Cortical Evoked Potentials in Children using Speech and Non-speech Stimuli

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Abstract

The present study was taken up with an objective to investigate the effect of age on latency, absolute amplitude and peak to peak amplitude of CAEPS components elicited by syllable /da/ and 500Hz tone burst stimuli. A total of 30 children with normal hearing sensitivity were taken. The age of the participants ranged from 2 to 15 years and they were divided into 3 groups (2 to 4.11, 5 to 9.11 and 10 to 15 years of age). CAEPS was recorded for both syllable /da/ and 500Hz tone burst at 80dBnHL. Latency of P1, N1, P2 and amplitude of P1-N1 and N1-p2 complex were noted. The data was compared between stimulus and across groups. The Peak latency of P1, N1 and P2 components declined with age. Peak amplitudes evoked by speech had greater amplitude than tone and also peak amplitude in younger children were greater than older group which was significant in this study. Thus it can be concluded that to evoke CAEPs in children, speech would be a better stimulus.

Key words: *CAEP, maturation, latency, amplitude.*

The slow, 'obligatory' cortical P1-N1-P2 evoked potentials are bioelectrical time locked responses that occur within 300 ms after the stimulus onset in adults. It is primarily determined by the physical properties of the stimulus and it invariably occurs when sound is detected by the subject (Hyde, 1997; Stapells, 2002). There are studies which have showed considerable changes in AEP morphology and latency along childhood and adolescence (Bruneau, Roux, Guerin, Barthelemy, & Lelord, 1997; Sharma et al., 1997).This existing evidence suggests that maturation of the AEPs and, thus, their underlying generators may have distinct maturational time courses (Kraus, McGee, Carrell, Sharma, Micco, & Nicol 1993).

It is more difficult to investigate auditory processing using behavioral measures (Pang & Taylor, 2000). So by recording age-related changes in the neurophysiological responses evoked by auditory stimulation, one can assess the maturation of the thalamo-cortical portions of the central auditory system (Vaughan & Arezzo, 1988).

The behavioral and perceptual correlates of infant and child ERPs are yet to be established. In children, especially in young children, behavioral data and ERP correlates are scarce since behavioral data are difficult to collect or unreliable. Cortical auditory evoked potentials (CAEPs) have been recorded using a wide range of stimuli including tones, clicks and speech stimuli. There are some evidence that CAEPs in infants evoked by different phonemes differ in latency and morphology (Kurtzberg, 1989).

CAEPs evoked by speech provide information about the biological processes underlying speech processing. The recording of cortical auditory evoked potentials (AEPs) to human speech sounds in infants have value as an index not only of the maturational state, but of the functional integrity of those regions of the cortex which process acoustically complex stimuli that are critical for the development of normal speech and language. Children's long-latency obligatory CAEPs are dominated by the P1 and N1 peaks, whereas those of adults are dominated by the P1–N1–P2 complex (Vaughan, 1975; Vaughan & Kurtzberg, 1989).

Wunderlich and Cone-Wesson (2006) reported that CAEP component latencies were relatively stable from birth to 6 years, but adults demonstrated significantly shorter latencies compared to infants and children. Words evoked significantly larger CAEPs in newborns compared to responses evoked by tones. Natural vowel sound generated CAEPS components (N1 and later waves) that are detected with consistently larger amplitude from the left hemisphere, whereas tonal stimuli produced symmetrical brain activity (Szymanski, Rowley, & Roberts, 1999).

There is dearth of information regarding the CAEPS recording using non-speech and speech stimuli in younger population. Most of the studies have either taken non-speech or speech stimuli to record CAEPS. Literature says that brief stimuli like clicks do not produce well defined waveform for the long latency auditory potentials. Hence tone burst and speech stimulus is used in this study for comparison.

Studies show that amplitude of N1and P2 complex is larger for speech evoked CAEP than for

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single frequency tonal stimuli, but latency value for N1 and P2 are usually earlier for tonal than speech stimuli (Cepohiene, Shestakova, Balan, Alku, Yiaguchi, & Näätänen 2001, Tiitinen, Sivonen, Alku, Virtanen, & Näätänen, 1999). So in order to find out which is the suitable stimuli to elicit response both tonal and speech signal are taken for study.

A comprehensive description of age-related AEP changes in neurologically intact and normal-hearing children will provide a useful reference for assessing suspected neuromaturational deficits or central auditory processing disorders in children. These data may also be useful in evaluating children with hearing disorders (e.g. unilateral deafness) or profoundly deaf children fitted with either hearing aids or cochlear implants (Ponton & Don, 1995, Ponton 1996a; 1996b).

The primary aim of the study was to investigate the effect of age on latency, absolute amplitude and peak to peak amplitude of CAEPS waves elicited by syllable /da/ and 500Hz tone burst stimuli. the secondary aim was to study whether there are any differences in CAEPS waves elicited by non-speech and speech stimulus, and to find the most effective stimuli among syllable /da/ and 500 Hz tone burst to elicit CAEPS response in children.

Method

A total of 30 children were taken. The age of the participants ranged from 2 to 15 years and they were divided into 3 groups (2 to 4.11, 5 to 9.11 and 10 to 15 years of age). Each group had 10 subjects. This groups were made based on the developmental changes observed by Sharma et al. 1997, Oades, Dittman-Balcar, and Zerbin 1997; Ponton, Eggermont, Kwong, and Don (2000). All the subjects participated in the study had normal hearing sensitivity This was assessed either using Visual reinforcement audiometry (VRA) or Auditory brain stem response (ABR) in young children and Pure tone audiometry in children who could give voluntary response. They had 100 percent speech identification scores (picture identification task for younger children and open set task was used for the older children). All of them had 'A' type tympanogram with presence of acoustic reflexs. TEOAEs were present in all the subjects. No relevant otological or neurological history or symptoms were present or expressed.

Test stimuli: 500Hz tone burst of 60 ms (20 ms rise and fall time and 20 ms plateau) and the syllable /da/. was used to record CAEPS. The syllable /da/ was spoken by a male speaker and digitally recorded using adobe audition (version 3.0) with the sampling frequency of 44,000Hz and 16 bit resolution. The naturally recorded /da/ syllable was then edited. The voice onset time, burst portion and a little portion of the vowel were retained to make the syllable duration approximately 150ms.

Procedure

A calibrated two channel diagnostic audiometer (OB-922) was used for visual reinforcement audiometry, pure tone and speech audiometry. The behavioral threshold at octaves frequencies from 250 Hz to 8 kHz for air conduction and 250Hz to 4 kHz for bone conduction were obtained. The thresholds were traced using modified Hughson and Westlake method (Carhart & Jerger, 1959).

Visual reinforcement audiometry was carried out to younger children who were not cooperative for pure tone audiometry. For elderly children speech identification scores (SIS) and Speech recognition threshold (SRT) were obtained using speech stimuli developed by Vandana (1998) and in case of younger group SIS was obtained using picture identification list in Kannada developed by Vandana (1998).

A calibrated Grason Stadler (GSI) Tympstar was used to carry out the tympanometry and reflexometry. It was carried out using 226Hz probe tone. Reflex eliciting tone of 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz were presented ipsilaterally and contralaterally to find out the presence or absence of acoustic reflexes. A significant change of admittance value of 0.03ml was considered as presence of reflex.

ILO 6 was used to record TEAOEs. It was measured using non-linear click trains of 260 sweeps. The overall TEOAEs amplitude of 6 dB SPL above the noise floor with the reproducibility >80% was considered as presence of TEOAEs.

An evoked potential system (Biologic navigator pro version 7.0) was used to record ABR and CAEPS. ABR was recorded for those children where pure tone threshold could not be obtained. It was made sure that impedance at each electrode was within 5 k Ω and inter electrode impedance was within 2 k Ω. Impedance for each electrode was also checked during testing, to make sure that patient movement did not dislodge the electrodes. Subjects with presence of wave V at 40 dB HL was considered as having normal hearing.

Table 1. Protocol used to record CAEPS

Stimulus parameter		Acquisition parameters	
Stimulus	500 Hz tone burst and syllable /da/	Mode of stimulation	Monoaural
Polarity	Alternate		
Number of sweeps	300	Filter setting	1 to 30Hz
Stimulus rate	1.1	Transducer	ER-3AInsert ear phone
Intensity	80 dB nHL	Analysis window	500 ms
		Notch filter	off
		Amplification	50,000

Recording of Auditory long latency response (CAEPS)

Older children were instructed to sit comfortably on a reclining chair and relax during the testing. They were also instructed to stay awake during the testing. They were asked to ignore the stimulus and restrict the movement of head, neck and eye during testing. For younger children CAEPS was recorded when they are quite and /or sleeping. CAEPS were recorded twice for the reproducibility for both speech and non-speech stimuli. The protocol used to record CAEPS is given in Table 1.

To arrive at the goal, the peak latency of P1, N1, P2, absolute amplitude of P1, N1, P2 waves and peak to peak amplitude of P1-N1, and N1-P2 component elicited by syllable /da/ and 500Hz tone burst were noted and compared.

Results

Waveform morphology: The overall results showed that younger children had longer latency and higher amplitude than the older group of children. As the age increased, the latency reduced and amplitude decreased.

CAEPS recorded from group 1: Nine subjects showed the presence of P1 and N1 components, where as four subjects P2 component for both syllable /da/ and 500 Hz tone had burst. CAEPS recorded for syllable /da/ and 500 Hz tone burst from a subject in group 1 is show in Figure 1.

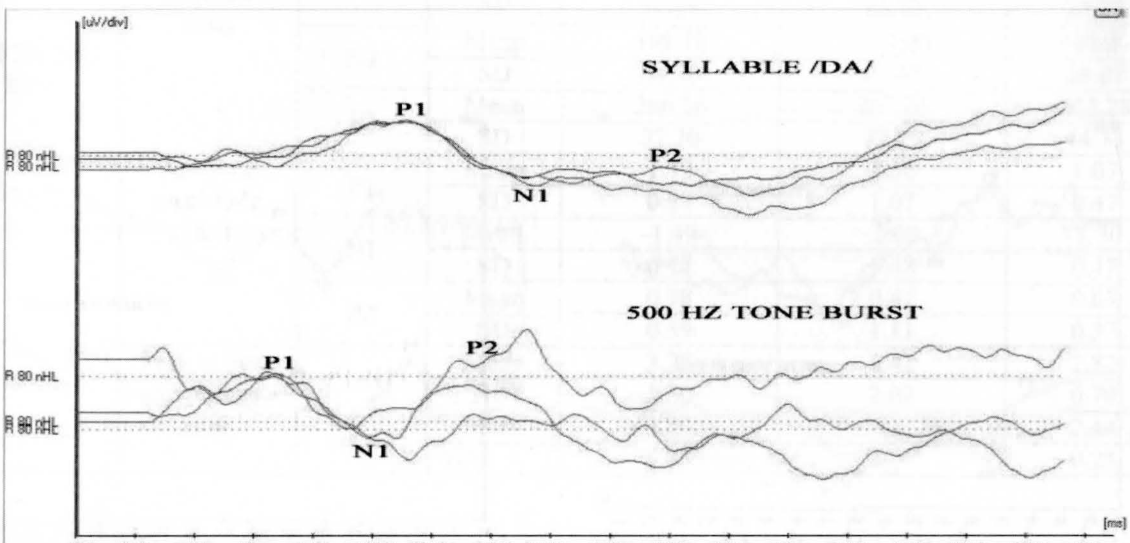

Figure1. CAEPS waves recorded using syllable /da/ and 500 Hz tone burst in a subject from group 1.

CAEPS recorded from group 2: A total of ten subjects participated in this group. CAEPs could be recorded in 8 subjects for both syllable /da/ and 500 Hz tone burst. All the eight subjects showed the presence of P1, N1, where as four subjects showed the presence of P2 component. CAEPS recorded for syllable /da/ and 500 Hz in a subject in group 2 is show in Figure 2.

CAEPS recorded from group 3: A total of ten subjects participated in this group. All the subjects showed the presence of P1 and N1 and P2 components for both syllable /da/ and 500 Hz tone burst. They all showed the shorter latency and reduced amplitude compare to the younger groups. CAEPS recorded for syllable /da/ and 500 Hz tone burst in a subject from group 3 is show in Figure 3.

Age related changes of ALLR elicited by syllable /da/ and 500Hz tone burst

P1 component: Table 3 shows that as the age increased, the P1 latency decreased and its absolute amplitude reduced for both the stimuli. Group 1 had longer latency and larger amplitude compared to group 2 and group 3 for both the stimuli. It can also be seen that P1 latency for 500 Hz tone burst was shorter and amplitude was reduced compared to syllable /da/.

ANOVA results indicated a significant group interaction for P1 latency for syllable /da/ [$F(2, 51) = 6.496$, $P<0.05$] and 500Hz tone burst [$F(2, 50) = 6.175$, $P<0.05$]. Similar results were also obtained for P1 amplitude for both syllable /da/ [$F(2, 51) = 5.129$, $P<0.05$] and 500 Hz tone burst [$F(2, 50) = 3.541$, $P<0.05$].

Duncan's post Hoc test showed that both P1 latency and amplitude was significantly different across groups at 0.05 significance level for both the stimulus. The details of test results are shown in the table 2.

Group 1 showed longer latency and larger amplitude compare to other two groups. Gropu 2 has shorter latency and amplitude was less compare to group 1 but latency was longer and amplitude was more compare to group 3.

Effect of stimulus on P1 latency and amplitude

Non-parametric, Wilcoxon Signed Ranks Test was administrated to see the effect of syllable /da/ and 500Hz tone burst on P1 latency and amplitude within the group.

The result is shown in the Table 4. From the Table 4, it can be seen that there was a significant difference between syllable /da/ and 500 Hz tone burst evoked P1 latency for group 2 and group 3. Where as, there was no significant difference noticed for group 1.

Also there was no significant difference between the amplitude of P1 elicited by syllable /da/ and 500 Hz tone burst for all the groups.

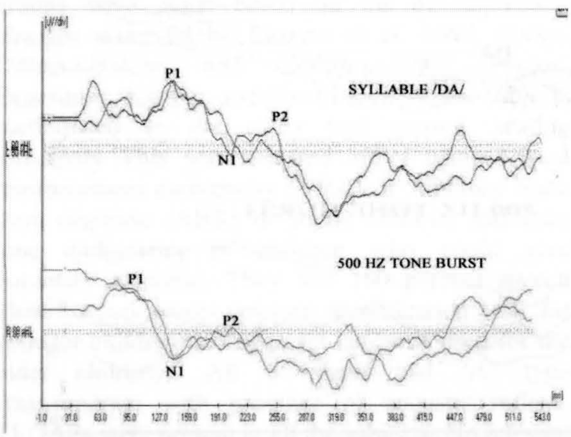

Figure 2. CAEPS waves recorded using syllable /da/ and 500 Hz tone burst in a subject from group 2

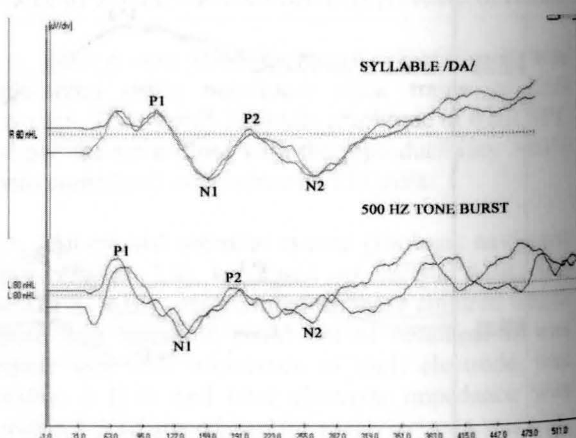

Figure 3. CAEPS waves recorded using syllable /da/ and 500 Hz tone burst in a subject from group 3.

Table 2. Duncan's post Hoc test results of P1 amplitude and latency for both the stimuli

Groups	P1 amplitude				P1 latency			
	Syllable /da/		500Hz tone burst		Syllable /da/		500Hz tone burst	
	1	2	1	2	1	2	1	2
10 to 15 years	1.0585		1.0755		115.14		101.34	
5 to 9.11 years		1.7769	1.3631	1.3631	121.76		111.06	
2 to 4.11 years		2.1133		1.7971		135.29		128.78

Table 3. Mean and standard deviation (SD) value of latency and amplitude of CAEP components elicited by /da/ and 500Hz tone burst across the groups

				Group 1	Group 2	Group 3
Syllable /da/	Latency (ms)	P1	Mean	135.29	121.76	115.14
			SD	22.69	15.43	12.88
		N1	Mean	229.12	191.10	166.89
			SD	43.95	52.80	25.76
		P2	Mean	281.00	257.54	244.92
			SD	33.39	63.64	31.13
	Absolute amplitude (µv)	P1	Mean	2.11	1.77	1.05
			SD	0.85	1.26	0.99
		N1	Mean	-2.35	-1.83	-1.45
			SD	1.55	1.67	0.84
		P2	Mean	0.31	0.61	0.75
			SD	1.08	1.16	0.50
	Peak to peak amplitude (µv)	P1-N1	Mean	4.44	3.54	2.47
			SD	2.09	2.16	0.67
		N1-P2	Mean	1.53	2.26	2.16
			SD	1.08	0.19	0.36
500 Hz tone burst	Latency (ms)	P1	Mean	128.78	111.06	101.34
			SD	31.89	22.89	14.93
		N1	Mean	198.78	183.00	149.54
			SD	34.70	53.43	24.07
		P2	Mean	266.36	267.25	212.28
			SD	37.20	72.79	44.33
	Absolute amplitude (µv)	P1	Mean	1.79	1.36	1.07
			SD	0.87	1.07	0.47
		N1	Mean	-1.49	-2.00	-1.70
			SD	0.77	1.48	0.47
		P2	Mean	0.78	0.42	0.63
			SD	0.59	1.11	0.37
	Peak to peak amplitude (µv)	P1-N1	Mean	3.28	3.42	2.82
			SD	1.30	2.02	0.79
		N1-P2	Mean	1.70	-	2.44
			SD	0.33	-	0.25

N1 component: From the Table 3, it can be seen that as the age increased, N1 latency decreased and its absolute amplitude reduced for syllable /da/ where as for tone burst no such specific pattern could be noticed. Group 1 had longer latency compare to group 2 and group 3 for both the stimuli where as amplitude of N1 elicited by /da/ was maximum for group 1. Latency for 500 Hz tone burst was shorter compare to syllable /da/. Where as, for tone burst, group 2 and group 3 showed larger amplitude than group 1.

Table 4. z-value and the significant level for CAEP latencies and amplitudes between the stimulus /da/ and 500 Hz tone burst within the group

		Z-value		
		Group 1	Group 2	Group 3
P1 wave	Latency	1.58	**2.01***	**3.28****
	Amplitude	1.82	1.64	0.19
N1 wave	Latency	**3.48****	1.08	**3.36****
	Amplitude	**2.77****	0.62	1.64
P2 wave	Latency	**2.20***	0.56	**3.11****
	Amplitude	0.84	0.21	0.00
P1-N1	Amplitude	**2.15***	0.20	1.49
N1-P2	Amplitude	0.53	-	**2.21***

*p<0.05; **p<0.01

ANOVA results indicated a significant group interaction for N1 latency [F (2, 51) = 10.810, P<0.05] for syllable /da/. A significant interaction was also observed across the group for N1 latency elicited by 500Hz tone burst [F(2,50) = 8.068, P<0.05]. Where as no significant difference for N1 amplitude elicited by syllable /da/ [F (2, 51) = 2.072, P>0.05] and 500 Hz tone burst [F (2, 50) = 1.156, P>0.05] was noticed.

Duncan's post Hoc test showed that N1 latency was significantly different between the groups at 0.05 significance level for both the stimulus. The details of the test results is shown in the Table 5.

Table 5. Duncan's post Hoc test result of N1 latency (ms) for both the stimuli.

Groups	Syllable /da/		500Hz tone burst	
	1	2	1	2
10 to 15 years	166.89	-	149.54	
5 to 9.11 years	191.10	-	-	183.00
2 to 4.11 years	-	229.12	-	198.78

Wilcoxon signed ranks test showed a significant difference between syllable /da/ and 500 Hz tone burst evoked N1latency for group 1 and group3 (table 4). No significant difference was noticed for both latency and amplitude in group 2 for both the stimulus. It can also be noticed that there was significant difference between the amplitude for N1 elicited by syllable /da/ and 500 Hz tone burst was seen only in group 1 and not in group 2 and 3.

P2 component: P2 latency decreased with age for both syllable /da/ and 500 Hz tone burst. Group 1 showed shorter amplitude for syllable /da/ compared to other two groups where as for tone burst, group 1 had larger amplitude compared to other two groups. Latency for 500 Hz tone burst was shorter and amplitude reduced compared to syllable /da/ except for group 2.

One way ANOVA was administered to see the effect of age on P2 latency and amplitude for each stimulus. ANOVA results indicated that there was no significant group interaction for P2 latency [F (2, 31) = 1.971, P>0.05] for syllable /da/ but a significant interaction was observed for P2 latency elicited by 500Hz tone burst [F (2, 29) = 4.239, P<0.05]. There was no significant group interaction observed for P2 amplitude for both the stimuli [F (2, 31) = 0.712, P>0.05, F (2, 30) = 0.648, P>0.05]

Duncan's post Hoc test showed that P2 latency obtained in group 3 was significantly different from other groups and there was no significant difference between group1 and group 2 P2 latency. The details of the test results are shown in Table 6.

Table 6. Duncan's post Hoc test results of P2 latency for 500Hz tone bursy stimulus

Groups	500Hz tone burst	
	1	2
10 to 15 years	212.2885	
5 to 9.11 years	-	266.3670
2 to 4.11 years	-	267.2567

P1-N1 complex amplitude: From the Table 3, it can be seen that as the age increased the P1-N1 complex amplitude reduced. Group 1 has larger P1-N1 complex amplitude than compared to group 2 and 3. It also observed that P1-N1 amplitude elicited by syllable /da/ has larger amplitude than evoked by 500 Hz tone burst.

One way ANOVA results indicated a significant group interaction for P1-N1 complex amplitude [F (2, 51) = 6.128, P<0.05] for syllable /da/ but no significant interaction was observed across the group for P1-N1complex amplitude elicited by 500Hz tone burst [F (2, 50) = 0.907, P>0.05]. The details of the Duncan's post Hoc test results are shown in Table 7.

Table 7. Duncan's post Hoc test results of P1-N1complex amplitude for syllable /da/

Groups	500Hz tone burst	
	1	2
10 to 15 years	2.82	2.82
5 to 9.11 years	-	3.42
2 to 4.11 years	-	3.28

Group 1 and 2, P1-N1 complex amplitude elicited by syllable /da/ differ significantly from group 3 amplitude.

Wilcoxon Signed Ranks Test shown a significant difference between syllable /da/ and 500 Hz tone burst evoked P1-N1 complex amplitude for group 1, where as there was no significant difference between stimuli for group 2 and 3 (Table 4).

N1-P2 complex amplitude: It can be seen in the table 3 N1-P2 complex amplitude had a lot of variability evoked by both syllable /da/ and 500 Hz tone burst. Only a few subjects in each group had recordable N1-P2 complex amplitude. In group 2, P2 could not be obtained for 500 Hz tone burst but present for stimuli /da/. Due to this variability in the data non-parametric test (Kruskal-Wallis test) was administered.

The result reveals that there was no significant difference across the groups (chi-square=5.691, degrees of freedom=2, P>0.05) for both the stimuli.

Wilcoxon Signed Ranks Test showed a significant difference between syllable /da/ and 500 Hz tone burst evoked N1-P2 complex amplitude for group 3. Whereas, no significant difference between stimuli for group 1 was noticed. In group 2, P2 could be obtained from a very few subjects for 500 Hz tone burst, hence not compared.

Discussion

The results of the present study showed that there were systematic age-related changes in magnitude and timing for P1, N1 and P2 components of CAEP, evoked by syllable /da/ and 500 Hz tone burst. It also showed that there was difference in waveform morphology elicited by syllable /da/ and 500 Hz tone burst.

Waveform morphology: The younger children had longer latency and higher amplitude than the older group of children. This is consistent with the results reported by various investigators (Kurtzberg, Hilpert, Kreuzer, & Vaughan, 1984; Sharma & Dorman, 2005). This is typically accounted for by the larger numbers of synapses, thinner skulls and feasibly, immaturity of functional specialization in children than adults (Huttenlocher, 1990).

In the current study, the Peak latency of P1, N1 and P2 component declined with age. This is consistent with the results reported by Bruneau, Roux, Guerin, Barthelemy, and Lelord, 1997, Cunningham et al (2000) Kraus, McGee, Carrell, Sharma, Micco and Nicol (1993).

Peak amplitudes evoked by speech had greater amplitude than tone and also peak amplitude in younger children was greater than older group which was significant in this study. Wunderlich and Cone-Wesson (2006) also observed greater amplitude for younger children than older children and also amplitude obtained for speech stimulus was more than non-speech stimulus.

Age related changes of CAEP component
P1 component

Latency: The result of the present study showed that as age increased the latency of P1 component decreased which is consistent with the results reported by Cunningham, Nicol, Zecker, & Kraus, (2000); Ponton, Eggermont, Kwong, and Don (2000); & Wunderlich et al., (2006). They observed younger group had longer latency than older group. Sharma et al., (1997) evoked CAEP using syllable /ba/ in 6 to 15 years and found that latency of P1 component reduced as age increased till 20 years of age.

The present study also observed was predominant P1 component in younger group (2 to 4.11 years). Similar findings were also observed in previous studies (Kushnerenko et al., 2002; Pang & Taylor, 2000). They reported that P1 component is most prominent peak found during early childhood.

In the present study we could see that younger group showed presence of large positive (latency) response evoked by syllable /da/ than 500 Hz tone burst which is consistent with the study done by Anu Sharma et al., (1997). However Szymanski, Rowley, & Roberts, 1999, Rowley, and Roberts, (1999) reported that there is no difference of P1 component latency for both speech and non-speech stimulus. Some studies reported that in children syllable /ba/, /ga/ and /da/ elicits shorter latency than non-phonetic sounds (Ceponiene, Alku, Westerfield, Torki & Townsend, 2005) which was in contrary to the findings of the present study.

Moore (2002) reported that neurofilaments with axons radiating into the deeper cortical layers IV, V and VI first appear between 4 – 12 months of age. By 2 years of age, a light plexus of vertical and horizontal axons was apparent in the deeper cortical layers and this plexus becomes progressively denser by 3 to 5 years (axonal density in the layers III to VI increased until about 5 years). This possibly could explain the maturational changes observed in latency of P1 across the groups.

Amplitude: The result of the present study showed that the amplitude of P1 component decreased with age. Ceponiene, Rinne and Näätänen (2002); Ceponiene, Alku, Aro, and Näätänen (2003) and Wunderlich and Cone-Wesson (2006) also observed larger P1amplitude in younger children and decreased P1 amplitude in older children. This decline in the P1 component as age increased is mainly reflects the decline of synaptic density which occurs in the auditory cortex during late childhood, reflects an ongoing process occurring throughout infancy and early childhood (Ponton, Eggermont, Kwong, & Don, 2000).

Present study also showed that speech evoked CAEP evoked larger amplitude than non-speech stimuli. Sharma et al, (1997) & Wunderlich and Cone-Wesson (2006) also reported more amplitude for speech evoked than non-speech CAEP.

However, Shtyrov, et al., (2000) and Szymanski et al, (1999) did not observe any difference of P1 component amplitude for both speech and non-speech stimulus.

N1 component

Latency: The results of the present study showed that as the age increased, the latency of N1 decreased for both syllable /da/ and 500 Hz tone burst. It was also seen that speech evoked N1 component had longer latency than 500 Hz tone burst. Wunderlich and Cone-Wesson (2006) reported that N1 latency decreased as age increased. Another study on CAEP by DeCreÂvoisier, Peronnet, Girod, Challet, and Revol (1975) reported that N1 obtained in younger children had longer latency than older children. These decreases in latency with age is supported by Rojas, Walker, Sheeder, Teale, and Reite (1998) who reported that there is a decrease in refractoriness of N1 component as age increases from at least 6–18 years.

Present study also showed that speech evoked CAEP had longer in latency than non-speech tone which was consistent with the earlier report (Diesch, Eulitz, Hampson, & Ross, 1996; Tiitinen, Sivonen, Alku, Virtanen, & Näätänen, 1999; Woods & Elmasian, 1986). They reported that speech (vowel) evoked responses had longer N1latency than non-speech (tones) stimulus. It was also reported by Boyd, Liasis, Bamiou, Campbell, Sirimanna, and Towell (2003) that N1 latency was longer on syllable than on tone trials.

There are some studies which reported that N1 has several generators in the upper cortical layers including primary and secondary auditory cortex in or near to the supratemporal plane (Ponton, Eggermont, Kwong, & Don, 2000). The axons in the upper cortical layers are sparsely distributed in childhood and it becomes more numerous at ages 2 and 3 than at 1 year. After 5 years of age, maturation of axons begin to appear in cortical layers 2 and 3 and by 12 years of age as their density is equivalent to that of young adults (Moore & Guan, 2001). These changes in the marginal layer of the auditory cortex might have attributed to maturational changes of N1 latency that was observed in this study.

Amplitude: The results of the present study showed that as the age increased, amplitude of N1 decreases for the syllable /da/, where as 500 Hz tone burst showed variability of N1 amplitude across groups. It was also seen that speech evoked N1 component had larger amplitude than 500 Hz tone burst. There is conflicting evidence to age related changes in amplitude of N1 component. Some studies have shown no age-related increase in N1 amplitude after 6 years of age (Ceponiene, Rinne & Näätänen (2002; Johnstone et al., 1996; Kraus, McGee, Carrell, Sharma, Micco, & Nicol, 1993; Sharma et al., 1997 and Tonnquist-Uhle´n, 1996). Others have found that, increased in N1 amplitude as age increased (Bruneau, Roux, Guerin, Barthelemy, & Lelord, 1997; Cunningham, Nicol, Zecker, & Kraus (2000); Ponton, Eggermont, Kwong, & 2000).

Present study also showed that speech evoked N1 amplitude is more than the non-speech evoked N1 amplitude is consistent with the earlier results (Ceponiene et al., 2001 and Tiitinen, Sivonen, Alku, Virtanen, & Näätänen, 1999). They reported that speech evoked N1 component was larger in amplitude than non-speech stimulus. It is reported by Woods and Elmasian (1986), that speech evoked (vowel) N1 amplitude is greater than the tones evoked N1 amplitude. Similar findings were also reported by Wunderlich and Cone-Wesson (2006). Boyd et al., (2003) also reported that speech sounds caused better dipolar organization of neural activity than tones which results speech better than tones.

However, Bruneau, Roux, Guerin, Barthelemy, and Lelord, 1997, (1997), Cunningham, Nicol, Zecker, and Kraus (2000); Tonnquist-Uhle´n et al.,(1995) and Ponton, Eggermont, Kwong, and Don (2000) reported that low tones evoked a larger N1 amplitude than words and also reported that older children showed larger amplitude than younger children. There were a few studies which observed no difference in N1 amplitude elicited by speech and non-speech stimulus (Szymanski, Rowley & Roberts, 1999). Similar findings were also reported by Ceponiene , et al., (2005).

P2 component

Latency: Present study showed decrease in P2 latency as age increased. Latency for 500 Hz tone burst was shorter compare to syllable /da/ but there was no such group interaction for both syllable /da/ and 500 Hz tone burst observed. It is consistent with studies done by Cunningham, Nicol, Zecker and Kraus (2000); Oades, Dittman-Balcar, & Zerbin, 1997); Ponton, Eggermont, Kwong, and Don (2000), Sharma et al., (1997) and Wunderlich et al., (2006). They reported that as the age increases P2 component latency reduced. Oades, Dittman-Balcar, and Zerbin 1997 also reported that the P2 latency decline with age.

In literature there are conflicting findings regarding the development of P2 peak. Studies by, Johnstone et al. (1996), Ponton, Eggermont, Kwong, and Don (2000) and Tonnquist- Uhlen, (1996) reported no age-related changes in P2 peak latency. Some researchers posit that auditory P2 emerges early in infancy (Kurtzberg, Hilpert, Kreuzer, & Vaughan, 1984), while others stated that it does not appear until 5–6 years of age (Ponton, Eggermont, Kwong, & Don, 2000).

Amplitude: The results obtained in the present study showed that amplitude elicited by speech is greater than non-speech which is consistent with earlier study (Tiitinen, Sivonen, Alku, Virtanen, & Näätänen, 1999). They reported that P2 amplitude was larger for speech (vowel) than tones. It was also reported that the peak amplitude of P2 in older group was larger than in younger group which was consistent with the study done by Wunderlich and Cone-Wesson (2006), who reported that amplitude of P2 component increased as age increased.

Several studies have reported a positive P2 amplitude gradient (Johnstone et al., 1996; Kraus, McGee, Carrell, Sharma, Micco, & Nicol, 1993; Oades, Dittman-Balcar, & Zerbin, 1997) while one study found no change in P2 amplitude in children between 5–15 years (Tonnquist Uhle´n, 1996). Another reported a negative gradient of amplitude (Ponton, Eggermont, Kwong, & Don, 2000. Woods and Elmasian (1986) reported that vowels elicited smaller P2 amplitude than simple tones which is in contrary to the results of the present study. Ceponiene, Rinne and Näätänen (2002) reported that younger children had larger P2 amplitude than older children which is also not in agreement with the results of the present study.

P1-N1 and N1-P2 complex amplitude

The P1-N1 complex amplitude decreased as age increased. Younger group had greater amplitude than older group, where as N1-P2 complex amplitude lot of variability between stimulus and also across groups.

This could be due to differential affect of age on P1 and N1 absolute amplitude. Wunderlich and Cone-Wesson (2006) reported a systematic decrease in the magnitude of P1 as age increased. Similar decrease in amplitude of P1 with age in order children was also reported by Ceponiene, Rinne and Näätänen (2002); Cunningham, Nicol, Zecker and Kraus, (2000), Oades, Dittman-Balcar, and Zerbin 1997, Ponton, Eggermont, Kwong, & Don, 2000 and Sharma et al., (1997).

Bruneau, Roux, Guerin, Barthelemy, and Lelord, 1997 observed increased in N1 amplitude with age. A similar finding was also reported by Pang and Taylor (2000) and Wunderlich and Cone-Wesson (2006). They reported a systematic increase in N1 amplitude from newborn to adulthood. Hence, the differential affect of age on P1 and N1absolute amplitude might have resulted in inconsistency of amplitude of P1-N1 complex in the present study.

The present study also observed that N1-P2 peak amplitude increased as age increased which is consistent with the observation made by Wunderlich and Cone-Wesson (2006). They have observed an increased in N1 and P2 amplitude as age increased. This increased in magnitude of N1 and P2 peak amplitude with age might have lead to the increase in N1-P2 complex amplitude with age which is observed in the current study.

Thus, it can be concluded from the results of the current study that the latency of the P1, N1 and P2 tend to decreased with age. Similar changes in latency can be expected for both speech and non-speech stimulus. P1 tend to show decrease in amplitude, whereas N1 and P2 amplitude may not increase or decline with age for both the stimulus. However, speech evoked response had larger amplitude and components were longer latency for all the components than the non-speech evoked CAEP components.

Conclusions

It can be concluded from the study that auditory cortical responses can be used to assess complex maturation of the thalamo-cortical portions of the central auditory system by measuring morphological changes in waveforms. Speech stimulus can be used to elicit CAEPs as it showed greater amplitude than tone burst for P1, N1 and P2 component. This would assist to observe morphological changes and also provides information about the biological processes underlying speech processing which is essential for speech and language development.

Implications

A comprehensive description of age-related AEP changes data obtained form current study would provide norms for normal-hearing children.It could also provide useful reference for assessing suspected neurologically insult and neuro-maturational deficits or central auditory processing disorders in children. It might be useful in evaluating children with hearing disorders or profoundly deaf children fitted with hearing aid or cochlear implants. It is a good electrophysiological tool to assess the benefit from the hearing aids especially in younger children who is not co-operative for behavioral testing. It might be used to monitor changes in the neural processing during/after rehabilitation. CAEPs differences between speech and non-speech are an indication of different underlying neural representations of speech sounds and non-speech stimuli and suggest that the information needed to differentiate the stimuli is available to the listener. It also helps in assessing neural encoding of speech signals.

References

Boyd, S., Liasis, A., Bamiou, D. E., Campbell, P., Sirimanna, T., & Towell, A. (2003). Auditory event-related potentials in the assessment of auditory processing disorders: A pilot study. *Neuropediatrics*, 34, 23–29.

Bruneau, N., Roux ,S., Gue´rin, C., Barthe´le´my, C., & Lelord, G. (1997). Temporal prominence of auditory evoked potentials (N1 wave) in 4–8-year-old children. *Psychophysiology*, 34, 32–38.

Carhart, R., & Jerger, J. F. (1959). Preferred method for clinical determination of pure tone thresholds. The Journal of Speech and Hearing Disorders, 24, 330-345.

Ceponiene, R., L, T., Alku, P., Aro, H., & Näätänen, R. (2003). Event related potential indices of auditory vowel processing in 3-year-old children. Clinical *Neurophysiology*, 114, 652–661.

Ceponiene, R., P. Alku. M., Westerfield. M. Torki, & J. Townsend. (2005). ERPs differentiate syllable and nonphonetic sound processing in children and adults. *Psychophysiology*, 42, 391–406.

Ceponiene, R., Rinne, T., & Näätänen, R. (2002). Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*, 113, 870-882.

Ceponiene, R., Shestakova, A., Balan, P., Alku, P., Yiaguchi, K., & Näätänen, R. (2001). Children's auditory event-related potentials index sound complexity and "speechness". *The International Journal of Neuroscience*, 109, 245-260.

Cunningham, J., Nicol, T., Zecker, S., & Kraus, N. (2000). Speech-evoked neurophysiologic responses in children with learning problems: development and behavioral correlates of perception. *Ear & Hearing*, 21, 554-568.

De Crévoisier, A., Peronnet, F., Girod, J., Challet, E., & Revol, M. (1975). Topography of auditory evoked potentials in the alert child. *Rev Electroencephalogr Neurophysiol Clin*, 5, 303-306.

Diesch, E., Eulitz, C., Hampson, S., & Ross, B. (1996). The neurotopography of vowels as mirrored by evoked magnetic field measurements. *Brain and Language*, 53, 143–168.

Huttenlocher, P. R. (1990). Morphometric study of human cerebral cortex development. Neuropsychologia, 28, 517–527.

Hyde. M. (1997). The N1 response and its applications. *Audiol Neurootol*, 2, 281-307.

Johnstone, S. J., Barry, R. J., Anderson, J. W., & Coyle, S. F., (1996). Age related changes in child and adolescent event-related potential component morphology, amplitude and latency to standard and target stimuli in an auditory oddball task. *International Journal of Psychophysiology*, 24, 223–238.

Kraus, N., McGee, T., Carrell, T., Sharma, A., Micco, A., & Nicol, T. (1993). Speech-evoked cortical potentials in children. *Journal of the American Academy of Audiology*, 4, 238-248.

Kurtzberg, D. (1989). Cortical event-related potentials assessment of auditory system function. *Seminars in Hearing*, 10, 252-261.

Kurtzberg, D., Hilpert, P. L., Kreuzer, J. A., & Vaughan, H. G. (1984). Differential maturation of cortical auditory evoked potentials to speech sounds in normal fullterm and very low-birth weight infants. *Developmental Medicine and Child Neurology*, 26, 466–475.

Kushnerenko, E., Ceponiene, R., Balan, P., Fellman, V., & Näätänen, R. (2002). Maturation of the auditory event-related potentials during the first year of life. *Neuroreport*, 13, 47–51.

Moore, J. K. (2002). Maturation of human auditory cortex: Implication for speech perception. *Annals of Otology, Rhinology and Laryngology*, 111, 7 - 10.

Oades, R. D., Dittman-Balcar, A., & Zerbin, D. (1997). Development and topography of auditory event-related potentials (ERPs): mismatch and processing negativity in individuals 8–22 years of age. *Psychophysiology*, 34, 677–693.

Pang, E. W., & Taylor, M. J. (2000). Tracing the development of the N1 from age 3 to adulthood: an examination of speech and non-speech stimuli. *Clinical Neurophysiology*, 111, 388–397.

Ponton, C, W., & Don, M. (1995). The mismatch negativity in cochlear implant users. *Ear and Hearing*, 16, 131-146.

Ponton, C. W., Don, M., Eggermont, J. J., Waring, M. D., & Masuda, A. (1996b). Maturation of human cortical auditory function: differences between normal hearing and cochlear implant children. *Ear and Hearing*, 17, 430-437.

Ponton, C. W., Don, M., Eggermont, J. J., Waring, M. D., Kwong, B., & Masuda, A. (1996). Auditory system plasticity in children after long periods of complete deafness. *NeuroReport*, 8, 61-65.

Ponton, C. W., Eggermont, J. J., Kwong, B., & Don, M. (2000). Maturation of human central auditory system activity: evidence from multi-channel evoked potentials. *Clinical Neurophysiology*, 111, 220–236.

Rojas, D.C., Walker, J.R., Sheeder, J.L., Teale, P.D., & Reite, M.L. (1998). Developmental changes in the refractoriness of the neuromagnetic M100 in children. *Neuroreport*, 9, 1543–1547.

Sharma, A., Kraus, N., McGee, T. J., & Nicol, T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalography and Clinical Neurophysiology*, 104, 540-545.

Stapells, D. R. (2002). Cortical event-related potentials to auditory stimuli. In J. Katz, R. F. Burkard, & L. Medwetsky (Eds.), *Handbook of clinical audiology*, (5th ed., pp. 378–406), Philadelphia, PA: Lippincott, Williams, and Wilkins.

Szymanski, M. D., Rowley, H. A., & Roberts, T. P. (1999). A hemispherically asymmetricalMEG response to vowels. *NeuroReport*, 10, 2481–2486.

Tiitinen, H., Sivonen, P., Alku, P., Virtanen, J., & Näätänen, R., (1999). Electromagnetic recordings reveal latency differences in speech and tone processing in humans. *Cognitive Brain Research*, 8, 355–363.

Tonnquist-Uhlen, I. (1996). Topography of auditory evoked long-latency potentials in children with severe language impairment: the P2 andN2 components. *Ear and Hearing*, 17, 314–326.

Vandana, S. (1998). *Speech identification test for Kannada speaking children*. Unpublished Independent Project, University of Mysore, Mysore in part fulfilment of Masters Degree in Audiology.

Vaughan, Jr., H. G. (1975). Electrophysiologic analysis of regional cortical maturation. *Biol. Psychiat.*, 10, 513-526.

Vaughan, Jr., H.G. & Kurtzberg, D. (1989). Electrophysiological indices of normal and aberrant cortical maturation. In P. Kellaway & J. L. Noebels (Eds.), *Developmental Neurophysiology*. Baltimore: Johns Hopkins Univ. Press.

Woods, D. L., & Elmasian, R. (1986). The habituation of event-related potentials to speech sounds and tones. *Electroencephalography and Clinical Neurophysiology*, 65, 447–459.

Wunderlich, J. L., & Cone-Wesson, B. K. (2006). Maturation of CAEP in infants and children: A review. *Hearing Research*, 212, 212-223.