Bone Conduction and Masking Effects on Cortical Auditory Evoked Potentials

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Abstract

Objectives: The P1 cortical auditory evoked potential (CAEP) is a biomarker of auditory cortical maturation, which has been mostly studied in individuals with hearing loss in the inner ear (sensorineural) using traditional air conduction testing. Fewer studies have investigated hearing loss at the level of the outer and/or middle ear (conductive), in which one treatment option is a bone conduction hearing aid that bypasses the outer and middle ear and often requires masking. Therefore, research is needed to investigate the impact of CAEPs via bone conduction. **Methods:** Bone conduction CAEPs without masking, with appropriate masking, and over-masking were compared to air conduction CAEPs in 20 normal hearing adults. Stimulus intensity was also varied in air and bone conduction conditions to reflect participants' most comfortable listening levels. **Results:** CAEP latencies did not significantly differ across conditions, but overall amplitudes for over-masking were reduced. Additionally, intensity of the stimulus did not significantly affect latencies or amplitudes. **Conclusion:** Cortical testing via bone conduction is similar to air conduction testing and therefore could be used to evaluate the development and maturation of the central auditory system in individuals who may be candidates for or use a bone conduction hearing aid. **Significance:** CAEP testing pre- and post-treatment with a bone conduction hearing aid could be useful in guiding clinical treatment decisions in the conductive hearing loss population.

1. **Introduction**

Cortical auditory evoked potentials (CAEP) are objective obligatory responses from the auditory cortex present from birth that have been demonstrated to reflect auditory cortical maturation (Dorman et al., 2007; Sharma et al., 2002a; Sharma et al., 2002b; Sharma et al., 2002c; Sharma et al., 2013; Sharma et al., 2005). Cortical auditory development is impacted by factors, such as appropriate and sufficient auditory stimulation. If a child does not receive enough auditory stimulation, as occurs with congenital hearing loss, then the auditory cortex may show atypical auditory cortical maturation (Dorman et al., 2007; Sharma et al., 2002a; Sharma et al., 2002b; Sharma et al., 2002c). As children develop and age, the CAEP response matures in morphology and latency due to underlying synaptogenesis and myelination. Typically, in adults the CAEP has three obligatory components: the P1, N1, and P2. These components are reliable biomarkers for the status of maturation in both primary and higher-order auditory cortices (Dorman et al., 2007; Sharma et al., 2013; Sharma et al., 2015). The P1 is the first positive peak and is generated in the primary auditory cortex. When a normal hearing baby is first born, their P1 will have a latency of about 300 ms (Ponton et al., 2000; Ponton & Eggermont, 2001; Sharma et al., 2002a; Sharma et al., 2002b; Sharma et al., 2002c). The P1 latency decreases throughout childhood until it reaches an adult latency of 50 to 70 ms (Ponton et al., 2000; Ponton & Eggermont, 2001). The N1 is the first negative peak, which first emerges as bifurcation of the P1 component. The N1 does not fully emerge until 6 to 7 years of age. Beginning in the second decade of life, the N1 will become more dominant (Ponton et al., 2000; Sharma et al., 2015). In adults with normal hearing, the N1 has a latency of around 100 ms (Wunderlich et al., 2006). The P2 is the second positive peak and occurs around 175 to 200 ms in normal hearing adults (Wunderlich et al., 2006). As the P1 response is present at birth, Sharma and colleagues have systematically documented the changes in the P1 CAEP response in normal hearing children and adults establishing age-based normative values and 95% confidence intervals for P1 latencies (Sharma et al., 2002a; Sharma et al., 2002b; Sharma et al., 2002c). These age-based normative values have been utilized to compare P1 responses from children with hearing loss to their normal hearing peers to determine if auditory cortical maturation is age-appropriate for the child (Cardon & Sharma, 2013; Sharma et al., 2002a; Sharma et al., 2002b; Sharma et al., 2002c, Sharma et al., 2005; Sharma et al., 2013).

Given these well documented maturational patterns of the CAEP response, the P1 component of the CAEP has also been used to evaluate the effects of hearing treatment on auditory cortical maturation in children with sensorineural hearing loss (i.e., hearing loss at the level of the inner ear) (Campbell et al., 2011; Cardon et al., 2012; Cardon & Sharma, 2013; Dorman et al., 2007; Sharma et al., 2005a; Sharma et al., 2005b; Sharma, et al., 2013). Studies have described auditory cortical development after hearing aid and cochlear implant treatment in groups of children with isolated sensorineural hearing loss (SNHL), SNHL and additional disabilities and auditory neuropathy (Campbell et al., 2011; Cardon & Sharma, 2013; Dorman et al, 2007; Pantelemon et al., 2019; Sharma et al., 2013). Clinical use of the P1 biomarker has also been effectively demonstrated in case studies. Campbell and colleagues (2011) describe three case studies of children with sensorineural hearing loss to demonstrate the effects of hearing treatment on the P1 biomarker. The first case was a child diagnosed with hearing loss at 0.87 years old and fit with bilateral hearing aids at 0.95 years old. CAEP responses from this child revealed a deprivation negativity. An unstimulated auditory pathway will often result in a deprivation negativity in CAEP responses, in which there is a large negativity before the P1 occurs at a delayed latency (Campbell et al., 2011). After 4.5 months of hearing aid usage, there was no longer a deprivation negativity and the P1 latency was within normal limits for their age. In the second case, a child was fitted with hearing aids at 0.13 years of age. After receiving bilateral hearing aids, their CAEP responses were measured and revealed a delayed P1 and deprivation negativity. These results suggested that the hearing aids were not stimulating the auditory cortex enough for an age-appropriate P1 response. The child then received a cochlear implant (CI) at 0.76 years old, and by 1 year of age, the child had a normal P1 latency (Campbell et al., 2011). Dorman and colleagues (2007) also presented two cases of children to demonstrate the effects of auditory deprivation before receiving CI. In the first case, the child had bilateral hearing loss and received bilateral hearing aids at 5 months old. After 7 months of hearing aid use, their CAEPs were recorded and the P1 was not detected, revealing that the hearing aids were not delivering a sufficient amount of sound for age-appropriate auditory cortical maturation. Since the hearing aids were not sufficient to provide the child with sound access, the child was implanted with a CI at 19 months old. Within 4 months of receiving the implant, the child's P1 was detected and within normal limits for their age, which corresponded with progress in speech and language development. This demonstrates that the CI was able to deliver enough auditory stimulation for the child to exhibit typical auditory cortical maturation for their age. In the second case, the child had bilateral severe-to-profound hearing loss with microtia (an underdeveloped outer ear), and atresia (absence of ear canal) also present in the right ear secondary to Goldenhar syndrome. At nine months old, the child received a hearing aid for their left ear and 17 months after the hearing aid was fitted, the CAEPs were recorded. However, there was not an identifiable P1, suggesting the hearing aid did not provide adequate auditory stimulation for age-appropriate development of the central auditory pathways. At two years and seven months, the child received a cochlear implant in the left ear. CAEP recordings post-implantation revealed that the P1 was still significantly delayed, consistent with reports of inconsistent implant use (Dorman et al., 2007). These case studies demonstrate the clinical utility of CAEP recordings in children with sensorineural hearing loss. Specifically, measuring CAEPs with hearing aid or CI amplification may assist clinicians in hearing loss treatment decisions and validation of appropriate hearing treatment. However, examination of CAEP responses is less established in children with conductive hearing loss (i.e., hearing loss at the level of the outer and/or middle ear) and has not been completed using these established stimuli and protocols which allow for the comparison of P1 CAEP responses to age-based normative values.

Conductive hearing loss occurs due to an impairment of the outer and/or middle ear in contrast to sensorineural hearing loss which is due to an impairment in the cochlea (inner ear) and/or auditory nerve. For example, conductive hearing loss can result from microtia and/or atresia, (which reflect abnormal development of the outer ear (pinna) and/or ear canal), and often co-occur and often present unilaterally (i.e., in one ear). Microtia and/or atresia may be treated with amplification delivered through a bone conduction hearing aid. This type of hearing aid allows for the delivery of sound through bone conduction pathways, bypassing the underdeveloped outer ear and middle ear, directly stimulating the cochlea through vibrations of the skull (Ikeda et al., 2021). Therefore, recent research has begun to explore the use of stimuli presentation through bone conduction transducers and their effects on CAEP components. Soares de Brito and Durante (2019) aimed to characterize CAEP responses using a bone oscillator headphone and tone bursts in neonates. Their results indicated no statistically significant differences in CAEP responses obtained via air conduction or bone conduction pathways, with P1 latencies occurring around 200–300 ms after stimulation. These findings suggest that bone conduction is a viable transducer for measuring CAEPs in neonates (Soares de Brito & Durante, 2019). Similar research has been conducted with normal hearing adults to investigate the reliability of both auditory brainstem responses (ABR) and CAEPs elicited from tone bursts presented through headphones to capture the response via the air conduction pathways compared to those recorded via the bone conduction pathways by directly streaming the tone bursts to a bone conduction hearing aid attached to a headband that was worn by the participants (Rahne et al., 2010). Results from 10 normal hearing adults found that direct stimulation to the bone conduction hearing aid produced clear and replicable responses for ABR and CAEPs. However, while ABR responses demonstrated latency shifts and stimulus artifact at higher intensity levels (i.e., 60 dB HL and 70 dB HL) that dissipated at lower intensity levels, CAEP responses did not show these limitations. The authors therefore concluded that CAEP testing may be particularly beneficial in individuals using bone conduction hearing aids (Rahne et al., 2010). Furthermore, CAEP testing in adults with acquired conductive hearing loss using a 1000 Hz tone burst presented through a bone oscillator has been completed showing differences in CAEP component amplitudes and latencies compared to normal hearing adults (Dabbous et al., 2024; Parry et al., 2019). While results from these studies are promising and demonstrate the feasibility of recording CAEP responses via the bone conduction pathway, further examination of CAEP responses elicited from speech sounds and presented through a bone oscillator are warranted.

Case studies utilizing a speech syllable have been described examining CAEP outcomes after hearing treatment in the pediatric conductive hearing loss population. Pantelemon and colleagues (2019) presented a case of a child with Goldenhar syndrome who presented with bilateral microtia with right ear stenosis (a narrow ear canal) and left ear atresia. The child presented with a moderate mixed hearing loss (hearing loss with both conductive and sensorineural components) in the right ear and severe mixed hearing loss in the left ear. After 27 months of bone conduction hearing aid use, this child exhibited normal P1 responses evoked via speaker while the child wore their bone conduction hearing aid (Pantelemon et al., 2019). A second case described a child with plurimalformative syndrome presenting with bilateral external auditory canal atresia, along with other middle ear bone malformations. The child was diagnosed with moderate conductive hearing loss in the left ear and severe mixed hearing loss in the right ear. The child used a bone conduction hearing aid attached to a soft band headband starting from 1.5 months old which was fully implanted at the age of 6. Approximately 6 months post-implantation, CAEPs were recorded via speaker with the child’s surgically implanted bone conduction hearing aid in use, revealing the P1 wave had normal morphology, but a potentially delayed latency (Pantelemon et al., 2020). These case studies demonstrate that CAEP testing pre- and post-bone conduction hearing aid use may be useful in guiding clinical decision making in a similar manner in which they are used for children with sensorineural hearing loss.

In the conductive hearing loss population, it is important to also consider how the presence of masking may impact CAEP responses given the high unilateral prevalence of conductive hearing loss. Masking is done by introducing an additional sound in one ear during the stimulation to the other ear to isolate one ear for testing. Recent studies have explored the effects of masking on air conduction recordings of CAEPs with studies reporting decreased amplitudes with masking (Cheng et al., 2024; Vander Werff et al., 2021). Additionally, previous studies have reported that the effects of masking may not be consistent across CAEP components (Cheng et al., 2024; Vander Werff et al., 2021). However, these studies presented the stimuli and masking noise via air conduction only. In contrast Soares de Brito and Durante (2019) included a bone conduction masking condition in their exploration of bone conduction CAEP responses in neonates and found no significant differences as a result of masking. Parry and colleagues (2019) further noted apprehension in testing adult unilateral conductive hearing loss participants in their normal hearing ear due to concerns of over-masking, in which the masking sound crosses back over to the ear being testing, reducing the validity of CAEP response. Therefore, exploration of the effects of both masking and over-masking on CAEP responses may aid in guiding the use of these objective CAEP responses in a clinical setting.

Despite a handful of previous studies, further systematic investigation of the impacts of bone oscillator presentation and masking on CAEP responses in the same group of subjects elicited from a speech stimulus with well-established age-based normative values is warranted prior to large scale studies of CAEP development in children with conductive losses. This could aid in the clinical uptake and use of objective CAEP measures. Therefore, the goal of this study was to evaluate sound presentation effects on CAEPs. Bone conduction CAEPs without masking, with appropriate masking, and over-masking were compared to air conduction on the CAEP components in normal hearing adults. We hypothesized that CAEP component latencies and amplitudes would not change as a function of air conduction compared to bone conduction but may differ in the presence of over-masking. Additionally, CAEP components were examined as a function of intensity level, as Rahne et al., (2010) noted stimulus artifacts may be present at high intensity levels when presenting via bone conduction. We predicted that there would be no latency effects as a function of intensity, but that amplitudes may decrease with intensity levels.

2. Materials and Methods

This study employed a cross-sectional design. The protocol was approved by the Institutional Review Boards at the University of Colorado Boulder. All subjects provided written informed consent prior to participation in the study.

2.1. Participants

Cortical auditory evoked potentials (CAEP) were recorded from 20 normal hearing adults between the ages of 18 to 38 years old (M = 24.8 years old). This study included 10 men and 10 women, with the average age of the men being 24.4 years (SD = 5.08) and the average age the women being 25.2 years (SD = 5.96) years. There were no statistical differences in age as a function of gender. Before CAEP testing, normal hearing was confirmed bilaterally in all participants through audiometric assessment, defined as hearing thresholds of 25 dB HL or better between the frequencies of 250 to 8000 Hz for both air and bone conduction. All participants presented without an air-bone gap greater than 10 dB HL. Figure 1 illustrates the averages of the participants' hearing thresholds for both air and bone conduction.

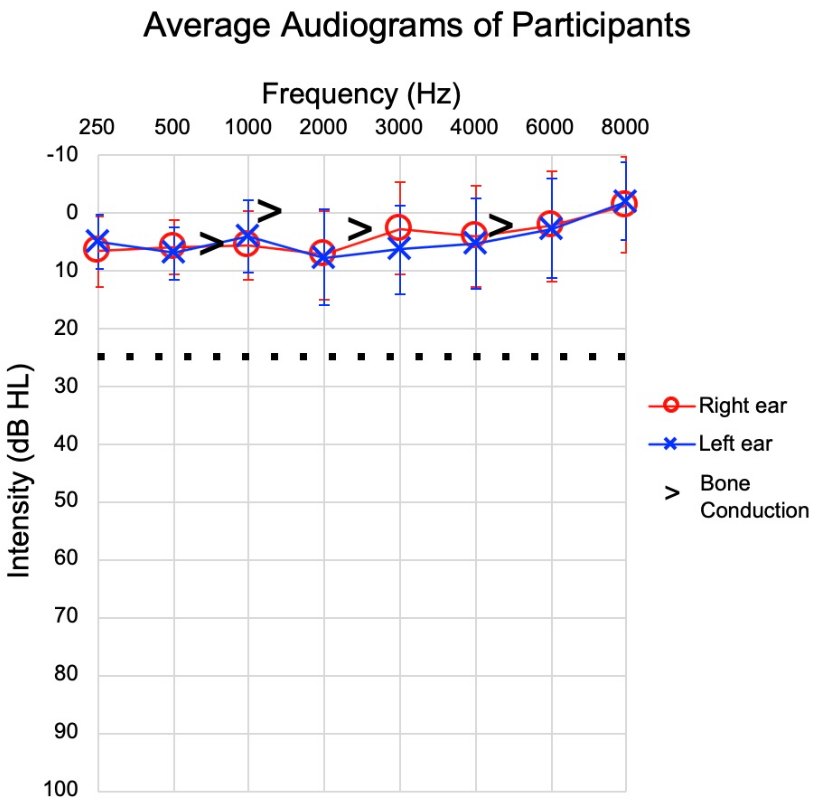

Figure 1. Average Audiogram for Participants. This audiogram shows the average of the participants' air conduction hearing thresholds and standard deviations for the right (red) and left (blue) ears, demonstrating their thresholds are within normal limits across all frequencies (25 dB HL or lower), which is represented with the dotted line.h Binaural average bone conduction thresholds are illustrated in black, with the difference between air conduction and bone conduction thresholds being within 10 dB HL.

2.2 Recording Cortical Auditory Evoked Potentials

CAEP responses were recorded in response to a well-established 90 ms synthesized speech syllable /ba/ (Sharma et al., 2002a; Sharma et al., 2002b; Sharma et al., 2002c). Testing for the participants occurred in an electromagnetically shielded sound booth where they were seated in a comfortable chair. The participant watched a movie on silent with subtitles during the recording. The test session, including audiometric assessments, electrode placement, and CAEP recording, took approximately 2 to 3 hours.

CAEP responses were measured using five electrodes. The active electrode was placed at Cz. The reference electrode was placed on the mastoid contralateral to the mastoid in which the bone oscillator was placed, and the ground electrode was placed on the forehead. Eye movements were monitored using two electrodes that were near the eye. Before placement of electrodes, the participants were instructed to put on an elastic headband on their head to secure the bone oscillator headphone, as previous pilot testing revealed this reduced stimulus artifact compared to the use of a traditional metal bone oscillator headband. Participants' heads were cleaned using rubbing alcohol and Nuprep abrasive paste to ensure impedances were less than 10 kOhms for all electrodes. Ten20 paste was used to place the electrodes on the participants which were then secured with medical tape. Cortical responses were recorded and analyzed using the Curry 7 software. Raw data was sampled at 1000 Hz and filtered from 0.1 to 100 Hz. Epochs contaminated by eye movements or external artifacts (over ±100 μV) were rejected. To ensure replicability, at least two trials of 250 sweeps were completed. A grand average of at least two replicable recordings was created from which peak latencies and peak amplitudes of each component (P1, N1, P2) for each individual participant was determined. Peak-to-peak amplitudes were calculated by subtracting the P1 amplitude and the N1 amplitudes (P1-N1 peak-to-peak amplitude) and the N1 amplitude and the P2 amplitude (N1-P2 peak-to-peak amplitude). Due to individual variability in peak amplitude magnitudes, only peak-to-peak amplitudes were utilized in this analysis.

The stimulus was delivered through soundfield speakers, through a bone oscillator headphone (RadioEar B71), and through a bone oscillator headphone with speech-shaped masking noise presented to the contralateral ear via insert earphone to create four testing conditions:

1. Speaker – Sound was presented at a fixed level of 65 dB HL through two loudspeakers positioned at a ±45° azimuth.
2. Bone Conduction – Sound was delivered through a bone oscillator headphone at a fixed level of 50 dB HL.
3. Appropriate Masking – An effective amount of masking of 45 dB HL (determined by measured hearing thresholds) was presented to one ear through an insert earphone, while the stimulus was delivered through the bone oscillator at a fixed level of 50 dB HL placed at the mastoid of the other ear.
4. Over-masking – Masking noise was increased to 70 dB HL played through an insert earphone which risked cross-hearing of the masking noise in the ear being tested (i.e., over-masking), meaning the masking noise could interfere with participants' ability to hear the target sound through the bone oscillator.

For all but one participant, the bone oscillator was placed on the right mastoid, while masking noise during the masking conditions was presented to the left ear. In one participant the bone oscillator was placed on the left mastoid and the masking noise was presented in the right ear due to partially occluding cerumen in the left ear.

Given that bone conduction artifacts may be present at higher stimulus intensity levels (Rahne et al., 2010) and that previous research has demonstrated that there are perceptual loudness differences between air conduction and bone conduction (Stenfelt & Håkansson, 2002; Stenfelt & Zeitooni, 2013), we examined the effects of intensity as a function of participants most comfortable listening level (MCL). To achieve this the stimulus was first set at 20 dB HL to start and while the stimulus was continuously playing, the participants were asked to indicate their comfort level of the stimulus using a loudness scale with 1 indicating the sound was very quiet, 6 being their MCL for the stimulus and 10 being very loud. Based on the participants' answer, the researcher would adjust the intensity by 5 to 10 dB HL until the participant indicated the stimulus was at their MCL. The testing at MCL was conducted with the speaker and the bone oscillator without masking conditions (i.e., conditions 1 and 2).

2.3 Statistical Analysis

The statistical analysis was conducted using R (version 4.2.1) and RStudio, an integrated development environment for R. A 3×3 repeated measures ANOVA was utilized to examine differences across the speaker, bone conduction, and two bone conduction with masking conditions for each CAEP component latency (P1, N1, P2). A 3×2 repeated measures ANOVA was employed to examine peak-to-peak amplitudes across these conditions. Furthermore, 3×2 repeated measures ANOVAs were used to examine the effect of keeping the intensity level of the stimulus constant for both speaker (65 dB HL) and bone conduction (50 dB HL) conditions separately compared to using an intensity identified by each participant as their most comfortable listening level (MCL) on each CAEP component latency. 2×2 repeated measures ANOVAs were used to examine the impact of these intensity differences on peak-to-peak amplitudes as well.

Assumptions of normality were assessed by examining the model residuals via quantile–quantile plots. CAEP component latencies underwent Box-Cox transformations when examining differences across the speaker, bone conduction, and two bone conduction with masking conditions, intensity effects in the speaker condition, and intensity effects in the bone conduction condition. CAEP peak-to-peak amplitudes underwent Box-Cox transformations when examining intensity effects in the bone conduction condition. QQ plots for the transformed data were reexamined. Overall, assumptions of normality in the central portions of the distribution were met, with only minor deviations in the tails. Previous research has shown that ANOVA is robust to modest violations of normality (Glass, Peckham, & Sanders, 1972; Lix, Keselman, & Keselman, 1996). Therefore, the analyses proceeded with the transformed data and the assumptions of normality were considered adequately met.

Assumptions of sphericity were assessed using Mauchly's Test of Sphericity. Mauchly’s test indicated that the assumption of sphericity was violated for latency and amplitude models when examining the effect of condition. Therefore, the Greenhouse-Geisser correction was applied to those results.

For significant ANOVA main effects and interactions, post-hoc analyses were completed utilizing non-parametric Wilcoxon Signed-Rank Tests, with Bonferroni-adjusted p-values.

3. Results

3.1 Stimulus Transducer Effects

3.1.1 Latency

Figure 2 illustrates the CAEP responses obtained in the speaker, bone conduction, bone conduction with appropriate masking, and bone conduction with over-masking conditions. A repeated-measures ANOVA revealed no significant main effect of condition ($F(3, 57) = 0.04$, $p = 0.958$), indicating that latencies do not differ across the four conditions. In addition, there was no significant interaction between CAEP components and conditions ($F(6, 114) = 0.26$, $p = 0.902$), suggesting that the latencies are consistent across conditions for each component. As expected, there was a significant main effect of component with the P1 occurring prior to the N1/P2 and the N1 occurring earlier than the P2 ($F(2, 38) = 215.49$, $p < 0.001$). Table 1 shows no significant difference in CAEP component latencies across conditions.

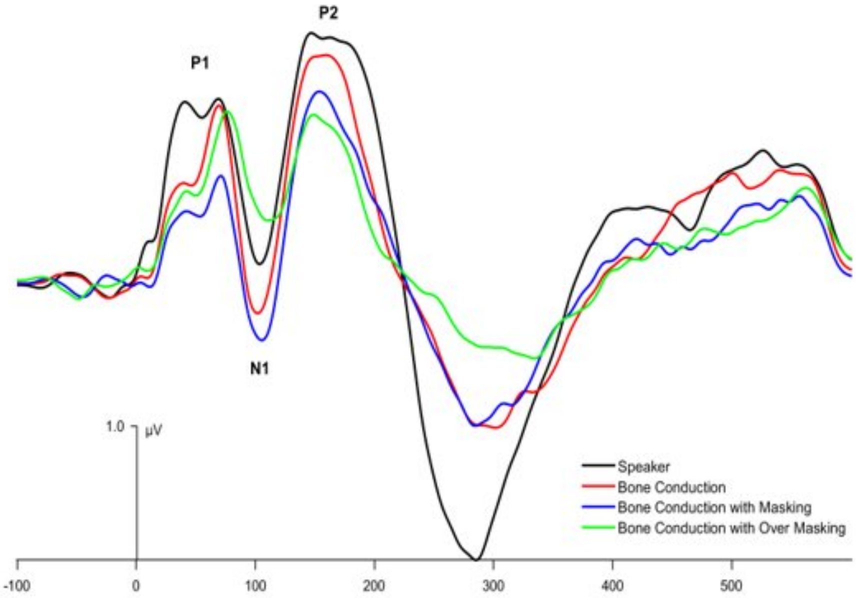

Figure 2. Average CAEP Responses as a Function of Stimulus Presentation Methods. CAEP responses were presented through soundfield speakers (black), through bone conduction (red), through bone conduction with appropriate masking (blue), and bone conduction with over-masking (green). There were no differences between the latencies (ms) of CAEP components (P1, N1, P2) across the different sound presentations. However, peak-to-peak amplitudes (μV) were significantly smaller in the over-masking condition compared to all other sound presentation conditions.

Latency (ms)			
	P1	**N1**	**P2**
Speaker	61.35 (15.62)	102.5 (13.89)	160.65 (28.30)
Bone Conduction	62.45 (18.89)	102.2 (16.16)	159.5 (27.56)
Appropriate Masking	63.4 (15.70)	100.55 (18.19)	161.0 (26.63)
Over-Masking	63.25 (19.26)	101.9 (21.41)	158.65 (31.88)

Table 1. Average Peak CAEP Latencies Across Sound Presentation Conditions. Average peak latencies and standard deviations are shown for each condition and component. There were no significant differences in the latencies of the P1, N1, or P2 component across sound presentations conditions (speaker, bone conduction, bone conduction with masking, and with over-masking).

3.1.2 Amplitude

A repeated-measures ANOVA with amplitude as the dependent variable revealed a significant effect of condition (F(3, 57) = 4.66, p = 0.026). Pairwise comparisons of the conditions revealed that overall, the peak-to-peak amplitudes were significantly smaller in the over-masked condition compared to the speaker (W=637, p=0.003), bone conduction without masking (W=706.5, $p<0.001$), and bone conduction with appropriate masking (W=678, $p<0.001$) conditions.

The main effect of component was not significant (F(1, 19) = 0.71, p = 0.708) nor was the interaction between component and condition (F(3, 57) = 1.57, p = 0.317). Therefore, as reflected in Table 2, the over-masked condition resulted in smaller peak-to-peak amplitudes for both the P1 to N1 peak-to-peak amplitude and N1 to P2 peak-to-peak amplitude compared to all other conditions.

Amplitude (µV)			
	P1 to N1	**N1 to P2**	**p-value**
Over-Masking	2.34 (0.99)	2.18 (1.03)	-
Speaker	3.3 (2.27)	3.84 (3.15)	*p=0.003***
Bone Conduction	2.95 (1.38)	3.38 (1.96)	*p<0.001****
Appropriate Masking	2.80 (1.25)	3.14 (1.60)	*p<0.001****

Table 2. Average Peak-to-Peak CAEP Amplitudes Compared to the Over-Masking Condition. Average peak-to-peak amplitudes and standard deviations are shown for each condition. Significantly smaller P1 to N1, or N1 to P2 peak-to-peak amplitudes were found in the over-masking condition compared to the speaker, bone conduction, and bone conduction with appropriate masking conditions.
** $p<0.01$
***$p<0.001$

3.2 Stimulus Intensity Level Effects

3.2.1 Speaker Condition

In the speaker condition, the MCL intensity selected by participants ranged from 25 to 75 dB HL. One participant selected an MCL above that used for the fixed intensity level of 65 dB HL. There was a significant main effect of component on latencies (F(2, 38) = 179.18, $p < 0.001$), but there was no significant main effect of condition (F(1, 19) = 1.53, p = 0.231), indicating that latencies did not differ as a function of intensity. Furthermore, there was no significant interaction between component and condition (F(2, 38) = 0.23, p = 0.795), confirming that for each component (P1, N1, P2) latency did not vary between the two intensity conditions (i.e., fixed at 65 dB HL and MCL).

The repeated measures ANOVA analysis for the comparison of peak-to-peak amplitudes obtained at a fixed intensity level of 65 dB HL and MCL revealed no statistically significant effects. These findings suggest that peak-to-peak amplitudes do not differ for the CAEP response as a function of moderate changes in intensity, as demonstrated in Figure 3.

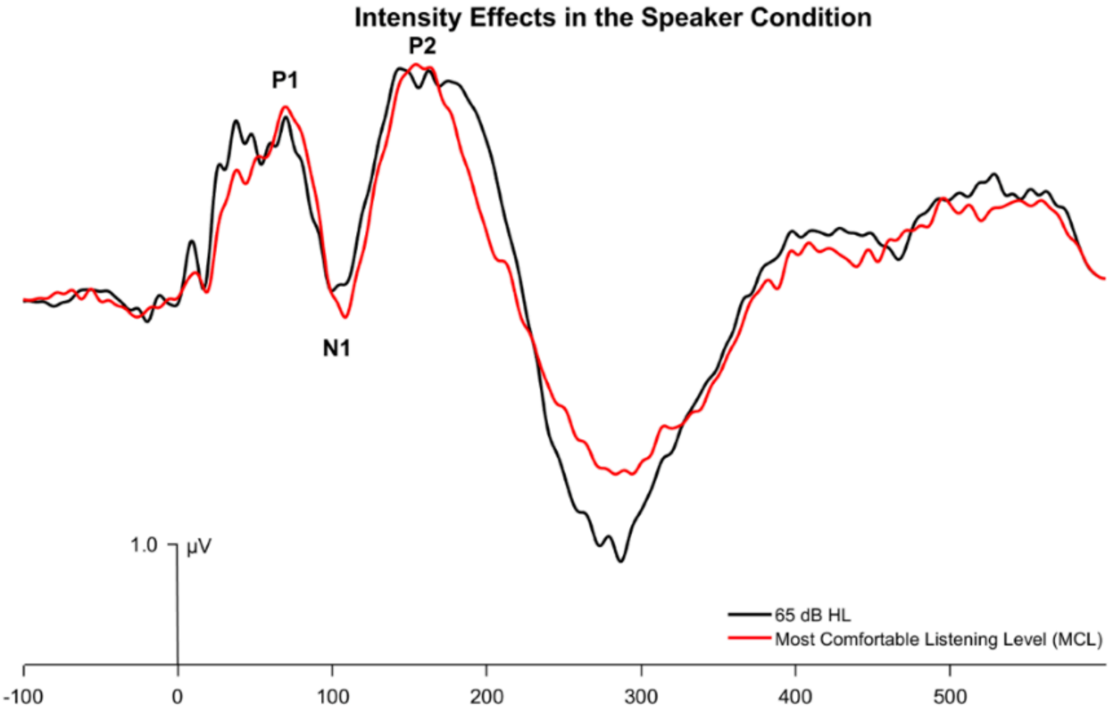

Figure 3. Intensity Effects in the Speaker Condition. CAEP responses were recorded at a fixed intensity of 65 dB HL (black) and at participants' MCL (red). There were no differences between the latencies (ms) and amplitudes (µV) of the CAEP components (P1, N1, P2) across the different sound intensities when delivering the stimulus through the sound field.

3.2.2 Bone Conduction Condition

In the bone conduction condition, the average MCL intensity selected by participants ranged from 20 to 60 dB HL. Seven participants indicated an MCL above that used for the fixed intensity level of 50 dB HL. In these participants, increased stimulus artifact was noted which partially obscured the P1 response as shown in Figure 4. Overall, when examining latencies, there was only a significant main effect of component (F(2, 38) = 195.45, $p < 0.001$). The latencies did not significantly change from the fixed intensity of 50 dB HL compared to participants selected MCL intensity levels.

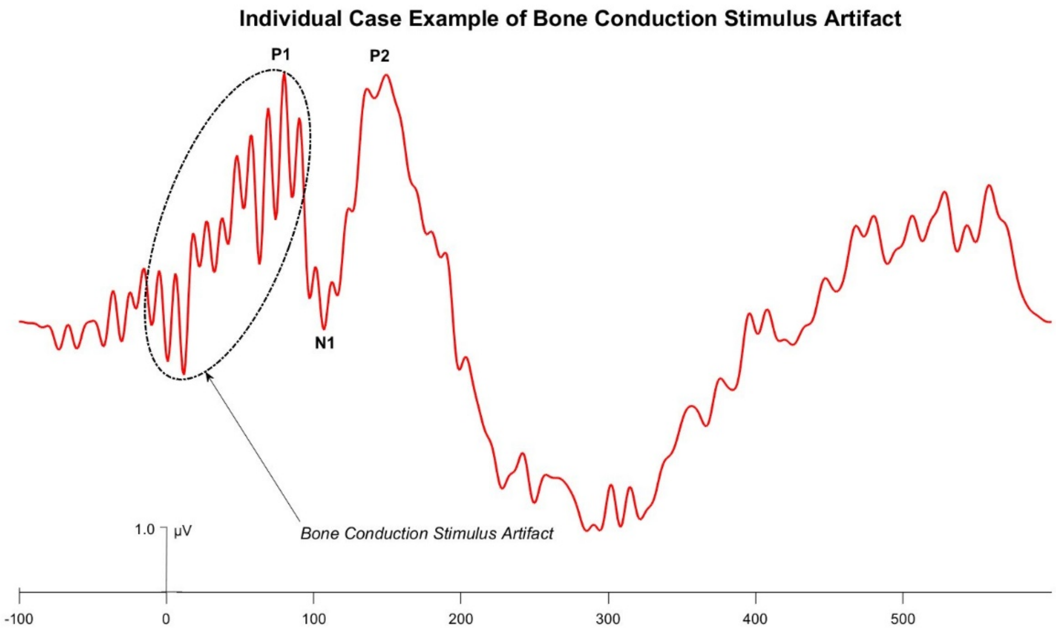

Figure 4. Individual Case Example of Bone Conduction Stimulus Artifact. The CAEP response (red) shown in this figure was recorded at a level of intensity chosen by the participant, corresponding to their MCL. The participant's MCL exceeded the fixed intensity of 50 dB HL, resulting in increased stimulus artifact. This artifact is particularly evident prior to and during the P1 response latency (ms).

Similarly, when examining amplitudes, no significant effects were noted indicating that the amplitudes of the CAEP components did not differ between the fixed intensity level and MCL when the stimulus was delivered via bone conduction (see Figure 5).

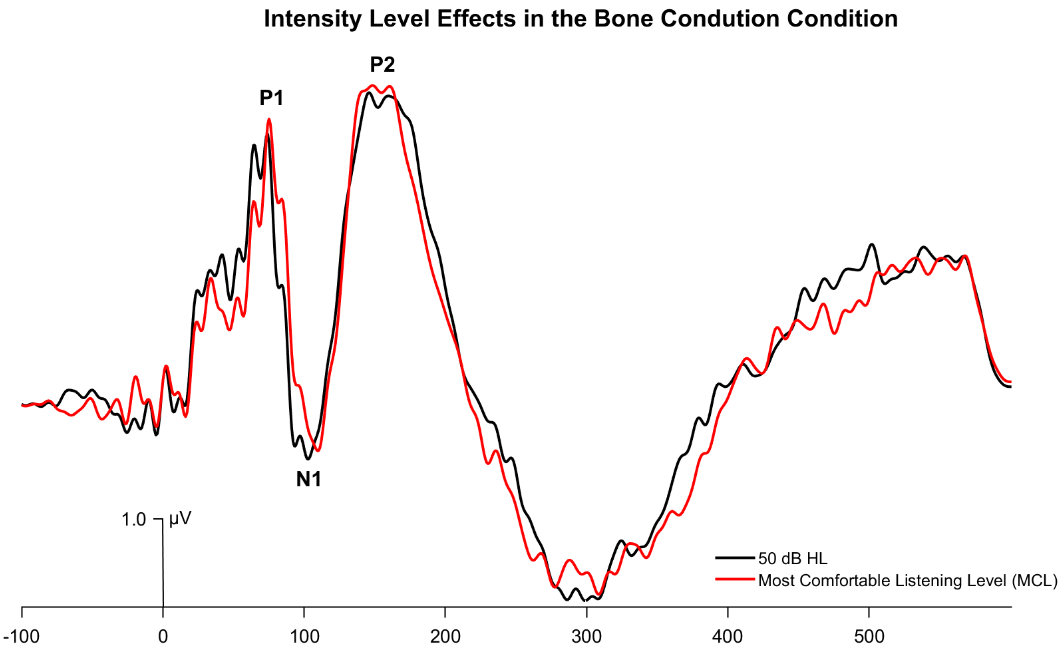

Figure 5. Intensity Effects in the Bone Conduction Condition. CAEP responses were recorded at a fixed intensity of 65 dB HL (black) and participants' MCL (red). There were no differences between the latencies (ms) and amplitudes (μV) of the CAEP components (P1, N1, P2) across the different sound intensities when delivering the stimulus via bone conduction.

4. Discussion

Overall, this study showed no significant differences in CAEP responses elicited with a speech sound when presented via sound field speakers or via bone conduction through a bone oscillator. CAEP component (P1, N1, P2) latencies were consistent across all stimulus presentation conditions including when presenting the stimulus with contralateral masking and varying the stimulus intensity. However, when examining the CAEP component amplitudes over-masking resulted in a reduced response. Interestingly, when comparing a fixed intensity level to individual MCL intensity levels amplitudes did not differ when presented via speakers or the bone oscillator.

The results of this study demonstrate that CAEP latencies were unaffected when the stimulus was presented via bone conduction using various levels of masking compared to a when the stimulus was presented via air conduction through a speaker. Similar to our study, Soares de Brito and Durante (2019) examined CAEP responses elicited via the bone conduction and air conduction pathways in neonates aged 6 to 28 days. After confirming normal hearing and no risk factors for hearing loss, the neonates were tested using tone bursts ranging from 500 Hz to 4000

Hz elicited via three ways: bone oscillator, bone oscillator with masking, and insert earphones. Consistent with our findings, their results showed no statistically significant differences in cortical responses across these presentation conditions, with age-appropriate latencies occurring around 200–300 ms after stimulation (Soares de Brito & Durante, 2019). Rahne and colleagues (2010) also noted a lack of CAEP latency shifts in normal hearing adults when comparing 1000 Hz and 2000 Hz tone bursts presented via the air conduction and bone conduction pathways. In that study, tone bursts were streamed directly to a bone conduction hearing aid attached to a headband worn by the normal hearing adults to elicit a CAEP response and compared to CAEP response collected with the stimuli presented via headphones (Rahne et al., 2010). Our study extends the results of previous studies showing the similarity of CAEP responses elicited via the bone conduction compared to the air conduction using a speech stimulus. Taken together, these findings suggest that CAEP responses produced via the bone conduction pathway are comparable to those elicited via air conduction across various types of stimuli, which may expand their clinical implementation. Importantly, established normative values with 95% confidence intervals have utilized P1 CAEP latencies with respect to age (Sharma et al., 2002a; Sharma et al., 2002b; Sharma et al., 2002c). Therefore, our results in conjunction with previous studies demonstrate the possibility of utilizing normative values to determine a child's age-appropriate auditory cortical maturation pre-and post-hearing treatment with bone conduction hearing aids.

Although the CAEP latencies in this study were found to be unaffected by masking, the amplitudes for the over-masking condition were reduced, consistent with previous research examining stimulus presentation and masking via air conduction pathways (Cheng et al., 2024; Vander Werff et al., 2021). In contrast, Soares de Boares and Durante (2019) found no effects of masking on P1 CAEP amplitudes. However, unlike the present study only one masking signal-to-noise ratio was employed in their study. Given that we saw no effects on amplitudes when using a more effective masking level, it could be that Soares de Boares and Durante did not reach over-masking levels in their study. Overall, our findings suggest that caution is needed when selecting the intensity of the masker. Given that microtia and/or atresia commonly occur unilaterally, future studies should carefully consider how to isolate the ear of interest when recording CAEP responses. In fact, Parry and colleagues (2019) examined CAEP elicited from a bone oscillator placed on the mastoid while presenting masking noise to the contralateral ear in

15 adults with unilateral conductive hearing impairment compared to 15 normal hearing adults. While not directly examined, the authors noted that they were unable to test the normal hearing ear in the adults with unilateral conductive hearing loss due to risks associated with over-masking. The authors suggested that over-masking could affect the validity of their findings. Taken together with the current results, it appears that over-masking could diminish significant amplitude findings in the conductive hearing loss population. However, it could be that different types of maskers also result in less effects on CAEP responses. A recent meta-analysis examining air conduction masking with an air conduction stimulus presentation indicates that different types of masking noise have less effects on CAEP results (Rocha et al., 2024). While not directly examined in our study, differential masker noise effects could also be present when using air conduction masking when presenting stimuli through the bone conduction pathway, as was done in this study. Therefore, future studies could explore how different types of masking noise affect CAEP morphology, particularly in clinical populations.

Our results indicated a lack of effect of intensity on CAEP responses. That is when comparing a fixed intensity level to individual MCL intensity the amplitudes and latencies did not differ when presented via speakers or the bone oscillator. In contrast, a recent CAEP study including normal hearing listeners reported significant N1 to P2 amplitude effects when decreasing the intensity by 20 dBA steps and only N1 latency effects from CAEP response obtained from Cz (Legris et al., 2022). Similarly, when investigating seven different intensity conditions that ranged from 30 dB HL to 90 dB HL in normal hearing listeners, stimulus intensity increases resulted in CAEP latency decreases and amplitude increases (Billings et al., 2007). Differences in intensity level effects reported from our results may be due to the fact that this study chose to investigate the impact of presenting at individual participants' MCL, which resulted in very modest changes in intensity in some participants, rather than decreasing the intensity by pre-specified step sizes. It is also important to note that some participants chose an MCL intensity level greater than that of the fixed intensity level conditions. Importantly, greater stimulus artifact was noted when increasing the intensity level during the bone conduction condition. In contrast, Rahne et al. (2010) noted a lack of stimulus artifact effects in CAEP responses compared to ABR responses elicited through bone conduction. While we were able to determine peak latency and amplitudes for all components when stimulating via the bone oscillator, our study did document the presence of stimulus artifact in some participants.

Differences in the presence of stimulus artifact may be due to the differences in transducers. While this study utilized a bone oscillator, Rahne and colleagues (2010) utilized a bone conduction hearing aid attached to a headband in which the stimulus was directly streamed to the bone conduction hearing aid, likely minimizing the stimulus artifact. Future research may wish to continue to examine bone conduction stimulus artifacts in more detail as a function of transducers, intensity, and filtering parameters.

Overall, the results of the present study show promise in the use of established age-based CAEP normative values independent of stimulus transducers. These results may suggest that future studies could examine children with microtia and/or atresia pre- and post-bone conduction hearing aid treatment. However, over-masking concerns may impede the ability of researchers and clinicians to obtain ear specific CAEP responses in some individuals in the unilateral conductive population. Future studies should continue to investigate the use of CAEPs in this population, which could become important in guiding clinical decision making.

5. Conclusions

The CAEP response is an objective biomarker of auditory cortical development. Previous research has established age-based normative values to compare auditory cortical maturation in clinical populations. While pre-and post-treatment CAEP outcomes have been well established in some clinical populations (i.e., individuals with sensorineural hearing loss), relatively less research has examined individuals with conductive hearing loss. Conductive hearing loss may be treated with a bone conduction hearing aid which delivers amplification through the bone conduction pathway. This study extends previous results by demonstrating that there is no significant difference between CAEP outcomes elicited via the air conduction and bone conduction pathways with an established stimulus associated with age-based normative values. However, careful consideration is needed when selecting appropriate masking intensity levels as over-masking may affect CAEP component amplitudes. Overall, the results from this study suggest that the P1 CAEP biomarker may be useful in the conductive hearing loss population and could be utilized pre- and post-bone conduction hearing aid treatment to monitor cortical auditory development.

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