# Maturation of Speech Evoked Cortical Auditory Evoked Potential

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## Abstract

*The present study aimed at (1) investigating developmental changes of CAEP in children age between birth to seven years and (2) also to investigate the effect of age on latency of different wave and peak to peak amplitude of speech evoked cortical auditory evoked potentials. 37 children from birth to 7 years of age with normal hearing participated in the study. CAEPS was recorded at varying intensity from 80 dB nHL to 20 dB nHL reducing in 20 dB steps at 1.1 repetitions/second. The result of the study showed systematic changes in latencies of CAEP components with age. The latency of P1, N1, P2 and N2 decreased and amplitude of N1-P2 complex increased systematically with age.*

***Key words**: Cortical auditory evoked potential (CAEP), maturation, development.*

The development of the auditory system follows a time course which is highly species specific. In humans, the ear begins its development very early in the life of embryo, approximately 15 days after fertilization. By birth, structures such as cochlea, ossicles of the middle ear reach adult size and shape. Whereas structures such as auricle, external auditory canal, tympanic membrane, middle ear cavity, eustachian tube, auditory nerve and central auditory nervous system continue to mature (Northern & Downs, 2002).

Development of central auditory system development takes place in two phases. During the first major phase of development, the neurons of the auditory system are generated, migrate to their adult locations, send out axons, undergo dendritic differentiation, and begin to establish synaptic connections. These developmental events lead to the very early establishment of the basic pattern of circuitry that would lead to characteristic of the adult. Second phase of development involves structural maturation after hearing onset. It involves stabilization of cell size and increase in volume of auditory structures. The increase in volume of auditory structures is due to continued growth of axons and dendrites, synaptogenesis, glial growth including myelination and angiogenesis (Cant, 1998).

There are several non-invasive audiological tests which can be used to monitor such development. Auditory brainstem responses are one of the most commonly used test to assess developmental changes at the brainstem level. CAEPs are used to monitor functioning of the central auditory pathways and to monitor development of auditory cortex.

The latency and morphology of the cortical, auditory evoked potential (CAEP) provides information about the maturation of central auditory pathways (Ponton, Eggermont, Kwong, & Don, 2000). In normal hearing children, the latency of the P1 wave decreases systematically as age increases (Ponton, Eggermont, Khosla, Kwong, & Don, 2002). For infants, the latency of the P1 can be as long as 300–400 ms and it reduces to 50 ms in adults. For a normal-hearing child the CAEP is dominated by the P1 neural response in a post-stimulus latency window of 50–300 ms whereas, adults CAEP are dominated by N1-P2 complex.

The research strongly suggests that young infants have the ability to perceive brief, rapidly changing temporal cues that are critical for discrimination of speech. Novak, Kurtzberg, Kreuzer, & Vaughan (1989) recorded CAEPs to formants extracted from synthesised CV syllables. They found no systematic effect of formant centre frequency on the responses recorded in the first 6 months of life. Kurtzberg, Hilpert, Kreuzer, Stone, & Vaughan (1986) found topographical differences in the CAEP of newborns that reflected frequency based differences in the place of articulation of consonants (/da/ vs. /ba/) and morphological differences that reflected voice onset time (/ta/ vs. /da/ and /ba/). Hence, CAEPs are used as an objective measure to investigate the neurophysiological processes that underlie the ability to perceive speech (Trembley, Piskosz, & Souza, 2003). Thus, CAEPs could be a potential tool to observe maturation of thalamic level and auditory cortex for speech.

Ponton et al. (2002) studied devoplemental changes of CAEP for children from 5 to 20 years of age. They found that P1 is most prominent in children 5 years and above. Sussman, Steinschneider, Gumenyuk, Grushko, and Lawson (2008) used speech stimulus to observe age related changes in children between 8 & 11 years. It can be noticed that all these studies were carried out in children age above 5 years. Several studies were carried out (Kraus, McGee, Carrell, Sharma, Micco, & Nicol, 1993; Sharma, Kraus, McGee, & Nicol, 1997;

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Sussman et al., 2008) using non speech and speech stimuli to observe changes in CAEPs over age by taking discrete age group. There are a very few studies where they have administered CAEP in children aged from birth to seven years of age to observe developmental changes. Hence, this study has been taken to see developmental changes of CAEP in children aged between birth and seven years.

CAEPs have been reported for clicks, tone bursts, and different types of speech signals including natural and synthetic vowels, syllables and words. Natural speech is preferred over non-speech stimuli as speech is more effective in identifying subtle neural processing problem in people with hearing impaired (Tremblay et al., 2003). Further, spectrally complex sounds such as speech evokes larger response than simple tones (Wunderlich, Cone-Wesson, & Sheperd, 2006). Ceponiene, Shestakova, Balan, Alku, Yiaguchi and Näätänen, (2001) investigated the CAEPs using speech and non speech stimulus and reported that the amplitude of N1-P2 complex was larger for speech sounds than for non-speech sounds. Hence, syllable /da/ was used to record the CAEPs in this study.

Stapells (2002) reported that P1 is prominent in infants at about 200 ms. Whereas, in adults the prominent peak is N1, a negative peak which occurs at a post-stimulus latency around 100 ms and P2, a positive peak at 175-200 ms (Wunderlich et al., 2006). Hence, the present study is focused to understand the change in wave morphology with age in paediatric population for speech stimulus i.e., occurrence or changes of dominant peak over age.

Many studies have demonstrated that peak latencies of cortical auditory evoked potential component are shorter in adults than in infants and children (Ponton et al., 2000; Wunderlich et al., 2006). Peak amplitudes increases with age for N1 and P2 components (Ponton et al., 2000; Wunderlich et al., 2006). Whereas, amplitude of P1 component also increases, reaches maximum amplitude at 1-3 year old (Ceponiene, Alku & Näätänen, 2003) and further amplitude declines with age (Ponton et al., 2000; Wunderlich et al., 2006). Hence, there is a need to understand the latency and amplitude changes as a function of age. Thus, the study was taken to investigate the differences in amplitude and latency changes as a function of age.

Auditory nervous system is not completely developed at birth. Hence, infants exhibit a lack of synchrony and this lack of synchrony would be more in infants with risk factors. Hence, CAEPS might be used as a tool to understand speech perception in infants. Thus, this study focused on establishing database for latencies and amplitude value for CAEP elicited by syllable /da/.

Hence, a systematic study on CAEPs from birth to 7 years would give a clear idea about developmental changes, change in morphology that occurs for the speech stimulus.

Aim of the study was to establish database for speech evoked cortical auditory evoked potentials for paediatric population from birth to 7 years of age at different intensities, to investigate the effect of age on morphology of cortical auditory evoked potentials to speech stimulus /da/, and to investigate the effect of age on latency of different wave and peak to peak amplitude of speech evoked cortical auditory evoked potentials.

## Method

**Subject:** A total of 37 children from birth to 7 years of age participated in the study. The subjects were divided into following groups:

Group A: 6 infants from birth to 6 months of age (mean age of 3.0 months).
Group B: 6 toddlers from 6 months to 12 months of age (mean age of 7.6 months).
Group C: 5 toddlers from 12 months to 24 months of age (mean age of 14.8 months).
Group D: 10 children from 2 years to 5 years of age (mean age of 45.7 months).
Group E: 10 children from 5 years to 7 years of age (mean age of 69.7 months).

**Subject selection criteria:** All the subjects had normal hearing sensitivity. Hearing sensitivity was ensured through pure-tone audiometry and immittance results for older children and behavioural observation audiometry (BOA), auditory brainstem response (ABR) and otoacoustic emissions (OAE) for younger children. Immittance showed normal middle function in all subjects. Transient click evoked oto-acoustic emissions (TEOAEs) were present in all the subjects. ABR was recorded at 40 dB nHL. Presence of any one of the ABR wave was considered as having normal hearing sensitivity.

**Stimulus used to record CAEPS:** The syllable /da/ recorded by Sumitha and Baraman, (2008) was used to record CAEPs. It consists of voice onset time, burst portion and a little portion of the vowel. The syllable duration was approximately 150 msec.

**Procedure**

**Behavioural observation audiometry:** A calibrated OB-922 clinical audiometer was used for behavioural observation audiometry and to estimate pure tone threshold. Behavioural observation audiometry was carried out in double room situation. Behavioural responses of the subjects were obtained in sound-

field condition using warble tones or narrow band noise of 500 Hz, 1 kHz, 2 kHz and 4 kHz and also for speech stimuli.

**Pure-tone audiometry:** Pure tone thresholds were obtained at octave frequencies between 250 Hz to 8000 Hz for air conduction and for bone conduction between 250 Hz to 4 kHz using OB 922 audiometer. The threshold was tracked using modified Hugson and Westlake method (Carhart & Jerger, 1959).

**Immittance evaluation:** A calibrated immittance meter (GSI- Tympstar) was used to assess middle ear status. The tympanometric measurements were carried out using 226 Hz probe tone at 85 dB SPL. For reflex measurements, the 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 reflexes.

**TEOAEs:** Transient click evoked oto-acoustic emissions (TEOAEs) was measured using ILO-V6 systems to check for integrity of the outer hair cells. TEOAEs were obtained using ILO-V6 instrument with a foam tip positioned in the external auditory canal so as to get a flat frequency spectrum across the frequency range. The stimulus used was click of 80 μs duration presented at 80 dB peak SPL. The stimuli were presented in non-linear mode and a total of 260 sweeps were averaged.

**Auditory brainstem response (ABR):** ABR and CAEPs was recorded using single channel IHS Evoked Potential System (v 3.22). The subjects were made to sleep to avoid the body movement. The non-inverting electrode was placed at Fz (high forehead) inverting electrode on the test ear mastoid and ground electrode was placed on the non test ear mastoid. Intra electrode and inter electrode impedance were maintained with in 5 kΩ and 2 kΩ respectively. ER-3A insert ear phones were used to present the stimulus for ABR. ABR was recorded using 100 μs click stimulus at 11.1 rate to obtain thresholds for infants, toddlers and young children on whom conditioned pure-tone threshold could not be obtained. The lowest level at which ABR was present (wave I or V) was considered as threshold.

Electrodes were not dislodged as, the same electrode montage was used to record CAEPS. Recording parameters used to record CAEPS is shown in Table 1. CAEPS were recorded twice at each intensity level for the reproducibility.

## Results

### Waveform morphology

Overall the result showed that the CAEPs were characterised by two components, a positive peak (P1) around 200 ms followed by a negative peak (N1) around 350 ms in toddlers below 2 years of age. The later components P2 and N2 were observed only in the older children age above 2 years.

*Table 1. Protocol used to record CAEPs*

| Stimulus parameter | | Acquisition Parameters | |
|---|---|---|---|
| Stimulus | Speech /da/ | Amplification | 50,000 |
| Duration | 150 ms | Filter setting | 1-30 Hz |
| Polarity | Alternate | Notch Filter | On |
| Transducer | ER-3A insert ear phones | | |
| Intensity | Variable starting at 80 dB nHL and was reduced in 20 dB nHL steps. | Electrode montage | Inverting (-) – test ear mastoid Non-Inverting (+) – Forehead Ground – Non test ear mastoid |
| Number of stimuli | 300 | Time analysis window | 500 msec with 50 msec baseline (per-stimulus recording) |
| Repetition rate | 1.1/s | Artifact rejection | 31 % |

In group 1, CAEPs could be recorded from 5 infants, CAEPs in one infant were noisy and hence was not considered for analysis. The characteristic positive peak (P1) was seen in all five infants at 80 dB nHL and at 60 dB nHL. P1 could be observed only and in 3 infants at 40 dB nHL. N1 was seen in 4 out of 5 infants at 80 dB nHL and 60 dB nHL. N1 was not observed at 40 dB nHL. CAEPs were not present at 20 dB nHL in all the infants.

In group 2, six P1 and N1 were recorded in all toddlers at 80 dB nHL and 60 dB nHL. At 40 dB nHL, P1 was present in 4 toddlers and N1 was present in only 2 toddler. At 20 dB nHL speech evoked P1 and N1 could not be recorded from any subject.

In group 3, P1 was present in all children at 80 dB nHL and 60 dB nHL. At 40 dB nHL, P1 was present in only 3 children. N1 was present in all children at 80 dB nHL. N1 was present in 3 children at 60 dB nHL and 40 dB nHL. CAEPs were absent at 20 dB nHL in all the children.

In group 4, P1 and N1 were present in all the children at 80 dB nHL. P1 and N1were present in 9 children at 60 dB nHL, 8 children at 40 dB nHL. Whereas at 20 dB nHL P1 was present in 7 children

*Table 2. Mean and standard deviation (SD) of P1, N1, P2 and N2 latency observed at different intensities across the groups*

| Intensity | Wave | Age | < 6 m | 6m-12 m | 1y-2y | 2y-5y | 5y-7y |
|---|---|---|---|---|---|---|---|
| 80 dB nHL | P1 | Mean | 204.88 | 222.66 | 191.44 | 126.74 | 105.04 |
| | | SD | 15.20 | 23.56 | 1.352 | 21.83 | 17.37 |
| | N1 | Mean | 362.85 | 372.00 | 347.80 | 215.64 | 164.05 |
| | | SD | 8.90 | 53.82 | 14.19 | 47.11 | 28.77 |
| | P2 | Mean | - | - | - | 244.28 | 215.60 |
| | | SD | - | - | - | 7.14 | 4.59 |
| | N2 | Mean | - | - | - | 293.13 | 287.36 |
| | | SD | - | - | - | 7.77 | 15.28 |
| 60 dB nHL | P1 | Mean | 218.80 | 242.93 | 217.00 | 143.28 | 133.92 |
| | | SD | 11.17 | 25.18 | 13.64 | 15.41 | 8.33 |
| | N1 | Mean | 407.10 | 382.26 | 360.40 | 232.11 | 221.92 |
| | | SD | 22.31 | 62.02 | 3.60 | 38.10 | 46.98 |
| | P2 | Mean | - | - | - | 261.86 | 261.60 |
| | | SD | - | - | - | 29.94 | 41.20 |
| | N2 | Mean | - | - | - | 319.60 | 310.56 |
| | | SD | - | - | - | - | 16.10 |
| 40 dB nHL | P1 | Mean | 253.00 | 273.60 | 250.33 | 169.60 | 152.17 |
| | | SD | 4.01 | 46.18 | 4.50 | 28.28 | 8.74 |
| | N1 | Mean | - | 410.50 | 387.06 | 271.22 | 252.11 |
| | | SD | - | 11.45 | 15.27 | 36.96 | 51.99 |
| | P2 | Mean | - | - | - | 265.60 | 283.60 |
| | | SD | - | - | - | - | 70.54 |
| | N2 | Mean | - | - | - | - | 322.40 |
| | | SD | - | - | - | - | 28.87 |
| 20 dB nHL | P1 | Mean | - | - | - | 195.20 | 185.80 |
| | | SD | - | - | - | 30.63 | 33.02 |
| | N1 | Mean | - | - | - | 298.13 | 304.00 |
| | | SD | - | - | - | 18.56 | 38.91 |
| | P2 | Mean | - | - | - | - | 430.40 |
| | | SD | - | - | - | - | - |
| | N2 | Mean | - | - | - | - | - |
| | | SD | - | - | - | - | - |

and N1 was present in 6 children. The later components of CAEPs, P2 and N2 were first observed in this group of children. 3 children at 80 dB nHL, only 1 child at 60 dB nHL and was not present in any children at 40 dB nHL. P2 and N2 were absent in all the children at 20 dB nHL.

In Group 5 P1 was present in all children at 80 dB nHL and 60 dB nHL, 9 children at 40 dB nHL and 8 children at 20 dB nHL. N1 was present in all children at both 80 dB nHL and 60 dB nHL, 7 children at 40 dB nHL and 6 children at 20 dB nHL. P2 was observed in six children at 80 dB nHL and 60 dB nHL. At 40 dB nHL 4 children had P2 wave and only one child showed P2 at 20 dB nHL. Similarly 5 children had N2 wave at 80 dB nHL and 60 dB nHL. At 40 dB nHL 4 children had N2 wave. However, none of them had N2 wave at 20 dB nHL.

**Age related changes in wave latency**

The mean, standard deviation and range of P1, N1, P2 and N2 latency were calculated for all the groups at each presentation level. This is shown in Table 2.

**P1 wave:** From the Table 2 it can be seen that as the age increased CAEPs P1 and N1 latency of decreased, except for group 2 which had maximum mean latency at each presentation level. Table also shows that, as the intensity of the stimulus was reduced the latency of all the waves increased in all the age groups. P1 and N1 was absent at 20 dB nHL in all the subjects in younger population aged below 2 years.

*Table 3. Chi-Square value, degrees of freedom and significant level for P1 latency observed at different intensities across the groups*

| Intensity (nHL) | 80 dB | 60 dB | 40 dB |
|---|---|---|---|
| Chi-Square value | 28.78 | 27.34 | 19.18 |
| df | 4 | 4 | 4 |
| p | 0.00 | 0.00 | 0.001 |

The Kruskal Wallis test was carried out for the comparison of P1 latency across the groups. The result indicated that P1 latency was significantly different across the groups at 80, 60 and 40 dB nHL. The results are shown in Table 3.

As Kruskal Wallis test showed significant differences at 80, 60 and 40 dB nHL, Mann-Whitney test was carried out in order to know whether there was any significant difference between the any two groups. The result can be seen in Table 4. It can be seen in the table that the P1 latency obtained in group 3, 4 and 5 reduced significantly from group 1 and group 2 at almost at all intensities. Whereas, P1 latency of group 1 and group 2 did not differ significantly from each other. In group 4, P1 latency did not differ significantly with group 5 at 60, 40 and 20 dB nHL also.

To compare P1 latency obtained at 20 dB nHL between the group 4 and 5 Mann Whitney test was administered. P1 latency did not differ significantly between the groups ($|z| = 1.27$, $P > 0.05$).

**N1 wave:** From the Table 2 it can be observed that as the age increased the latency of N1 component decreased, except for group 2 which had maximum latency at all presentation level. Table also shows that, as the intensity of the stimulus reduced the latency of N1 increased for all the age groups. N1 was absent at 40 dB nHL and 20 dB nHL in group 1, and was absent at 20dB nHL in all the subjects in group 2 and group 3. The Kruskal Wallis test was carried out for the comparison of N1 latency across the groups. The result indicated that N1 was significantly different across the groups at 80 dB nHL, 60 dB nHL, and 40 dB nHL. The details are shown in Table 5. Mann Whitney test was carried out to seem which two groups N1 latency differed significantly from each other. The details of the test results are shown in Table 6.

It can be seen in the table 6 that N1 latency of group 5 reduced significantly from group 1, 2, 3 and 4 at almost at all intensities. Whereas N1 latency of group 1 and group 2 did not differ significantly from each other. Also N1 latency in group 4 did not differ significantly with N1 latencies in group 5 at 80 and 40 dB nHL.

**P2 and N2 wave:** From the Table 2, it can be seen that P2 and N2 was absent in all the subjects and at all presentation levels for younger children age less than 2 years. P2 and N2 were first noticed in children aged from 2 years. From the Table 2 it can also be seen that as the age increased the latency of P2 and N2 component of the CAEP decreased at all levels of stimulation. The latency of both P2 and N2 increased with decrease in presentation level for both the groups (group 4 & group 5).

*Table 4. z-value and the significant level for P1 latency between the groups at 80 dB nHL, 60 dB nHL and 40 dB nHL*

| Group Intensity | | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| 1 | 80 | $\|z\| = 1.28$ | $\|z\| = 1.98$ * | $\|z\| = 3.06$ * | $\|z\| = 3.06$ * |
| | 60 | $\|z\| = 1.46$ | $\|z\| = 0.10$ | $\|z\| = 3.00$ * | $\|z\| = 3.07$ * |
| | 40 | $\|z\| = 0$ | $\|z\| = 1.96$ * | $\|z\| = 2.45$ * | $\|z\| = 2.50$ * |
| 2 | 80 | - | $\|z\| = 2.10$ * | $\|z\| = 3.25$ * | $\|z\| = 3.29$ * |
| | 60 | - | $\|z\| = 2.19$ * | $\|z\| = 3.18$ * | $\|z\| = 3.26$ * |
| | 40 | - | $\|z\| = 0.35$ | $\|z\| = 2.55$ * | $\|z\| = 2.78$ * |
| 3 | 80 | - | - | $\|z\| = 3.06$ * | $\|z\| = 3.06$ * |
| | 60 | - | - | $\|z\| = 3.00$ * | $\|z\| = 3.07$ |
| | 40 | - | - | $\|z\| = 2.45$ * | $\|z\| = 2.50$ * |
| 4 | 80 | - | - | - | $\|z\| = 2.19$ * |
| | 60 | - | - | - | $\|z\| = 1.76$ |
| | 40 | - | - | - | $\|z\| = 1.59$ |

* $p < 0.05$

*Table 5. Chi-Square value, degrees of freedom and significant level for N1 observed at different intensities across the groups*

| | 80 dB nHL | 60 dB nHL | 40 dB nHL |
|---|---|---|---|
| Chi-Square value | 26.008 | 22.932 | 11.368 |
| df | 4 | 4 | 3 |
| p | 0.000 | 0.000 | 0.010 |

*Table 6. z-value and the significant level for N1 latency between the groups at different intensities*

| Group | Intensity | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| 1 | 80 | \|z\| =0.10 | \|z\|=2.44* | \|z\|=2.83 * | \|z\|=2.71 * |
| | 60 | \|z\| =0.42 | \|z\| = 0 | \|z\|= 2.78 * | \|z\|=2.82 * |
| | 40 | - | - | - | - |
| 2 | 80 | - | \|z\|=0.91 * | \|z\|= 3.25 * | \|z\|=3.09 * |
| | 60 | - | \|z\| = 0 | \|z\|= 3.18 * | \|z\|=3.25 * |
| | 40 | - | - | - | - |
| 3 | 80 | - | - | \|z\|= 3.06 | \|z\|=2.92 * |
| | 60 | - | - | \|z\|=2.50 * | \|z\|=2.53 * |
| | 40 | - | - | \|z\|=2.44 * | \|z\|=2.39 * |
| 4 | 80 | - | - | - | \|z\|=2.13 |
| | 60 | - | - | - | \|z\|=2.13 * |
| | 40 | - | - | - | \|z\|=0.86 |

* $p < 0.05$

*Table 7. z-value and the significant level of P2 and N2 latency at different intensities between the groups*

| Wave | Group | 80 dB nHL | 60 dB nHL |
|---|---|---|---|
| P2 | 4* 5 | \|z\| = 2.46<br>P < 0.05* | \|z\| = 0.25<br>P > 0.05 |
| N2 | 4* 5 | \|z\| = 0.90<br>P > 0.05 | - |

** $p < 0.05$*

Mann Whitney test was carried out to compare P2 and N2 latency between the groups. The result showed a significant difference in P2 latency at 80 dB nHL and no significant difference at 60 dB nHL between the groups. N2 latency did not differ significantly at 80dB nHL between the groups (Table 7).

**Age related changes in P1-N1 amplitude**

From the Table 8 it can be seen that as the intensity of the stimulus was reduced the amplitude of the P1-N1 reduced systematically for all the age groups. However, P1-N1 complex did not show any systematic increase or decrease in its amplitude with the age.

Kruskal Wallis test showed significant difference in amplitude of P1-N1 complex across the group only at 80 dB nHL. The results are shown in Table 9. To know if there was a significant difference for P1-N1 amplitude at 80 dB nHL between the groups Mann-Whitney test was administered. From the table 10 it can be seen that amplitude of P1-N1 complex of group 4 is significantly smaller from group 1, 2 and 3 at 80 dB nHL.

**N1-P2 amplitude**

It can be seen from the Table 11 that as the intensity of the stimulus reduced the amplitude of the responses reduced systematically for both the groups.

Further, N1-P2 complex showed a systematic increase in amplitude with the age. Mann-Whitney test showed a significant difference in amplitude of N1-P2 complex between the group 4 and group 5 at 80 dB nHL and no significant difference at 60 dB nHL.

*Table 8. Mean, and standard deviation (SD), of P1-N1 amplitude observed at different intensities across the groups*

| Intensity | Age | < 6m | 6 m-12 m | 1y - 2y | 2y - 5y | 5y - 7y |
|---|---|---|---|---|---|---|
| 80 dB nHL | M | 7.75 | 7.55 | 10.51 | 4.92 | 6.39 |
| | SD | 2.14 | 2.62 | 3.08 | 1.45 | 2.74 |
| 60 dB nHL | M | 5.52 | 6.10 | 6.01 | 4.34 | 7.65 |
| | SD | 1.37 | 2.42 | 0.70 | 1.09 | 3.62 |
| 40 dB nHL | M | - | 5.80 | 3.90 | 4.55 | 6.47 |
| | SD | - | 0.25 | 0.09 | 1.93 | 1.77 |
| 20 dB nHL | M | - | - | - | 3.88 | 5.89 |
| | SD | - | - | - | 1.20 | 3.36 |

*Table 9. Chi-Square value, degrees of freedom and significant level for P1-N1 amplitude observed at different intensities across the groups*

| P1-N1 | 80 dB nHL | 60 dB nHL | 40 dB nHL |
|---|---|---|---|
| Chi-Square value | 12.333 | 5.945 | 5.93 |
| df | 4 | 4 | 2 |
| p | 0.015 | 0.203 | 0.051 |

*Table 10. z-value and the significant level for P1-N1 amplitude at 80 dB nHL between the groups*

| Group | 2 | 3 | 4 | 5 |
|---|---|---|---|---|
| 1 | \|z\|=0.21 | \|z\|=1.47 | \|z\|=2.40 * | \|z\| =0.67 |
| 2 | - | \|z\|=1.64 | \|z\|=2.17 * | \|z\| =0.51 |
| 3 | - | - | \|z =2.94 * | \|z\| =1.75 |
| 4 | - | - | - | \|z\| =1.06 |

* $p < 0.05$

*Table 11. Mean, and standard deviation (SD) of N1-P2 amplitude observed at different intensities across the groups*

| Intensity (dB nHL) | Age | 2y - 5y | 5y - 7y |
|---|---|---|---|
| 80 | Mean | 2.11 | 3.47 |
| | SD | 3.07 | 1.66 |
| 60 | Mean | 1.75 | 2.63 |
| | SD | 1.72 | 5.34 |
| 40 | Mean | 1.32 | 2.22 |
| | SD | - | 5.80 |
| 20 | Mean | - | 5.48 |
| | SD | - | - |

*Table 12. z-value and the significant level for N1-P2 amplitude at 80 dB nHL and 60 dB nHL between the groups*

| Group | 80 dB nHL | 60 dB nHL |
|---|---|---|
| 4* 5 | \|z\| = 1.715 * | \|z\| = 0.258 |

* $P < 0.05$

## Discussion

The result of the study showed that there were systematic age-related changes in latencies for all components of the CAEP evoked by syllable /da/. The systematic changes of CAEP observed in the study are in consonance with the result observed by Bruneau, Roux, Guérin, Barthélémy, and Lelord (1997), Ceponiene et al. (2003), Sharma et al. (1997) Sussman et al. (2008) Wunderlich et al. (2006). These changes provide insights into the maturation of the neural generators of the CAEP.

**Waveform morphology**

The result of the study showed that children below two years of age had a characteristic positive peak around 200 ms followed by a negative peak around 350 ms. A similar observation was also made by various investigators (Barnet, Ohkich, Weiss, & Shanks, 1975; Sharma & Dorman, 2006; Shucard & Thomas et al., 1987). Later components i.e., P2 and N2 were first observed in group 3 i.e. age range between 2 – 5 years of age. This result is in agreement with the results obtained by Wunderlich et al. (2006). They reported that all the components of CAEPs, P1-N1-P2-N2, were present in toddlers with a mean age of 24 months. In contrast Ponton et al. (2000) reported P2 and N2 to be present in children greater than 5 years of age.

**Age-related changes in response latency**

**P1 wave:** The result showed that the P1occured at about 200 ms in infants and toddler below two years of age. This is consistent with the previous studies on CAEPs in infants aged less than 12 months (Rapin & Graziani, 1967). They also reported a positive P1 peak around 200 ms in children age below two year. A similar finding was also reported by Molfese (2000) who observed a large positive wave at around 200 ms (P1) in a 16 month old child. In Group *4, P1 was observed around 125 ms*, which is in consonance with the results reported by Ceponiene et al., (2003). In a group of 3 year old children they observed CAEP responses dominated by a large P1 wave at around 130 ms. P1 latency for group 5 was around 105 ms and similar results were also reported by (Cunningham et al., 2000; Oades et al., 1997; Ponton et al., 2000, 1996; Sharma et al., 1997).

A significant decrease in latency of P1 in children older than 2 years was observed in the current study. Similar results are also reported by Cunningham et al. 2000; Oades et al. 1997; Ponton et al. 2000, 1996; Sharma et al. 1997; and Wunderlich et al. 2006. Kushnerenko, Ceponiene, Balan, Fellman, and Näätänen, (2002) reported that latency of P1significantly decreased from 3 to 6 months and, further, to 9 months of age. However, in this study, P1 did not differ significantly between the infants in group 1 and toddler in group 2. A similar finding for children in the first year of life has been reported by Shucard et al., (1987). However, investigators have also shown a decrease in P1 latency within first year of life (Kushnerenko, Ceponiene, Balan, Fellman, & Näätänen, 2002; Wunderlich et al., 2006).

It is believed that P1 is generated in the deeper cortical layer, of the lateral portion of Heschl's gyrus. 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) (Moore & Guan, 2001). This could be the reason why significant changes in P1 latency was not noticed between group 1 and group 2. But P1 latency decreased significantly after 2 years of age.

**N1 wave:** The results of the current study showed that the N1occurs at about 360 ms in infants. This is consistent with the previous studies of CAEPs in infants which reported presence of N1 latency around 300–550 ms (Barnet et al., 1975; Shucard et al.,

1987). In children between 1 and 2 years of age N1 latency observed was around 345 ms. In Group 4, N1 was observed around 215 ms. Ceponiene et al., (2003) also observed in a group of 3 year old children who showed a response dominated by a large P1 around 130 ms followed by two negative waves at 250 and 450 ms. N1 latency for group 5 was around 120 - 200 ms and similar results were reported by various investigators for five to six year old children (Bruneau, Roux, Guérin, Barthélémy, and Lelord, 1997; Cunningham et al., 2000; Johnstone, Barry, Anderson, & Coyle, 1996; Ponton et al., 2000).

The result showed a general decline in N1 latency with age in children older than one year. Similar findings were also reported by various authors (Bruneau, Roux, Guérin, Barthélémy, and Lelord, 1997; Cunningham et al., 2000; Kraus et al., 1993; Oades, Dittman-Balcar, & Zerbin, 1997; Ponton et al., 2000; Sharma et al., 1997; Shucard et al., 1987). Latency of N1 was not significantly different in infants less than 1 year old in the present study and this finding is in consonance with the results obtained in various studies (Shucard et al., 1987; Wunderlich et al., 2006).

N1 has several generators in the upper cortical layers including primary and secondary auditory cortex in or near the supratemporal plane (Ponton et al., 2002). 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, mature axons begin to appear in cortical layers 2 and 3 and by 12 years of age their density is equivalent to that of young adults (Moore & Guan, 2001). These changes in the marginal layer of the auditory cortex might be attributed to maturational changes of N1 latency that was observed in this study and also suggest the possible reason why significant changes of N1 latency observed after 1 year of age.

**P2 and N2 wave:** P2 latency was significantly reduced at 80 dB nHL in group 5 in comparison to group 4. A similar decline in P2 latency with age was reported by Oades et al. (1997). Whereas, most studies reported no age-related changes in P2 peak latency (Johnstone et al., 1996; Ponton et al., 2000; Tonnquist- Uhlen, 1996).

Latency of N2 was not significantly different in children between 2 to 7 years of age. In literature there are conflicting findings regarding the development of peak latency of N2 with some studies showing a decline (Cunningham et al., 2000; Oades et al., 1997), others observed no change in N2 latency (Johnstone et al., 1996; Tonnquist- Uhlen, 1996). Ponton et al., (2000) observed an increase in latency with age. N2 component of CAEPs has been linked to higher level, discriminative processes (Cunningham et al., 2000; Johnstone, et al., 1996) and is sensitive to task demands and attention (Näätänen & Picton, 1986). Hence, the lack of age related changes in N2 latency in the present study might be due to the task used to elicit CAEP which did not tap the attention levels and discrimination ability of the participants.

**Age-related changes in response amplitude**

**P1-N1 amplitude:** P1-N1 complex amplitude did not show any systematic increase or decrease in amplitude across the age. This could be due to differential affect of age on P1 and N1 absolute amplitude. Wunderlich et al. (2006) reported a systematic decrease in the magnitude of P1 after the first year of life. Similar decrease in amplitude of P1 with age in older children was also reported by Ceponiene et al. (2002), Cunningham, Zecker, and Kraus, (2000), Oades Dittman-Balcar, & Zerbin, (1997), Ponton et al. (2000) and Sharma et al. (1997).

Bruneau, Roux, Guérin, Barthélémy, and Lelord (1997) observed an increase in N1 amplitude with age. A similar finding was also reported by Pang and Taylor (2000). Wunderlich et al. (2006) reported a systematic increase in N1 amplitude from newborn to adulthood. Hence, the differential affect of age on P1 and N1 absolute amplitude might have resulted in inconsistency of amplitude of P1-N1 complex in the present study.

**N1-P2 amplitude:** The result of the present study showed that, N1-P2 complex amplitude increased systematically with age. This finding is in agreement with the observation made by Wunderlich et al. (2006). They reported that absolute amplitude of N1 and P2 was small in newborns and toddlers and larger in the children and adults. Due to increase in N1 and P2 absolute amplitude with age N1-P2 complex amplitude has increased with age, which is observed in the current study.

## Conclusions

The results of the current study showed systematic changes in latencies of components of CAEPs with age. Morphology of CAEPs recorded from infants was different from those of older children. Younger children less than two years of age had characteristic biphasic response, a positive peak (P1) around 200 ms followed by a negative peak (N1) around 350ms after the stimulus onset. P2 and N2 of the CAEPs were first observed in children older than two years of age. Further, the latency of P1, N1, P2 and N2 decreased and amplitude of N1-P2 complex increased systematically with age. This variation in CAEP wave latency and amplitude is to be considered while dealing with the clinical group. Thus it can be concluded from the study that CAEPs can be used to observe maturation of auditory cortex.

The use of speech stimuli could give two fold advantages i.e. to observe maturational changes and also systematic changes in perceptual abilities of a individual with age.

**Implications of the study**

The data obtained from the study provides normal values which can be used as norms for CAEPs elicited by speech. This would be useful to identify abnormal CAEPs and also to observe maturational delay.

The electrophysiological measures such as CAEPs are important because they can be used to evaluate the benefits of hearing aids and cochlear implants in infants, young children, and adults in whom behavioural responses cannot be obtained while fitting amplification devices. These data can be used as reference to observe the benefits.

Changes in morphology of wave after rehabilitation might provide insight about the neural plasticity or maturation of central auditory pathway after rehabilitation. The data obtained from the study can also be used as baseline to notice post rehabilitation changes after hearing aid fitting and cochlear implantation.

Results of CAEPs results obtained in this study can be used as an electrophysiological measure of speech encoding ability in the auditory system of children below 7 years of age.

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