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Auditory cortical stimulability in non habilitated individuals – An evidence from CAEPs

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ABSTRACT

Objective: The effect of long term auditory deprivation on Cortical Auditory Evoked Potentials (CAEPs) especially in human models is not well explored. Hence, the current study was aimed to investigate the effects of long-term auditory deprivation and stimulability of auditory cortex in non habilitated congenitally deaf adolescents and adults using CAEPs.

Methods and Results: A total of 27 non-habilitated congenitally deaf adolescents/adults with age and gender matched normal hearing adolescents/adults participated in the study. The congenitally deaf group was fitted with high gain hearing aids (first fit). Further, the CAEPs were recorded. The obtained CAEP components were assessed for group effect, source and topographical differences. The between group analysis for CAEP responses showed a significant difference only for P2 latency and amplitude. The source analysis revealed that, in the normal hearing group for CAEPs, the sources were within the temporal regions. However, in the congenitally deaf group, along with the temporal cortex, the bilateral prefrontal cortex also was activated.

Conclusion: The findings revealed that it is possible to stimulate and evoke a matured CAEP response from a long deprived auditory system with adequate acoustic stimulation. The presence of CAEP responses is indicative of the functionality of the innate auditory pathway and the crossmodal plasticity in long auditory deprived individuals.

1. Introduction

Evidence from the literature reveals that there are specific areas mapped in the central nervous system for different functions. These areas entail adequate stimulation to mature and lack of it would lead to deprivation. Auditory maturation also depends on various factors such as length of auditory deprivation, age of onset, specific auditory experience, speech and language acquisition. The researchers have consistently reported that CAEPs responses would be abnormal or absent in individuals with auditory deprivation (Dorman et al., Knudsen, 2004; Kral et al., 2002b; Ruben, 1999; Sharma et al., 1997; Sharma et al., 2005a,b). However, in literature, there are few studies which have reported of matured CAEP responses in congenitally deprived hearing impaired individuals. (Lammers et al., 2015) reported matured CAEP responses in cochlear implanted adults soon after the rehabilitation. (Abraham et al., 2015, 2017) reported of matured adult like CAEP responses in congenitally deaf adults followed by first fit of hearing aid. The maturational patterns of CAEPs are quite well established up to 13 years of age. This assumption is not yet tested in long deprived individuals beyond this age. Thus, this hypothesis, which is mostly tested on animals and younger hearing impaired should be put to test before extrapolating it to the adult population.

There are evidences from literature which suggest a mixed representation of both auditory and visual areas in adult brain (Chen et al., 2016; Finney et al., 2001, 2003; Pockett et al., 2013). Further, recent evidence indicates that an auditory stimulus can evoke potentials over visual cortex even in normal hearing individuals (Pockett et al., 2013) strongly indicating that prelingually deafened adults could possibly show matured auditory responses due to this multimodal representation of auditory and visual cortex.

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There are not much published clinical studies to test this assumption that, children and adults with hearing impairment can have matured CAEP after critical age in spite of not being provided with auditory rehabilitation. Further, there are not many studies explain the crossmodal plasticity, especially the role of visual cortex over auditory areas in congenitally long deprived individuals using EEG.

Hence, there is a need to study, whether the CAEP responses are present in congenitally long deprived non habilitated adolescents and adults and the possibility of evoking matured CAEPs in them. Further, to understand the role of cross modal stimulation; especially, the role of visual stimulation on auditory cortex. Thus, it is presumed will help to understand why there is limited benefit from aural rehabilitation, especially after the critical period. In addition, the current study would also help to understand the stimulability of auditory cortex and crossmodal plasticity after prolonged period of deafness.

2. Method

The study was carried out using the cross sectional-observational research design. The participants who met the inclusion criteria were recruited for the study using purposive sampling method. In the congenitally deaf group, those who were having past or present history of middle ear pathology, who received any type of aural rehabilitation, those receiving benefit with the aid and who were using oral mode of communication were excluded from the study. The participants were recruited into two groups, Group I: non habilitated prelingual hearing impaired adolescents and non habilitated prelingual hearing impaired adults (Congenitally deaf group – 27 participants); Group II: normal hearing age and gender matched adolescents and normal hearing age and gender matched adults (Normal hearing group– 27 participants). Age range of the participants was 13–45 years.

All the measurements were carried out in acoustically treated rooms with permissible noise levels as recommended by ANSI (American National Standards Institute, 1991). The ear canal and tympanic membrane were examined using an otoscope for the presence of perforation, cerumen, infection, debris and foreign body in the ear canal. A duly calibrated diagnostic audiometer was used to obtain all the participant's Air Conduction (AC) and Bone Conduction (BC) thresholds. The status of the middle ear was assessed with a duly calibrated middle ear analyzer using tympanometry and reflexometry. The group I was fitted with a digital hearing aid (extra strong class). The participant's demographic details and audiograms (250 Hz–8 KHz for AC and 250 to 4 KHZ for BC) of the test ear were entered in the hearing aid specific programming software and saved. The hearing aid was programmed (Best Fit) based on the participant's audiogram thresholds.

Prior to the hearing aid fitting, an electro acoustic measurement was carried out to examine the working condition of the aid and to cross verify with manufactures specifications using Fonix7000 (version 1.63, Frye Electronics, USA). After the hearing aid fitting, an insertion gain measurement was carried out to ensure the target real ear SPL from the hearing aid. The benefit with the hearing aid was further measured using a functional gain measurement where, the participant was made to sit in a free field and warble tones were presented from the audiometer through a speaker. The participant was instructed to respond for the sound by raising the finger whenever the sound was heard. The responses were expected to be within the speech spectrum (at least 1 kHz below 50 dB HL). If the responses were exceeding the speech spectrum, the gain of the hearing aid was increased and re assessed; yet again, if the thresholds were falling above the speech spectrum, the subjects were excluded from the study. The participants hearing thresholds are shown in Fig. 1.

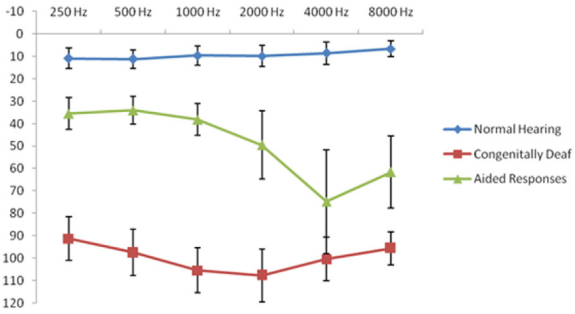


Fig. 1. Pure tone average (dB HL) across the frequencies for normal hearing and aided and unaided conditions in the congenitally deaf group.

2.1. Electrophysiological testing

2.1.1. ERP recording

ERPs were recorded from 32 Ag/AgCl electrode cap and arranged based on 10–20 electrode montage classification system. The location of the electrodes was midline Fz, FCz, Cz, CPz,Pz (5 electrodes), frontal FP1/FP2, F7/F8, F3/F4, FT7/FT8 (8 electrodes), central C3/C4, CP3/CP4 (4 electrodes), parietal P3/P4, P7/P8 (4 electrodes), occipital O1, O2, Oz (3 electrodes) temporal T7/T8, TP7/TP8 (4 electrodes) and ground (GND) electrode. These electrodes were referenced to GND electrode in the cap and right and left mastoid. Two bipolar channels, HEOG and VEOG were used to monitor horizontal and vertical eye TM movements along with the scalp EEG. The electrodes were connected to SynAmps (Compumedics, Neuroscan, Charlotte, NC, USA) amplifier for amplifying the EEG from the electrodes. EEG was recorded using 'ACQUIRE' module in 'NeuroScan' system and TM channels were continuously digitized at a rate of 256 Hz by a SynAmps amplifier and a 50 Hz notch filter was applied to all the channels. Further, BESA 6.1 software was used for the offline processing of the prerecorded EEG.

The stimuli was generated and presented through 'SOUND' module of STIM 2 CompumedicsNeuroscan SystemsTM software (CompumedicsNeuroscan, Charlotte, NC, USA) with a sampling rate of 44,100 Hz. The duration of the stimulus was 130 ms and the stimulus was windowed with Blackman window of 15% length at both the ends to avoid formation of clicks. Transducer used was speaker (Martin Audio C115, UK) which was connected to an amplifier (CROWN D75, USA). The speaker was placed at the ear level at 45° angle and 3 feet distance. The stimuli were coded with a trigger value for sorting the evoked responses. The total number of stimuli presented was 100 with an Inter Stimulus Interval (ISI) of 1000 ms at 30 dB SL of the participants 1 kHz threshold (in the normal hearing group) and aided 1 kHz pure tone threshold (in the deaf group). SLM was used to measure the real time sound level.

2.2. Statistical analysis

The significant differences in the CAEP responses were analyzed using descriptive statistical methods and cluster probability permutation statistics. The statistical analysis was performed using SPSS15.0 (SPSS Inc., Chicago, USA). Repeated measure of ANOVA was used for within group comparison and MANOVA was used to assess between group difference for latency and amplitude. Further, the CAEP responses were subjected to cluster permutation statistics and the source images were subjected to image cluster permutation statistics using BESA statistics 2.0. This analysis was carried out between congenitally deaf and normal hearing individuals using a non-parametric independent *t*-test with 1000 permutations.

3. Results

3.1. CAEP in normal hearing and congenitally deaf group

The CAEPs were present in all the participants between 80 - 130 ms latency range for N1 and 150–250 ms for P2. The congenitally deaf group revealed a bifid P2, whereas, the normal hearing group showed a single peak response. The grand averaged CAEPs from all the channels in both these groups are shown in Fig. 2A and the selected electrodes (ROI) are shown in Fig. 2B.

3.1.1. N1 latency

The N1 CEAP component was observed between 80 and 130 ms latency range in both groups. The mean and standard deviation of N1 component is shown in Table 1.

In the normal hearing group, the N1 latency significantly differed between the Oz, Cz, and Fz electrodes (F (2, 52) = 3.7, p = 0.029). However, the post-hoc pairwise comparison with Bonferroni correction failed to reveal any significant difference between these electrodes. The N1 latency at Oz, T7, and T8 did not differ significantly from each other (F (2, 52) = 1.07, p = 0.350). In the congenitally deaf group, the comparison of N1 latency between Oz, Cz, and Fz electrodes did not show any significant difference (F (2, 52) = 2.83, p = 0.068). However, the Oz, T7, and T8 electrodes showed a significant difference (F (2, 52) = 3.89, p = 0.037). Further, the post-hoc analysis with Bonferroni correction did not reveal any significant difference among the electrodes. The between-group analysis of the N1 latency failed to show any significant difference between Oz, Cz, and Fz electrodes (F (1, 50) = 1.49, p = 0.227; Wilk's Λ = 0.918). Similar results were observed at Oz, T7, and T8 electrodes (F (1, 50) = 2.40, p = 0.078; Wilk's Λ = 0.874).

3.1.2. P2 latency

The P2 component was bifid in the congenitally deaf group and the first peak was considered for the measurement of latency and amplitude. The first peak was observed between 150 and 200 ms latency range and the second peak observed between 200 and 250 ms. The mean and standard deviations are shown in Table 2.

In the congenitally deaf group, the comparison of P2 latency across Oz, Cz, and Fz electrodes revealed a significant difference (F (2, 52) = 7.54, p = 0.001). The post-hoc analysis using Bonferroni correction showed that the P2 latency at Oz was significantly longer than Cz electrode site (p = 0.001). However, the comparison between Oz, T7, and T8 did not reach a statistical significance (F (2, 52) = 0.191, p = 0.755). In the normal hearing group, the P2 latency across Oz, Cz, and Fz showed a significant difference (F (2, 52) = 9.37, p = 0.002). Further, the post-hoc analysis using Bonferroni correction showed that the latency at Oz was significantly longer than Cz (p = 0.009) and Fz (p = 0.013). Similarly, Oz, T7 and T8 too showed a significant difference (F (2, 52) = 12.21, p = 0.001) and the P2 latency was significantly longer at Oz electrode site compared to T7 (p = 0.007) and T8 (p = 0.001). The group had a significant effect on P2 latency at Oz, Cz and Fz electrode sites (F (1, 50) = 4.22, p = 0.010; Wilk's Λ = 0.798) where, the normal hearing group showed a shorter latency than congenitally deaf group at Oz (p = 0.013) and Fz (p = 0.035) electrode sites. The comparison of Oz, T7 and T8 showed a significant difference (F (1,50 = 13.02, p = 0.001; Wilk's Λ = 0.561) and the pair-wise comparison using Bonferroni Post-hoc test revealed that the congenitally deaf group had a significantly longer latency at Oz (p = 0.013), T7 (p = 0.001) and T8 (p = 0.001).

3.1.3. N1 amplitude

The N1 amplitude varied considerably across the participants including both the groups. In both the groups, the sphericity was violated for Oz, Cz, Fz and Oz, T7, T8 electrode groups. Hence, a repeated measures of ANOVA with Greenhouse-Geisser correction was applied. The mean and standard deviations are shown in Table 3.

In the normal hearing group, the electrode effect was significant on N1 amplitude across Oz, Cz and Fz (F (1.38, 36.12) = 56.04, p = 0.001) electrodes and the post-hoc analysis using Bonferroni correction showed that the amplitude was higher at Oz than Cz (p = 0.001) and Fz (p = 0.001). The other electrode comparison that is Oz, T7 and T8 did not reach a statistical significance (F (1.75, 45.54) = 2.80, p = 0.078). The repeated measures analysis in the congenitally deaf group showed a significant difference for the N1 amplitude on the Oz, Cz, Fz (F (1.34, 35.02) = 58.61, p = 0.001); where, the amplitude at Cz was significantly larger than Oz (p = 0.001) and Fz (p = 0.018). The Oz, T7 and T8 (F (1.63, 42.38) = 9.32, p = 0.001) comparisons too showed a significant difference. Further, the post-hoc analysis revealed a significantly higher amplitude at Oz electrode site T7 (p = 0.002) and T8 (p = 0.001). The comparison of N1 amplitudes between the participant groups at Oz, Cz, Fz and Oz, T7 and T8 failed to show significant difference (F (1, 50) = 1.92, p = 0.138; Wilk's Λ = 0.89) and (F (1, 50) = 2.36, p = 0.082; Wilk's Λ = 0.87), respectively.

3.1.4. P2 amplitude

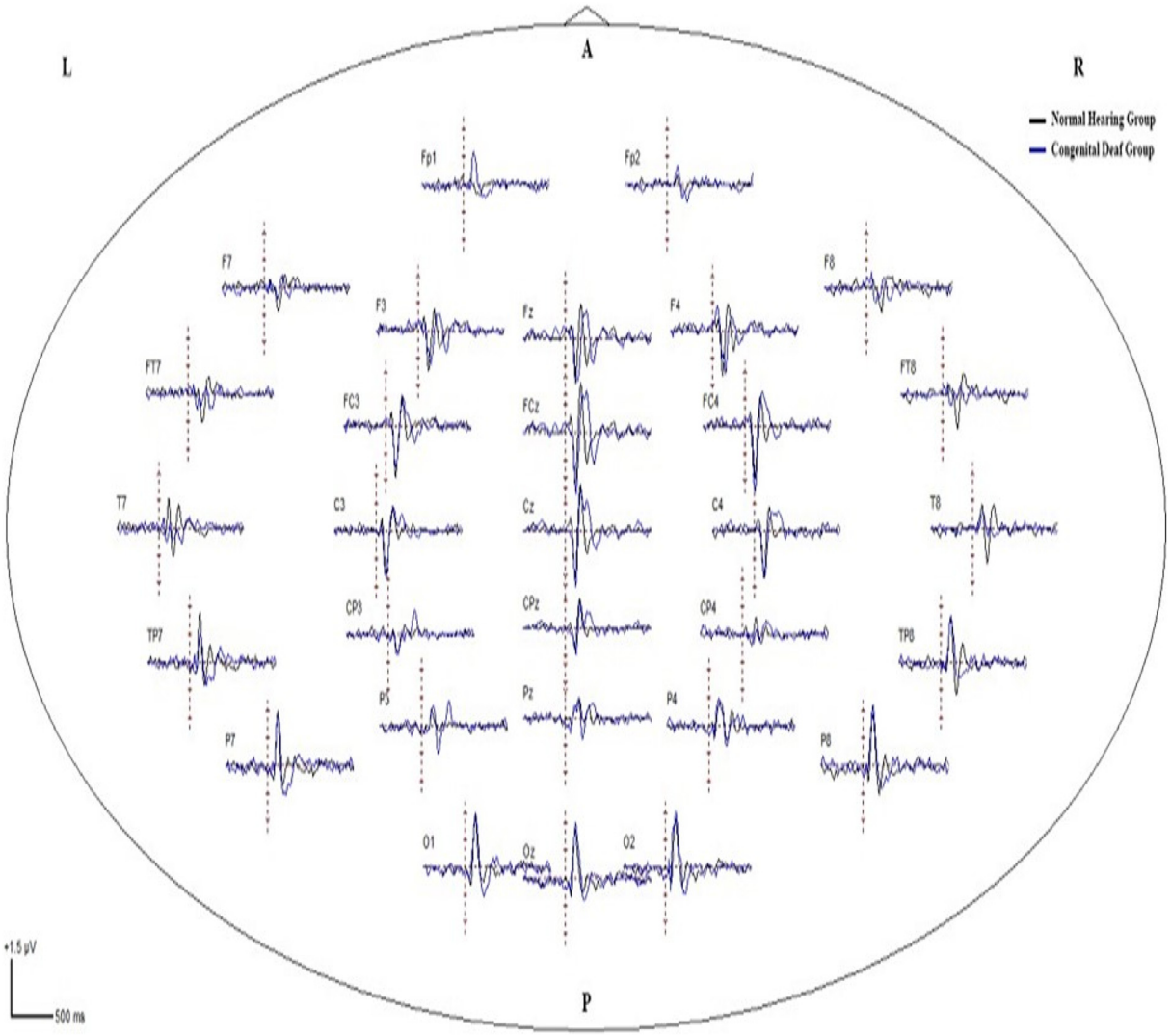
Similar to N1 amplitude, the P2 amplitude also varied between the participants. In the congenitally deaf group, the sphericity was violated for Oz, T7 and T8 comparison, whereas, in the normal hearing group the violation was between Oz, Cz and Fz. Hence, Greenhouse-Geisser correction was applied prior to the analysis. The mean and standard deviations of P2 amplitude is shown in Table 4.

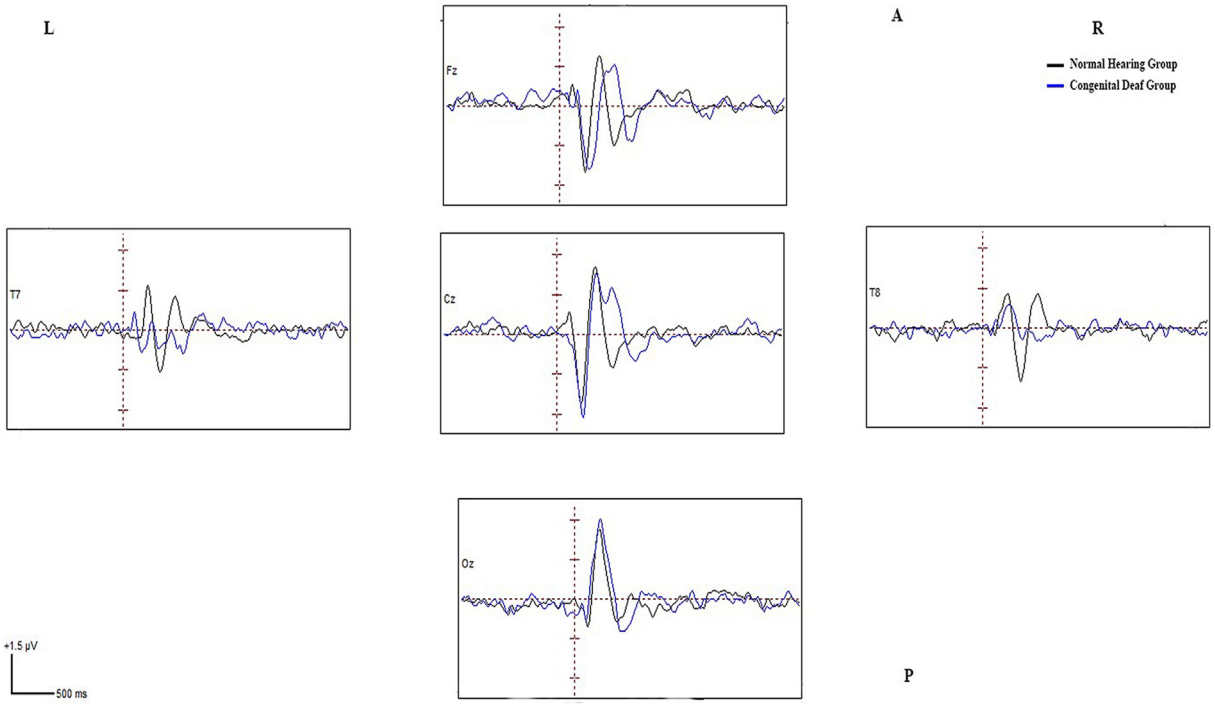
In the normal hearing group, the P2 amplitude showed a significant difference between Oz, Cz, and Fz (F (1.47, 38.29) = 22.25, p = 0.001) and Oz, T7, and T8 (F (2,52 = 11.81, p = 0.001). The post-hoc analysis using Bonferroni correction showed that the amplitude was significantly higher at Cz (p = 0.001) and T8 (p = 0.001). In the congenitally deaf group, P2 amplitude comparison between the electrode showed only a significant difference between Oz, Cz and Fz (F (2, 52) = 7.16, p = 0.002). Further, the post-hoc comparison showed that the amplitude found to be significantly lesser at Oz. However, Oz, T7 and T8 did not reach a significance (F (1.59, 41.45) = 0.115, p = 0.848). The group had a significant effect on P2 amplitude at Oz, Cz and Fz electrode sites (F (1, 50) = 3.30, p = 0.028; Wilk's Λ = 0.835). Further, the pair wise comparison showed a significantly higher amplitude at Fz (p = 0.021) electrode in normal hearing group. Also, the Oz, T7 and T8 electrode comparisons showed a significance (F (1, 50) = 5.76, p = 0.002; Wilk's Λ = 0.743), and the post-hoc analysis using Bonferroni correction revealed that the amplitude at T8 (p = 0.021) site was significantly larger in normal hearing group.

3.2. Source analysis of CAEPs

The source analysis was performed on grand averaged CAEP waveforms of the normal and the congenitally deaf groups using CLARA in BESA source analysis module. The time range for the analysis was from −50 to 500 ms and the highest peaks in the Global Field Power (GFP) were considered for the source localization. In the normal hearing group, the activity was peaked at 112 ms and the source activity was in the right and left temporal regions as shown in Fig. 3A. Similarly, the second highest source of activity peaked at 176 ms in the right-left temporal regions and the sources are shown in Fig. 3B.

In the congenitally deaf group, the CAEP responses showed three peak activities at 112 ms, 204 ms and 248 ms, respectively.


L
A
R
Normal Hearing Group
Congenital Deaf Group
Fp1
Fp2
F7
F3
Fz
F4
F8
FT7
FC3
FCz
FC4
FT8
T7
C3
Cz
C4
T8
CP3
CPz
CP4
TP7
TP8
P3
Pz
P4
P7
P8
O1
Oz
O2
+1.5 µV
500 ms
P


L
A
R
Normal Hearing Group
Congenital Deaf Group
Fz
T7
Cz
T8
Oz
+1.5 µV
500 ms
P

The first source (at 112 ms) localized to the right, the left temporal cortex, and another source of activation was in the bilateral prefrontal cortex (Fig. 4A). The second activity (at 204 ms) also showed a similar pattern of activation like normal hearing group except for an activity in the prefrontal cortex (Fig. 4B). The third activity (at 248 ms) was in the prefrontal cortex and the activity was seen only in the congenitally deaf group (Fig. 4C). Thus, the major difference observed between the two participant groups was the presence of the third peak at 248 ms in GFP and an additional consistent activity in the bilateral prefrontal cortex across the peak time points.

3.3. Permutation statistics of CAEPs

The group difference in the scalp distribution between the congenitally deaf and the normal hearing groups was analyzed using BESA Statistics for a −500 to 1000 ms time window with 1000 permutations and a p value of 0.05. The cluster permutation statistics was performed using a two-tailed independent t-test. Similarly, the CLARA source images for the CAEP were subjected to cluster permutation statistics using two-tailed independent t-test.

The results of the permutation cluster analysis for CAEPs between the deaf group and the normal hearing group showed that 11 clusters were similar between them. However, out of 11 clusters, five clusters reached a statistical significance. The test results are shown in Fig. 5A and Fig. 5B. The significant clusters were located on the midline (Fz, FCz, Cz and CPz), right fronto-central (F3, FC3 and C3), left frontal (F4, FC4 and C4), right temporal (FT7, T7, TP7and P7) and left temporal (F8, FT8, T8, TP8 and P8). However, the ROI was Fz, Cz, T7 and T8, hence only these electrode sites were reported in the result section. The clusters at the T8, Fz and Cz sites showed a highly significant difference ($p = 0.001$), ($p = 0.003$), and ($p = 0.001$), respectively, at around 250 ms and the T7 site showed a less significant cluster ($p = 0.008$) at around 250 ms for the between-group comparison. However, in comparison to other sites only T8 showed a significant difference at two time ranges, which was around 100 ms and 250 ms, respectively.

The statistical analysis with CLARA images for CAEPs in the congenitally deaf group vs the normal hearing group did not show any statistical significance. However, only one cluster showed a trend of significance ($p = 0.57$) and the Talairach coordinates for the cluster was (X = −31.5, Y = −2.9, Z = 16.7). Further, the group difference between the subjects showed activation in the right prefrontal cortex and the activation was higher in the deaf group than in the normal hearing group. The cluster and the group differences are shown in Fig. 6.

4. Discussion

The maturation of auditory cortex is highly bound to adequate acoustic stimulation and age (Knudsen, 2004; Kral et al., 2002; Kral et al., 2002; Ruben, 1999; Sharma et al., 2005a,b). These maturational changes can be traced using CAEPs. The maturational changes of CAEP responses are well documented in normal hearing individuals and in aural rehabilitation with respect to the age and the related changes occurring in CAEP responses (Eggermont, 1985; Eggermont and Ponton, 2003; Kushnerenko et al., 2002; Ponton et al., 2002; Sharma et al., 2004). These CAEP responses mature in a comprehensive manner within the period of critical age with adequate auditory stimulation. The absence of acoustic stimulation will lead to an immature central auditory system. After the critical age, especially after seven years, the absence of auditory stimulation reveals an absent or an abnormal pattern of CAEP response (Dorman et al., 2007; Knudsen, 2004; Kral et al., 2002b; Ruben, 1999; Sharma et al., 1997, 2005).

The current study results showed consistent and reliable matured adult-like CAEP responses in long deprived congenitally deaf adolescents and adults. These responses were consistent across the participants and the N1−P2 responses were well within the normative. On the morphological examination of latency and amplitude, the responses were comparable to the normal hearing group with few observable morphological differences. The congenitally deaf, long deprived adolescents and adults group showed slightly early latency and higher N1 amplitude in the central and frontal electrode sites. However, there was no significant difference in the statistical analysis. It was also observed that the deaf individuals showed a bifid P2 in the frontal and central electrode sites (F3, F4, FC3, FC4, C3, C4, Fz, FCz, Cz and CPz). In addition, the P2 latency and amplitude showed a significant difference between the groups.

The findings from the current study of the presence of matured adult-like CAEPs are in line with the previously reported studies in late implanted adults (Gordon et al., 2008; Lammers et al., 2015). (Lammers et al., 2015) reported matured CAEP responses in late implanted adults soon after the implantation. Similarly, (Gordon et al., 2008) reported matured CAEP responses in late implanted children and having poor speech perception. Studies have used the N1 component to measure the stimulability of the auditory cortex and to measure the auditory threshold. Evidence from these studies supports the stimulability of the primary auditory cortex even in long deprived congenitally deaf (Lammers et al., 2015; Land et al., 2016). The presence of CAEP responses, especially the N1 component is indicative of the stimulability of the auditory cortex, that is, the innate auditory pathway is still intact for stimulating the auditory cortex to an extent even after long period of auditory deprivation (Andrej Kral et al., 2006; Lammers et al., 2015). This auditory cortical activation was evident in the source analysis, where the activity was centred in the bilateral temporal regions (Fig. 5). This source of activity in the temporal regions confirms that the responses were auditory and the auditory cortex receives the stimulation.

(Gordon et al., 2008; Lammers et al., 2015) reported of an early latency and increased amplitude for the N1 component in long auditory deprived individuals. In the current study, compared to the normal hearing group, the latency was prolonged, and the amplitudes were higher in the congenitally deaf group. This latency difference might be due to the variation in the mode of stimulation used in the current study (Hearing aid versus direct stimulation of the auditory nerve through cochlear implant). When we evoke a CAEP response using a hearing aid, the latency of the response is affected by the compression and other characteristics of the hearing aid. This might have caused a slight prolongation in the CAEP components (Billings et al., 2007, 2011).

The amplitude of the N1 component was observed to be higher in the deaf group. This might have been caused by the activation of a large number of unspecified and unspecialized neurons than normal hearing individuals. Since the auditory system is deprived of stimulation for a long period of time the auditory cortex loses the tonotopicity and cortical decoupling takes place (Kral et al., 2000, 2002b, 2005; Kral and Eggermont, 2007). This leads to the auditory cortex to have reduced corticocortical connections and less top down inhibition (Klinke et al., 1999; Kral et al., 2000, 2002b). Hence, the abnormal synaptic connections fires in a larger scale

Fig. 2. The grand average CAEP response from normal hearing and congenitally deaf groups (A). The grand average CAEP response from normal hearing and congenitally deaf groups at Fz, Cz, Oz, T7 and T8 electrode sites (B).

Table 1
The mean and standard deviation (SD) of CAEP N1 latency at Cz, Fz, T7, T8 and Oz electrode sites in the normal hearing and the congenitally deaf groups.

N1 Latency (ms)		
	Normal Hearing Group	Congenitally Deaf Group
Electrode	Mean ± SD	Mean ± SD
Cz	109.18 ± 6.35	113.70 ± 9.48
Fz	112.51 ± 6.45	114.88 ± 10.01
T7	108.62 ± 6.52	115.03 ± 10.77
T8	110.62 ± 8.49	114.29 ± 10.13
Oz	108.44 ± 6.89	110.55 ± 6.89

Table 2
The mean and standard deviation of CAEP P2 latency at Cz, Fz, T7, T8 and Oz electrode sites in the normal hearing and the congenitally deaf group.

P2 Latency (ms)		
	Normal Hearing Group	Congenitally Deaf Group
Electrode	Mean ± SD	Mean ± SD
Cz	175.07 ± 10.34	178.14 ± 19.68
Fz	177.55 ± 6.66	186.66 ± 20.80
T7	174.14 ± 13.63	195.55 ± 23.84
T8	171.70 ± 7.51	196.51 ± 21.59
Oz	187.77 ± 14.71	198.44 ± 15.77

Table 3
The mean and standard deviation of CAEP N1 amplitude at Cz, Fz, T7, T8, and Oz electrode sites in the normal hearing and the congenitally deaf groups.

N1 Amplitude (μV)		
	Normal Hearing Group	Congenitally Deaf Group
Electrode	Mean ± SD	Mean ± SD
Cz	−0.75 ± 0.59	−1.21 ± 0.93
Fz	−0.66 ± 0.47	−0.86 ± 0.75
T7	0.60 ± 0.74	0.088 ± 0.78
T8	0.45 ± 0.42	0.31 ± 0.49
Oz	0.80 ± 0.57	0.82 ± 0.57

Table 4
The mean and standard deviation of CAEP P2 amplitude at Cz, Fz, T7, T8 and Oz electrode sites in the normal hearing and the congenitally deaf groups.

P2 Amplitude (μV)		
	Normal Hearing Group	Congenitally Deaf Group
Electrode	Mean ± SD	Mean ± SD
Cz	0.92 ± 0.71	0.80 ± 1.16
Fz	0.72 ± 0.58	0.19 ± 1.00
T7	−0.52 ± 0.53	−0.28 ± 0.85
T8	−0.73 ± 0.34	−0.21 ± 0.64
Oz	−0.19 ± 0.57	−0.23 ± 0.61

might have led to higher amplitude in deaf individuals.

Interestingly, the P2 component of the CAEP response showed a bifid and broad P2 component in the congenitally deaf group (in 22 participants). Also, the latency were prolonged and amplitude (P2b) were slightly higher than the normal hearing group. These results are also in line with the previously reported findings in adults with post lingual sensory neural hearing loss (Bertoli et al., 2011; Campbell and Sharma, 2013). (Campbell and Sharma, 2013) reported that adults with post lingual mild to moderate sensory neural hearing loss showed an increased P2 amplitude and prolongation in latency. Similarly, (Bertoli et al., 2011) reported comparable findings in hearing aid users with mild to moderate hearing loss. This increased amplitude and the prolongation of the P2 component is observed to be due to the effortful listening, inefficient cortical processing, reduced central inhibition, impaired higher order auditory processing, and neural firing of a large number of unspecialized neurons (Kral et al., 2000, 2002b). Even though these results were reported in acquired hearing loss, these findings can be applied to the current study results. Since there was no adequate auditory stimulation or no stimulation from birth it's our presumption that they too might show similar central characteristics.

In the source analysis, bilateral temporal activation was found in the congenitally deaf group at 112 ms and 204 ms, which reflects the N1 and P2 activity. The presence of these activities within the auditory cortex clearly indicated the stimulability of auditory cortex and that the tonotopicity is still preserved in the long-deprived cortex. Land et al. (2016) reported that even though there was a cross-modal reorganization, the higher auditory cortical areas were able to be stimulated even after a long deprivation in congenitally

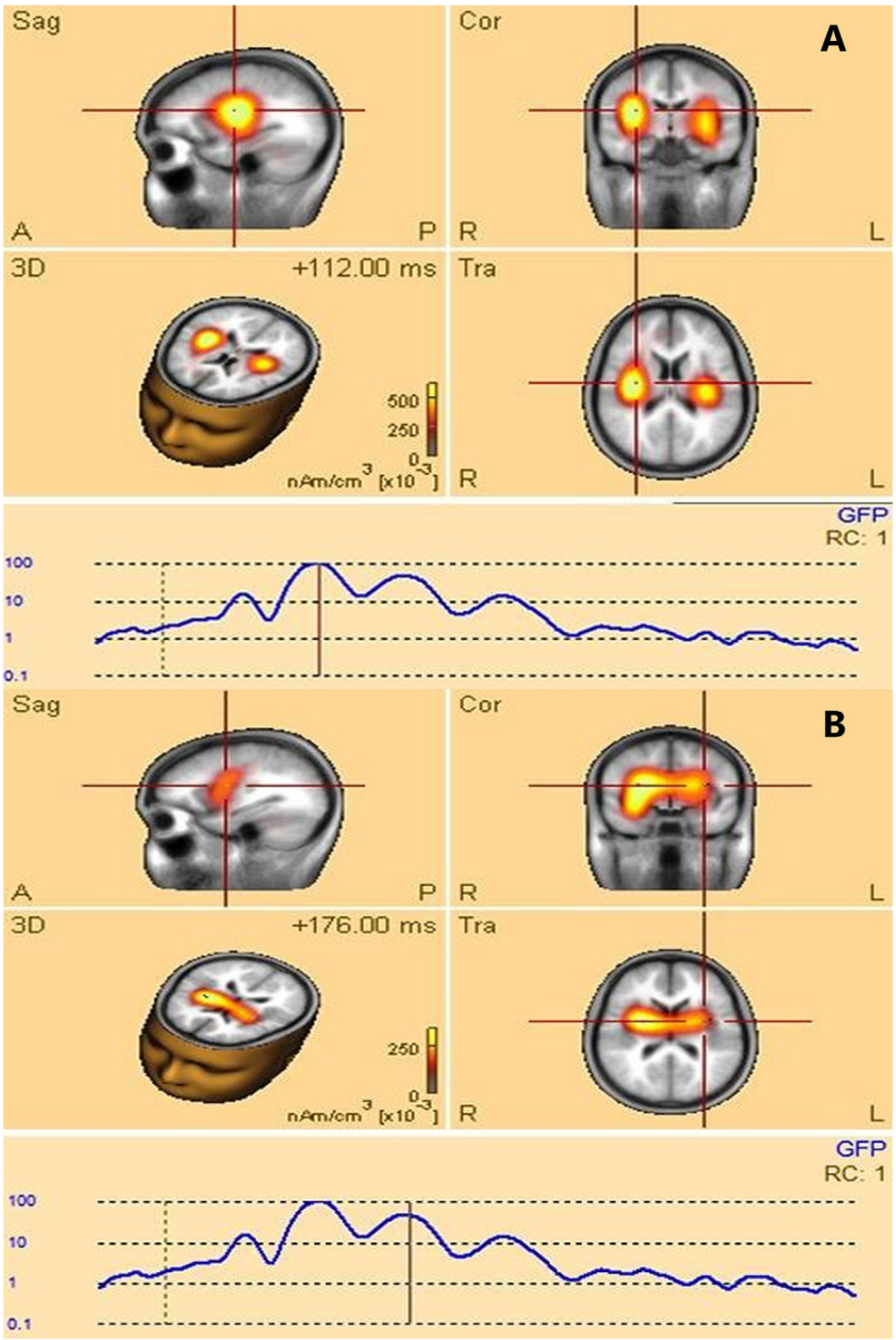


Fig. 3. The sources at 112 ms in normal hearing participants for grand averaged CAEP response (A). The sources at 176 ms in normal hearing participants for grand averaged CAEP response (B).

deaf cats. Another study by Striem-Amit et al. (2016), investigated the functional connectivity and tonotopicity in congenitally deaf adults using fMRI. It revealed that the tonotopicity and functional connectivity patterns were still preserved in the auditory cortex for high and low frequency regions even after a long period of auditory deprivation.

Researchers have (Barone et al., 2013; A Kral, 2013) reported that the congenitally deaf cat showed auditory responses in the DZ of auditory cortex even after long period of auditory deprivation. (Lomber et al., 2010) reported that the congenitally deaf cats showed cross-modal reorganization in the auditory cortex, however, the functional properties were preserved. These findings support that even after a prolonged period of deprivation, it is possible to evoke a CAEP response from congenitally deaf adults for

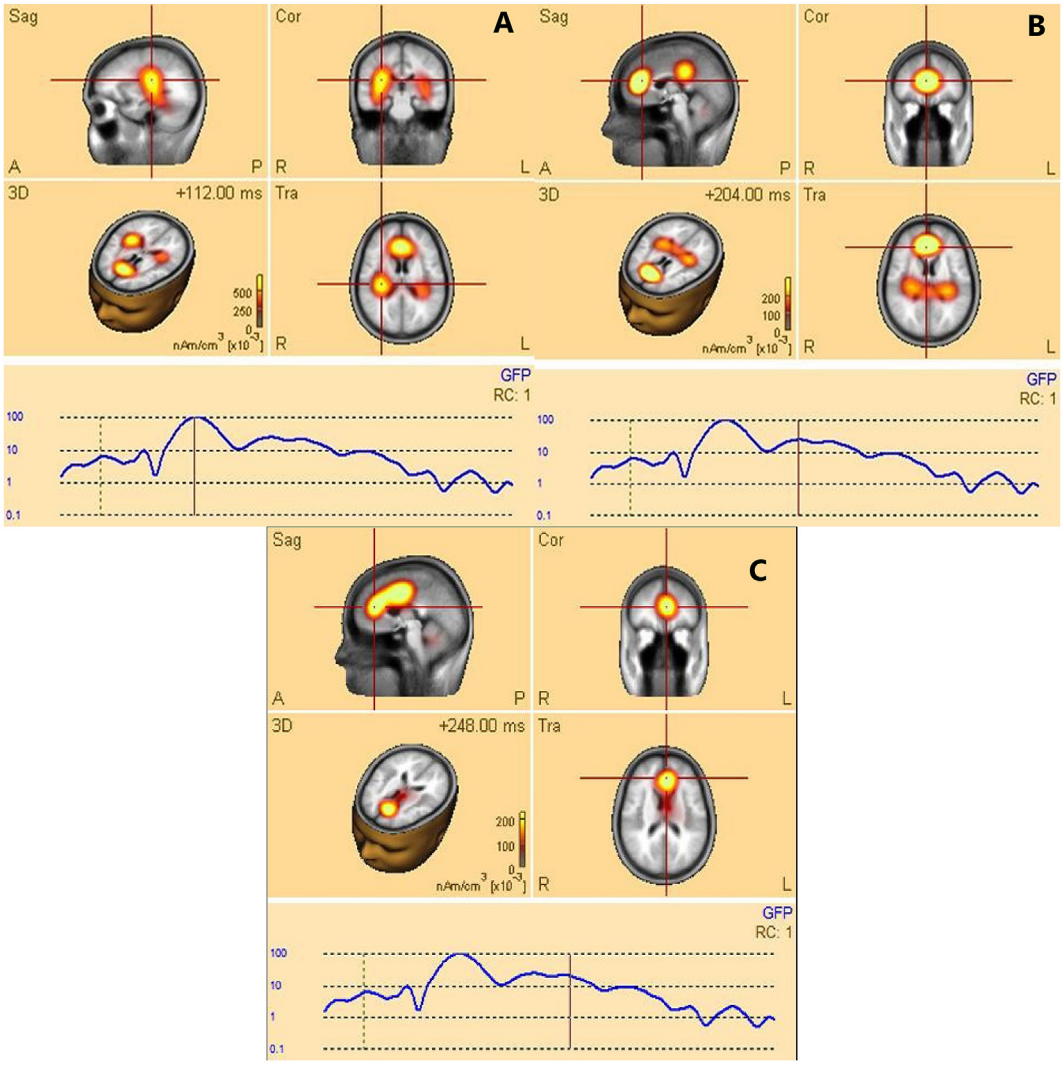


Fig. 4. The sources at 112 ms in congenitally deaf group for grand averaged CAEP response (A). The sources at 204 ms in congenitally deaf group for grand averaged CAEP response (B). The sources at 248 ms in congenitally deaf group for grand averaged CAEP response (C).

simple acoustic stimuli like pure tones which may not require a complex processing in the higher centres. Also, the CAEP responses indicate that auditory cortex preserves its auditory characteristics to an extent, however, there might be a possibility of cross-modal reorganization expected.

In the source analysis results, it was observed that the congenitally long deprived individuals showed a frontal/prefrontal activity for CAEP. These activations were not observed in the normal hearing group. This frontal activation may be an indication of cortical reorganization or an extra cognitive effort given to the stimulus to understand the upcoming sensory stimuli. Campbell and Sharma (2013) reported that there was a frontal activity in mild to moderate sensorineural hearing loss individuals for a passive auditory listening task. The authors reported that the frontal activations are a result of the compensatory effect of hearing loss (re-allocation of the auditory cortical region for auditory stimuli) and these activations are related to the increased listening effort.

The fMRI studies (Davis and Johnsrude, 2003; Okada et al., 2010; Peelle et al., 2010) on older adults have revealed that the frontal regions are activated in response to a complex or in an effortful listening task. In the congenitally deaf group, an effortful listening was morphologically reflected in the CAEP responses as well by the presence of a bifid P2. The first P2 peak (P2a) at 204 msec showed an activity in the temporal and frontal regions and the second peak (P2b) showed an activity only in the frontal regions. Hence, the current study also hypothesizes that the congenitally deaf individuals show a compensatory effect due to hearing loss. The upcoming sensory stimulus is processed with an extra effort for auditory stimuli than normal hearing individuals.

5. Conclusion

The current study findings reveals the possibility of evoking a matured adult like CAEPs among non habilitated congenitally deaf adolescents and adults especially after the critical period with the first time stimulation. The CAEP parameters were well within the normative. This shows that the auditory cortex is still preserved with some innate auditory characteristics by receiving innervations from other highly used sensory areas. Hence, the CAEP responses may not reflect the true benefit from the rehabilitation after the critical age.

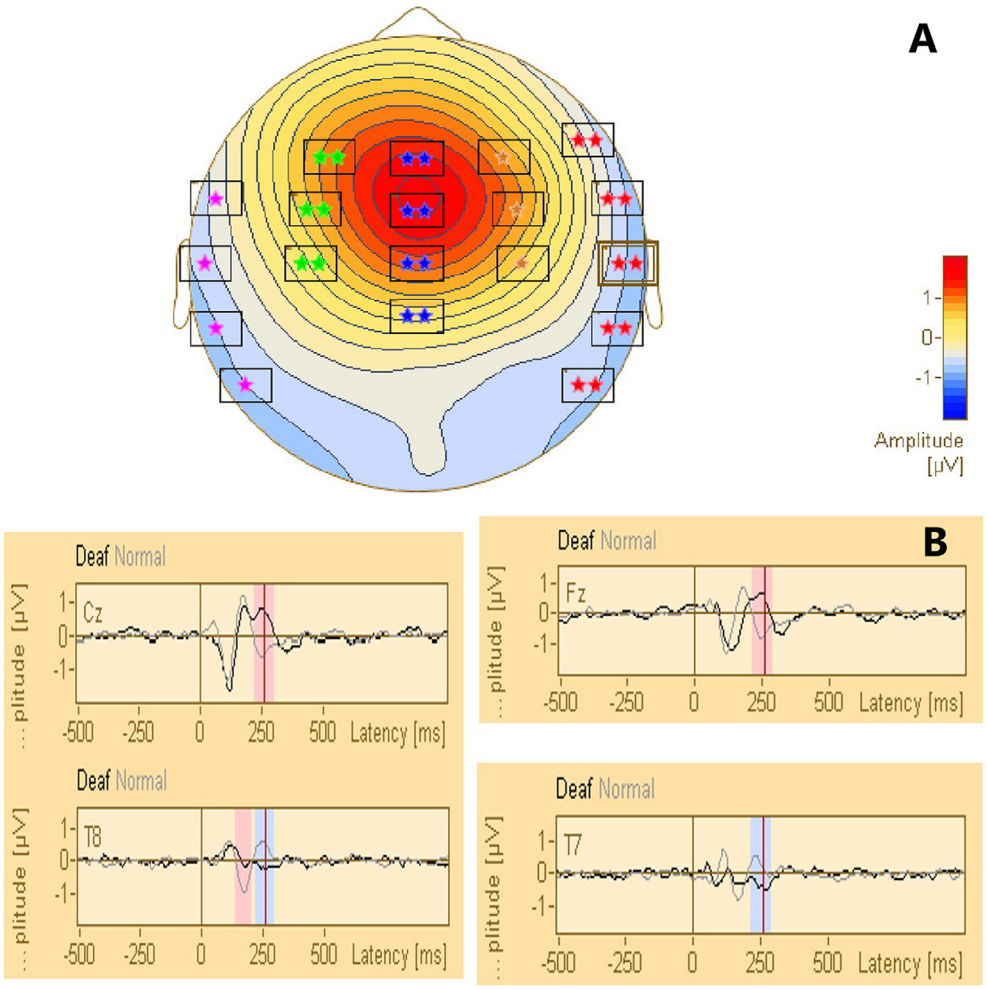


Fig. 5. The statistically significant clusters between congenitally deaf and normal hearing individuals for cluster permutation statistics (A). The time range with significant difference for CAEP between the congenitally deaf and the normal hearing group (B).

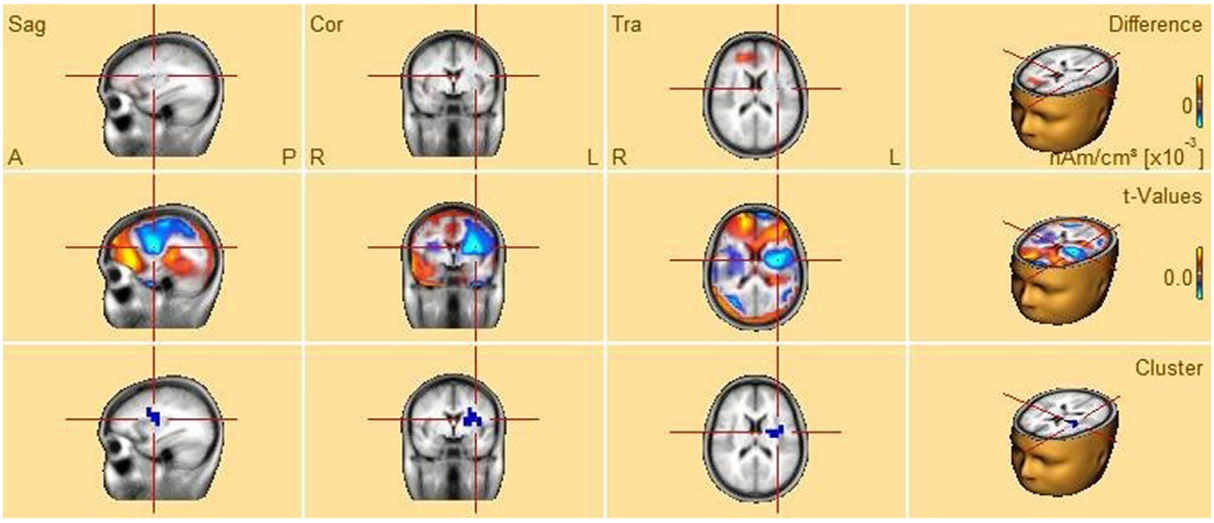


Fig. 6. The CAEPs image cluster permutation statistics between the congenitally deaf and the normal hearing group.

Declaration of competing interest

No potential conflict of interest relevant to this article was reported by the authors.

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