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Central Auditory Processing Disorder Profile in Premature and Term Infants

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Abstract

Objective—The aim of this study is to compare central auditory processing disorder (CAPD) profile between children born prematurely and at term.

Methods—A retrospective study involving children 7 to 13 years of age who were referred for CAPD evaluation over the past 3 years. Parental reports and medical records were used to collect information. Children with a score ≥ two standard deviations below the mean for at least one ear on at least two different CAPD tests were considered to have CAPD.

Results—A total of 82 children were evaluated for CAPD of which 22 met exclusion criteria, resulting in 60 children with CAPD (15 premature and 45 term). Premature children had higher prevalence of cesarean section delivery and neonatal jaundice compared with term children. Premature children had a higher total number of failed CAPD tests compared with the term children. Among CAPD tests, there was an increased frequency of abnormal Phonemic Synthesis test (PST) and decreased frequency of abnormal Staggered Spondaic Word test (SSW) among premature children compared with term children.

Conclusion—Premature children differ in CAPD profile compared with term children. Findings suggest possible etiological differences for CAPD such as jaundice or differential susceptibility of premature children for altered PST and SSW performance when compared with the term children.

Keywords

speech and language delay; central auditory nervous system; jaundice; attention deficit hyperactivity disorder

The efficiency and effectiveness with which the central nervous system utilizes auditory information is referred to as central auditory processing (CAP). According to the consensus

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statement of the task force on CAP, the central auditory processes are responsible for sound localization and lateralization; auditory discrimination; auditory pattern recognition; temporal aspects of audition, including temporal resolution, temporal masking, temporal integration, and temporal ordering; and proper auditory performance in the presence of competing and degraded acoustic signals. An observed deficiency in one or more of the aforementioned is defined as central auditory processing disorder (CAPD) by the American Speech-Language-Hearing Association (ASHA, 2005) Working Group.[1]

Anatomically, the central auditory nervous system (CANS) includes nuclei and pathways in the brain stem, basal ganglia, thalamus, primary and association areas of cortex, and corpus callosum.[2] Direct toxicity to one of the areas of the CANS and/or neuromaturational delay during the critical period of the CANS development often results in auditory sensory deprivation which is thought to be the underlying pathogenesis for CAPD.[1,3] Therefore, CAPD is a heterogeneous disorder with varied presentation. The prevalence and etiology of CAPD in children are unclear.[1,3] Depending on etiology factors and associated underlying pathogenesis, the presentation and prevalence of CAPD may differ. Because children prematurely born and those born at term differ in neonatal morbidities, the difference in auditory processing profile between children born prematurely and those born at term may shed light on putative etiological factors; however, this has not been well studied. We hypothesized that premature infants will differ in abnormal CAPD profile compared with children born at term. The objective was to compare performance on behavioral CAPD tests between children born prematurely and at term who were diagnosed with CAPD. The study was approved by the Institutional Research Review Board and parental consent was obtained for the study.

Methods

Study Design

A retrospective observational study was performed to evaluate differences in abnormal auditory processing profiles between premature and term infants who were later diagnosed with CAPD during school age.

Study Population

Children 7 to 13 years of age who were referred for CAPD evaluation at the University of Rochester Medical Center, Department of Audiology, over the past 3 years and diagnosed with CAPD were considered for this study. Children with bilateral deafness or blindness were not considered for this study as it is difficult to evaluate these infants. Children younger than 7 years were also not included due to limited availability and variability of normative data in the literature because of task complexity and response demands of the task. We were interested in neonatal factors other than birth asphyxia and therefore infants with head injury requiring admission to the hospital, malignancy, birth asphyxia (Apgar score < 3 at 5 minutes), postneonatal culture proven sepsis, postneonatal encephalitis, postneonatal meningitis, or chromosomal disorders were excluded. Children who had the history of adoption were also excluded due to lack of medical information. Children who

were born < 37 weeks' gestational age were considered as children born prematurely while children born at ≥ 37weeks' gestational age were considered as term children.

Auditory Processing Disorder Evaluation and Definition

All children were evaluated by an audiologist in a quiet sound suite using standardized and validated behavioral CAPD tests which assesses a variety of auditory processes as well as various regions and levels within the CANS. The behavioral central auditory tests used were staggered spondaic word (SSW) test, filtered word test (FWT), dichotic digit test (DDT), pitch pattern sequence test (PPST), phonemic synthesis test (PST), auditory continuous performance test (ACPT), and synthetic sentence identification test (SSIT). SSW is a dichotic speech test that assesses binaural integration. FWT assesses auditory closure and auditory memory. The DDT assesses auditory memory and binaural integration. The PPST is a key measure of prosodic auditory processing. PST assesses auditory memory and decoding ability. ACPT is a test of auditory attention and impulsivity vigilance. SSIT is a test that assesses speech in noise function. SSIT was presented in as SSI-CCM (contralateral competing message) and no reading was required as the subjects were instructed to repeat any or all of the target (nonsense sentence) in the test ear. Failure to repeat the target sentence or a portion of it, along with including any portion of the contralateral competing message (story about Davy Crockett) in the verbal response was scored as incorrect. Children with a score two standard deviations or more below the mean for at least one ear on at least two different behavioral central auditory tests were considered to have CAPD.[1]

Each of the seven behavioral CAPD tests (SSW, FWT, DDT, PPST, PST, ACPT, and SSIT) was scored as either a pass or fail. The number of tests that an individual participant failed was summed and a total CAPD score was recorded. A score of two would indicate that an individual participant failed two CAPD tests and a score of seven would indicate that an individual participant failed all seven CAPD tests. The group of children born prematurely was compared with the group of term children for the total number of failed (or abnormal) behavioral CAPD test (range, 2–7). Second, the frequency of failed (or abnormal) individual behavioral CAPD test was compared between these two groups.

Medical records were used to collect information on perinatal and early childhood medical conditions and associated neurodevelopmental comorbidities. For missing information on early childhood, medical conditions and associated neurodevelopmental comorbidities including speech and language delay, autism spectrum disorder, and attention deficit hyperactivity disorder (ADHD), a standardized questionnaire was used to collect information from parents. Neonatal jaundice was defined as unconjugated hyperbilirubinemia requiring phototherapy.

Statistical Analyses

Student *t*-test was used to analyze continuous variables using Stata (Stata Corporation, College Station, TX). A Chi-square or Fisher exact test as appropriate was used to analyze nominal variables. All tests were two-sided and a $p < 0.05$ was considered statistically significant.

Results

A total of 82 children were evaluated for CAPD over a 3-year period. Of the 82 children, 11 children had normal evaluation on CAPD test. Of the remaining 71 children, 11 met exclusion criteria (seven with history of adoption, one with head injury, one with birth asphyxia, one with malignancy, and one with chromosomal disorder) and were not included in the study. Of the remaining 60 children diagnosed with CAPD, 15 were born prematurely (group 1) and 45 were born at term (group 2). There was no significant difference in demographics and common medical conditions except for gestational age, birth weight, mode of delivery, and neonatal jaundice between the two groups of children (Table 1). Children born prematurely were born more frequently via cesarean section and had a higher prevalence of neonatal jaundice compared with term children. Although less common than premature infants, one-third of term children were born via cesarean section and had recurrent middle ear infections, whereas 49% of term children had neonatal jaundice. There was high prevalence of speech and language delay and ADHD among children with CAPD. There was no significant difference in prevalence of speech and language delay, autism spectrum disorder, and ADHD between the two groups (Table 1).

There was a significant difference in the total number of failed (or abnormal) behavioral CAPD tests between the two groups with children born prematurely with higher total number of failed CAPD test compared with term children. The mean length of stay for premature infants in the neonatal intensive care unit was 40 days (range, 3–136 days). We found no correlation between length of stay and total number of abnormal APD tests ($r = -0.16$, $p = 0.4$). There was no difference in frequency of failed (or abnormal) FWT, DDT, PPST, ACPT, and SSIT between the two groups. However, there was a significant difference in the frequency of failed SSW and PST test between the two groups. Term children had significantly higher frequency of abnormal SSW measures when compared with children born prematurely. Children born prematurely had significantly higher frequency of abnormal PST compared with term children (Table 2). Table 3 compares the age corrected scores on individual components of the SSW and PST and the mean age the tests were performed between the two groups. Premature infants were a year older compared with the term infants at the time of CAPD evaluation and therefore the scores on individual subcomponents were corrected for the age at assessment. The score for an individual subcomponent of the SSW and PST for each individual subject is calculated by subtracting the observed score from the normal age expected score. A positive sign is given if the score is in the expected direction of improvement with age, whereas a negative sign is given if the score is not in the direction of improvement with age. In general, SSW subcomponent scores decrease with age while PST scores increase with age. As shown in Table 3, the term children performed less well on the majority of individual subcomponents of SSW; however, the difference in scores of individual subcomponents of SSW was not significant between the two groups. There was a trend for significance with lower (poor) PST scores among preterm children compared with term children.

Discussion

The precise causes for CAPD remain unclear; however, the current literature suggests that exposure to unknown exogenous factors during critical periods of brain development may play important roles in the development of CAPD. Because CAPD can result from adverse effects to one of the sites along the auditory pathway from the brain stem to auditory cortex, several etiological factors may be involved, but each individual etiological factor would have specific presentation depending on the site of lesion along the auditory pathway. It is therefore plausible that CAPD is a syndrome of multiple and separate disorders resulting from a range of etiologies rather than a unitary disorder. One plausible way to identify etiology factors is to identify a more homogenous phenotype of CAPD and then evaluate the association of CAPD subtypes with putative etiological factors. There are many differences between premature infants and term infants. Premature infants are not only exposed to more and varied medical problems that can adversely affect brain development but also the premature brain is generally more susceptible to injury from neurotoxins than the mature brain. Therefore, the difference in CAPD profiles between premature and term children may suggest either exposure to different putative etiologic factors or difference in vulnerability to the same exposure factors. Our findings, which we believe is the first report, suggest that premature infants with CAPD have higher prevalence of abnormal PST but lower prevalence of abnormal SSW when compared with term children. PST primarily evaluates decoding deficits whereas the SSW is a broader test evaluating decoding deficit, auditory memory, and organizational aspect of auditory processing abilities. Term children performed less well on most of the components of SSW compared with children born prematurely. The performance on these individual subcomponents of SSW explains the findings of more failed SSW (a composite outcome) among term children compared with children born prematurely.

We found a high prevalence of other neuromorbiditis such as speech and language delay and ADHD among our subject population which is consistent in the literature.[4] Although there was no significant difference in the prevalence of speech and language delay, there was a trend for higher frequency of speech and language delay in our premature infants as compared with term infants with CAPD. The coexistence of speech and language delay in children with CAPD may suggest more global brain dysfunction. It may also suggest the presence of the common etiologic factor responsible for both CAPD and speech and language delay. Identification of such an important etiological or biological marker can enhance our ability to further characterize CAPD subtypes and also provide ways to prevent CAPD.

We found that premature infants with CAPD have more total number of failed CAPD tests compared with term children with CAPD which is suggestive of more global CANS dysfunction from exposure to one or more neurotoxins. Neonatal jaundice has been shown to affect auditory pathway at multiple levels and may explain the higher total number of failed CAPD tests.[5–8] Neonatal jaundice may also be associated with speech and language delay which was found to be more common among premature infants.[9] To corroborate a possible association, jaundice requiring phototherapy was more common among premature infants as compared with term infants. Because of frequent coexistence of language

disorder, current recommendation is to have children suspected for CAPD to be evaluated by both a speech–language pathologist and an audiologist to determine areas of concern and weakness so as to determine the most appropriate intervention. The higher number of failed CAPD tests among premature infants compared with term infants also suggest that premature infants may require less number of CAPD tests during evaluation to diagnose CAPD. To diagnose CAPD, a test battery approach is recommended to evaluate four principal processes: closure task, dichotic ability, the temporal ordering task, and binaural interaction. Our findings suggest that SSW, FWT, DDT, and PST are tests commonly failed by premature infants and this information may be useful to the audiologist in choosing the test battery for evaluation in premature infants.

It is unclear why term children developed CAPD and had a higher prevalence of abnormal SSW. A large number of term children had significant jaundice requiring phototherapy, were delivered via C-section (presumably because of medical problems and anticipated risk to the fetus and mother), and/or had multiple ear infections during early childhood indicating that these term children were not normal. Jaundice and recurrent middle ear infections have been associated with peripheral hearing disorders and therefore may explain the findings of CAPD in term children.[6–8] These medical conditions may also explain the high prevalence of speech and language delay, autism, and ADHD noted in term children with CAPD.[9,10]

Most of the children with CAPD in our study were males which is not surprising. Male children are at higher risk of abnormal neurodevelopmental disorders compared with female children. Although this may be gender specific, it is more likely to be related to the increased frequency of neonatal morbidities among male infants compared with female infants. However, the finding that most of the children with CAPD were Caucasian is intriguing. Although the URMC Audiology is located in a large urban area, most of the children that were referred for evaluation of CAPD were Caucasian. It is possible that children of other races are less susceptible to CAPD or less likely to be identified and referred for CAPD evaluation. We suspect that the lower referral rate for other races is more likely to result from poorer access to testing including transportation and insurance coverage, or lower parental or school personal expectations.

The major limitation is that the findings are based on a retrospective study. Although this investigation was retrospective and may have some information bias, we gathered both parental information as well as reviewed medical records to categorize the risk factors. We specifically evaluated putative risk factors that may be seen in both the groups such as recurrent ear infections and jaundice. We excluded children with possible risk factors such as history of head injury, malignancy, meningitis, etc. Because neonatal morbidities such as intraventricular hemorrhage and cystic periventricular leukomalacia are rarely seen in term infants, these factors were not considered in our analyses. Second, we thoughtfully used phototherapy as a surrogate measure of significant jaundice requiring treatment to control for the difference in susceptibility to bilirubin-induced neurotoxicity based on total serum bilirubin concentration between premature and term infants. The degree of jaundice associated with bilirubin-induced neurotoxicity varies as a function of gestational age.[11] Premature infants are at a higher risk of bilirubin-induced neurotoxicity at a lower level of jaundice compared with term infants and therefore phototherapy is indicated at a lower level

of jaundice in premature infants compared with term infants. However, phototherapy is based on total serum bilirubin concentration which may be an inadequate measure of bilirubin toxicity as recent evidence favors the use of unbound bilirubin as a biochemical measure to predict bilirubin toxicity.[5,7] The participants were evaluated for CAPD by experienced audiologists using a recommended test battery. The high prevalence of language delay and ADHD noted in our subject population with CAPD is consistent with the literature and provide validity to our findings.

In summary, there is a significant difference in CAPD profile between children born prematurely and those born at term. The associated comorbidities and difference in CAPD profile may help us identify CAPD subtypes that may be unique to specific population and or etiology. Large prospective studies are urgently required to delineate CAPD subtypes and correlate to biological plausible etiologic factors. Such studies may help us to identify putative etiologic factors such as jaundice and inform underlying mechanisms. Identification of etiological factors is critical before preventive intervention can be implemented to decrease the prevalence of CAPD.

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Table 1

Demographic, clinical risk factors, and comorbidities in term and preterm children

	Term children (*n* = 45)	**Preterm children (*n* = 15)**	***p***
Gestational age (wk)[a]	39 ± 1	31 ± 4	0.00
Birth weight (g)[a]	3,425 ± 630	2,009 ± 908	0.00
Race (% Caucasians)	60	80	0.2
Gender (% male)	67	80	0.5
Mode of delivery (% C-section)	29	66	0.01
Neonatal jaundice (%)	49	94	0.002
Type of feeding during neonatal period (% predominantly breast milk)	34	75	0.2
Recurrent ear infections (≥ 5 during the first 3 y)	35	40	0.7
History of speech delay (%)	69	80	0.7
History of autism spectrum disorder (%)	8	6	1
History of attention deficit hyperactivity disorder (%)	27	20	0.7

[a]Mean ± standard deviation.

Table 2

Performance on CAPD test battery between children born prematurely and term

	Term children (*n* = 45)	**Preterm children (*n* = 15)**	***p***
Total no. of abnormal tests (mean ± SD)	3.6 ± 1.2	4.4 ± 1.4	0.04
Failed staggered spondaic word test (%)	98	80	0.04
Failed filtered word test (%)	95	93	1
Failed dichotic digit test (%)	60	67	0.7
Failed pitch pattern sequence test (%)	26	25	1
Failed phonemic synthesis test (%)	27	67	0.01
Failed auditory continuous performance test (%)	55	54	0.9
Failed synthetic sentence identification (%)	36	54	0.3

Abbreviations: CAPD, central auditory processing disorder; SD, standard deviation

Table 3

Staggered sporadic word test and phonemic synthesis test scores between term and preterm children

	Term children (*n* = 45)	**Preterm children (*n* = 15)**	***p***
Age tested (y)[a]	8.1 ± 1.6	9.0 ± 2.1	0.11
Subcomponents of staggered spondaic word test			
Total no. of errors[a]	−12.3 ± 16	−10.6 ± 12.9	0.7
RNC[a]	−0.66 ± 3	−0.6 ± 2.4	0.7
RC[a]	−2.57 ± 5	−2.53 ± 6.6	0.8
LC[a]	−6.3 ± 7.1	−6.1 ± 6.3	0.9
LNC[a]	−1.1 ± 3.2	−0.2 ± 1.2	0.2
Ear effect	−0.488 ± 3.2	−1.2 ± 2.2	0.28
Order effect	−0.622 ± 4.33	0 ± 3.66	0.54
Reversals[a]	0.06 ± 3.8	−2.4 ± 7.2	0.08
PST			
PST score[a]	3.37 ± 4.8	1.8 ± 4.1	0.13

Abbreviations: LC, left competing; LNC, left noncompeting; PST, phonemic synthesis test; RC, right competing; RNC, right noncompeting;

[a]Mean ± standard deviation.