57 non-APD children were compared. They reported similar symptoms and had similarly high rates of co-morbid learning problems. No aetiological factor (including history of otitis media, adverse obstetric history or familial history of listening problems) predicted APD group membership.

Conclusions: Children identified with APD on the basis of commonly used APD tests can not be distinguished on the basis of presenting features or the aetiological factors examined here. One explanation is that learning problems exist independently of auditory processing difficulties and the aetiological factors do not have a strong causal role in APD. However, no gold standard for APD testing exists and an alternative explanation is that the commonly used APD tests selection criteria for this study were based on may be unreliable.

Introduction

APD is diagnosed on the basis of difficulty identifying or discriminating sounds despite normal peripheral hearing. Difficulty understanding speech in noise is the most common

manifestation. Although it does not feature in mainstream diagnostic classifications such as DSM-IV or ICD-10, APD is becoming more widely diagnosed. However, controversy exists over definition of APD, diagnosis, association with learning problems and causation. We studied a sample of children referred to a specialist APD clinic to describe and compare the presenting features of APD and non-APD children as well as examine the role of various possible aetiological factors in APD.

Chermak and Musiek (1997) noted that cases with an obvious neurological etiology are in the minority (~5%). For the remainder, the concept of ‘neuro-developmental disorder’ or ‘maturational lag’ is usually invoked, but the etiology of this is not clear and there is little evidence for the role of any particular risk factor. Inherited factors and/or obstetric complications may relate to neuro-developmental auditory processing problems while auditory deprivation due to otitis media has been suggested to underlie a maturational delay (Bamiou, Musiek, & Luxon, 2001). Musiek, Gollegly and Ross (1985) reported that a genetic contribution was suspected for some children with APD of ‘neuro-developmental origin’, with many having family histories of learning disabilities. Note, however, that Musiek et al did not distinguish between learning problems such as specific language impairment (SLI), for which there is good evidence for heritability (Bishop, 2006) and more specific auditory problems.

The first part of this article seeks to characterise children diagnosed with APD in comparison to children for whom an APD diagnosis is excluded (non-APD) in terms of terms of age, sex, co-morbid conditions and reported symptoms. The second part explores factors that may be significant in the aetiology of auditory processing problems.

Methods

Data were analysed retrospectively from a research database containing demographic details, case history, symptoms, co-morbid conditions, audiological test results and suspected aetiology. Information was entered into the database from medical case notes.

Participants

At the time of writing, data were available for 149 children referred to a specialist APD clinic at Great Ormond Street Hospital (GOSH) in London. All children underwent pure tone audiometry, otoscopy, and tympanometry. Those with peripheral hearing outside clinical norms ($N = 5$) were excluded from further analysis. Additional tests of auditory processing were administered as described below. APD was defined as a composite SCAN test score of less than -1 SD below the mean, with reference to US population-based performance norms, and failure on one or more tests of auditory processing described below (RGDT, GIN, PPT or DPT). 'Non-APD' children were those who had a SCAN composite score greater than -1 SD below the mean or normal scores on the tests of auditory processing or both. The SCAN composite score was an average of the first two SCAN subtests; Auditory Figure-Ground (AFG) and Filtered Words (FW). For two cases that were missing the FW subtest, the SCAN composite was taken to be the score for the AFG subtest only. Children were not classified if they were missing the SCAN, or if they had not done any of the other tests of auditory processing. Using these criteria, 32 children were identified as APD (14 females, 18 males), with 57 being classed as non-APD (17 females, 40 males). 55 children could not be classified as they had incomplete test results.

Sample sizes of 32 APD cases and 57 non-APDs provide a power of 84% to detect a moderate effect size of 0.6.

1. Assessments used for diagnosis of APD

SCAN

The SCAN is a US-produced standardised test of auditory processing, and is the most commonly used instrument for diagnosis of APD (Hind, 2006). It is composed of four subtests including i) discrimination of monaurally presented single words against background noise, ii) acoustically degraded single words and iii) dichotically presented single words and iv) sentence stimuli. The child version, the SCAN-C (Keith, 2000b) is for use with children aged 5 to 11 and the SCAN-A (Keith, 1994) for those aged 11 plus.

Random Gap Detection Test (RGDT)

The RGDT (Keith, 2000a) is a standardised test that assesses an individual's gap detection threshold of tones and white noise. The test includes stimuli at four frequencies (500, 1000, 2000, and 4000 Hz) and white noise clicks of 50 microseconds duration. This test provides an index of auditory temporal resolution. In children, an overall gap detection threshold greater than 20 ms constitutes failure (Keith, 2000a). This point is slightly below -2 SD, based on US population-based normative data for children 5 to 11:11 published with the RGDT.

Gaps in Noise Test (GIN)

The GIN (Musiek et al., 2005) is another measure of auditory temporal resolution. The test assesses an individual's gap detection threshold in white noise. Comparative performance data exist for adult normal-hearing listeners and adults with confirmed neurological involvement of the auditory nervous system.

Pitch Patterns Sequence Test (PPT) and Duration Patterns Sequence Test (DPT)

The PPT (Musiek, 1994; Pinheiro, 1977) and DPT (Musiek, 1994; Musiek, Baran, & Pinheiro, 1990) are both measures of auditory pattern identification. The PPS consists of series of three tones presented at either of two pitches, for example 'high high low' or 'low low high'. The DPS consists of series of three tones that vary in duration rather than pitch, for example, 'two short, one long' or 'one long, one short, one long'. Individuals are asked to describe the pattern of pitches presented. US population-based normative data are provided for children aged 6 through 9 for the PPS, though only adult performance norms are available for the DPS.

2. Dependant variable measures

Symptoms

Presenting symptoms were based on parental report during the initial case history interview. Additionally, listening difficulties were quantified using the following two questionnaires.

Children's Auditory Performance Scale (CHAPS) and Fisher's Auditory Checklist

The CHAPS (Smoski, Brunt, & Tannahill, 1998) is a screening questionnaire for listening difficulties. Parents or teachers are asked to compare the child's listening in a range of

conditions such as 'Multiple Inputs', 'Ideal' or 'Noise'. The CHAPS provides average scores for each condition as well as a total score. The CHAPS recommends referral for APD evaluation if the average total score or any of the average scores for each condition are lower than -0.05.

Fisher's auditory checklist (Fisher, 1976) is a screening questionnaire for listening difficulties that can be completed by parents or teachers. It is comprised of the list of 25 statements, such as "*Says "Huh?" and "What?" at least five or more times per day*" and "*Experiences problems with sound discrimination*". However, many of these statements are not specific for APD, such as "*Has a short attention span*", "*Has a language problem (morphology, syntax, vocabulary, phonology)*" and "*Has an articulation (phonology) problem*". The number of items checked is scored as a percentage, which can be compared against norms for 5 to 11 year olds. The authors recommend referral to an audiologist for APD examination if a child's score is poorer than 72%, close to below one standard deviation below the mean.

Aetiological Measures

Otitis media

In order to examine history of OME as a risk factor for later diagnosis of APD, a positive history of chronic OME was identified if the child had had one or more sets of ventilating tubes inserted during their lifetime.

Obstetric Complications

Parents of children referred to GOSH for APD testing received a standard case history interview, including a relevant obstetric history. Obstetric history obtained from this interview was re-coded according to the Obstetric Enquiry Schedule (OES) (Bolton et al., 1994), as used by Merricks and colleagues (2004). The OES systematically records information about the pre-, peri- and early neonatal periods. Non-optimal events are coded and summed to provide an overall measure (the optimality score) of obstetric conditions. A score of zero reflects an optimal birth with increasing scores reflecting increasingly sub-optimal births. Earlier research using the OES found good reliability on the basis of maternal report compared to obstetric records ($r = .72$) (Bolton et al., 1997).

Familial contributions

A positive history of listening difficulties was identified if a member of the immediate biological family had listening difficulties, excluding peripheral hearing loss. Positive family history of a developmental disorder was identified if an immediate family member had an autistic spectrum disorder (ASD), dyslexia, language problems, attention deficit hyperactivity disorder (ADHD) or dyspraxia.

Results

Presenting Features

Proportion of males and females and average age were not significantly different between groups. The average age of the APD children was 10.3 (*sd* 2.6) and 10.1 for the non-APD group (*sd* 2.0). Average SCAN composite scores, average parental Fisher's checklist percentile and average parental CHAPS scores are shown in Table 1. There was no

significant difference between average percentile score on Fisher's checklist or on total CHAPS score between groups.

Insert Table 1 here

Symptoms

Table 2 contains a list of symptoms that were reported during clinical interview in order from most to least common as well as the average number of symptoms reported by group. Symptoms that occurred in less than 10% of APD children are omitted. Difficulties with hearing speech in noise, difficulties with spoken instructions, difficulties with reading or spelling, poor concentration and memory, hyper-sensitivity to sounds and needing the TV loud were the most characteristic symptoms of children with APD. However, these symptoms were not specific to those with APD; there were no significant differences in occurrence of any symptoms between non-APD and APD children. The average number of reported symptoms was also similar for both APD and non-APD children, with an average of between 3 and 4 symptoms reported.

Insert Table 2 here

Co-morbid conditions

Table 3 shows the proportion of APD and non-APD children whose parents also reported that they had a condition co-morbid with listening difficulties. Categories are not mutually exclusive. Also shown are prevalence estimates in the general population; these will vary depending on criterion for identification and are therefore not intended for direct statistical

comparison. Rather, they are included to give the reader a feel for the characteristics of children referred for APD testing compared to children in the general population.

Very few children had a formal diagnosis of 'SLI' or 'language impairment'. Instead, a history of having received speech therapy for a language-based problem was examined. Language and literacy problems were most common among APD children. A substantial minority of APD children had ADHD or autism. Autism in particular is over represented in the children referred to GOSH for APD testing, compared to the prevalence in the general population. Prevalence of epilepsy and genetic disorders is also high within children referred for APD testing, though this may be explained by the APD clinic's involvement in epilepsy and Gaucher disease research studies. A significant proportion in both groups (~20%) had multiple conditions. Comparative frequency analyses found no significant differences in the incidence of any condition for the APD and non-APD subgroups.

Insert Table 3 here

Aetiological factors

For the aetiological analysis, two children with an identifiable neurological cause for their auditory processing problems (Landau-Kleffner syndrome and Gaucher-related brainstem abnormalities) were excluded from the APD group.

Otitis Media

29% of APD children (5 of 17 with complete AP test data) compared to 10% of non-APD children (4 of 38) had a history of chronic OME. In a large national UK birth cohort study, 10.9 to 12.1% of children had at least one episode of otitis media with effusion or hearing difficulty by age 5 (Bennett & Haggard, 1998). There seems to be a possible trend for higher rates of OME among children with APD than in the general population, though there was no difference in the proportion of children with a history of chronic OME between APD and non-APDs (Fisher's exact test $p = 0.27$).

Obstetric complications

In order to compare rates of obstetric complications (OCs) among children with APD and a comparable group of children, a post-hoc comparison with a sample of children with SLI and their non-affected siblings from the study of Merricks and colleagues (2004) was carried out. The mothers of 5 children out of the 148 in the GOSH sample (3.4%) were identified as having had toxæmia. This compares to 6% in the Merricks et al sample and estimates of 5 to 7% of pregnancies in the general population (Roberts & Cooper, 2001). Within the GOSH sample, there was no difference between APD children and non-APDs in the incidence of probable toxæmia ($U = 888.0$, ns, $r = -.06$) or in general rate of OCs ($U = 812.0$, ns, $r = -.10$).

Further analysis of the relationship between OCs, toxæmia and APD using logistic regression to provide odds ratios found no evidence for an association between either OCs or toxæmia and auditory processing problems (see Table 4).

Insert Table 4 here

Familial contributions

41% (13 out of 32) of parents of APD children reported a close relative with a developmental disorder (Autism or ASD, dyslexia, language problems, ADHD or dyspraxia), compared to 23% of non-APD children (13 out of 57). 16% (5 of 32) reported listening difficulties (excluding peripheral hearing loss) (non-APD; 21%, 12 of 57). Proportions of familial listening difficulties and learning difficulties were not significantly different between non-APD and APD children. Family history of listening difficulties was not a significant predictor APD group membership, $\chi^2 (1) = 0.4$, ns, $R^2 = 0.00$ (Cox & Snell), 0.01 (Nagelkerke), Beta = 0.36 (0.58 expected), ns, CI 0.46 to 4.53.

Effect of changing case criteria

Research with the SCAN suggested UK children score significantly more poorly than the US-based norms (Dawes & Bishop, in press; Marriage, King, Briggs, & Lutman, 2001). Raw SCAN scores were not available to enable us to adjust GOSH children's scores in line with method suggested by Dawes and Bishop (in press). Rather, as UK children scored close to one standard deviation poorer than US norms on the total SCAN score, participants were classified as APD if they scored lower than -2 standard deviations on the composite SCAN score (rather than -1 *sd* that the above analysis was based on) and failed one or more other tests of auditory processing (RGDT, GIN, PPT or DPT). This more conservative

criterion yielded 23 children with APD with the remaining 66 classed as non-APD. The above analyses were re-run. Results were very similar to the above, with no differences between APD and non-APD groups in terms of type and number of reported symptoms, co-morbid conditions, CHAPS or Fishers checklist scores, developmental history, family history or history of OME. Neither OCs, toxaemia nor OME history were significant predictors of APD group membership.

Discussion

Among children referred for investigation of APD, there were no differences between those who did and did not receive an APD diagnosis, either in symptoms or co-morbid conditions. The CHAPS and Fisher's APD screening questionnaires also did not differentiate between groups. A high proportion of both APD and non-APDs had co-morbid conditions, especially those related to language and literacy. Overall, non-APD and APD children did not differ in any respect apart from performance on tests of auditory processing used to define the groups. It is possible that for some children, auditory processing difficulties may co-exist with and exacerbate learning problems. However, if this is the case, there seem to be no readily identifiable differences case history or behavioral symptoms that can discriminate between those with and without auditory problems.

An alternative possibility is that the diagnostic criteria used to identify auditory processing problems are not reliable. Reliability of the GIN, RGDT, PPT and DPT is not known, though moderate retest reliability is reported for the SCAN-C with individual subtest

reliability ranging from 0.65 to 0.82 (Keith, 2000b). However, our recent work with children with APD suggests that SCAN reliability may be lower for children with APD. In research conducted at the Experimental Psychology Department at Oxford University, 9 children with APD recruited through Audiology clinics were given the SCAN-C less than 9 months after it was administered at each child's respective referring centre (median time interval, 4.5 months). 4 of these 9 children scored within 1 standard deviation of their initial score while 5 children scored more than 1 standard deviation different (4 better, 1 worse). Concerns over the reliability of auditory processing tests have been voiced previously (Cacace & McFarland, 1995). Cacace and McFarland noted that that reliability should be established for both different age groups of normally developing children and specific populations (ie APD). The SCAN-C has the former but not the latter. Cacace and McFarland also noted that the reliability of most auditory processing tests is unknown. Without reliable auditory tests, a reliable clinical diagnosis can not be obtained and APD research can not progress. Reliability of tests is a crucial factor in future APD research.

In terms of aetiology, there was no evidence for a role for toxemia or other adverse obstetric events as risk factors for a diagnosis of APD. There was also no evidence for a history of OME or a family history of listening difficulties as risk factors. These findings need to be interpreted with caution, however, as this study has a number of limitations. Firstly, this study may underestimate the incidence of OCs in the children seen for APD assessment. While Merricks et al interviewed mothers and specifically asked for a response to each item on the OES, in the current study, obstetric information was re-coded using the OES from APD consultation case notes. The current study may thus miss relevant obstetric data. A possible indication of this is that the rate of toxemia in the current study (3.4%)

seems low in comparison to both the rate in Merricks et al (6%) and that estimated for the general population (5 to 7%; Roberts & Cooper, 2001).

The rate of language problems for both APD and non-APD children was high compared to the general population. In the field of language disorders, inherited factors have been recognized as aetiologically significant (Bishop, 2006). In the case of APD, though familial learning problems appeared high among children referred for AP testing, they did not differ between APD and non-APD groups. It should also be borne in mind that the index of familiarity included a wide range of developmental disorders, and the rate of familiarity that would be found in the general population on this index is not known.

Finally, all the children in the GOSH sample were referred for APD testing because of unexplained listening difficulties and assessment was carried out following the latest ASHA recommendations (2005), but there is no agreement on a gold standard for APD diagnosis. Though APD in this sample was defined using common AP tests and relatively conservative criteria (children had to score poorly on more than one test of auditory processing to be identified as APD), this may not be the ideal way to diagnose APD. In future, a better method of identifying central auditory processing problems in both children and their family members may lead to different estimates of the role of familiarity, obstetric factors and OME in risk for APD as well as better understanding of auditory processing problems and their impact on children's development and daily life.

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Table(s)

Table 1. Average auditory checklist scores and SCAN scores by group

	Group	N	Mean	Std. Deviation
Fishers %*	APD	8	48.0	26.4
	Non-APD	21	47.8	16.9
Total CHAPS#	APD	10	-2.1	1.0
	Non-APD	21	-2.0	2.9
SCAN composite†	APD	32	2.8	1.6
	Non-APD	57	5.1	2.1

*A score of 72% or lower (close to -1sd) on Fisher’s checklist is the recommended cut-off score for referral for APD examination.

#Total scores lower than - 0.05 should be referred for APD investigation, according to CHAPS guidelines.

†SCAN composite scores are standardized with a mean of 10 and a standard deviation of 3.

Table(s)

Table 2. Incidence and type of reported symptoms for children diagnosed with APD and those for whom APD has been excluded

Symptom	APD (N = 32)	Non-APD (N = 57)
Difficulties with speech in noise	20 (66%)	42 (76%)
Reading problems	15 (47%)	18 (32%)
Difficulties with spoken instructions	11 (34%)	28 (49%)
Spelling problems	12 (37%)	17 (30%)
Concentration problems	7 (22%)	12 (21%)
Memory problems	7 (22%)	14 (25%)
Hyperacusis	6 (19%)	13 (23%)
Needs TV loud	6 (19%)	3 (5%)
Pragmatic/social problems	4 (13%)	11 (19%)
Average number of symptoms reported	3.4 (1.8)	3.8 (1.8)

Table 3. Incidence and type of co-morbid conditions for children diagnosed with APD and those for whom APD has been excluded

Condition	APD (N = 32)	Non-APD (N = 55)	General population estimate
Dyslexia	8 (25%)	7 (12%)	5.3% to 11.8% (Katusic, Colligan, Barbaresi, Schiad, & Jacobsen, 2001)
Seen a SLT for language	4 (13%)	6 (11%)	7.4% (SLI) (Tomblin et al., 1997)
Articulation problem	0	4 (7%)	3.8% (at age 6) (Shriberg, Tomblin, & McSweeny, 1999)
ADHD	3 (9%)	5 (9%)	6% (St Sauver, Barbaresi, Katusic, Weaver, & Jacobsen, 2004)
Dyspraxia	2 (6%)	7 (12%)	13.5% (Kadesjo & Gillberg, 1999)
Autism/ASD	3 (9%)	5 (9%)	<<1% (Barbaresi, Slavica, Colligan, Weaver, & Jacobsen, 2005) (Powell et al., 2000)
Genetic syndrome*	1 (3%)	3 (5%)	<<1% (Gaucher disease) <<1% (Lysosomal storage disorders in total) (Meikle, Hopwood, Clague, & Carey, 1999)
Epilepsy*	4 (13%)	3 (5%)	<<1% (Cockerell, Eckle, Goodridge, Sander, & Shorvon, 1995)
Comorbid conditions (two or more of the above)	7 (22%)	11 (20%)	

*Note: Children with epilepsy and Gaucher disease were referred for APD testing as part of other GOSH research studies. Children with epilepsy and genetic syndromes may thus be over-represented.

Table(s)

Table 4. Median optimality score and incidence of toxaemia in each participant group. Odds ratio for the risk of being a case compared to a control for each group.

		Obstetric optimality			Toxaemia	
		Median optimality	Inter-quartile range	Odds ratio (CI)	Possible Toxaemia	Odds ratio (CI)
GOSH	APD (N = 32)	0	2	1.18 (0.91, 1.53)	3% (1 case)	1.07 (0.49, 2.31)
	Non-APDs (N = 57)	0	1		0% (no cases)	
Merricks etal (2004)	Controls (N = 61)	1	2	1.15 (0.87, 1.52)	5% (3 cases)	2.25 (0.37, 13.8)
	SLI cases (N = 37)	2	2		8% (3 cases)	
	All (probands and sibs) (N = 194)	1	1.5		6% (12 cases)	