SPEECH-EVOKED CORTICAL POTENTIAL IN CHILDREN WITH NORMAL HEARING SENSITIVITY

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A Dissertation Submitted in Part Fulfilment For Degree Of

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MANASAGANGOTHRI, MYSORE-570006

MAY, 2014


Dedicated to Maa, Papa and Anu
For their endless love and support

CERTIFICATE

This is to certify that this dissertation entitled "**Speech Evoked Cortical Potential in Children With Normal Hearing Sensitivity (3-9years)**" is the bonafide work submitted in part fulfillment for the degree of Master of Science (Audiology) of the student with Registration No. 12AUD003. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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CERTIFICATE

This is to certify that this dissertation entitled "**Speech Evoked Cortical Potential in Children with Normal Hearing Sensitivity (3-9 years)**" has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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DECLARATION

This dissertation entitled "**Speech Evoked Cortical Potential in Children With Normal Hearing Sensitivity (3-9 years)**" is the result of my own study under the guidance of Dr. Prawin Kumar, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier in any other University for the award of any Diploma or Degree.

Mysore

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INTRODUCTION

When an individual hears a sound, acoustic information is transformed from a mechanical vibration into an electrical signal in the inner ear. This electrical signal is further processed and then transferred through the auditory pathway in both a temporal and spatial manner. The electrical activity can be recorded on the scalp using electroencephalography (EEG) and averaged to detect the neural responses that occur specifically in response to the sound. Event related potentials (ERPs) are time-locked brain responses to some "event", which can include acoustic, visual, or some other sensory stimulus.

Late latency auditory evoked potentials, historically the first discovered, are cortical in origin and are much larger and lower in frequency than early and middle latency potentials. This potential is highly dependent upon stimulus type, recording location and technique, patient age and state. The late latency responses may differ dramatically in morphology and timing due to above mentioned variability.

Cortical Auditory Evoked Potentials (CAEP) is used to monitor the functioning of central auditory pathways and also to monitor the development of auditory cortex. CAEPs are believed to reflect the activities of excitatory post-synaptic potentials at the level of thalamus and higher auditory cortex (Panton & Don, 2003; Wunderlich& Cone-Wesson, 2006).

CAEPs consists of a series of positive and negative peaks (P1/N1 complex) occurring between 80 and 150 ms after stimulus onset. P1 and N1 components of cortical potentials primarily reflect sensory encoding of auditory stimulus attributes and precede more endogenous components such as N2 and P3 which are concerned with attention and cognition. These potentials are generated by multiple temporally overlapping subcortical and cortical sources (Chen & Buchwald, 1986; Naatanen&Picton, 1987). These components are passively elicited in that the subject is not required to perform a task and is asked simply to

remain alert. Since they are not influenced by behavioural and performance related demands, these evoked responses provide a reliable objective measure of cortical auditory function in children.

Development of the peripheral auditory system (ear and auditory brainstem) is complete in early childhood (Eggermont, 1989). In contrast, central auditory pathways of the human brain exhibit progressive anatomical and physiological changes through early childhood (Kraus et al., 1985; Courchesne 1990; Huttenlocher, 1979). This maturation is likely to have impact on speech and oral language skills, which are primarily acquired through auditory modality. CAEPs reflect maturation of the human brain through change in their latency, amplitude and morphology (Eggermont, 1989; Courchesne, 1990).

A study by Golding et al. (2007) on 21 aided children with severe to profound hearing loss in age range 8 weeks to 3 years showed significant relationship between CAEP and functional outcomes for aided infants. The results show variability as the comparison is made with the functional outcome measures. While there has been substantial progress in our understanding of CAEP development (Wunderlich& Cone-Wesson, 2006), there remains several areas which require investigation. The characteristics of the CAEP in the toddler years (1-3 years) are largely uncharted and those in the early years of childhood (4-6 years) require further systematic examination.

NEED FOR THE STUDY

There are several studies highlighting the importance of CAEPs in assessing and monitoring the progress of children with hearing impairment (Dillon et al., 2012; Golding et al., 2007; Hinduja, Kushari&Vanaja, 2005, Shruti &Vanaja, 2007). However, there are variability's observed in different studies while recording CAEPs. This could be because of different factors which may be responsible for changes in latency and amplitude of CAEPs.

In spite of different variables affecting cortical potential recording, in different audiological set up there is a need to have their own normative to compare with the clinical populations. Further, presentation of speech stimuli is having own importance in comparison to other kinds of stimuli like click or tone bursts while recording CAEPs. Hence, present study is taken up to develop normative in Indian population with three different types of speech stimuli (/m/, /g/ & /t/) representing low, mid and high frequency region of speech audibility.

The detection of adult cortical auditory evoked potentials (CAEPs) can be challenging when the stimulus is just audible. Golding et al. (2009) compared the effectiveness of responses detected based on statistical procedure and expert examiners in (1) detecting the presence of CAEPs when stimuli were present, and (2) reporting the absence of CAEPs when no stimuli were present, was investigated.

CAEPs recorded from ten adults, using two speech-based stimuli at5 stimulus presentation levels, and non-stimulus conditions were recorded and given to four experienced examiners who were asked to determine if responses to auditory stimulation could be observed, and their degree of certainty in making their decision. These recordings were also converted to multiple dependent variables and Hotelling's T2 was applied to calculate the probability that the mean value of any linear combination of these variables was significantly different from zero. The results showed that Hotelling's T2 was equally sensitive to the best of individual experienced examiners in differentiating a CAEP from random noise. It is reasonable to assume that the difference in response detection for a novice examiner and Hotelling's T2 would be even greater. Further, present study will also check the effect of different intensity levels on these different speech stimuli.

AIM OF THE STUDY

The present study is aimed to develop the normative in Indian population for speech evoked cortical potential in children with normal hearing in the age range of 3-9 years.

OBJECTIVE OF THE STUDY

1) To determine the effect of intensity on speech evoked cortical potential in children of different age groups.
2) To determine the effect of different speech stimulus on speech evoked cortical potential.
3) To determine the effect of age on speech evoked cortical potential in children with normal hearing.

REVIEW OF LITERATURE

Auditory evoked potentials (AEPs)are a time-locked voltage changes in the presence of a sound stimulus. These voltage changes are generated by auditory system neurons, from the cochlear nerve up to the cortex, occurring in response to the repetition of a sound. The electrical activity to each stimulus is recorded and averaged, to lower the signal to noise ratio of the AEP, as a single response is low in amplitude and embedded in the background noise. The auditory brainstem response (ABR) is an early onset AEP, followed by the middle latency response (MLR) and the late latency response (LLR).

The late latency response (LLR) is an AEP that is made up of several different peaks, the P1, N1, P2 and N2. The LLR occurs approximately 50ms after stimulus onset, and begins with a positive peak of the P1, followed by the negative peak N1, positive peak P2 and negative peak N2. The N1 response is a large negative peak occurring between 80-140ms post stimulus onset, while the P2 is a large positive peak occurring between 140-250ms post stimulus onset (Boutros et al., 2004). The various peaks of the LLR originate from different areas of the neural network. The P1 originates from the secondary auditory cortex, the N1 has multiple generators in the primary auditory cortex, the frontal lobes, and midbrain, the P2 originates from the thalamic reticular activating system, and the N2 has non-specific subcortical origins as reviewed by (Bishop, Hardiman, Uwer, & Von Suchodoletz, 2007; Burkard, Don, &Eggermont, 2007).

Research on AEP changes that occur over time, in response to a train of stimuli, and inter-individual differences in this response, has focused mainly on the cortical level. As a stimulus is repeated, the amplitude of N1 decreases, a phenomenon known as the N1 response decrement or sensory gating. For N1 group data, some report that the greatest N1

decrement occurs at the second presentation of the stimulus within the stimulus train, and this decrement increases as the interval between stimuli decreases (Zhang, Eliassen, Anderson, Scheifele, & Brown, 2009). Inter-individual differences in AEPs, rather than group data, are valuable for evaluating variability with respect to temporal aspects of auditory neural processing. Significant variation between subjects is observed in the N1 response decrement pattern, when individual rather than group data are considered (Budd, Barry, Gordon, Rennie, &Michie, 1998; Rosburg, Zimmerer, &Huonker, 2010; Sable, Low, Maclin, Fabiani, &Gratton, 2004; Soros, Michael, Tollkotter, & Pfleiderer, 2006). Several different mechanisms associated with the cortical level neural circuitry have been proposed as being responsible for the N1 decrement, including differences in the refractory period of neurons (Rosburg, Zimmerer, &Huonker, 2010), latent inhibition (Sable, Low, Maclin, Fabiani, &Gratton, 2004), or habituation (Budd, Barry, Gordon, Rennie, &Michie, 1998). The ability of neurons to generate action potentials in response to repeated stimuli depends on several factors associated with neural adaptation (synaptic transmission and the neural refractory period), and inhibition. Neural adaptation is the change that occurs in response to a constant or repeating stimulus. Pre- and post-synaptic mechanisms can also be involved, and inhibition can serve to decrease the neural response to a constant or repeating stimulus by negative feedback mechanisms.

Normal Development of the P1 Response

Latency of the P1 wave is thought to reflect the sum of synaptic transmission delays throughout the central auditory pathways (Eggermont, Ponton, Don, Waring & Kwong 1997). Latency changes in the P1, as a function of increasing age, reflect the maturation of the central auditory pathways occurring (at least in part) in response to auditory stimulation. Sharma and colleagues have gathered data describing the developmental trajectory of the P1 response throughout infancy and childhood, and other researchers have further described

patterns of development that lead to changes in P1 latency and morphology (Sharma, Kraus, McGee & Nicol, 1997; Ceponiene, Rinne& Näätänen, 2002; Sharma, Dorman & Spahr, 2002; Moore & Linthicum, 2007). The N1-P2 cortical response has been investigated in normal hearing individuals (Adler & Adler, 1989; Tremblay, Billings, Friesen, & Souza, 2006) as a means of quantifying the detection of acoustic cues. The response can be accurately and efficiently obtained using an electrode placed at the vertex (Vaughan & Ritter, 1970).

Cortical auditory evoked potentials have most frequently been elicited using tonal stimuli; although speech stimuli have been utilized recently in individuals with normal hearing (Agung, Purdy, McMahon, &Newall, 2006; Tremblay, et al., 2006; Tremblay, Kelly, et al., 2005; Micco, et al., 1995Friesen, Martin, & Wright, 2003) and in cochlear implant recipients (Groenen, Beynon, Snik,& Broek, 2001).Stimuli that vary in the spectral and temporal domains best reflect natural speech and provide a realistic representation of speech processing at the cortical level.

Normal Development of the N1 Response

Some studies have reported that the latency of N1 decreases as age increases up to 16 years (Tonnquist-Uhlen et al., 1995) and up to 20 years (Johnson, 1989). The N1 amplitude increases up to 15 years of age(Martin et al., 1988). Conversely, other studies have reported little or no age-related changes in N1 latency (Ohlrichet al., 1978; Martin et al., 1988) and amplitude (Fuchigami et al., 1993). Recent studies have also suggested that the component analogous to the adult N1 may not emerge until 8 or 10 years of age (Csepe, 1995; Ponton et al., 1996).Various investigators have reported unreliable N1 responses in

young children aged 5 to 7 years (Goodling et al. 1978) that becomes progressively consistent as the age increases(Ponton et al. 2000) or adolescence (Sharma et al. 1997)

Normal Development of the P2 Response

Developmental changes reported for P2 responses elicited by simple stimuli have generally been minimal. But Ponton et al. (1997) reported that at birth and up to 7 years of age P2 is absent and the response is dominated by large late P1 response. Some researchers have reported that P2 latency increases with age (Goodling et al.1978), whereas others have reported no maturational changes in P2 response (Barrett et al.1997).

Maturation of the cortical auditory evoked potential in infants and young children

The P1 waveform changes in a complex manner in children. P1 decreases systematically in latency and/or amplitude to reach adult values almost at the age of 14-15years (Ponton, Eggermont, Kwong, & Don, 2000) or 20 years (Sharma, Kraus, McGee, & Nicol, 1997). The maturation of CAEPs has been investigated in children who received their cochlear implant between 18 months and six years of age, with the average age of implantation being 4.5 years (Ponton, Don, Eggermont, Waring, & Masuda, 1997).

The CAEPs, and in particular, the peak latency of P1, appeared to mature at the same rate as in children with normal hearing but were approximately delayed by the corresponding length of auditory deprivation (Ponton et al., 1996). This finding emphasizes that once adequate auditory stimulation is provided, the central auditory pathway continues to develop, but it is delayed by the duration of deafness, suggesting a limited form of auditory plasticity.

Other studies further suggest that the plasticity of central auditory pathways is maximal only for a restricted period of about 3.5 years in early childhood (Sharma, Dorman, & Kral, 2005; Sharma, Dorman, & Spahr, 2002). If the hearing system is stimulated within that

period, the P1 morphology and latency reach age-normal values within 3 to 6 months following the beginning of auditory stimulation. By contrast, if the auditory system does not receive adequate stimulation for more than 7 years, then most children exhibit a delayed P1 latency and an abnormal large P1, even after years of implant use (Sharma, Dorman, &Kral , 2005; Sharma, Dorman, &Spahr, 2002).

Julia et al in 2006 studied the Maturation of the cortical auditory evoked potential in infants and young children. Evoked potential (CAEP) in humans. The participants in this experiment were 10 newborns (<7 days), 19 toddlers (13–41 months), 20 children (4–6 years) and 9 adults (18–45 years). CAEPs were obtained in response to low (400 Hz) and high (3000 Hz) tones and to the word token /bæd/, all presented at 60 dB HL, at a rate of 0.22 Hz. Latency and amplitude measures were made for CAEP components P1, N1, P2 and N2 as a function of participant age, stimulus CAEP component latencies were relatively stable from birth to 6 years, but adults demonstrated significantly shorter latencies compared to infants and children. Components P1 and N2 decreased in amplitude, while components N1 and P2 increased in amplitude from birth to adulthood. Words evoked significantly larger CAEPs in newborns compared to responses evoked by tones, but in other age groups the effects of stimulus type on component amplitudes and latencies were less consistent.

There was evidence of immature tonotopic organisation of the generators of N1 when responses from infants and young children were compared to those of adults. The scalp distribution of components N1 and P2 was clearly different in newborns and toddlers compared to children and adults. In the younger groups, both N1 and P2 were uniformly distributed across the scalp but in children and adults these components showed more focal distributions, with evidence of response laterality increasing with maturity. The results of the present study describe CAEPs recorded from multiple scalp electrodes, for tones and speech stimuli, in infants and children from birth to 6 years of age. Frequency-related differences in

component amplitude were apparent at all ages reflecting development of tonotopic organisation of the CAEP neural generators.

Effect of Intensity

Roth et al. 1980, 1982 and 1984 in a series of studies concluded that P3 showed a marked intensity effect. It is said that P3 behaved like a exogenous component in that stimulus intensity was the most powerful variable in determining its amplitude. They also reported that P3 amplitude differed significantly among 3 intensity levels. However, amplitude of P3 did not vary with intensity in a linear manner. Papanicolau et al. 1985 found that latency of P3 appeared to be linearly related to stimulus intensity i.e., latency increased systematically as the stimulus intensity decreased.

Billings, Tremblay et al. in 2007 studied the Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. P1, N1, P2, and N2 cortical evoked potentials were recorded in sound field from 13 normal-hearing young adults in response to a 1000-Hz tone presented at seven stimulus intensity levels. Peak amplitudes increased and peak latencies decreased with increasing intensity for unaided and aided conditions. However, there was no significant effect of amplification on latencies or amplitudes. Taken together, these results demonstrate that 20 dB of hearing aid gain affects neural responses differently than 20 dB of stimulus intensity change. Hearing aid signal processing is discussed as a possible contributor to these results. This study demonstrates (1) the importance of controlling for stimulus intensity when evoking responses in aided conditions, and (2) the need to better understand the interaction between the hearing aid and the CAS.

Purdy et al.(2013) studied the effects of stimulus level on speech-evoked obligatory cortical auditory evoked potentials in infants with normal hearing for a low frequency (/m/) and high frequency (/t/) speech sound. CAEPs were recorded for two natural speech tokens, /m/ and /t/. Participants were 16 infants aged 3-8months with no risk factors for hearing

impairment, no parental concern regarding hearing. They were also having normal tympanogram and present otoacoustic emissions. Infants were either tested at levels of 30, 50, and 70dB SPL or at 40, 60, and 80dB SPL, in counterbalanced order.

Input-output functions showed different effects of increasing sound level between stimuli. There were minimal changes in latency with increase in level for /t/. For /m/, there were approximately 50-60ms latency increases at soft compared to loud levels. Amplitudes saturated at moderate-high levels (60-80dB SPL) for both stimuli. They concluded that Infants' CAEP input-output functions differ for /t/ versus /m/ and differ from those previously reported for adults for other stimuli. Effects of stimulus and level on CAEPs should be considered when using CAEPs for hearing aid or cochlear implant evaluation in infants.

Speech-evoked CAEPs provide an objective measure of central auditory processing. Possible differences in CAEP growth between infants and adults suggest developmental effects on intensity coding by the auditory cortex. Koravand, Jutras, and Lassonde (2012) found larger P1 amplitude and shorter N2 latency in children with hearing loss comparatively to children with normal hearing. The findings indicate that the level of presentation could affect differently the two components

Effect of Stimuli

Several researchers used different stimuli to elicit CAEP responses such as click, tone pips, tone bursts, speech stimuli (Davis et al., 1966;Rapin et al., 1966).Further, They observed that rise time and fall time of 20mswere more effective in eliciting cortical potential. Most commonly used stimuli for clinical assessment are the long duration stimuli. The use of long duration stimuli reduces the spread of cochlear excitation and maintains frequency specificity (Hyde, 1997). It is well known that tonal stimuli gives limited

information about perception of speech. Hence, speech stimuli are preferred for neural representation of stimuli at cortical levels.

Late latency responses in normal hearing individuals

Tremblay and Kraus (2002) studied the effect of auditory training on cortical neural activity. Seven normal-hearing young adults were trained to identify two synthetic speech variants of the syllable /ba/. As subjects learned to correctly identify the two stimuli, changes in P1, N1, and P2 amplitudes were observed. Of particular interest is that P1, N1, and P2 components of the N1-P2 complex responded differently to listening training. That is, significant changes in P1 and N1 amplitude were recorded over the right but not the left hemisphere. In contrast, increases in P2 were observed bilaterally. Their results indicated that training-related changes in neural activity are reflected in far-field aggregate neural responses and that distinct patterns of neural change, perhaps reflecting hemispheric specialization, likely represent different aspects of auditory function.

Peggy, Diane and David, 2005 studied the effects of sensorineural hearing loss and personal hearing aids on cortical event related potential and behavioural measures of speech sound processing. They recorded Cortical ERPs /ba/ and /da/ speech stimuli presented at 65 and 80 dBSPL from 20 normal-hearing adults and 14 adults with sensorineural hearing losses. The degree of sensorineural impairment at 1000 to 2000 Hz ranged from moderate losses (50-74 dB HL) to severe-profound losses (75-120 dB HL). The speech stimuli were presented in an oddball paradigm and cortical ERPs were recorded in both active and passive listening conditions at both stimulus intensities.

The adults with hearing impairments were tested in the unaided and aided conditions at each intensity level. Results indicated that the use of personal hearing aids substantially improved the detectability of all the cortical ERPs and behavioural d-prime performance scores at both stimulus intensities. This was especially true for individuals with severe-profound hearing losses. At 65 dB SPL, mean ERP amplitudes and d-prime sensitivity scores were all significantly higher or better in the aided versus unaided condition. At 80 dB SPL, only the N1 amplitudes and d-prime sensitivity scores were significantly better in the aided condition.

Even though the majority of the hearing-impaired subjects showed increased amplitudes, decreased latencies, and better waveform morphology in the aided condition, the amount of response change (improvements) seen in these measures showed considerable variability across subjects. When compared to the responses obtained from the normal-hearing subjects, both hearing-impaired groups had significantly prolonged e latencies at both intensity levels.

They concluded that hearing-impaired individual brains process speech stimuli with greater accuracy and in a more effective manner when these individuals use their personal hearing aids. This is especially true at the lower stimulus intensity. The effects of sensorineural hearing loss and personal hearing aids on cortical ERPs and behavioral measures of discrimination are dependent upon the degree of sensorineural loss, the intensity of the stimuli and the level of cortical auditory processing that the response measure is assessing.

Reliability of Late Latency responses

Tremblay et al in 2003 studied the test-retest reliability of cortical evoked potentials using naturally produced speech sounds. They obtained auditory evoked potentials from

seven normal-hearing young adults in response to four naturally produced speech tokens (/bi/, /pi/, /ʃi/, and /si/). Stimuli were tokens from the standardized UCLA version of the Nonsense Syllable Test (NST) (Dubno & Schaefer, 1992). Using a repeated measures design, subjects were tested and then retested within an 8-day period. Results indicated that auditory cortical evoked potentials elicited by naturally produced speech sounds were reliably recorded in individuals. Also, naturally produced speech tokens, representing different acoustic cues, evoked distinct neural response patterns.

They concluded that cortical evoked potentials elicited by naturally produced speech sounds can be reliably recorded in individuals. Naturally produced speech tokens, representing different acoustic cues, evoke distinct neural response patterns. Given the reliability of the response, this work has potential application to the study of neural processing of speech in individuals with communication disorders as well as changes over time after various types of auditory rehabilitation.

METHOD

Participants

Twenty four children (40 ears) with normal hearing in the age ranges of 3 to 9 years were selected for the study. Further, they were divided into three different age groups (3 to 5 years, 5 to 7 years, & 7 to 9 years). There were only 5 children (9 ears) in the age range of 3-5 years; 9 children (13 ears) in the age range of 5-7 years; and 10 children (18 ears) in the age range of 7-9 years participated in the study.

Subject inclusion criteria

- Participants were having hearing sensitivity within normal limits (≤ 15 dB HL) for octaves frequencies from 250 to 8000 Hz and/or presence of wave V in auditory brainstem response at 30 dBnHL.
- Normal middle ear functioning as indicated by immittance evaluation.
- No history of otologic and neurologic problems and no illness at the day of testing.
- Participants were excluded from the study if they were found to have clinically abnormal click-evoked ABR findings and abnormal middle ear functioning.
- Retro-cochlear pathology was ruled out using ABR and OAEs.

Testing environment

All the behavioural as well as electrophysiological tests were carried out in a sound treated room where the noise levels will be as per the guidelines in ANSI S3.1 (1991).

Instrumentation

- Calibrated double channel clinical audiometer (Orbitor-922) was used for visual reinforcement audiometry (VRA) or Conditioned audiometry (using insert earphones), or Pure Tone Audiometry.
- Calibrated GSI-Tympstar Immittance meter was used for tympanometry and reflexometry.
- Biologic Navigator Pro EP (version 7.07) was used for ABR threshold estimation.
- HEARLab (version 1.0) system was used for recording of speech evoked cortical potentials.

Procedure

- Conditioned audiometry/ visual reinforcement audiometry/ pure tone audiometry were carried at octaves 250 to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction.
- Immittance audiometry was carried out with a probe frequency of 226 Hz. Further, ipsilateral and contralateral acoustic reflex thresholds were measured at 500, 1000, 2000, and 4000 Hz.
- The electrophysiological testing was carried out which include click-evoked ABR to verify normal hearing sensitivity of the participants wherever required. For ABR recording, client was seated in a reclining chair. The skin surface at the two mastoids (M1, M2) and forehead (Fz) was cleaned with skin abrasive, to obtain skin impedance of less than 5 kΩ for all electrodes. The electrodes were placed with the help of skin conduction paste and surgical plaster was used to secure them tightly in the respective places. Participants were instructed to relax and refrain from extraneous body movements to minimize artifacts. The testing was done monaurally.
- For speech evoked cortical potential recording, HEARlab (version 1.0) evoked potential system was used. The participant was seated at the test position with his/her head approximately 1 meter from the loudspeaker positioned at 0^0 azimuths. The child was encouraged to sit quietly in the test position using distractions such as age appropriate toys and silent movies. Stimulus was presented with a fixed inter-stimulus interval of 1125 ms

(offset to onset). The electrode sites were prepared using cotton applicator and electrode gel to obtain impedance <5 kΩ. Disposable self-adhesive button electrodes were used. This cortical potential recording system uses an automatic statistical detection procedure which does not require a subjective response interpretation from the operator. The system generated p-value was determined the presence and absence of response. Testing was done using default setting of the HEARlab system. There were three speech Stimuli at three different intensities were used to record cortical potential. The details protocol is mentioned in table 1.

Table 1: *Parameters for click evoked ABR and speech evoked cortical potential*

Parameters	Click evoked ABR	Speech evoked cortical potential
Stimulus	Click (100 µs duration)	/m/ (30 ms), /g/ (30 ms) and /t/ (30 ms)
Electrode Placement	Reference - M1 Active - Fz Ground- M2	Reference: M1/M2 Active: Cz Ground: Fz

Intensity	80 dB nHL	55 dB SPL, 65 dB SPL, and 75 dB SPL
Transducer	Insert earphones	Loudspeaker
Transducer Position	None	0 degree azimuth
Ear	Monaurally	Monaurally
Polarity	Alternating	Alternating
Filter setting	100 - 3000 Hz.	1-30 Hz
Repetition rate	30.1/sec	1.1/sec
Total no. of sweeps	2000	200
Impedance	< 5 kΩ	< 5 kΩ
No. of Channels	One	One
Analysis Time	10 Sec	500 ms

RESULTS AND DISCUSSION

The main aim of the study was to find out the effect of intensity and different stimuli on latency and amplitude measures of cortical potential in individuals with normal hearing for different age groups. To establish this goal, peak latency and amplitude of P1 and N1 waves were measured and analyzed further using below mentioned statistical tools.

Speech evoked cortical potential was successfully recorded from children with normal hearing sensitivity using three different speech stimuli (/m/,/t/, & /g/) at three different intensity (75 dBSPL,65 dB SPL& 55 dB SPL) levels. There were three different age groups (3-5 years, 5-7 years, & 7-9 years) of children in the age range of 3-9 years were selected for the study (figure 1).

To analyze the data collected from different age groups of children with normal hearing, descriptive statistics, repeated measure analysis of variance (ANOVA), and Bonferroni pair wise compassion were done. Descriptive statistics was done to find out mean and standard deviation (SD) for all the parameters for all the three age groups. Repeated measure ANOVA was done to see the effects of different ears, different speech stimuli and different intensity and their interaction. Further, Bonferroni pair wise comparison was done to check the differences between different speech stimuli as well as different intensity levels with reference to different age groups. In addition to that, non-parametric tests like Kruskal Wallis test was done for 7-9 years of age group children since the sample size was very less for wave P1 and N2.

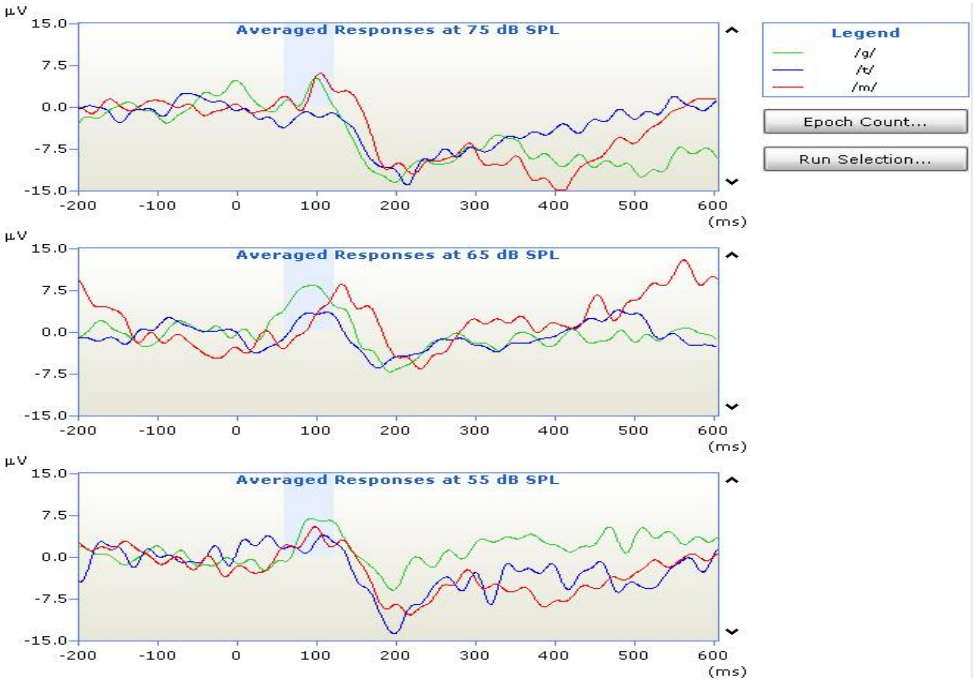

Figure 1: A sample waveform of speech evoked cortical potential at different intensity using different speech stimuli from a child with normal hearing

Table 2: Mean and Standard deviation (SD) for Latency and amplitude measure for 3-5 years age group children with normal hearing

Stimuli	Intensity (N)	Latency measure (ms)				Amplitude measure (µV)			
		P1		N1		P1		N1	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
/m/	75 (9)	101.22	13.43	198.22	20.98	5.51	3.71	-3.71	4.83
	65 (9)	98.44	18.74	164.11	53.64	3.98	2.92	-5.93	6.14
	55 (7)	97.85	16.49	181.71	40.28	3.72	3.66	0.46	5.11
/t/	75 (9)	82.22	18.72	197.55	18.55	3.38	4.54	-4.57	4.67
	65 (9)	83.22	11.68	170.55	27.02	3.94	1.96	-3.38	3.62
	55 (7)	75.00	37.27	180.85	36.44	4.00	3.85	-1.45	6.10
/g/	75 (9)	98.77	10.43	198.55	15.45	5.20	3.68	-5.72	5.15
	65 (9)	77.11	13.89	165.22	35.50	2.96	2.43	-3.67	5.00105
	55 (7)	88.14	19.58	170.71	29.07	4.20	2.82	0.44	6.13

N: Number of ears; SD: Standard deviation; µV: Microvolt; ms: millisecond

Speech evoked cortical potentials responses were successfully recorded from all the children with normal hearing for three different age groups. The wave P1 and N1 were marked based on visual inspection as offline analysis. Further, wave P2 and N2 was also observed for most of the children in the age range of 7-9 years. However, in other two younger age groups (3-5 years & 5-7 years), wave P2 and N2 were not traceable in most of the children.

Cortical potential outcomes for children with normal hearing (3-5 years)

Wave P1 latency

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave P1 latency [F (2, 46) = 0.780], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 3-5 years. Further, there were no interaction observed for different speech stimuli with ears [F (2, 46) = 1.536], $p>0.05$] for latency measures. In addition to that, different speech stimuli did not show significant interaction with different intensity [F (4, 46) =1.638], $p>0.05$] for individuals with normal hearing in the age range of 3-5 years.

Wave P1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave P1 amplitude [F (2, 46) = 0.647], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 3-5 years. Further, there were no interaction observed for different speech stimuli with ears [F (2, 46) = 0.610], $p>0.05$] for amplitude measures. In addition to that, amplitude measures of different speech stimuli did not show significant interaction for different intensity levels [F (4, 46) = 0.662], $p>0.05$] for individuals with normal hearing in the age range of 3-5 years.

Wave N1 latency

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave N1 latency [F (2, 42) = 0.088], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 3-5 years. Further, there were no interaction observed for different speech stimuli with ears [F (2, 42) = 0.139], $p>0.05$] for latency measures. In addition to that, different speech stimuli did not show significant interaction with different intensity [F (4, 42) = 0.268], $p>0.05$] for individuals with normal hearing in the age range of 3-5 years.

Wave N1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave N1 amplitude [F (2, 42) = 3.798], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 3-5 years. Further, there were interaction observed for different speech stimuli with ears [F (2, 42) = 4.224], $p<0.05$] for amplitude measures. However, amplitude measures of different speech stimuli did not show significant interaction for different intensity levels [F (4, 42) = 1.449], $p>0.05$] for individuals with normal hearing in the age range of 3-5 years.

Cortical potential outcomes for children with normal hearing (5-7 years)

The mean and standard deviation (SD) of latency and amplitude measures in individuals with normal hearing in the age range of 5-7 years are mentioned in table 2. Further, table 2 also mentioned about mean and SD for different speech stimuli (/m/, /t/, & /g/) at three different intensity levels (75 dB, 65 dB & 55 dB).

Table 3: Mean and standard deviation (SD) of latency and amplitude measures in the age range of 5-7 years

Stimuli	Intensity (N)	Latency measure (ms)				Amplitude measure (µV)			
		P1		N1		P1		N1	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
/m/	75 13)	95.23	12.65	199.15	36.55	6.93	4.55	-1.43	7.96
	65 11)	96.90	14.32	192.54	46.33	7.82	4.60	-6.40	8.96
	55 (6)	88.83	11.53	207.66	28.32	10.23	3.35	-.71	6.70
/t/	75 (13)	91.23	17.54	202.92	37.10	7.21	5.66	-6.05	6.90
	65 (11)	91.54	12.66	194.72	47.97	5.22	4.54	-6.51	6.92
	55 (6)	90.50	19.71	204.33	29.68	2.94	5.86	-2.25	8.64
/g/	75 (13)	92.53	15.46	209.00	39.02	6.65	4.72	-3.51	6.54
	65 (11)	96.27	16.95	176.81	48.49	6.53	4.57	-6.91	4.37
	55 (6)	89.33	12.54	201.33	32.79	8.06	3.77	-2.55	2.85

N: Number of ears; SD: Standard deviation; µV: Microvolt; ms: millisecond

Wave P1 latency

Repeated measures ANOVA shows significant main effect across different speech stimuli for wave P1 latency [F (2, 56) = 6.013], $p<0.05$] of cortical potentials in individuals with normal hearing in the age range of 5-7 years. Further, there were interaction observed for different speech stimuli with ears [F (2, 56) = 5.468], $p<0.05$] for latency measures. However, different speech stimuli did not show significant interaction with different intensity [F (4, 56) = 1.179], $p>0.05$]for individuals with normal hearing in the age range of 5-7 years.

Wave P1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave p1 amplitude [F (2, 56) = 0.596], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 5-7 years. Further, there were no interaction observed for different speech stimuli with ears [F (2, 56) =1.587], $p>0.05$] for amplitude measures. In addition to that, amplitude measures of different speech stimuli did not show significant interaction for different intensity levels [F (4, 56) = 2.405], $p>0.05$]for individuals with normal hearing in the age range of 5-7 years.

Wave N1 latency

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave N1 latency [F (2, 52) = 1.377], $p>0.05$]of cortical potentials in individuals with normal hearing in the age range of 5-7 years. Further, there were no interaction observed for different speech stimuli with ears [F (2, 52) = 0.921], $p>0.05$]for latency measures. In addition to that, different speech stimuli did not show significant interaction with different intensity [F (4, 52) = 2.482], $p>0.05$] for individuals with normal hearing in the age range of 5-7 years.

Wave N1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave N1 amplitude [F (2, 52) = 0.157], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 5-7 years. Further, there were interaction observed for different speech stimuli with ears [F (2, 52) = 0.181], $p>0.05$]for amplitude measures. However, amplitude measures of different speech stimuli did not show significant interaction for different intensity levels [F (4, 52) = 0.406], $p>0.05$] for individuals with normal hearing in the age range of 5-7 years.

Cortical potential outcomes for children with normal hearing (7-9 years)

While recording CAEPs in 7-9 years of age group children with normal hearing, it was observed that there were most of children whom early peaks were traceable i.e. P1,and N1 (table 4). However, there were children whom later components (P2& N2) could be measured. But since sample size was very small for wave P2 and N2, non-parametric test (Kruskal Wallis test) was done to find out the differences noticed with reference to intensity as well as for different speech stimuli (table 5).

Stimuli	**Intensity**	**Latency measure (ms)**		**Amplitude measure (µV)**	
	(N)	**P1**	**N1**	**P1**	**N1**

		Mean	SD	Mean	SD	Mean	SD	Mean	SD
	75 18)	77.94	11.58	149.27	49.49	2.47	2.27	-1.63	7.55
/m/	65 (15)	80.60	12.02	147.93	43.96	3.69	2.62	-1.95	5.93
	55 (16)	83.56	13.60	157.43	45.97	4.59	4.11	-.38	5.11
	75 (9)	81.77	12.82	151.38	45.56	2.76	2.29	-2.21	6.12
/t/	65 (9)	74.73	15.31	142.46	38.58	3.94	2.07	-.37	4.82
	55 (7)	82.75	17.77	152.06	38.47	3.07	3.73	-1.75	5.62
	75 (9)	74.22	10.38	148.16	44.86	3.47	1.65	-3.16	4.22
/g/	65 (9)	79.73	14.35	149.53	47.81	4.84	2.59	.30	4.82
	55 (7)	85.00	20.31	155.75	52.31	2.21	4.12	-1.88	4.92

Table 4: Mean and standard deviation (SD) of latency and amplitude measures in the age range of 7-9 years (wave P1 & N1)

Wave P1 latency

Repeated measures ANOVA shows significant main effect across different speech stimuli for wave P1 latency [F (2, 94) = 0.012], $p>0.05$]of cortical potentials in individuals with normal hearing in the age range of 7-9 years. Further, there were interaction observed for different speech stimuli with ears [F (2, 94) = 0.40], $p>0.05$] for latency measures. However, different speech stimuli did not show significant interaction with different intensity [F (4, 94) =1.891], $p>0.05$] for individuals with normal hearing in the age range of 7-9 years.

Wave P1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave P1 amplitude [F (2, 52) = 1.377], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 7-9 years. Further, there were no

interaction observed for different speech stimuli with ears [F (2, 52) = 0.921], $p>0.05$] for amplitude measures. In addition to that, different speech stimuli did not show significant interaction with different intensity [F (4, 92) = 3.386], $p<0.05$] for individuals with normal hearing in the age range of 7-9 years.

Table 5: Mean and standard deviation (SD) of latency and amplitude measures in the age range of 7-9 years (wave P2 & N2)

Stimuli	**Intensity**	**Latency measure (ms)**				**Amplitude measure (Microvolt)**			
		P2		**N2**		**P2**		**N2**	
		Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
/m/	75	167.20	66.66	248.50	13.67	4.79	5.02	-1.85	4.06

	65	190.80	37.18	251.33	12.74	1.32	1.97	-2.70	2.47
	55	174.50	20.85	256.60	20.31	3.28	2.14	-3.41	1.41
	75	176.85	35.70	225.83	11.10	2.54	2.27	-2.70	2.60
/t/	65	178.62	27.70	243.60	7.46	1.83	0.47	-5.10	2.06
	55	174.20	25.30	236.50	12.36	2.39	1.73	-4.68	1.86
	75	187.20	33.35	248.16	19.06	2.08	4.92	-3.09	2.36
/g/	65	180.87	30.74	239.25	14.61	2.64	2.53	-2.53	0.69
	55	170.00	34.78	257.00	13.85	2.12	1.23	-7.22	3.34

Wave N1 latency

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave N1 latency [F (2, 90) = 0.726], $p>0.05$]of cortical potentials in individuals with normal hearing in the age range of 7-9 years. Further, there were no interaction observed for different speech stimuli with ears [F (2, 90) = 1.144], $p>0.05$] for latency measures. In addition to that, different speech stimuli did not show significant interaction with different intensity [F (4, 90) = 0.312], $p>0.05$] for individuals with normal hearing in the age range of 7-9 years.

Wave N1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave N1 amplitude [F (2, 92) = 1.077], $p>0.05$] of cortical potentials in individuals with normal hearing in the age range of 7-9 years. Further, there were interaction observed for different speech stimuli with ears [F (2, 92) = 1.125], $p>0.05$] for amplitude measures. However, amplitude measures of different speech stimuli did not show significant

interaction for different intensity levels [F (4, 92) = 1.152], p>0.05] for individuals with normal hearing in the age range of 7-9years.

Wave P2 and N2 latency and amplitude measures

Kruskal Wallis show no significant difference with reference to intensity for both latency and amplitude measures of late latency responses except N2 latency for /t/ sound at 0.05 levels. Further, there were no significant differences for different speech stimuli for both latency and amplitude measures (table 5).

The observations of responses present or absent were validated using both automatic analysis as well as visual inspection technique. Golding et al (2009) and Carter et al (2010) compared the detection of CAEPs using automatic statistics and visual detection technique. Results showed that automatic statistical analysis is equally sensitive to the best of individual experienced examiners in differentiating CAEP responses from random noise. However, present study preferred visual inspection technique for detection of responses and measure latency and amplitude of different components of CAEPs. These responses were measured by two experienced audiologist having an exposure of cortical potential recording. Since there were few instances where at lower presentation level responses were marked as absent through visual techniques. However, automatic analysis techniques showed responses as present. Hence, visual identification of peaks was preferred in present study.

CAEPs recorded in children is quite different then adults in terms of morphology of waveforms, presence of different peaks and their latency and amplitude measures as reported by Wunderlich and Cone-Wesson (2006) in a article reviewed on CAEPs maturation in infant and children. In adults, CAEP responses are observed as classical multiphasic (P1-N1-P2-N2) with N1 and P2 being the dominant features whereas the infant waveform are typically biphasic and becoming more complex over a span of years. Earlier peaks, P1 and N1 are

observed in younger ages but less frequently evoked (Little et al., 1999; Molfese, 2000; Phlirch& Barnet, 1972; Rapin&Graziani, 1968) and relatively smaller then P2 and N2. It is also seen that along with the morphological changes, there are alteration in the scalp topography, peak amplitudes and latencies of different CAEP components.

Ceponiene et al. (2003) studied speech evoked CAEPs in 3 year olds children which showed a responses dominated by a large P1 around 130 ms followed by negative waves at 250 ms. In contrast another study done by Molfese and Hess (1978, 1988) in 3- 4 year old children observed a negative wave around 170-200 ms, a positive wave at 290-300 ms and a negative wave at 420-450 ms when inter stimulus interval used were 4-10 seconds.

Effect of Age on Latency and Amplitude Measures

Wave P1 latency

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave P1 latency [F (2, 212) = 1.229], $p>0.05$] of cortical potentials in individuals with normal hearing. Further, there were no interaction observed for different speech stimuli with reference to different intensity levels [F (2, 212) = 1.315], $p>0.05$]. However, different speech stimuli did show significant interaction with age [F (4, 212) =3.697], $p<0.05$] for individuals with normal hearing. Figure 1 shows the mean and 95% confidence interval (CI) for wave P1 latency for different age groups. From figure 1, it is very clear that for /m/, /t/ and /g/ stimuli, the P1 latency is lowest for older age groups (7-9 years) in comparison to the other two younger groups (3-5 years & 5-7 years).

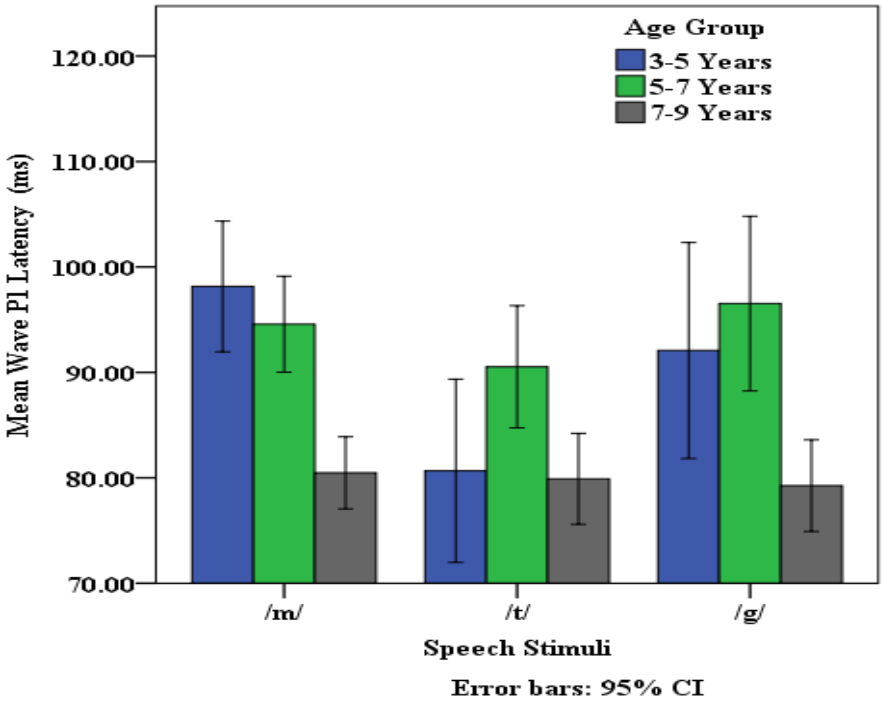

Figure 1: Mean and 95% confidence Interval (CI) for wave P1 latency for different age groups

Wave P1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave p1 amplitude [F (2, 210) = 0.936, p>0.05] of cortical potentials in individuals with normal hearing. Further, there were no interaction observed for different speech stimuli with reference to different intensity levels [F (2, 210) =2.781], p>0.05]for amplitude measures. In addition to that, different speech stimuli did not show significant interaction with age [F (4, 210) = 1.237, p>0.05] for individuals with normal hearing. Figure 2 shows the mean and 95% confidence interval (CI) for wave P1 amplitude for different age groups. From figure 2, it is very clear that for /m/, /t/ and /g/ stimuli, the P1 amplitude is highest for middle age groups (5-7 years) in comparison to the other two age groups (3-5 years & 7-9 years).

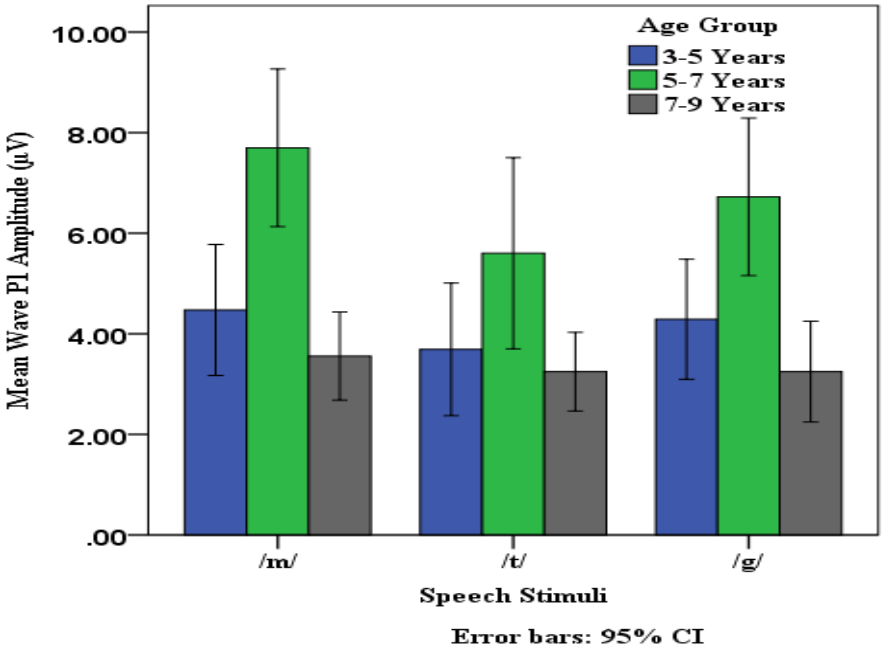

Figure 2: Mean and 95% confidence Interval (CI) for wave P1 amplitude for different age groups

Wave N1 latency

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave n1 latency [F (2, 200) = 0.447, p>0.05] of cortical potentials in individuals with normal hearing. Further, there were no interaction observed for different speech stimuli with reference to different intensity levels [F (2, 200) = 0.788, p>0.05] for latency measures. In addition to that, different speech stimuli did not show significant interaction with age [F(4, 200) = 0.406, p>0.05] for individuals with normal hearing. Figure 3 shows the mean and 95% confidence interval (CI) for wave N1 latency for different age groups. From figure 3, it is very clear that for /m/, /t/ and /g/ stimuli, the N1 latency is lowest (better) for older age groups (7-9 years) in comparison to the other two younger groups (3-5 years & 5-7 years).

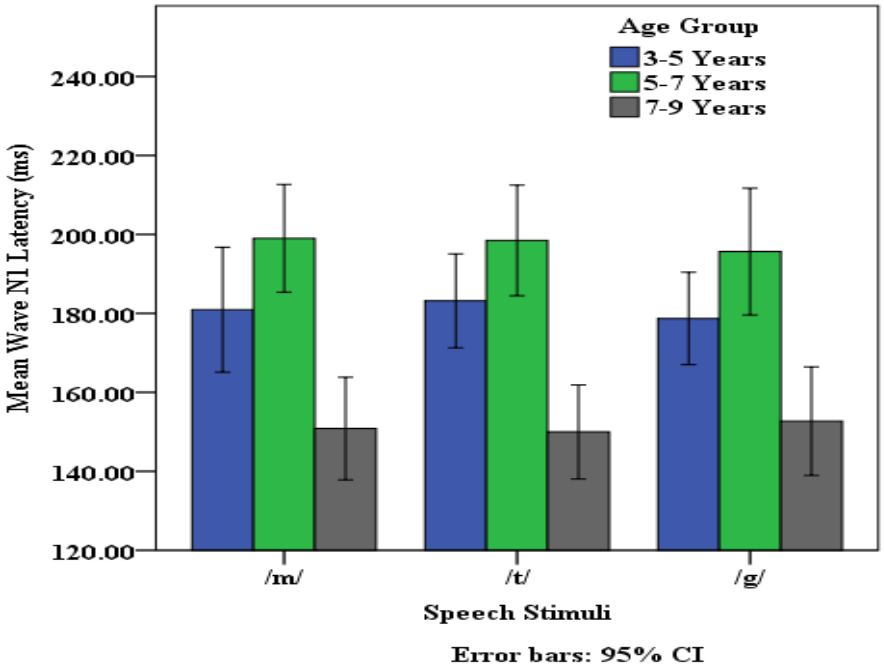

Figure 3: Mean and 95% confidence Interval (CI) for wave N1 latency for different age groups

Wave N1 amplitude

Repeated measures ANOVA shows no significant main effect across different speech stimuli for wave N1 amplitude [F (2, 202) = 0.408, p>0.05] of cortical potentials in individuals with normal hearing. Further, there were no interaction observed for different speech stimuli at different intensity levels [F (2, 202) = 0.119, *p*>0.05] for amplitude measures. In addition to that, amplitude measures of different speech stimuli also did not show significant interaction with age [F (4, 202) = 0.573, *p*>0.05] for individuals with normal hearing. Figure 4 shows the mean and 95% confidence interval (CI) for wave N1 amplitude for different age groups. From figure 4, it is very clear that for /m/, /t/ and /g/ stimuli, the n1 amplitude is lowest for older age groups (7-9 years) in comparison to the other two younger groups (3-5 years & 5-7 years).

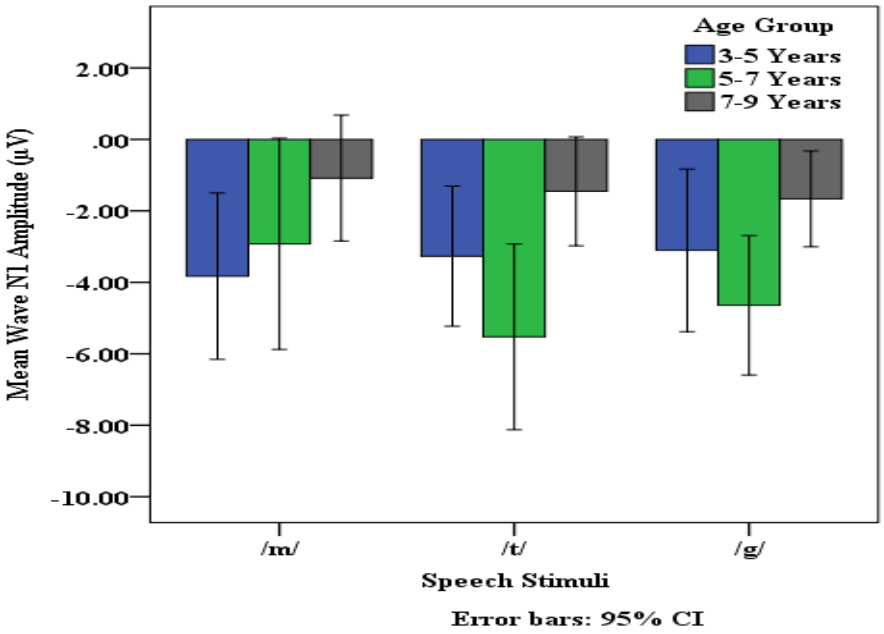

Figure 4: Mean and 95% confidence Interval (CI) for wave N1 amplitude for different age groups

In the present study, when three age groups were evaluated for speech evoked cortical potential, it was observed that for children in group 3-5 years and 5-7 years there was no significant difference on latency and amplitude of P1 and N1 waves with respect to varying intensity level, but in 7-9 years age group, there was significant difference found in latency and amplitude of P1 and N1 waves. Similar finding was observed by previous researchers (Gilley et al., 2005).

Study done by Gilley, Sharma, Dorman and Martin (2005) observed developmental changes in different components of CAEPs responses. They measured CAEPs in 50 normal hearing children in the age range of 3-12 years using speech stimuli with change in inter stimulus intervals. The results showed there is a significant change in the CAEPs as a function of age and stimulus rate. They observed wave P1 latency in the range of 96 to 76 ms and p1 amplitude in the range of 2.6 microvolt to 1.9 microvolt. Similarly wave N1 latency

was observed in the range of 119 to 106 ms and N1 amplitude in the range of 0.9 microvolt to -0.5 microvolt. Post hoc analysis showed no significant differences in P1 latency between two youngest age groups (3-4 and 5-6 years) at any inter stimulus interval conditions. However, P1 amplitude showed significant differences in these age groups.

Julia et al (2006) studied the maturation of the cortical auditory evoked potential in infants and young children. The participants in this experiment were 10 newborns (<7 days), 19 toddlers (13–41 months), 20 children (4–6 years) and 9 adults (18–45 years). They reported changes in latency and amplitude measures for CAEP components P1, N1, P2 and N2 as a function of participant age. They also noticed stimulus CAEP component latencies were relatively stable from birth to 6 years, but adults demonstrated significantly shorter latencies compared to infants and children. Components P1 and N2 decreased in amplitude, while components N1 and P2 increased in amplitude from birth to adulthood.

Previous studies provide conflict finding in terms of deductibility of the N1 responses in school age children. Sharma et al (1997) reported N1 responses in 61 % of children aged 6-7 years and 69% of children aged 10-12 years. Similarly, Cunnigham et al (2000) reported wave N1 responses in 45 % of children aged 5-7 years and 55% of children aged 11-12 years. However, we find N1 responses in all children with normal hearing in three different age groups (3-5 years, 5-7 years, & 7-9 years). The differences could be because of differences in methodology and research design in terms of methodology adopted such as the presentation of speech stimuli, and presentation levels. Hence, differences could be noticed because of those above mentioned factors.

Dun et al (2012) measured the sensitivity of CAEP detection in infants with hearing impairment in response to short speech (/m/, /g/ & /t/) sounds. They were evaluated for CAEP using automatic statistical detection paradigm at different sensation levels (0 to 20 dB) for infants in the age range of 8 months to 30 months. The results revealed that the presence

or absence of CAEPs can provide indication of the audibility of the speech sounds for infants with sensorineural hearing impairment. They did not observed significant difference between different speech stimuli for both latency and amplitude measures. Similar finding is observed in the present study. In addition to that, even no morphological CAEP differences were found between speech sounds. They said it could be because of low number of data points. P1 latency was significantly correlated with age by Dun et al (2012). In a similar line, Kushnerenko et al (2002) measured CAEPS in infants with normal hearing in the age range of 0-12 months with inter stimulus intervals (ISI) of 750 ms at 70 dB SPL using longer speech stimuli (200 ms). The results revealed wave P1 latency and amplitude was correlated with age, as age increases the latency were shorter and amplitude were higher. However, they noticed only wave N2 amplitude correlated well with age.

Chang et al (2012) measured the CAEPs in infants with sensorineural hearing impairment using speech stimuli at different intensity levels. The present study results shows higher sensation levels lead to greater number of present CAEP responses being detected. Further, more CAEPs responses were detected for aided then unaided condition. They also observed that /g/ and /t/ stimuli presented at higher estimated sensation levels evoked more statistically significant CAEP responses, while /m/ sounds was not loud enough to elicit CAEP responses.

The probable reason for changes in infant CAEP reflects changes in the intracortical synaptic organization of the auditory cortex, specifically as reported by Vaughan and Kurtzberg (1989), it is due to the development of synaptic connections on pyramidal cells in layer III of the cortex. In addition, Huttenlocher and Dabholkar (1997) observed that there is a rapid increase in synaptic density in the auditory cortex. Further, as children grow older, the formation of myelin along the axon increases the conduction velocity of a signal transmission, and consequently affects the timing of subsequent signal propagation (Sabatini

&Regehr, 1999; Salamy, 1978; Sanes et al., 2000). It is also presumed that since the latency and synchrony of the neural signal are dependent on myelination, the evoked potentials will reveal shorter latencies, increased amplitudes and more defined waveform morphology with maturation (Musiek et al., 1988).

Thus, it can be concluded that immature central auditory system, incomplete myelination and synaptogenesis will lead to longer neuronal refractory periods and lower cortical excitability (Surwillo, 1981). Moore and Guan (2001) reported a steady increase in axonal density until 5 years of age. Further, they also observed that auditory cortex begins to develop a more complex network of axons throughout the superficial layers and becomes adult like density by about 11 years of age.

There is few limitation with the present study in which HEARLab system was used to record speech evoked CAEPs. This system is having limitation in monitoring ocular EEG monitoring, because of single channel facility. Hence, artifact was not monitored for eye blinking. However, artifact rejection criteria were adopted to reject all epochs that exceeds a specific value, which shows excessive noise present in recording.

SUMMARY AND CONCLUSION

The present study was aimed to develop the normative in Indian population for speech evoked cortical potential in children with normal hearing in the age range of 3-9 years. Twenty four children (40 ears) with normal hearing were divided into three different age groups (3 to 5 years, 5 to 7 years, & 7 to 9 years). There were only 5 children (9 ears) in the age range of 3-5 years; 9 children (13 ears) in the age range of 5-7 years; and 10 children (18 ears) in the age range of 7-9 years participated in the study.

Speech evoked cortical potential was successfully recorded from children with normal hearing sensitivity using three different speech stimuli (/m/,/t/, & /g/) at three different intensity (75 dB SPL,65 dB SPL& 55 dB SPL) levels. To analyze the data collected from different age groups of children with normal hearing, descriptive statistics, repeated measure analysis of variance (ANOVA), Bonferroni pair wise compassion and Kruskal Wallis tests were done.

Repeated measure ANOVA did not show significant differences for wave P1 and N1 for latency and amplitude measures for children with normal hearing in the age range of 3-5 years. For 5-7 years of age group children with normal hearing, repeated measure ANOVA did not show significant differences for wave P1 and N1 for latency and amplitude measures except wave P1 latency at 0.05 levels. In 7-9 years of age group children with normal

hearing, repeated measure ANOVA did not show significant differences for wave P1 and N1 for latency and amplitude measures. Further, Kruskal Wallis test was done for wave P2 and N2 responses because of small sample size. The results showed no significant differences at different intensity levels. Hence, to conclude in this present study the differences with reference to intensity and speech stimuli was not reflected distinctly in terms of Latency and amplitude measures.

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