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Electrophysiological Examination of Ambient Speech Processing in Children With Cochlear Implants

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ABSTRACT

Purpose: This research examined the expression of cortical auditory evoked potentials in a cohort of children who received cochlear implants (CIs) for treatment of congenital deafness (n = 28) and typically hearing controls (n = 28).
Method: We make use of a novel electroencephalography paradigm that permits the assessment of auditory responses to ambiently presented speech and evaluates the contributions of concurrent visual stimulation on this activity.
Results: Our findings show group differences in the expression of auditory sensory and perceptual event-related potential components occurring in 80- to 200-ms and 200- to 300-ms time windows, with reductions in amplitude and a greater latency difference for CI-using children. Relative to typically hearing children, current source density analysis showed muted responses to concurrent visual stimulation in CI-using children, suggesting less cortical specialization and/or reduced responsiveness to auditory information that limits the detection of the interaction between sensory systems.
Conclusion: These findings indicate that even in the face of early interventions, CI-using children may exhibit disruptions in the development of auditory and multisensory processing.

Early auditory deprivation in cases of severe to profound congenital hearing loss has been associated with changes in primary sensory processing and increasingly higher level processes, including attention and learning (Kral & Eggermont, 2007; Pisoni et al., 2016, Sharma et al., 2009). Kral et al. (2016), for instance, have argued that early deprivation sets into motion a series of changes that influence the effective connectivity among functional–neural systems well beyond those of primary auditory cortex. Such widespread changes may, in part, reflect the effects of cross-modal plasticity in early auditory cortices. In cross-modal plasticity, sensory and perceptual processes typically subserved by relatively modality-specific neural processing become attuned to sensory signals from intact modalities including vision and somatosensation. While research in animal models provides some evidence for conscripted changes in auditory association regions (Kok et al., 2014; Land et al., 2016; Lomber et al., 2010), data exhibiting cross-modal cortical reorganization in humans have been more limited, especially in pediatric populations (see the study of Bell et al., 2019, for a recent review).

In an effort to better understand the scope of cross-modal cortical reorganization, we examine the expression of cortical auditory evoked potentials (CAEPs) in typically developing (TD) children and children with cochlear implants (CIs). We make use of a novel electroencephalography (EEG) paradigm that permits rapid, reliable, and noninvasive assessment of neural activity along both auditory and visual pathways (Backer et al., 2019). A

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particularly unique contribution of this research includes the assessment of auditory signals from passively presented ambient speech and the effects of concurrent processing of audio and visual stimulation in these populations.

CAEPs are a cascade of responses that include the P1, N1, P2, and N2 components that occur within approximately 50–300 ms after sound onset (Davis et al., 1966; Vaughan & Ritter, 1970). In addition to simple auditory stimuli such as clicks (e.g., Arslan et al., 1984) and tones (e.g., Davis et al., 1966), speech also elicits CAEPs (e.g., Kraus et al., 1993). Adultlike CAEP morphology emerges around 10–13 years of age (Ponton et al., 2000; Shahin et al., 2010). The putative generators of the CAEPs include the primary and nonprimary auditory cortices, along with the association and possibly frontal cortices (Hari et al., 1980; Kanno et al., 2000; Näätänen & Picton, 1987; Picton et al., 1999; Scherg & Von Cramon, 1985; Shahin et al., 2007; Vaughan & Ritter, 1970). CAEPs in young children are dominated by P1 and N2 components, as opposed to the mature P1–N1–P2–N2 component morphology observed in adults (Čeponienė et al., 2002; Shahin et al., 2010). In addition to age, stimulus rate and refractory periods are also important in the expression of CAEPs (Gilley et al., 2005; 2006; Sussman et al., 2008). For example, Gilley et al. note the importance of consideration of age and refractory periods in the expression and detection of P1 and N1 CAEPs, as clear differences owing to stimulus train durations have been observed across typical and atypical developmental populations (Gilley et al., 2005, 2006). Sussman et al. (2008) reported changes in CAEP morphology as a function of age and rates of presentation.

In general, faster rates of presentation result in the suppression of discrete components, especially the maturing auditory components such as the N1. Importantly, the expressions of P1 and N2 were detectable in their youngest subjects (ages 8–11 years) with stimulus onset asynchrony that ranged from 200 to 800 ms. Given these considerations, we restrict our investigation to auditory P1 and N2 components.

Auditory P1

The P1 CAEP has been described as a biomarker of primary auditory cortex development in the deaf (Sharma et al., 2015). P1 latency has been shown to decrease with age in normal-hearing children (Eggermont, 1988; Eggermont et al., 1997; Gilley et al., 2005; Liégeois-Chauvel et al., 1994; Sharma et al., 1997, 2002). In previous work with deaf children implanted with CIs prior to 3.5 years old, Sharma et al. (2002) showed normal P1 latency and morphology by 7–8 months post implant. However, the normalization of P1 latency following CI implantation is not certain, as Corina et al. (2017) have reported latency and amplitude differences in some early implanted children despite at least 8 months of experience with their CIs. These data suggest that even with early implantation and adequate experience, some children with CIs will nevertheless exhibit atypical P1 latencies, potentially reflecting an aberrant maturation of cortical function. Whether such differences underlie cross-modal changes is not well understood.

Auditory N2

The mature morphology of the CAEPs in adults comprise the P1–N1–P2–N2 (Ponton et al., 2000). In young children, CAEPs are dominated by P1 and N2 components, with the N2 being the dominant negative component (Čeponienė et al., 2001). The N2 emerges around 6 months, stabilizing in amplitude and latency by 2 years (Shafer et al., 2015) and may be obtained in passive listening conditions with stimuli presented at regular intervals. Ponton et al. (2000) found that the N2 amplitude increases from age 4 to 10 years and thereafter decreases to reach adult values by age 17 years. Children's N2 is largely insensitive to stimulus rate (Čeponienė et al., 1998) while N2 amplitude changes as a function of the acoustic sound content. The N2 is larger in response to complex rather than simple tones and vowels (Bruder et al., 2011; Čeponienė et al., 2001) and to low-pitched tones rather than high-pitched tones (Korpilahti et al., 2002) and is larger in response to syllables than nonspeech analogs (Čeponienė et al., 2005, 2008). In the studies using syllables, N2 and N4 behaved similarly and were suggested to reflect higher order sound analysis, such as the content recognition of syllables, scanning for access to semantic representations, or short-term memory retrieval (Čeponienė et al., 2001, 2005, 2008). Diminished N2 and N4 peaks have been found in children with developmental dysphasia (Korpilahti, 1996; Korpilahti & Lang, 1994), reading impairment (Neville et al., 1993), and language impairment (Čeponienė et al., 2005).

Multisensory Processing

The ability to register sensory signals from different modalities is a basic neural function that underlies our perceptual experiences. Under some circumstances, covarying signals from different modalities may be integrated into meaningful percepts (e.g., the integration of auditory and visual information in naturalistic speech perception), whereas other cotemporal signals may be monitored and treated as separate information (e.g., the sight of a stop sign while listening to music while driving).

Multisensory processing in the cortex has been assumed to occur in specialized cortical modules relatively late in the processing hierarchy and only after

unimodal sensory processing in the so-called "sensory-specific" areas (Thesen et al., 2004). However, functional imaging and EEG studies suggest that the senses can influence each other even at the earliest levels of cortical processing, that is, at the level of the primary sensory cortices (see the study of Thesen et al., 2004, for a review; see also the studies of Molholm et al., 2002, and Shahin et al., 2018). Behavioral impairments of specific sensory systems have been known to impact faithful integration of complementary multimodal signals. In the case of deafness, studies have shown that the timing and duration of auditory sensory deprivation modulate integrative effects of audiovisual (AV) processing in the service of speech recognition (Bergeson et al., 2005; Gilley et al., 2008; Schorr et al., 2005; Stevenson et al., 2017). While the majority of studies investigating multisensory processing in this population have focused upon higher level processes such as word recognition, as noted by Stevenson et al. (2017), there is a dearth of work on low-level multisensory sensory perception in CI users. This study provides a step in this direction and makes use of a novel paradigm to investigate the effects of concurrent visual stimulation during auditory processing in pediatric users.

Ambient Speech

This study makes use of a passive EEG paradigm under conditions of ambient speech perception. Traditionally, auditory and speech perception studies have used highly controlled listening conditions to investigate effects of interest. Laboratory results can be difficult to relate to real-world experience, and little work has exploited the ways in which naturalistic stimuli can be leveraged to investigate task-irrelevant overheard speech. It is of further interest to note that a great deal of speech exposure when learning a language is ambient; hence, it is important to gauge how receptive the brain is to this modality. The ability to process and attend to background speech is by no means a given, particularly in complex auditory environments, and is frequently problematic for individuals with hearing loss (Shinn-Cunningham & Best, 2008). The compulsive processing of background linguistic information may not be automatic for children who are hard of hearing or who have been fitted with CIs. Prior work by Backer et al. (2019) reported reliable P1 and N1 ERP components followed by a sustained negativity in typically hearing adults in response to ambient presented speech. Here, we extend these findings to a TD pediatric population and in deaf children using CIs.

This Study

This study makes use of an EEG paradigm that permits assessment of auditory signals from passively presented ambient speech. We evaluate the utility of ambient speech in eliciting characteristic auditory potentials through assessments of P1 and N2 CAEPs in a sample of TD children and congenitally deaf CI-using children. In addition, we gauge the contributions of concurrent visual stimulation during auditory processing in these populations.

We entertain several hypotheses. To the extent that children with profound hearing loss who have subsequently received CIs show lingering effects of auditory deprivation, such as less mature sensory systems, we may expect to see reduced and/or delayed P1 auditory evoked potentials relative to typically hearing children. Moreover, if higher order sound analysis of speech stimuli is hampered in CI-using children, we may see morphological differences in the expression of the N2 component. Finally, if auditory sensory and perceptual processes exhibit cross-modal changes that show bias toward visual information processing, we expect that the neural and topographic signatures of auditory processing during concurrent visual processing will differ from TD children. While the direction of such changes has yet to be understood, we speculate that there will be additional processing costs associated with auditory processing in the face of concurrent visual stimulation in this population. This may manifest as delayed and/or exaggerated auditory components under dual-stimulus conditions (i.e., co-occurring audio and visual stimulation) relative to auditory stimulation alone. Such a pattern has been reported for CAEPs in healthy adults processing tone trains and flashing checkerboards (İşoğlu-Alkaç et al., 2007). However, it is also possible that, for CI-using children relative to the TD children, there will be less modulation of ERP components during co-occurring conditions if reduced responsiveness to auditory information limits the interaction between sensory systems. Probing the effects of concurrent visual stimulation during auditory processing on early P1 and later N2 components may provide insights into the scope of cortical reorganization evident in this population.

Method

Stimuli

We adopted a passive EEG task in order to permit a wide range of ages to be tested. Children viewed silent cartoons displayed in the center of the screen while they saw peripherally presented flashing checkerboards and heard auditorily presented sentences. The audio stimuli were drawn from 180 unique sentences from the Harvard/IEEE Corpus (IEEE, 1969) and edited into simple short sentences that are 1.5 s in duration. The sentences were processed to create chirped-speech (aka "cheech," for TD

children) or clicked-speech (aka "cleech," for CI-using children) as detailed in patent listing (Miller et al., 2015; see below for further details). These sentences were concatenated along with 1.5-s silences (for visual-only trials, see below) into a 1-min continuous presentation. There were forty 1.5-s trials/minute, with every other trial being audio-only (AO; 20 total) and with the interleaving trials either AV (10) or visual-only (10). Each minute of audio stimuli was followed immediately by a second minute of the same stimuli with phase inverted to control for any electrical artifact induction from the loudspeaker, thus creating a 2-min block. Six of these 2-min blocks were presented in total, each with novel sentences and randomized AV/visual trial ordering and with short breaks between each. Across the entire 12-min experiment, there were a total of 240 AO trials, 120 AV trials, and 120 video-only trials. Presentation software (Neurobehavioral Systems version 18.2) was used to deliver stimuli.

To create cleech/cheech stimuli used in the testing of the CI-using children and TD children, respectively, speech sentences (sampled at 22050 Hz) were first pitch-flattened to 82 Hz using the Praat software (Boersma & Weenink, 2021). Then, portions of the voiced speech energy were replaced by energy-matched chirps (i.e., transient sounds that increase rapidly in frequency) or clicks. The introduction of this chirp/click energy into the speech acoustics serves to optimize auditory responses recorded at the scalp, particularly the early ones, by aligning them temporally. In the case of TD children, this occurs because the time course of the chirps accommodates for the basilar membrane delay, which otherwise serves to smear the timing of transduction across frequencies, leading to smaller early (auditory brainstem) responses. In CI-using children, the basilar membrane delay is not involved in sound transduction; hence, an energy-matched click stimulus best approximates the same synchronous stimulation across frequencies in the auditory nerve (Dau et al., 2000; Elberling & Don, 2008). Specifically, the chirps/clicks were combined with speech through frequency multiplexing: chirps/clicks and speech occupy alternating, interleaved frequency bands spanning one octave each. The resulting cheech/cleech stimulus comprised speech energy occupying 0–250 Hz, 500–1000 Hz, and 2000–4000 Hz, with chirp/click energy occupying 250–500 Hz, 1000–2000 Hz, and 4000–11025 Hz; see Figure 1). Moreover, the chirps/clicks were temporally aligned with individual glottal pulses in the speech, thereby coinciding with each voiced event. The mean and median time between subsequent voiced, chirped/clicked periods was 314 and 268 ms, respectively. In this way, cheech/cleech is perceived as a single speech stream because the chirps/clicks align acoustically and perceptually with the natural voicing. The perception of the rapidly presented chirps/clicks within the cheech can be described as a rattling characteristic that blends perceptually with the voice. Thus, the resulting speech has a robotic monotone quality, but it is highly intelligible with clear linguistic content.[1] The wider utility of this manipulation is discussed in detail in the study of Backer et al. (2019). In this article, we describe the CAEPs that result from time-locking the EEG to the voicing onsets in the cheech/cleech, specifically to the first chirp/click in each voiced period (M = 2.67 voiced periods per trial). To further isolate auditory-only events that were not contaminated by preceding visual events, we averaged EEG voice onset periods from the auditory-only trials that occurred at least 500 ms after the offset of any visual flashing.

On the AV 1.5-s trials, periods of concurrent visual stimulation accompanied the auditory stimulus. As shown in Figure 2, the visual stimuli had an inner ring composed of eight equally spaced checks and an outer ring composed of 16 equally spaced checks. The checks within the inner and outer rings flickered sinusoidally at a rate of 7.5 and 12 Hz, respectively, with the alternate checks within each ring flickering in counterphase. Although auditory–visual trials were characterized by simultaneous occurrences of flashing visual stimuli and auditory sentences, as noted previously, there is no expectation that these independent signals would be necessarily integrated. In this study, we compare auditory-only and AV trials; visual-only trials will be treated in a separate report.

In summary, six 2-min stimulus blocks were presented, consisting of auditory-only trials (1.5-s duration sentences, 240 trails), visual-flashing checkerboards (1.5-s duration, 120 trials), and combined audio and visual trials (1.5-s duration, 120 trials), while children passively viewed centrally presented entertaining silent cartoons.

All testing sessions took place in a quiet, dimly lit room. Measurements of two screen luminance values of the components of the visual display (white and gray) were measured using a Konica-Minolta CS-100A meter (white: M = 107 cd/m^2; gray: M = 63.98 cd/m^2) for each testing session to ensure similar lighting across all sessions. An HP Z24i monitor was used to view both cartoons and the visual stimuli. Audio was presented free-field from a single stand-mounted Auvio 05A13 loudspeaker. We used a NuForce Icon amplifier to drive the loudspeaker, which was positioned above the display monitor. The distance

[1]In a sentence reproduction task using the sentential stimuli used in this study, adult listeners (n = 25) were asked to transcribe auditorily presented sentences under three conditions: pitch-flattened clear speech, cheech processed, and cleech processed. Overall subjects were most accurate transcribing the clear pitch flattened speech (95.39%) and less accurate with cleech and cheech processed speech (83.67% and 83.61%, respectively). In both the cleech and cheech conditions, errors arose primarily in the reporting of short closed-class words "the," "a," "we," "they," and "he."

Figure 1. Time by frequency speech spectrogram illustrating the multiplexing of chirps with spectral representation of speech energies and periods of voicing.

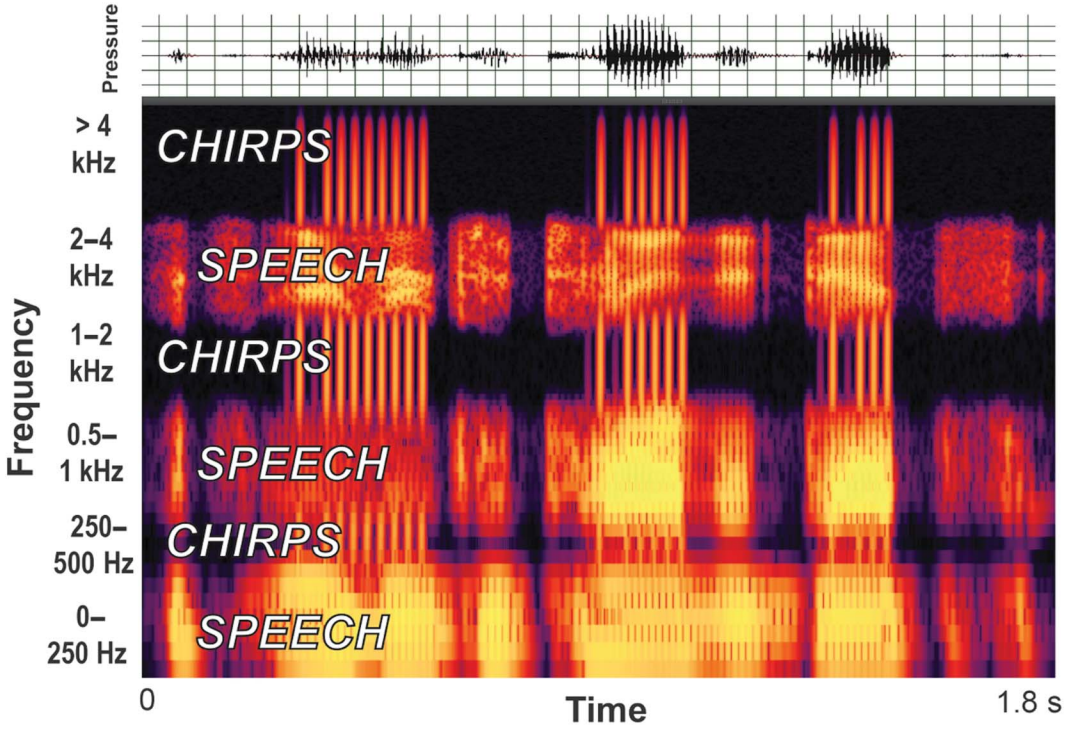
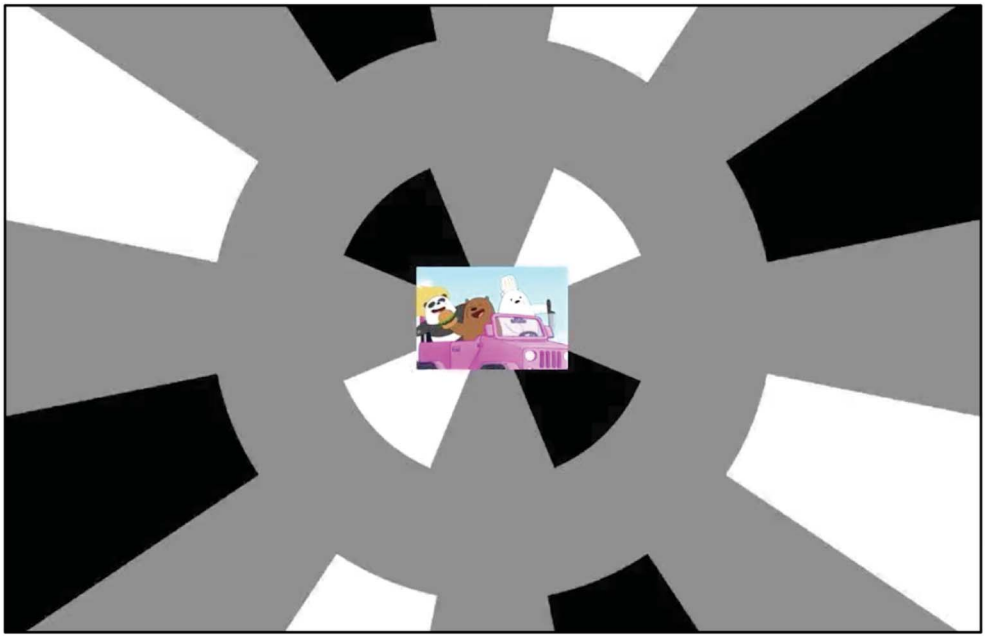


Figure 2. Concurrent visual stimulus display with central cartoon flanked by an inner ring of eight flashing checks (7.5 Hz) and an outer ring of 16 flashing checks (12 Hz).



between the monitor and the child was measured and set to 28 in.

Sound level measured for each session (Radio Shack SPL meter 33–2055) was 60 dB for typical-hearing children and 65 dB for CI-using children. Sound level was checked prior to each testing session and whenever we switched from testing CI children to TD children and vice versa. An experimenter was seated to the right side of the child. This helped encourage participants to remain seated and attend to the stimuli. No response was required from participants.

Participants

A total of 56 children participated in this study, 28 typical hearing (TD; 12 male, mean age = 77 months, range: 31–122 months) and 28 deaf children using CIs (20 male, mean age = 74 months, range: 46–128 months). Parents of all participants filled out background questionnaires regarding their children's hearing, vision, language history, and other diagnoses. All TD children were reported to have normal or corrected to normal vision and normal hearing levels in both ears. All deaf children were first identified through hospital administered early hearing detection and intervention screening and received their CI by 31 months. The majority of children in the CI group had bilateral implants, except for two, both male, both with CI on the left side; these children had moderate or severe hearing loss in their right ear and wore hearing aids, one of these children was not eligible for a CI on their other ear due to an anatomical anomaly. CIs and hearing aids were active during our testing sessions. Causes of deafness for CI-using children were Connexin 26 gene mutation (five), enlarged vestibular aqueduct (two), Pierre Robin syndrome (one), Pendred syndrome (one), and Waardenburg syndrome (one). The rest (18) were unknown, nine of them with family members that were Deaf or Hard of Hearing. Manufacturers of the CIs were AB Naida (nine), MED-EL (nine), and Cochlear Nucleus (10). Table 1 reports demographic characteristics of the subjects. Daily checks were performed at the schools to ensure that CIs were working, where the child had to repeat or identify Ling, Madell, Hewitt sounds, with vision denied for each side. Weekly checks were performed by teachers on students' equipment. CI-using children in our studies receive quarterly booth testing to ensure that all students have access to sounds across the frequency spectrum and complete speech perception testing. Children with any additional medical diagnosis (e.g., autism and cerebral palsy) were excluded from both groups. No participants were considered cognitively impaired. There were two children from each group that were diagnosed with attention deficit disorder. This study was carried out in accordance with the recommendations of University of California, Davis, Institutional Review Board Administration, Social & Behavioral Committee C, Davis, CA, protocol 806455–6 with written informed consent from all the parents/guardians of the subjects.

Table 1. CI-using children subject demographics.

Subject	Age	Gender	Age at first implant	CIs
1	46	M	7	2
2	51	M	15	2
3	54	M	13	2
4	60	M	10	2
5	60	M	12	2
6	61	M	12	2
7	63	M	18	2
8	65	M	20	1
9	65	M	14	2
10	66	M	13	2
11	70	M	10	2
12	72	M	12	2
13	74	F	27	2
14	75	F	31	2
15	75	M	16	1
16	75	F	30	2
17	75	M	13	2
18	76	M	13	2
19	78	M	14	2
20	78	F	31	2
21	79	F	18	2
22	80	F	28	2
23	81	M	10	2
24	86	M	14	2
25	90	M	14	2
26	91	F	10	2
27	103	F	24	2
28	128	M	12	2

Note. Age variables are presented in months. CIs = cochlear implants; M = male; F = female.

Data Recording and Analysis

Continuous EEG was recorded during the six 2-min blocks using a BioSemi Active Two system (BioSemi Inc.), at a sampling rate of 16 kHz, from 21 scalp sites (19 in the cap and two externals applied to the left and right mastoids) using the standard 10/20 locations. EEG was recorded with the common mode sense active electrode as the effective ground using a Driven Right Leg passive electrode to clamp subject voltage to the amplifier reference value and re-referenced offline (http://www.biosemi.com/faq/cms&drl.htm).

Event-Related Potentials

EEG was downsampled to 512 Hz and band-pass-filtered at 0.1–30 Hz (eight-order Butterworth), then re-referenced to averaged mastoids. Independent component analysis (ICA; Infomax; EEGLAB v14.1.2; Delorme & Makeig, 2004) was used to remove eye and CI artifacts. Additional artifact rejection was performed automatically

on the basis of threshold excluding all trials that exceeded +/−120 μV using ERPLAB (Lopez-Calderon & Luck, 2014) v8.25. Channels clearly corrupted by CI electrical artifact—typically P7, P8, T7, and T8—or poor signal were removed and interpolated (spherical). Measurements of event-related potential (ERP) waveforms were for local peak latency and amplitudes using ERP measurement tool in ERPLAB v8.25. We adopted commonly used time windows for the measurements of the P1 (80–200 ms) and N2 (200–300 ms) components.

Current Source Density

Current source density (CSD) maps were generated with the EEG data of all participants. The CSD sets were computed from the mean amplitude waveforms from 19 channels across the scalp for each subject. This function was performed using the ERPLAB plugin (Lopez-Calderon & Luck, 2014) for EEGLAB (Delorme & Makeig, 2004). As discussed subsequently, we take this fact into consideration when interpreting our CSD results.

Statistical Analysis

For the statistical analysis, we made use of repeated-measures analysis of variance (ANOVA) with the within-subject factors of Condition (AO and AV), Site (Fz, Cz, and Pz), and Group (TD and CI-using children) as a between-subject factor. To quantify the P1 data, we evaluated peak amplitude and latency in the 80- to 200-ms time window. For the N2, we evaluated peak amplitude and latency in a 200- to 300-ms time window. Greenhouse–Geisser correction was applied to compensate for violations of the sphericity assumption. Planned comparisons are evaluated with paired *t* tests, and Tukey's honest significant difference test is used to assess post hoc comparisons.

Results

Visual inspection of the data revealed a prominent positive component (P1) between approximately 50 and 200 ms followed by a broader negative component (N2) between 200 and 400 ms (see Figures 3A and 3B). This latter component was prominent in the TD children's data but largely absent in the CI-using children. This latter component further differs in morphology from the sustained N1 negative component observed in our earlier study of adults (Backer et al., 2019). As noted previously, in children, the N2 is the most predominant negative peak of auditory evoked potentials, whereas in adults, the N1 component dominates (Čeponienė et al., 2002).

P1 Amplitude

The ANOVA revealed a main effect of Condition, $F(1, 54) = 8.037$, $p < .006$, $\eta_p^2 = .130$; Group, $F(1, 54) = 15.813$, $p < .001$, $\eta_p^2 = .227$; and Site, $F(1.815, 98.025) = 44.164$, $p < .001$, $\eta_p^2 = .450$, as well as a Site × Group interaction, $F(1.815, 98.025) = 6.137$, $p < .003$, $\eta_p^2 = .102$. No other higher order interactions were significant. The main effect of Condition revealed that, overall, the responses to AV stimuli were more positive than AO stimuli (AV = 3.394 μV, AO = 2.419 μV). The main effect of Group revealed that TD children had larger P1s than CI-using children (TD: $M = 3.725$ μV; CI: $M = 2.088$ μV). The main effect of Site revealed a hierarchy of responses such that positivities were greatest at site Fz (3.499 μV) followed by Cz (3.177 μV) with smallest response at Pz (2.043 μV). Pairwise comparison revealed that the differences between sites Fz and Cz were marginally significant ($p < .055$), whereas site Pz was significantly different from Fz and Cz (all ps $< .001$). The Site × Group interaction

Figure 3. Auditory evoked potential components (P1 and N2) recorded at site Fz in response to ambient speech (Panel A) and ambient speech presented with concurrent visual stimulation (Panel B) in typically developing children and children using cochlear implants. The gray lines are responses from typically developing children; the red lines are responses from cochlear implant using children. Peak of the component in the 80- to 200-ms time window is labeled P1. Peak of the component in the 200- to 300-ms time window is labeled N2.

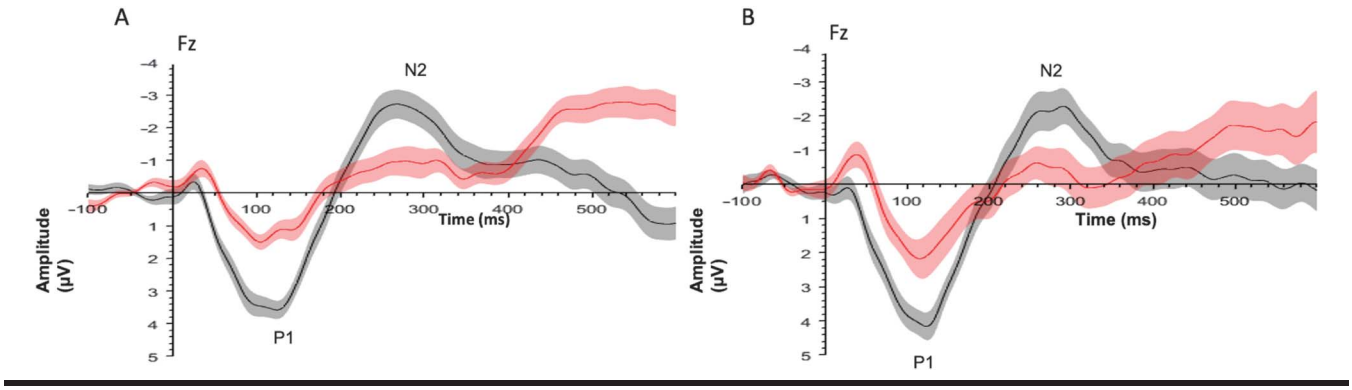
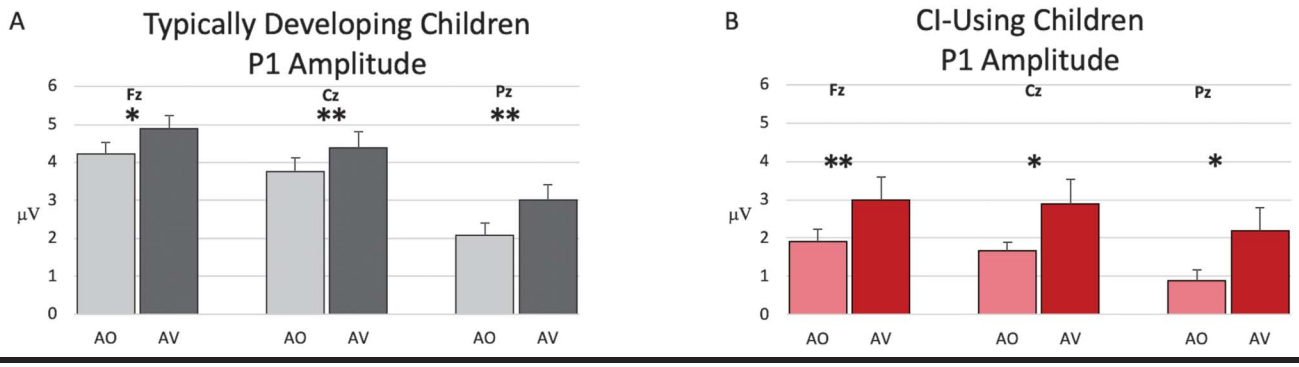


Figure 4. P1 peak amplitudes during AO and AV trials for typically developing and CI-using children, illustrating the main effects of Condition, Group, and Site and individual modulations at each midline site. (A) Peak P1 amplitudes and standard errors at midline sites (Fz, Cz, and Pz) in typically developing children. Light gray bars are responses during AO trials; dark gray bars are responses during AV trials. (B) Peak P1 amplitudes and standard errors at midline sites (Fz, Cz, and Pz) in CI-using children. Light red bars are responses during AO trials; dark red bars are responses during AV trials. AO = audio-only; AV = audiovisual; CI = cochlear implant. *$p < .05$; **$p < .068$.



indicated significant pairwise differences in amplitude for each site for TD children (TD: M = Fz 4.554 μV, Cz 4.076 μV, Pz 2.545 μV, ps < .045). For CI-using children, the P1 amplitude at site Fz was not significantly different from site Cz (p = .475), whereas Pz differed significantly from both Fz and Cz (CI: M = Fz 2.445 μV, Cz 2.278 μV, Pz 1.542 μV, $p < .001$). The Site × Group interaction further reflects the fact that TD children's amplitudes were significantly larger than the CI-using children's amplitudes especially at frontal sites Fz and Cz ($p < .001$) with a less significant difference at site Pz (p = .016).

Based on our questions of interest regarding the influence of concurrent visual stimulation on these auditory signals, we performed group-specific planned comparison of the AV and AO conditions at each midline site. Figure 4 illustrates amplitude differences between AO and AV trials. Tables 2 and 3 report the amplitude differences and *SD* between AV and AO trials, *t*-statistics and *p* value (one-sided), and Cohen's *d* effect size for each group.

As indicated in Tables 2 and 3, the peak amplitude differences between AO and AV conditions show significant trends between conditions, with AV trials of greater magnitude than AO trials. Cohen's *d* values indicate very large effect sizes overall that are numerically greater for the CI-using children relative to TD children.

P1 Latency

The ANOVA of peak latency values revealed a main effect of Site, $F(1.829, 98.752) = 7.235$, $p < .002$, $\eta_p^2 = .118$. A marginal Group × Condition interaction, $F(1, 54) = 3.757$, $p = .058$, $\eta_p^2 = .065$, and a marginal three-way Condition × Site × Group interaction, $F(1.768, 95.452) = 3.122$, $p = .055$, $\eta_p^2 = .055$. The main effect of Site revealed that latencies were shortest at site Fz (M = 122.140 ms), relative to site Cz (M = 126.046 ms), with the longest latency at site Pz (M = 133.789 ms). Exploratory analysis of the Group × Condition interaction revealed that, in CI-using subjects, P1 latencies were not significantly different between AO and AV conditions (CI: AO, M = 131.836; AV, M = 128.953; p = .617). In contrast for TD children, the P1 latency for the AO condition was significantly earlier than the AV Condition (TD: AO, M = 117.839; AV, M = 130.673; p = .029). Further clarification of this trend is warranted by the three-way interaction whereby the mean latency for the CI-using group at site Cz during AO trials was significantly longer than TD subjects' latency at this same site (CI: M = 134.068; TD: M = 113.143; p = .012). This pattern was also observed for site Pz (CI: M = 143.973; TD: M = 121.932; p = .007). This between-group difference during AO trials was not observed at site Fz (CI:

Table 2. P1 amplitude differences at midline sites as a function of condition, *SD*s, *t* score, *p* value, and Cohen's *d* effect size.

		Typically developing children				
Site	**Condition**	**Difference (μV)**	***SD***	***t*(27)**	***p***	**Cohen's *d***
Fz	AV–AO	.669	1.97	1.79	.042	1.97
Cz	AV–AO	.626	2.16	1.53	.068	2.16
Pz	AV–AO	.930	2.53	1.94	.062	2.53

Note. AV = audiovisual; AO = audio-only.

Table 3. P1 amplitude differences at midline sites as a function of condition, *SD*s, *t* score, *p* value, and Cohen's *d* effect size.

CI-using children						
Site	**Condition**	**Difference (μV)**	***SD***	***t*(27)**	***p***	**Cohen's *d***
Fz	AV–AO	1.078	3.42	1.669	.053	3.42
Cz	AV–AO	1.230	3.30	1.974	.029	3.30
Pz	AV–AO	1.310	3.76	1.840	.038	3.76

Note. CI = cochlear implant; AV = audiovisual; AO = audio-only.

M = 117.467; TD: M = 118.4; p = .889). In contrast to the AO condition, between-group peak latencies during the AV condition did not differ significantly from each other at all sites (all ps > .346). See Figure 5.

CSD 80–200 ms

CSD representations reflect the second spatial derivative of the scalp voltage and thus provide a conservative, more localized description of neural current generators than typical voltage maps (Tenke & Kayser, 2012). By quantifying current sources and sinks as opposed to the magnitude of voltage deviations, CSD minimizes the effects of volume currents and may highlight further group differences. CSD maps for TD children in the 80- to 200-ms P1 window during the AO condition exhibit bilateral frontal and posterior–occipital current sources with bilateral posterior–parietal sinks. Under concurrent visual stimulation, this pattern becomes strengthened and more asymmetrical with a broader right temporal–parietal source focus and a weak posterior–occipital sink. The CSD maps for CI children in the 80- to 200-ms window indicate a more attenuated response. During the AO condition, we observe a bilateral frontal source that is highly asymmetric (left > right) and asymmetrical bilateral parietal sink (left > right) and a weak posterior–occipital source. During concurrent audio and visual stimulations, the CI children's pattern shows a broadened left–frontal and central source with an asymmetric bilateral parietal–occipital sink (left > right). See Figure 6.

N2 Amplitude

Peak amplitude measurements in the N2 window of 200–300 ms revealed a main effect of Site, F(1.612, 87.035) = 21.753, p < .001, η_p^2 = .287, a marginally significant effect of Group, F(1, 54) = 3.943, p < .052, η_p^2 = .068, and a Site × Group interaction, F(1.612, 87.035) = 5.991, p < .006, η_p^2 = .100. There were no other main effects or higher order interactions. The main effect of Site indicated the greatest negativity at site Fz (M = −2.277 μV) followed by Cz (M = −2.130 μV) and Pz (M = −1.269 μV). The marginally significant effect of Group indicated that TD children showed more negative peak amplitudes in the N2 window than CI-using children (TD:

Figure 5. P1 latencies for each midline site for typically developing and CI-using children. Note that for typically developing children, the latencies are sensitive to condition manipulations (i.e., AO vs. AV) in contrast to CI-using children show nonsignificant latency modulations between conditions. (A) P1 latencies at midline sites (Pz, Cz, and Fz) in typically developing children. Light gray bars are responses during AO trials; dark gray bars are responses during AV trials. (B) P1 latencies at midline–central sites (Pz, Cz, and Fz) in CI-using children. Light red bars are responses during AO trials; dark red bars are responses during AV trials. AO = audio-only; AV = audiovisual; CI = cochlear implant; n.s. = not significant.

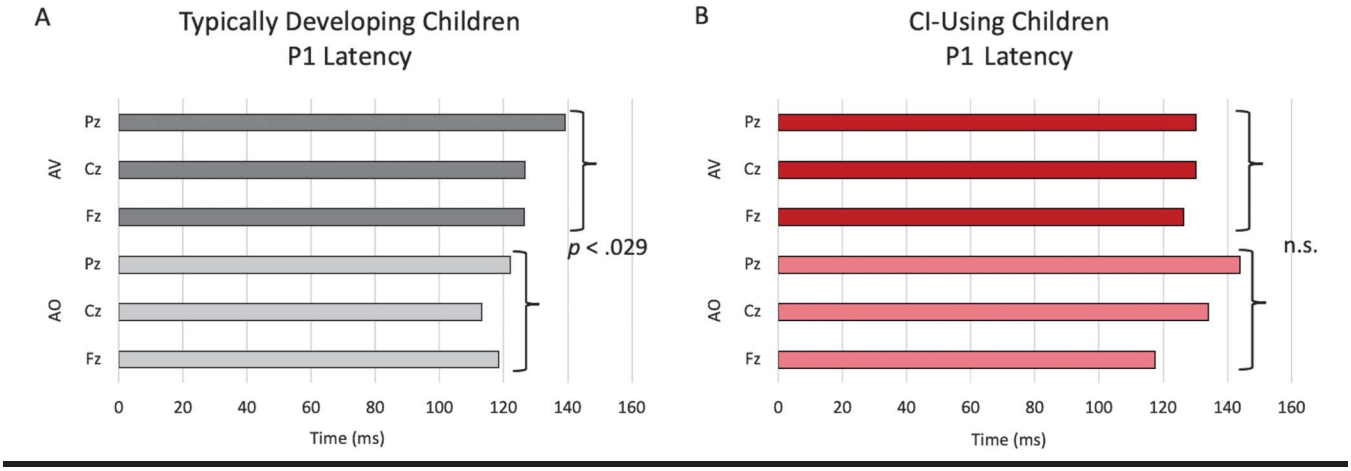
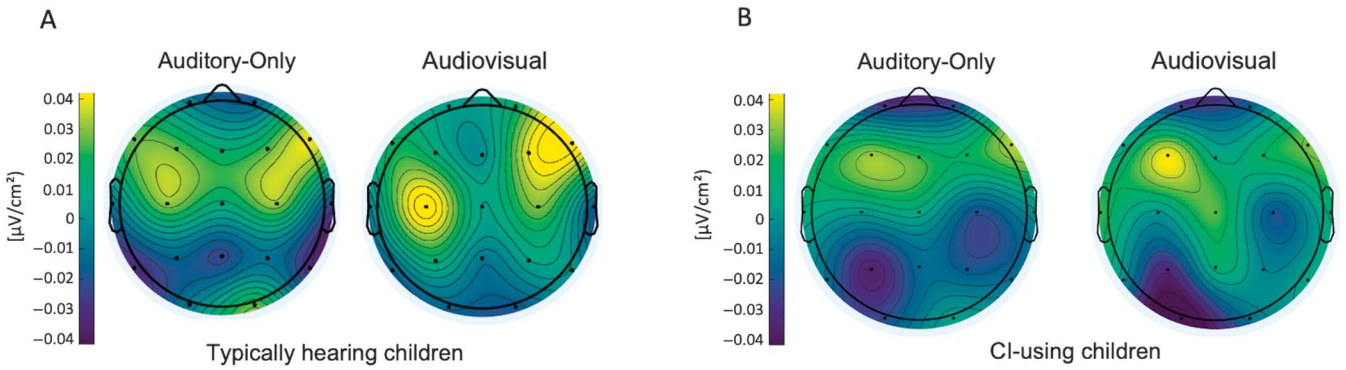


Figure 6. Current source density renderings from an 80- to 200-ms time window. Colors correspond to second spatial derivative of the scalp voltage. Positive values (yellow) represent current flow from the brain to the scalp (i.e., sources); negative values (blue) represent current flow from the scalp to the brain (i.e., sinks). (A) Children with typical hearing. Left side represents data from the auditory-only condition; right side represents data from the audiovisual condition. (B) CI-using children. Left side represents data from the auditory-only condition; right side represents data from the audiovisual condition. CI = cochlear implant.



M = −2.339 μV; CI: M = −1.445 μV). The analysis of the Site × Group interaction revealed that TD children's amplitudes at sites Fz were significantly more negative than those observed for CI-using children (TD: M = −2.997; CI, M = −1.5576; $p < .006$). A trend for this same pattern was observed at site Cz (TD: M = −2.601; CI, M = −1.660; $p < .065$). The amplitudes were not significantly different from one another at site Pz (TD: M = −1.419; CI: M = −1.119; $p = .516$). See Figure 7.

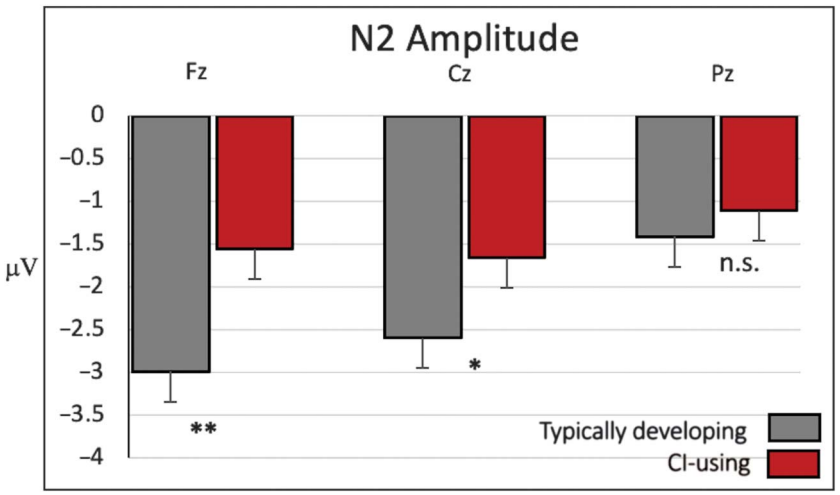
N2 Latency

Peak latency measurements evaluated using ANOVA revealed no significant main effects or interactions (all ps > .156).

CSD 200–300 ms

CSD maps for TD children in the 200- to 300-ms window for the auditory-only condition revealed a bilateral frontal sink with a weaker central source that extended posteriorly and included weak bilateral parietal–occipital sources. During the concurrent audio and visual stimulations, we observed prominent bilateral parietal sources with prominent bilateral frontal (left > right) and posterior–occipital sinks. CSD maps for CI children in the 200- to 300-ms auditory-only condition window indicate a bilateral frontal (left > right) source and a prominent occipital source. This is bounded by frontal–central and bilateral parietal sinks. During audio and visual stimulations, there is a shift of the occipital source to a central–parietal

Figure 7. Peak amplitudes and standard errors of N2 components recorded at midline sites (Fz, Cz, and Pz). Gray bars represent values from typically developing children; red bars represent values from CI-using children. CI = cochlear implant; n.s. = not significant. *p = .065; **p < .006.



region while the asymmetric frontal (left > right) source pattern is maintained. See Figure 8.

Discussion

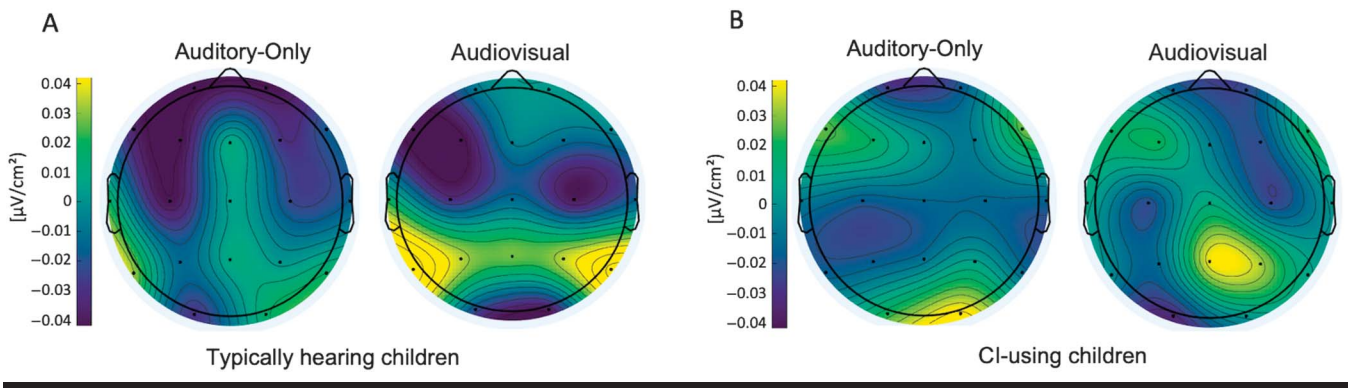
Our data indicate the presence of a positive component between 80 and 200 ms with a frontocentral focus elicited from initial voicing onsets of ambiently presented speech. This component is consistent with the description of an auditory evoked P1 and replicates findings from a study of adult participants reported in the study of Backer et al. (2019). We should note that the latencies of canonical ERP components evoked by continuous speech are typically later than those for isolated sounds preceded by silence. Group differences were observed in the magnitude of the P1 component, whereby TD children showed a greater positivity than CI-using children during auditory-only conditions. Significant between-group differences in P1 latency were observed at central site Cz and midline parietal site Pz, whereby the TD children exhibited shorter peak latencies than CI-using children.

The effect of concurrent visual stimulation on the auditory P1 component resulted in increased amplitudes for each group. Interestingly, the effect size was more pronounced in the CI-using children relative to the TD children at central midline sites. However, while concurrent visual stimulation led to increased P1 latencies in the TD children, the already-longer P1 latencies of the CI users observed in the auditory-only condition were not further modulated by concurrent visual stimulation. The pattern of increased CAEP amplitude and latency found in our TD children during combined auditory and visual processing appears similar to the effects reported in adults by İşoğlu-Alkaç et al. (2007) using a simpler paradigm of tone trains accompanied with and without a flashing checkerboard. The lack of a latency modulation in the CI-using children may be an indication of reduced synaptic transmission within auditory pathways, but despite this processing delay, the CI-using children do exhibit a responsiveness to the additional visual stimuli, indicative of processing cost.

The corresponding CSD topographic analysis in the P1 time window revealed group differences with TD children exhibiting a bilateral frontal current source and a weak posterior–occipital source. This pattern resembles that reported by Sussman et al. (2008) in their study of P1 maturation in pediatric populations. In contrast, CI children showed an asymmetric left frontal source accompanied by bilateral parietal posterior sinks (left > right). However, due to the occurrence of interpolated data for a subset of CI-using children, we must be cautious in our interpretation of posterior parietal differences across groups. Topographic differences were further indicated in consideration of the ERPs. For example, during the auditory-only condition, the P1 component showed more differentiated amplitudes across frontal, central, and parietal sites for TD children (Fz > Cz > Pz). In contrast, CI-using children showed overall smaller amplitude differences with less differentiation across Fz, Cz, and Pz electrode sites (Fz = Cz, >Pz). These topographic differences may reflect cortical reorganization owing to early auditory deprivation in CI-using children (Kral et al., 2019).

In previous work with deaf children implanted with CIs prior to 3.5 years old, Sharma et al. (2002, 2015) showed normalization of P1 latency and morphology by 7–8 months post implant. In the present sample, all 28 CI users were implanted by 2.6 years (31 months) and were more than 36 months post implant. Consistent with Sharma et al.'s reports of normalization with early

Figure 8. Current source density renderings from a 200- to 300-ms time window. Colors correspond to second spatial derivative of the scalp voltage. Positive values (yellow) represent current flow from the brain to the scalp (i.e., sources); negative values (blue) represent current flow from the scalp to the brain (i.e., sinks). (A) Children with typical hearing. Left side represents data from the auditory-only condition; right side represents data from the audiovisual condition. (B) CI-using children. Left side represents data from the auditory-only condition; right side represents data from the audiovisual condition. CI = cochlear implant.



cochlear implantation, we observe P1 latency values recorded at site Cz largely commensurate with those reported by Sharma et al. (2002; CI: M = 134.06, SD = 36.33, range: 78.13–199.2; TD: M = 113.14, SD = 22.63, range: 78.13–156.25). However, in the present data, CI users' latencies at Cz were statistically longer than those of TD children and more variability is noted. These differences may be a reflection of the ambient speech paradigm used in this study compared to the auditory oddball paradigm used by Sharma et al. (2002).

In further quantification of morphological differences, TD children's P1 amplitudes were overall larger than CI-using children especially at central and frontal sites. Amplitude differences may reflect perceived loudness of the auditory stimuli (Bertoli et al., 2011) and/or the degree of attentional engagement (Luck et al., 1990; Mangun, 1995). Recall in the present experiment, auditory stimuli were presented ambiently without a requirement to attend. The amplitude differences we observe may be a reflection of less automatic engagement to the ambient speech signal in children with CIs. As discussed below, data from the N2 component provide further support for this hypothesis.

Examination of the N2 time window indicates the presence of an N2 component between 200 and 300 ms, which was largely absent in CI-using children. While there were no significant effects in the expression of the latency of this component across groups, clear differences in the magnitude of the response were observed, with TD children exhibiting greater negativities especially at frontocentral sites. This pattern is consistent with prior developmental research, which shows that the N2 becomes increasingly prominent in the ERP waveform during early childhood and shows a topographic pattern where amplitudes follow a (Fz > Cz > Pz) pattern (Čeponienė et al., 1998, 2002; Ponton et al., 2000; Sussman et al., 2008).

The N2 component has been associated with early speech processing that includes phonetic discrimination and phonological processes that serve as an interface of lexical forms (Deacon et al., 1991; Polich, 1985; Schmitt et al., 2001; van den Brink & Hagoort, 2004; van den Brink et al., 2001). The present data indicate significant amplitude effects at site Fz in the N2 time window in TD children, but not in children with CI, suggesting that the ambiently presented stimuli may not enact phonetic discrimination and/or automatic registration of phonological–lexical mappings thought to serve as a precursor to lexical recognition. Studies have reported prolonged latencies in the expression of N2 components in adults with CIs. This was associated with the performance on a speech recognition task and was modulated by the presence of background noise (Finke et al., 2016). Consistent with the premise that these individuals have difficulties mapping acoustic–phonetic features to lexical representations, prolonged N2 latency has been previously observed for lower signal-to-noise ratios in typically hearing children. This indicates that adverse listening conditions lead to delayed N2 latency (Almeqbel & McMahon, 2015). Furthermore, the detection of the speech-evoked N2 has been shown to be variable in children with CIs (Gabr & Serag, 2018). Longer N2 latencies and lower N2 amplitudes have been observed in CI-using children with poor performance on speech audiometry measures, compared with age-matched TD children (Munivrana & Mildner, 2013), suggesting that deficits in performance may be partly explained by deficits in discrimination and cognitive processing (Bakhos et al., 2018).

In contrast to the modulations of concurrent visual stimulation in the auditory P1 ERP data, we did not observe concurrent visual effects on the N2 auditory ERP components. This may indicate that our responses to ambient speech may be tapping into fairly specific linguistic processing operations and not domain-general attentional or integrative multimodal processes. However, it is interesting to note that, for the TD children, the CSD solutions in the N2 windows show qualitative differences with clear condition-specific modulations. While the TD AO CSD maps show prominent bilateral frontal sinks, consistent with data from the study of Sussman et al. (2008), during the AV condition, we see the emergence of clear bilateral parietal sources. A topographically similar source is noted in the same 200- to 300-ms time window in a study of novel auditory word learning when accompanied by a visually depicted semantic referent (François et al., 2017).

In contrast, in CI-using children, we see only a slight modulation from the P1 CSD patterns. Specifically, we see a maintenance of an attenuated left–frontal source that was observed in the P1 time window. However, we do see the emergence of an occipital source both in the auditory-only and visual-only conditions. The weak N2 ERP in CI-using children requires that we interpret the CSD topographies with caution.

Taken together, the TD children appear to process the early ambient auditory sensory stimuli in the P1 window (i.e., 80–200 ms) efficiently and exhibit both amplitude and latency modulations based on concurrent visual stimulation. Current sources reflect a rather stable bilateral–frontal profile. As auditory processing continues to evolve during the N2 window (200–300 ms), ERPs are not strongly modulated by the AV stimulation; however, CSD maps show a clear shift in current source that is not evidenced in the CI-using children. The differentiated topographic responses in both the ERP components and CSD maps in TD children may reflect a more nimble and automatic deployment of resources in complex environments.

In contrast, CI-using children appear to exhibit less temporally efficient ambient auditory processing as

indexed by the relatively attenuated P1 amplitudes and extended P1 latencies. Nevertheless, in spite of the less temporally efficient processing, there is a responsiveness to concurrent visual stimulation, which modulates P1 amplitudes. CSD maps largely indicate a left–frontal source that broadens centrally during visual stimulation. During the N2 window (200–300 ms), we see little evidence of a clear N2 component in the ERP waveforms. This may indicate a lack of a further linguistic elaboration of the ambient auditory stimulus. Similarly, while the CSD maps show an emergence of occipital sources during both auditory-only and AV conditions in this time window, we observe the continued presence of an attenuated left–frontal source originally observed in the P1 window; overall, this may be an indication of less auditory specialization.

We made several predictions regarding the expected effects. We predicted that CI-using children may show reduced and delayed auditory evoked potentials relative to typically hearing children owing potentially to less mature sensory systems. This prediction was confirmed as we observed decreased P1 ERP amplitudes and near lack of an N2 component. Comparing across groups, the lack of substantive changes in CSD maps across the two time windows in CI-using subjects may reflect a more entrenched processing system. These data are consistent with the premise that early auditory deprivation may have long-lasting effects on auditory processing despite early interventions. Kral et al. (2019) have posited that there may be a lack of top–down stabilization within auditory cortical regions that is dependent upon preservation of bottom–up signals early in development. The present data may be a reflection of this developmental disequilibrium. We hypothesized that there may be additional costs associated with auditory processing in the face of concurrent visual stimulation for this population. We observed an enhancement in P1 amplitude under dual-modality conditions but little change in latency. This may reflect temporal inefficiency in the registration of these ambient auditory signals. Nevertheless, concurrent visual information did appear to affect the gain of the auditory signal. We observed site-specific and topographic CSD differences across these groups. Whereas TD children exhibited more focal bilateral regional current flow, which showed topographic changes as a function of concurrent visual stimulation, CI-using children showed an asymmetric left–frontal source focus and less differentiation, suggesting less cortical specialization and/or reduced responsiveness to auditory information, which limits the detection of the interaction between sensory systems.

There are several limitations to this study, which must be acknowledged. Our testing of children (both CI-using and TD controls) often occurred at our partner schools and a community service center, rather than in a strict clinical environment. This does introduce unexpected challenges in the control of the testing environment. We do take measures to mitigate these environmental factors such as calibration of sound pressure level and monitor luminance. The use of a BioSemi Active Two EEG system greatly reduces stray electrical interference and does not require a shielded room.

While the differences in the construction of cleech and cheech stimuli are theoretically well motivated, we have only limited data directly comparing the effects of these manipulations on EEG. Based on unpublished observations of data from adults and children, we can state that CAEPs in the late time windows reported in this article look highly similar; however, more work is required to better understand the effects of the clicks and chirps for earlier occurring components (e.g., wave 5 and middle latency responses). In addition, it is important to recognize that the time-locked auditory events in our study have mean ISI of 314 ms and a median of 268 ms. These relatively fast periods might reduce the detectability of CAEPs that are typically observed in adolescence and especially in adult subjects (i.e., P1–N1–P2–N2); however, as Sussman et al. report (2008), even at rates as fast as 200 ms, identifiable P1–N2 are reliably present in 8- to 11-year-olds and are quite stable in amplitude during childhood. Gilley et al. report reductions in the amplitude of P1 components at 360-ms ISI compared to 2,000-ms ISI, which are attributed to difference in refractory periods. However, the latency of the P1 component was not affected by these ISI manipulations in 3- to 6-year-old children.

In the treatment of the EEG data from CI-using children, there are also known electrical artifacts caused by the CI processor. We make use of ICA to isolate and remove clear CI artifacts when they are present. However, the use of ICA for CI artifact removal may also impact waveform morphology. We have purposely limited our ERP analysis to three common midline sites (Fz, Cz, and Pz) that are informative for assessments of CAEP. In contrast, the CSD maps are based upon all scalp sites; however, this required the use of interpolated data for those parietal electrode sites where CI hardware impacted the integrity of the recordings. This may limit the fidelity of current sources rendered in posterior–parietal regions.

Conclusions

This study used a novel multisensory paradigm to assess auditory processing in TD children and children with CIs. The data suggest early responsiveness to ambiently presented auditory stimuli as indexed by the presence of the P1 frontal–central component in both populations. However, in contrast to CI-using children, TD children showed larger P1 amplitude effects and shorter P1 latencies. For the TD children, relative to the auditory-only

condition, concurrent audio and visual stimulation modulated both P1 amplitude and latency (leading to larger and later P1 peak amplitudes). In contrast, this same manipulation only affected P1 amplitude in CI-using children. If we take the longer P1 latency in TD to reflect an adaptive process in the face of competing visual stimulation, this may imply that CI-using children do not demonstrate adaptive processing in the presence of cross modal distraction. This finding might be a reflection of differences in cortical development of the auditory system. Examination of a later N2 component revealed further differences. This component was less evident in CI-using children suggesting that, compared to TD children, CI children may not engage in further linguistic elaboration of ambient speech. Finally, CSD solutions in the P1 and N2 time windows revealed further group differences that may be reflective of reduced specialization of auditory processing in CI-using children. Taken together, these data suggest that despite early interventions, congenitally deaf children may exhibit disruptions in the development of auditory and multisensory processing.

Data Availability Statement

The data sets generated during and/or analyzed during this study are available from the corresponding author on reasonable request.

Acknowledgments

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