Published in final edited form as:

Semin Hear. 2011 May 1; 32(2): 147–155. doi:10.1055/s-0031-1277236.

Clinical Application of the P1 Cortical Auditory Evoked Potential Biomarker in Children with Sensorineural Hearing Loss and Auditory Neuropathy Spectrum Disorder

Julia Dee Campbell, Au.D, Garrett Cardon, Au.D, and Anu Sharma, Ph.D

Brain and Behavior Laboratory, Speech, Language, & Hearing Sciences, 2501 Kittredge Loop Road, 409 UCB, University of Colorado, Boulder, CO 80309-0409, Fax: 303-492-3274

Abstract

The P1 component of the cortical auditory evoked potential (CAEP) shows clearly documented age-related decreases in latency and changes in morphology in normal hearing children, providing a biomarker for development of the auditory cortical pathways in humans. In hearing-impaired children, auditory deprivation may affect the normal age-related changes in central auditory maturation. Appropriate early intervention with amplification and/or electrical stimulation can provide the necessary stimulation needed to drive progress in central auditory maturation and auditory skill development, however objective measures are needed to evaluate the effectiveness of these treatments in infants and young children. We describe three pediatric cases, where we explored the clinical utility of the P1 as an objective biomarker of auditory cortical development after early intervention. We assessed development of P1 CAEP latency and morphology in two children with sensorineural hearing loss (SNHL) who received intervention with hearing aids (*case 1*) and cochlear implants (*case 2*) and a child with Auditory Neuropathy Spectrum Disorder (ANSD) (*case 3*). Overall, we find that the P1 CAEP serves as useful tool for assessing the effectiveness of early intervention treatment and clinical management of pediatric hearing-impaired patients.

Keywords

Cochlear implant; P1; biomarker; hearing aid; children; cortical auditory evoked potential; Auditory Neuropathy Spectrum Disorder; hearing impairment; pediatric

Introduction

Systematic age-related changes in the latency, amplitude and morphology of the obligatory cortical auditory evoked potential (CAEP) have been used for decades to evaluate changes in the development and functioning of the central auditory pathways in childhood^{1,2,3,4,5,6,7}. In young children, the obligatory CAEP is dominated by a large positive peak that occurs between 100 and 300 ms following the presentation of an auditory stimulus. This peak is known as the P1 component of the CAEP. The P1 is thought to arise as a result of synaptic activity in primary auditory cortex, thalamo-cortical projections, and intercortical recurrent activity^{8, 9,10}, providing information regarding early-stage auditory cortical processing.

The P1 latency is a useful biomarker of central auditory development since it is dynamic in nature. For example, in infants with normal hearing the P1 peak typically occurs at a latency

of approximately 300 ms. This latency value gradually decreases until the end of the second decade of life, at which time the P1 is typically observed around 60 ms. The decrease in peak latency is thought to reflect maturational changes in the cortex, such as improved synaptic connectivity, decreased refractory periods, and improved myelination, much of which occurs as a result of auditory stimulation^{10,11}. The amplitude and morphology of the CAEP waveform also vary with age, such that the P1 decreases significantly in amplitude by adolescence, giving way to a robust negativity followed by a large positivity (i.e., N1 and P2 components respectively)^{6,7}.

Sharma and colleagues have reported on the normal developmental trajectory for P1 latency. By fitting 95% confidence intervals to this trajectory, a normal range of development was established^{1,2,3,4,12,13}. Children who received appropriate auditory stimulation before age 3.5 years (via cochlear implants or hearing aids) showed normal development of P1 latencies, even if latencies were initially delayed. In contrast, children who were provided appropriate stimulation after the age of 7 years typically never exhibited normal P1 responses^{1,2,3,4,12}. These findings are relevant for at least three reasons. First, adequate stimulation of the auditory cortex through auditory prostheses in individuals with hearing loss is paramount to normal auditory cortical development. Adequate stimulation includes early identification and treatment, appropriate setting of hearing aids and cochlear implants, as well as sufficient experience with these devices. Second, P1 responses provide an index of normal central auditory maturation for use in evaluating normal cortical development in children with hearing loss. Finally, a sensitive period, or finite time frame of optimal cortical plasticity, has been documented in the studies described above. Intervention must occur during this short time period in childhood in order for normal cortical maturation to take place.

The P1 CAEP has also proven useful in the assessment of central auditory maturation in children with ANSD. Several studies have demonstrated that CAEPs are not only recordable in children with ANSD, but are also highly correlated with behavioral outcome^{14,15,16}. For example, Sharma and colleagues demonstrated that P1 latency, amplitude, and morphology served to divide a group of 21 children with ANSD into three distinct categories: 1) Children with normal P1 latency, amplitude and morphology; 2) Children with normal P1 morphology, but delayed latency and decreased amplitude; 3) Children with abnormal P1 response. When the mean scores on a measure of auditory skill development (IT-MAIS¹⁷) for each group were compared, it was reported that children with normal P1 responses showed superior auditory skill development over those who had delayed or abnormal P1 responses. Thus, the P1 may be a useful predictor of behavioral outcome in young children with ANSD. Additionally, children who received intervention with hearing aids early in life were more likely to show normal P1 responses and good behavioral outcome¹⁶.

As we have described above, the absence of appropriate auditory stimulation during a short sensitive period, early in childhood negatively affects cortical maturation. Lack of appropriate cortical maturation is correlated with abnormalities in speech and language development^{12,13,18,19}. Given that central auditory system plasticity peaks in the early years of life^{1,2,3,11}, there is a limited time frame within which to provide appropriate early intervention. Of course, it is not sufficient to simply provide early intervention. The effectiveness of these treatments needs to be rigorously evaluated in infants and young children. The P1 CAEP can be non-invasively recorded without any behavioral response from the subject, and therefore, represents an objective measurement technique for assessing the central auditory development in infants and young children. The maturational status of the central auditory pathways may serve as a marker of the effectiveness of early intervention. That is, children fitted appropriately with amplification or electrical stimulation ought to show, at the very least, normal development of the central auditory pathways.

In this report, three cases (two children with SNHL and one child with ANSD) are described to illustrate the clinical application of the P1 as a biomarker for central auditory development. These cases highlight the utility of the P1 as an objective clinical tool that may supplement conventional audiologic test measures in clinical management of pediatric hearing-impaired patients.

Methods

We present data from three hearing-impaired children, all male. Research protocols were in accordance with the University of Texas at Dallas and University of Colorado at Boulder Institutional Review Board guidelines.

P1 CAEP Recordings

CAEPs were recorded in response to a synthesized speech syllable /ba/ which had a total duration of 90 ms and was presented at an interstimulus interval of 610 ms (see Sharma et al., 2005 for stimulus details⁴). Note: Noise bursts or pure tones may also be used to elicit the P1 response; however it is important to consider the frequency spectrum of the stimuli in conjunction with the patient's degree and configuration of hearing loss when using novel stimuli. The stimulus was presented via loudspeaker placed at an angle of 45 degrees to the ear of interest. For bilateral responses, presentation occurred via a speaker placed at an angle of 45 degrees to each ear. Hearing aids and cochlear implants were set to the usual settings. The stimulus was presented at a comfortable loudness level (approximately 65 dB HL, $\pm$ 5 dB), unless the subject required a higher level of intensity due to the severity of auditory thresholds. In all cases, every effort was made to ensure that stimulus presentation levels were made suprathreshold by at least 20 dB, and audibility of the stimuli was ascertained through audiological records and behavioral observation of the subject.

Subjects were seated on their parent's lap in a comfortable reclining chair in a sound booth. Children were able to watch a movie on a television monitor placed directly in front of the chair in the sound booth. Audio levels were muted. CAEPs were collected using a Compumedics Neuroscan, Inc. electroencephalography (EEG) system. Silver/silver chloride cup electrodes were used for recordings, with the active electrode placed at Cz and the reference placed on one mastoid. Eye-blinks were monitored using a bipolar electrode montage (lateral outer canthus referenced to superior orbit), with eye-blinks rejected online during the test session. When performing clinical P1 testing, it is imperative to monitor for eye-blinks and ensure this artifact does not contaminate the recording. For children who received cochlear implants, multiple electrodes in addition to the Cz and mastoid sites were arranged across the forehead in order to minimize the artifact recorded on the scalp from the cochlear implants²⁰.

The recording window included a 100 ms pre-stimulus and 600 ms post-stimulus time, and the sampling rate was set at 1.0 kHz. Responses were filtered from 0.1 Hz to 100 Hz. Additional artifact rejection was performed offline at a threshold of $\pm$ 100 μ V. Two or more runs of at least 200 sweeps each were collected for each subject in order to determine waveform replicability, then grand-averaged. The peak of the P1 response was computed for each child and compared with the 95% confidence intervals for normal development of the P1 response^{1,2,3,12}.

Interpretation of the Waveforms

Assessment of the P1 peak in the CAEP response was performed by two or more clinical audiologists, so that agreement on peak reliability would be obtained. Low-pass filtering (30 Hz at 12 dB/octave) was performed after the P1 peak had been ascertained, and then only for

use in data presentation. In our experience, we have found that care must be taken when low-pass filtering or ‘smoothing’ the CAEP waveform, as this transform may alter the appearance of the CAEP resulting in P1 responses that are inaccurately picked. For example, if artifact, such as that generated by a cochlear implant during CAEP recordings is low-pass filtered, it may closely resemble a P1 response that does not actually exist. Furthermore, consideration of the ages of our subjects in waveform interpretation was of great importance as the morphology of the CAEP waveform changes as age increases. In older children and adolescents (approximately 7–11 years), the P1 bifurcates as the N1 develops, creating a P1-N1-P2 complex^{6,7}. To the untrained observer, the N1 of an older child may resemble a deprivation negativity¹³ (see Figures 1A and 2A) and vice versa. Thus, it is necessary for those interested in conducting P1 testing to become familiar not only with CAEP morphology in normal-hearing and hearing-impaired children, but with developmental effects resulting in morphological changes on the CAEP waveform.

Minimization of Artifact

Recordings of cochlear implant artifact, as well as the inclusion of unwanted eye-blinks, can cause serious misinterpretation in determining the P1 peak response. The aforementioned method of online eye-blink removal followed by artifact rejection offline typically produces CAEP waveforms free of eye-related myogenic artifact. However, it is also helpful to monitor the patient’s ongoing EEG during P1 testing to ensure that any eye-blinks are indeed rejected.

Of more serious concern is the cochlear implant artifact generated in CAEP recordings. When acoustic energy is transduced into electrical stimulation by a cochlear implant, an electrical field is generated over much of the scalp. This artifact may then contaminate and obscure possible P1 peaks in CAEP recordings, confounding interpretation. Our lab has dealt with the issue of cochlear implant artifact for several years, and has found specific methods that have proven effective in obtaining artifact-free P1 recordings in cochlear implant patients²⁰. The method of greatest clinical use, which was utilized in case 2 of this report, is the placement of multiple electrodes along the forehead in addition to the Cz and mastoid sites. This technique allows for a greater number of recording sites that may lie outside of the cochlear implant artifact field. However, the generation of artifact varies from patient to patient, making it near impossible to predict the optimal electrode site of recording. When performing P1 testing in a patient with cochlear implants, identification and subsequent rejection of the artifactual recording is a priority.

Case 1: Evaluation of early intervention with amplification—This male patient (AB) was referred for further testing after failing newborn hearing screening in one ear. Diagnostic auditory brainstem response (ABR) testing was performed at 0.87 years of age and revealed SNHL in the moderate range for the left ear and in the severe range for the right ear. The etiology of the hearing loss was reported to be unknown. AB was fit with bilateral hearing aids at 0.95 years, well within the sensitive period for central auditory system development^{1,2,3,12}. CAEP testing was performed at 0.95, 1.34, 2.43, 2.98 and 3.85 years of age in order to monitor central auditory development. Fig. 1A shows soundfield recordings which revealed a replicable and robust P1 response with a delayed latency was recorded at 0.95 years, the same age at which the patient was first fit with hearing aids. A deprivation negativity, which is a large negative response occurring before the P1 and considered a hallmark of an unstimulated central auditory pathway^{1,2,3,4,13}, was present, confirming the lack of adequate stimulation provided to the central auditory pathways. At age 1.34 years, after 4.5 months of hearing aid use, aided soundfield recordings showed the absence of the deprivation negativity and a robust P1 response whose latency was within normal limits for P1 latency development. Subsequent P1 recordings at 2.43, 2.98 and 3.85

years of age demonstrated a continued decrease in P1 peak latency, indicating normal longitudinal central auditory maturation via appropriate amplification (Fig. 1C).

Behavioral unaided thresholds for this patient at 2.98 years showed hearing loss in the severe-profound range bilaterally and aided thresholds in the mild-moderate range. Our findings of normal cortical maturation at an early age complemented AB's generally good results on a test of speech perception. For example, his speech perception scores at 3.85 years of age were 91% for the Multisyllabic Lexical Neighborhood Test²¹.

Case 2: Evaluation of cochlear implant candidacy and fitting—A male patient (CD) was referred to our laboratory after failing newborn hearing screening bilaterally. CD underwent diagnostic ABR testing at 2 weeks of age. ABR responses were absent bilaterally for 500, 1000, 2000, and 4000 Hz (maximum presentation level was 85 dB nHL), suggestive of a profound hearing loss. Distortion product otoacoustic emissions (DPOAEs) were absent bilaterally at all frequencies (2000–8000 Hz). Genetic testing revealed the cause of hearing loss to be a Connexin 26 defect; no history of hearing loss had been present in the family prior to this child. Powerful behind-the-ear hearing aids were fit bilaterally at 0.13 years of age, and P1 CAEP testing was conducted at ages 0.17, 0.27, 0.5 and 1 year to evaluate progress in central auditory development as a result of stimulation via amplification.

Aided soundfield testing revealed a deprivation negativity and a delayed P1 response at 0.17, 0.27 and 0.5 years of age (Fig 2A), suggesting that acoustic stimulation via hearing aids was inadequate for development of the central auditory pathways. At 0.5 years, aided thresholds were found to be in the severe-profound range. At this point, the family had decided to pursue simultaneous bilateral cochlear implantation. At 0.76 years (well within the sensitive period for central auditory development), this case was one of the youngest to receive bilateral simultaneous implantation in the state of Colorado.

At age 1 year, after less than 3 months of cochlear implant use, CAEP responses were recorded in response to the /ba/, presented separately to the right and left cochlear implant in soundfield. Both implants were set to the usual settings. A replicable and robust P1 peak response was identified for both the right and left ears (Fig. 2B). The latency for the P1 peak was within normal limits (Fig. 2C), suggesting that the cochlear implants were providing the necessary stimulation (not previously provided by the hearing aids) for normal development of the central auditory pathways. We will continue to monitor the central auditory development in this patient, as well as obtain measures of speech and language outcomes at appropriate ages.

Case 3: Management of pediatric Auditory Neuropathy Spectrum Disorder (ANSD)—This male child (RV) was born slightly premature and placed on mechanical ventilation at birth. Shortly after birth, he showed signs of jaundice and underwent antibiotic treatments with ototoxic drugs for possible meningitis. Initial ABR recordings showed an absent ABR response and a robust cochlear microphonic (CM) that inverted polarity when the click stimulus was reversed in polarity suggestive of a diagnosis of ANSD²². However, when the patient was 4 months of age a replicable wave V was noted in the ABR report, although it was delayed. A robust, inverting CM was also present in this recording. The patient's DPOAEs have always been present. These clinical findings lead to a diagnosis of ANSD by the patient's audiological team.

When the child was old enough to perform the task (7 months of age), behavioral audiometry was performed. Over several testing sessions, between the ages of 0.61 and 1.68 years, RV's auditory thresholds fluctuated between normal hearing and a mild to moderate hearing loss. Because of this fluctuation, hearing aid fitting was delayed until consistent

auditory thresholds could be obtained. His parents have regularly reported that this child's hearing abilities sometimes change from day to day, again, consistent with his diagnosis of ANSD. This patient has received weekly visits from an early interventionist who worked with both the patient and his parents to ensure proper early childhood development and service provision since birth. At age 1.76 years, this patient received a hearing aid in the right ear. However, parents and service providers report that the patient does not tolerate the device well and, therefore, he has used it inconsistently.

P1 testing was performed at 0.98 years of age. Stimuli were presented via soundfield to both ears simultaneously. P1 CAEP response is shown in Fig. 3A. Replicable and robust waveforms revealed P1 peak latencies in the normal range for this patient's age (Fig. 3B). The normal P1 indicates that, despite a neural dys-synchrony at the level of the VIII nerve, this patient's cortex had received adequate stimulation to drive normal cortical development. Given these results, one might expect this patient's behavioral performance to show reasonably positive outcomes¹⁶.

In order to evaluate behavioral performance, the Infant Toddler Meaningful Auditory Integration Scale (IT-MAIS¹⁷) was administered several times throughout this patient's history. The IT-MAIS is a clinician-directed parent interview that focuses on auditory skill development. This patient received a score of 34/40 at 1.87 years, which is consistent with results from patients with ANSD who showed good behavioral outcomes¹⁶. This observation is supplemented by clinical reports, which suggest that RV's speech and language development was determined to be age-appropriate. We will continue to monitor all aspects of this patient's development closely.

Discussion

We describe three cases in which the P1 CAEP was useful in assessment of early intervention and clinical management in children with SNHL and ANSD. The P1 has a unique role in identifying the extent to which the central auditory pathways may have benefited from amplification or implantation by reflecting the developmental trajectory for maturation of a given patient's central auditory system over the course of the treatment. For example, initial P1 results in cases 1 and 2 revealed that the P1 latency was delayed, and a deprivation negativity was present suggestive of unstimulated central auditory pathways. However, for case 1, the deprivation negativity disappeared and the P1 latency decreased to normal limits following sufficient (4.5 months of) experience with appropriate amplification. This result is consistent with our previous (unpublished) reports that when adequate stimulation is provided via a hearing aid P1 latencies decrease to within normal limits in a 3–5 month timeframe. Therefore, it is important to monitor the P1 prior to, or, at the time of fitting hearing aids (as a baseline) and then after several months of consistent use with the amplification device. The latency decrease in this timeframe provides an indication of the extent to which the central pathways have progressed in development due to the auditory stimulation.

In the case of patient 2, P1 latency recorded with hearing aids in place did not decrease, even though the child had been fit with powerful behind-the-ear hearing aids for approximately four and half months. After less than three months of experience with bilateral cochlear implants, P1 latencies decreased to within normal limits, suggesting that the cochlear implant was able to provide adequate stimulation for development of the central auditory pathways not provided by the hearing aids.

The P1 results for case 2 are in keeping with a larger (unpublished) study of 116 children under 2 years of age in whom we evaluated the sensitivity and specificity of the P1 response

in determining cochlear implant candidacy. In this data set, we found that the P1 biomarker had a sensitivity rate of (89%) and a specificity rate of (85%) when compared to the gold standard of the traditional comprehensive audiological battery in making cochlear implant candidacy decisions. Interpretation of this study must be viewed with caution as the clinicians often had access to the P1 data while making candidacy decisions. Nonetheless, these data provide some indication of the close correspondence of the P1 biomarker with behavioral testing for cochlear implant candidacy decisions.

The current report also describes the utility of CAEP measurements in a child with ANSD (Case 3). Behavioral testing provided highly unreliable and variable audiometric results, which fluctuated in the mild-moderate hearing loss range. Although, parents and professionals familiar with the case reported good auditory performance as well as speech and language development, given the diagnosis of ANSD, it was unclear whether the child was receiving the necessary auditory input required for normal auditory cortical development. Normal P1 results in this case served as a confirmation that the auditory cortex was being adequately stimulated. The combination of normal P1 results and reasonably good behavioral performance for case 3 are consistent with recent research supporting the claim that the maturational status of the P1 response and behavioral performance are correlated in children with ANSD^{14,15,16}.

While it is true that behavioral outcome is still the gold standard for measuring a patient's progress and the overall goal of clinical management, the P1 offers the benefit of immediate knowledge concerning cortical developmental status, often well before a child is of age for reliable speech perception testing. The cases we have presented here are examples of the benefit that children receive when they are provided with appropriate intervention early in life. However, while the P1 is useful as a marker of a plastic neural system, it cannot encompass the complex influences that lead to expert use of oral speech and language. As Geers (2006)²³ reports, many factors influence speech understanding and oral language development, including the amount and type of rehabilitation. None-the-less it is likely that the neural processes that constrain P1 latency also influence the complex auditory functions that underlie speech perception and oral language acquisition.

Conclusion

The P1 biomarker may have a role to play within the battery of audiological test procedures. When interpreted properly, P1 results may provide useful information regarding maturation of the central auditory pathways in children with hearing impairment. This information, when used cautiously in conjunction with other test results, may provide clinicians with better direction as they make clinical decisions about appropriate intervention and management of their pediatric patients.

Acknowledgments

This research was supported by NIH grant R01 DC006257 to A.S.

References

1. Sharma A, Dorman M, Spahr A, Todd NW. Early cochlear implantation in children allows normal development of central auditory pathways. *Ann Otol Rhinol Laryngol Suppl.* 2002a; 189:38–41. [PubMed: 12018346]
2. Sharma A, Dorman MF, Spahr AJ. Rapid development of cortical auditory evoked potentials after early cochlear implantation. *Neuroreport.* 2002b; 13:1365–1368. [PubMed: 12151804]

3. Sharma A, Dorman MF, Spahr AJ. A sensitive period for the development of the central auditory system in children with cochlear implants: implications for age of implantation. *Ear Hear.* 2002c; 23:532–539. [PubMed: 12476090]
4. Sharma A, Dorman MF, Kral A. The influence of a sensitive period on central auditory development in children with unilateral and bilateral cochlear implants. *Hear Res.* 2005a; 203:134–143. [PubMed: 15855038]
5. Ceponiene R, Rinne T, Naatanen R. Maturation of cortical sound processing as indexed by event-related potentials. *Clin Neurophysiol.* 2002; 113:870–882. [PubMed: 12048046]
6. Gilley PM, Sharma A, Dorman M, Martin K. Developmental changes in refractoriness of the cortical auditory evoked potential. *Clin Neurophysiol.* 2005; 116(3):648–657. [PubMed: 15721079]
7. Sharma A, Kraus N, McGee TJ, Nicol TG. Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalogr Clin Neurophysiol.* 1997; 104:540–545. [PubMed: 9402896]
8. Ponton CW, Eggermont JJ, Kwong B, Don M. Maturation of human central auditory system activity: evidence from multi-channel evoked potentials. *Clin Neurophysiol.* 2000; 111:220–236. [PubMed: 10680557]
9. Kral A, Eggermont JJ. What's to lose and what's to learn: development under auditory deprivation, cochlear implants and limits of cortical plasticity. *Brain Res Rev.* 2007; 56:259–269. [PubMed: 17950463]
10. Eggermont JJ, Ponton CW. Auditory-evoked potential studies of cortical maturation in normal hearing and implanted children: correlations with changes in structure and speech perception. *Acta Otolaryngol.* 2003; 123:249–252. [PubMed: 12701751]
11. Huttenlocher PR, Dabholkar AS. Regional differences in synaptogenesis in human cerebral cortex. *J Comp Neurol.* 1997; 387:167–178. [PubMed: 9336221]
12. Sharma A, Dorman MF. Central auditory development in children with cochlear implants: clinical implications. *Adv Otorhinolaryngol.* 2006; 64:66–88. [PubMed: 16891837]
13. Sharma A, Martin K, Roland P, et al. P1 latency as a biomarker for central auditory development in children with hearing impairment. *J Am Acad Audiol.* 2005b; 16:564–573. [PubMed: 16295243]
14. Kraus N, Bradlow AR, Cheatham MA, et al. Consequences of neural asynchrony: a case of auditory neuropathy. *J Assoc Res Otolaryngol.* 2000; 1:33–45. [PubMed: 11548236]
15. Rance G, Cone-Wesson B, Wunderlich J, Dowell R. Speech perception and cortical event related potentials in children with auditory neuropathy. *Ear Hear.* 2002; 23:239–253. [PubMed: 12072616]
16. Sharma A, Cardon G, Martin K, Roland P. Cortical maturation and behavioral outcomes in children with auditory neuropathy spectrum disorder. *Int J Audiol.* (in press).
17. Zimmerman-Phillips S, Robbins AM, Osberger MJ. Assessing cochlear implant benefit in very young children. *Ann Otol Rhinol Laryngol.* 2000; 109:42–44.
18. Sharma A, Nash AA, Dorman M. Cortical development, plasticity and re-organization in children with cochlear implants. *J Commun Disord.* 2009; 42:272–279. [PubMed: 19380150]
19. Sharma, A.; Dorman, M.; Biever, A.; Gilley, P.; Nash, A.; Campbell, J. A sensitive period for cortical development and plasticity in children with sequential bilateral cochlear implants. Anaheim, CA. Poster presented at: Annual Meeting of the Association for Research in Otolaryngology; February 2010; (2010, February).
20. Gilley PM, Sharma A, Dorman M, Finley CC, Panch AS, Martin K. Minimization of cochlear implant stimulus artifact in cortical auditory evoked potentials. *Clin Neurophysiol.* 2006; 117:1772–1782. [PubMed: 16807102]
21. Kirk K, Pisoni DB, Osberger MJ. Lexical effects on spoken word recognition by pediatric cochlear implant users. *Ear Hear.* 1995; 16:470–481. [PubMed: 8654902]
22. Berlin CI, Bordelon J, St John P, et al. Reversing click polarity may uncover auditory neuropathy in infants. *Ear Hear.* 1998; 19:37–47. [PubMed: 9504271]
23. Geers AE. Factors influencing spoken language outcomes in children following early cochlear implantation. *Adv Otorhinolaryngol.* 2006; 64:50–65. [PubMed: 16891836]

Learning Objective

As a result of this activity, the participant should be able to 1) discuss the effects of hearing loss on central auditory maturation in children and 2) discuss the clinical utility of the P1 CAEP as a biomarker in hearing-impaired children.

Figure 1. Case 1. (A) Aided P1 response at the time of initial hearing aid fitting (age 0.95 years). (B) Aided P1 response following 4.5 months after hearing aid fitting (age 1.34 years). (C) P1 latencies at ages 1.34, 2.43, 2.98 and 3.85 years compared to 95% confidence intervals for normal development of the P1 response.³

Figure 2.

Case 2. (A) Aided P1 response following 4 months of hearing aid (HA) use (age 0.5 years). (B) P1 response recorded for the right and left ears, respectively, after cochlear implant use of less than 3 months at age 1 year. (C) P1 latencies at ages 0.5 and 1 year compared with 95% confidence intervals (CI) for normal development of the P1 response.³

Figure 3. Case 3. (A) Unaided P1 response. (B) P1 latency at 0.98 years of age compared to 95% confidence intervals for normal development of the P1 response.³