AIDED CORTICAL ASSESSMENT IN CHILDREN WITH SEVERE TO PROFOUND HEARING IMPAIRMENT (3 TO 6 YEARS)

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Register No: 12AUD011

A Dissertation in Part Fulfilment of Final Year

Master of Science (Audiology)

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ALL INDIA INSTITUTE OF SPEECH AND HEARING

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MAY, 2014

Dedicated to

My beloved Papa, Mummy, Sister& Brothers

For their endless love, support and encouragement

&

Almighty

CERTIFICATE

This is to certify that this dissertation entitled "**Aided cortical assessment in children with severe to profound hearing impairment (3 to 6 years)**" is the bonafide work submitted in part fulfillment for the degree of Master of Science (Audiology) of the student with Registration No. 12AUD011. 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.

Mysore

May, 2014

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CERTIFICATE

This is to certify that this dissertation entitled "**Aided cortical assessment in children with severe to profound hearing impairment (3 to 6 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.

Mysore

May, 2014

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DECLARATION

This dissertation entitled "**Aided cortical assessment in children with severe to profound hearing impairment (3 to 6 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.

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INTRODUCTION

Cortical Auditory evoked potentials (CAEPs) represent summed neural activity in the auditory cortex in response to sound (Carter, Dillon, Seymour, Seeto, & Dun, 2013). CAEPs can be classified as obligatory "or "discriminative". Discriminative potentials are evoked by a change from frequent 'standard' stimulus to an infrequent 'deviant' stimulus. Obligatory AEPs are evoked by repeated sounds such as clicks, brief tones, or speech stimuli. They are usually classified in terms of their latencies or, the time of occurrence after presentation of a stimulus. The obligatory CAEP is also called auditory late latency responses (ALLR) (Hall, 1992). The impact of sensorineural hearing loss is greater for the later evoked potentials that reflect higher level stimulus processing. AEPs reflect maturation of the human brain through change in their latency, amplitude and morphology (Eggermont, 1989; Courchesne, 1990).

A small number of stimuli, presented at a slow rate of 1 or 2 pulse per second are used to generate a cortical response and stimuli used are broadband clicks, tones and speech segments. Tonal stimuli with duration of 100 to 200 ms, can be used to generate an obligatory cortical response as a means of estimating auditory threshold in adults who are unable or unwilling to participate in normal behavioural testing since the 1960s (Davis, 1965; Coles & Mason, 1984; Hyde, Alberti, Matsumoto, & Yao-Li Li, 1986; Rickards, DeVidi, & McMahon, 1996; Wunderlich, Cone-Wesson, & Shepherd 2006). There are some evidence that CAEPs in infants evoked by speech phonemes differ in latency and morphology (Kurtzberg, 1989). CAEPs differences between speech stimuli are an indication of different underlying neural representation

of speech sounds and suggest that information needed to differentiate the stimuli is available to the listener (Purdy et al., 2005).

CAEPs consists of a series of positive and negative peaks (P1/N1 complex) occurring between 80 and 500 ms after stimulus onset. The typical adult response (i.e., over 20 years of age), consists of a dominant negative wave, known as N1, with a latency of 80 - 120 ms and N2 in the latency 180 to 200ms. This feature is preceded and followed by positive components in most cases i.e., P1 has a latency of 50 to 70 ms, and P2 has a latency of 150 - 200 ms (Davis, 1965).

The amplitudes and latencies of these CAEP components can vary substantially between and within adults depending on the level of alertness and/or heightened levels of background noise associated with restlessness. The average newborn infant CAEP is dominated by a series of positive peaks with a prominent peak at 200 to 300 msec after the introduction of the stimulus. There are however substantial individual differences in the shape of this response that makes response detection by human eye alone, very challenging (Rapin & Granziani, 1967). P1 and N1 components 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. P1 and N1 are generated by multiple temporally overlapping subcortical and cortical sources (Chen & Buchwald, 1986; Naatanen & Picton, 1987; Sharma, Kraus, McGeeb & Nicol, 1997). 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 (Sharma et al, 1997).

Cortical potentials are generated by multiple brain regions including primary auditory cortex, auditory association areas, frontal cortex and subcortical regions (Oates, Kurtzberg & Stapells, 2002) that mature at different rates. There are complex changes in the morphology, scalp distribution and amplitude and latency of the P1-N1- P2 waves with maturation (Cunningham, Nicol, Zecker & Kraus 2000; Ponton, Eggermont, Kwong & Don, 2000).

CAEP morphology is dependent on age, sleep state, attention, stimulus, presentation parameters, and electrode recording position. In awake and alert children up to the age around 6 years, a reliable CAEP can be recorded from the vertex at a rate of about one a second generally which consists of a positive peak ranging from about 250 ms (at birth) to 100 ms (in childhood), followed by a low-amplitude negative deflection ranging from 450-600 ms (at birth) to 200 ms (in childhood). The decrease of the latency is explained by the development of the auditory system over time and is also dependent on the duration a person has been exposed to sound. From around the 8^{th} year of life, the appearance of an extra negative deflection N1 separates the positive deflection into peaks P1 and P2. This change continues until adulthood, where the CAEP has a distinct P1-N1-P2-N2 pattern (Dun, Carter, & Dillon, 2012).

Applications of CAEP in threshold estimation are limited to difficult to test population (Korzack, Krutzberg & Stapells, 2005), as CAEP is affected by sleep and alertness, it is not widely used for threshold estimation. CAEPS have been used to provide functional measure of the benefit provided by hearing aids (Korzack, Krutzberg & Stapells, 2005). Recording CAEPs can provide evidence of speech detection at the cortical level in the auditory system. Robust CAEPs can be observed to verify the audibility of speech stimuli presented at conversational level in infants

and young children fitted with hearing aids (Kurtzberg, 1989; Steinschneider, Kurtzberg, & Vaughan, 1992).

An investigation by Hinduja, Kusari and Vanaja (2005) showed that auditory late latency responses of individuals with a hearing aid showed larger amplitude and shorter latency when the aided thresholds were within the speech spectrum than when they were not within the speech spectrum. A few studies show the amplitude and latency changes with auditory experience in children and adult with hearing impairment using amplification devices (Kraus, Smith, Reed, Stein, & Cartee, 1995; Purdy et al, 2001; Tremblay, Kelly & Kraus, 2002).

It would be possible to use CAEPs recording to measure the adequacy of amplification provided to children with hearing impairment to achieve listening skills. This research was designed to incorporate cortical responses in hearing aid benefit assessment in children with hearing impairment.

NEED FOR THE STUDY

From the series of research conducted at National Acoustic Laboratories (NAL), HEARLab system was developed for fitting and validating the hearing aids. To make the procedure more objective and to maintain uniformity in the analysis, statistical procedure was adopted in the system. Previous studies as mentioned earlier have been done mainly on age groups of large range which resulted in inter-subject variability and also effect of intensity has not been explored widely (Shruthi &Vanaja, 2007; Dun, Carter & Dillon, 2012; Golding et al., 2007; Wunderlich, Cone-Wesson & Shepherd, 2006).

CAEPs in 3 to 6 year old aided children have not been studied exclusively. Dun, Carter & Dillon (2012) studied sensitivity of CAEP is the age 8 to 30 months,

Golding et al (2007) studied relationship between obligatory CAEPs and functional measures in children of 8 weeks to 3 year, Shruthi and Vanaja (2007) studied CAEP in both aided and unaided condition in children with hearing impairment in the age range of 5-7 years and so forth. Hence there is a need to study aided speech evoked CAEPs in this age group.

The P1-N1-P2 evoked neural response pattern is influenced by the acoustic content of the evoking signal. However hearing aids alter the content of the signal. This makes hearing aids a separate source of variables. There is a need to know more about the representation of signals processed by hearing aid at the cortical level. Hence it is essential to study the CAEPs in aided conditions. Further, three different speech stimuli, /ma/ which has spectral energy in low frequency, /ga/ which has energy in mid frequency and /ta/ which has energy in high frequency, will be used in this study in both aided and unaided conditions. In addition to that, different intensities will be considered to study the signal processing at threshold and suprathreshold levels.

The present study focuses on CAEPs elicited by consonant-vowel syllables to examine normal maturation of the central auditory areas in aided children with severe to profound hearing loss. Understanding the normal patterns of maturation of AEPs evoked by speech sounds may aid in the development of electrophysiological techniques for diagnosing abnormal central auditory maturation coincident with speech, language and hearing impairments.

AIM OF THE STUDY

The aim of the study was to obtain the aided CAEPs in children with severe to profound hearing impairment in the age range of 3 to 6 years.

OBJECTIVE OF THE STUDY

- To determine the latency and amplitude of CAEPs at different intensities (55, 65 and 75 dB SPL) in aided conditions in children with severe to profound hearing impairment.
- To determine the effect of speech sounds i.e. /m/, /g/, and /t/ on latency and amplitude of CAEPs in aided conditions.

REVIEW OF LITERATURE

CAEPs are measures of the brain's response to sensory stimuli that reflect synchronous neural activity along the auditory centers of the cortical pathway. The adult response (i.e., over 20 years of age), consists of a dominant negative wave, known as N1, with a latency of 80 - 120 ms and N2 in the latency 180 to 200 ms. This feature is preceded and followed by positive components in most cases (i.e., P1 has a latency of 50 to 70 ms, and P2 has a latency of 150 - 200 ms (Davis, 1965). The P1 component of cortical auditory evoked potential has been established as a biomarker for assessing the maturation of the central auditory system in children (Sharma et al., 2005).

The auditory late response has multiple generators in primary and secondary auditory cortices, including Heschl's gyrus, the planum temporale, and the superior temporal lobe (Naatanen & Picton, 1987). Latency and amplitude characteristics of the response have been shown to differ based on stimulus parameters. Several studies have demonstrated increased N1-P2 latency and decreased amplitude with decreased stimulus intensity (Billings, Tremblay, Souza, & Binns, 2007; Rapin & Graziani, 1967). The interval between stimulus presentations has also been shown to singularly affect response amplitudes, with longer interstimulus intervals corresponding to increased response amplitudes (Friesen & Picton, 2010; Tremblay, Billings, & Rohila, 2004).

Maturation of cortical auditory evoked potential in infants and young children

Wunderlich, Cone-Wesson & Shepherd, (2006) studied the maturation of the cortical auditory evoked potential in infants and young children. The participants in this study 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. Results showed 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. 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. They concluded frequency-related differences in component amplitude were apparent at all ages reflecting development of tonotopic organisation of the CAEP neural generators.

Sharma et al. (1997) studied developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. Synthesized consonant-vowel syllable /ba/ was used to elicit cortical AEPs in 86 children with normal hearing in the age range of 6 to 15 years and ten normal adults. The adult response consists of a prominent negativity (N1) at about 100 ms, preceded by a smaller P1 component at about 50 ms. In contrast, the children response is characterized by a large P1 response at about 100 ms and decreases significantly by 20 years of age. In children P1 is followed by N1b at 200ms and in older children earlier N1a is seen which decrease with age. Results show that child P1 develops systematically into the adult response and maturational changes in the central auditory system are complex and extend well into the second decade of life.

Maturation of CAEP in normal hearing infants, children and adults

Kraus et al. (1993) studied speech evoked cortical potentials in children (7-11 years) and compared responses to 10 adults. Responses were elicited using

synthesized stimulus /da/ and /ga/ with duration of 90 ms which consisted of 5 formants and differed in the onset frequency of second and third formant. The well-defined N1/P2 complex of adult response was not found in children. Peaks P1, N1 and P2 differed morphologically in children and adults. P1 and N1 latencies were longer and P2 amplitude was smaller in the children. P1 tends to dominate the P1/N1 complex, N1 latency is less well defined and P2 is smaller in children as compared to adults. They concluded latency and morphology of p1, N1 and P2 may provide measure of maturation of central pathways.

Purdy et al. 2005 studied obligatory CAEPs to speech and tonal stimuli in 20 infants (3-7 months) and 14 adults with normal hearing. The tonal and speech stimuli used were 500,1k, 2k and 4k Hz tone bursts and /t/, /k/, /d/ and /g/ speech sounds respectively in adults and presented at 65 dB SPL. Infants were tested with 500 Hz and 2 kHz tonal stimuli and, /g/, /t/ and /m/ speech stimuli that was included to achieve greater spectral contrast across speech stimuli. Adult waveforms showed the well-documented P1, N1 and P2 peaks that occurred at 57, 106, and 198 ms, on average, across stimuli and electrode montages. However Infant waveforms showed a broad positivity “P1” at 202 ms following by a late negativity “Nl” at 367 ms, on average. They concluded that CAEPs can be recorded reliably to a range of speech and tonal stimuli in both infants and adults with normal hearing.

Cone and Whitaker (2013) studied the dynamics of infant cortical auditory evoked potentials for tone and speech tokens. CAEPs were measured for 36 infants, between the ages of 4 and 12 months and 9 young adults (19-24 years) with normal hearing. First, CAEPs amplitude and latency input-output functions were obtained for 4 tone bursts and 7 speech tokens. The tone bursts stimuli were 50 ms duration tokens of pure tones at 0.5, 1.0, 2.0 and 4.0 kHz. Similarly the speech sound tokens, (/a/, /i/,

/o/, /u/, /m/, /s/, and /ʃ/), were created from natural speech samples with 50 ms in duration. CAEPs were obtained for tone burst and speech token stimuli at 10 dB level decrements in descending order from 70 dB SPL (All CAEP tests were completed while the infants were awake and engaged in quiet play). For the second experiment, observer-based psychophysical methods were used to establish perceptual threshold for the same speech sound and tone tokens. The results indicated that CAEP component latencies in infants were prolonged by 100-150 ms in comparison to adults. CAEP latency-intensity input output functions were steeper in infants compared to adults. CAEP amplitude growth functions with respect to stimulus SPL are adult-like at this age, particularly for the earliest component (P1-N1). Infant perceptual thresholds were elevated with respect to those found in adults. Furthermore, perceptual thresholds were higher, on average, than levels at which CAEPs could be obtained. When CAEP amplitudes were plotted with respect to perceptual threshold (dB SL), the infant CAEP amplitude growth slopes were steeper than in adults with normal hearing.

They concluded that although CAEP latencies indicate immaturity in neural transmission at the level of the cortex, amplitude growth with respect to stimulus SPL is adult-like at this age, particularly for the earliest component, P1-N1. It is possible to use tonal or speech sound tokens to evoke CAEPs in an awake, passively alert infant, and thus determine whether these sounds activate the auditory cortex. This could be beneficial in the verification of hearing aid or cochlear implant benefit.

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.

Different Stimuli used to measures CAEPs

There are different types of stimuli that can evoke CAEPs. All differ in their spectral and temporal parameters. The stimuli used can be speech, clicks or tones.

Tones: Tonal stimuli have been used to elicit CAEPs. (Davis, Bowers & Harish, 1968). Rise/fall time of greater than 20 ms and duration of hundreds of milliseconds are effective in eliciting the CAEP and low frequency tones elicit larger CAEPs than high frequency tones (Jacobson, Lombardi, Gibbens, Ahmad, & Newman, 1992).

Clicks: clicks elicit a neural response from a large number of auditory neurons Synchronously because of its rapid onset and broadband spectrum so are likely to produce robust responses. Click corresponds to best region of hearing above 500 Hz so not possible to characterize the shape of hearing loss (Katz, Burkard & Medwetsky, 2002).

Toneburst: Tone burst with long rise/fall time and plateau time from 500 to 4000 Hz can be used for threshold estimation (Katz, Burkard & Medwetsky, 2002). Taguchi, Picton, Orpin & Goodman, (1969) used tone-bursts as stimuli to assess CAEPs in 220 sleeping newborn infants. They observed reliable CAEPs.

Speech: speech is complex time varying signal that are poorly approximated by non-speech stimuli such as click, tones, and noise bands. The naturally produced speech stimuli show good test-retest reliability when recorded from the same individual. Natural speech can be used to assess changes in neural activity over time after hearing aid fitting, cochlear implantation or auditory training (Tremblay, Friesen, Martin & Wright, 2003)

Cortical auditory evoked potential measures in Normal hearing children

CAEP responses amplitude increases in a linear fashion as stimulus intensity increases, whereas latency decreased over the same intensity range (Antinoro, Skinner & Jones, 1969). Latency for the N1 or P2 component changes relatively little as intensity increases, except at intensity levels very close to auditory threshold (Picton, Hillyard & Galambos, 1976). Studies show that largest amplitude increase occur within the first 20 to 30 dB above auditory thresholds, and then increase more gradually with increasing intensity level and reaches plateau above approximately 75 dB (Beagley & Knight, 1967). The trend of increasing CAEP amplitudes and decreasing latencies with increasing sensation level has been confirmed in other studies with adults and normally hearing infants (Ross, Lutkenhoner, Pantev & Hoke, 1999 & Taguchi et al., 1969).

A study by Purdy, Sharma, Munro and Morgan (2013) in 16 infants (3-8 months) using speech sounds /m/ and /t/ done at 30, 50 and 70 dB SPL showed robust cortical auditory evoked potential (CAEP) amplitudes at low stimulus levels in infants. The effect of stimulus level on CAEP latencies differed between speech stimuli. There were minimal changes in latency with increase in level for /t/ and for /m/, there were approximately 50–60ms latency increase at soft compared to loud levels. Amplitudes saturated at moderate–high levels (60–80 dB SPL) for both stimuli in Infants.

Carter, Golding, Dillion, and Seymour (2010) studied the effectiveness of an automated statistic versus experienced examiners in detecting the presence of infant CAEPs when stimuli (/m/ and /t/) were present in 87 infant. CAEPs were recorded to three sensation levels, 10, 20 and 30 dB relative to the behavioural thresholds and to non stimulus trials. Results showed that as the stimulus level increased, the sensitivity

index increased for both methods of response detection, but neither reached the maximum possible value with a sensation level of 30 dB.

Alain, Woods & Covarrubias (1997) showed that increasing the stimulus duration up to approximately 30 to 40 ms leads to increased N1 amplitude with minimal changes in amplitude. There is a significant reduction in CAEP threshold as a function of signal duration (Eddins & Peterson, 1999). Stimulus duration are variable in CAEP studies using naturally produced speech stimuli with the duration ranging from 300 ms (Ostroff et al., 1998) to 756 ms (Tremblay et al., 2003)

Golding et al. (2006) studied the effect of stimulus duration on CAEPs in normal hearing infants aged 3 to 7 months, using natural speech segment /m/ and /t/. Results showed no significant change in the latency of P1 with changes in duration, but minor increase in amplitude when duration was increased from short to medium length. They concluded that there was no clear advantage of using stimulus durations beyond 35 ms.

Generally, studies reporting an age-related decrease in N2 amplitude have used relatively long (>1.3 s) inter-stimulus- intervals (Enoki, Sanada, Yoshinaga, Oka & Ohtahara, 1993; Johnstone, Barry, Anderson, & Coyle, 1996). Whereas studies finding no change or a complex pattern used shorter (<1.0 s) inter stimulus intervals (Tonnquist-Uhle´N, Borg & Spens, 1995). Although N2 in children is not particularly sensitive to manipulation of inter stimulus intervals (Ceponiene, Cheour & Naatanen, 1998), the use of a long inter stimulus intervals may allow age-related changes in amplitude to become evident because of the greater difference between child and adult values. When inter stimulus intervals was longer than 1.2–2.4 s, an adult like N1m can be recorded in most children from 3 to 15 years (Paetau, Ahonen, Salonen & Sams, 1995).

Golding et al. (2006) studied the effect of interstimulus interval in ten normal-hearing infants, aged 3 to 7 months, using the natural speech segments /m/ and /t/. Results showed that as the ISI increased, P1 latency was constant but its amplitude increased nonlinearly for /t/ only. They concluded that for the selected speech stimuli there was no clear advantage in using stimulus durations beyond 35 ms and ISIs beyond 1125 ms in infant assessments.

Cortical auditory evoked potential measures in children with hearing impairment

Dun, Carter & Dillion (2012) studied sensitivity of CAEP detection for hearing impaired infants in response to short speech sounds. 25 sensorineural hearing impaired infants (8 to 30 months) were tested once, 18 aided and 7 unaided. Behavioural thresholds of speech stimuli /m/, /g/, and /t/ were determined using visual reinforcement orientation audiometry. Afterwards, the same speech stimuli were presented at 55, 65, and 75 dB sound pressure level, and CAEPs were recorded. An automatic statistical detection paradigm was used for CAEP detection. For sensation levels above 0, 10, and 20 dB respectively, detection sensitivities were equal to 72±10, 75±10, and 78±12%. In 79% of the cases, automatic detection P-value became smaller when the sensation level was increased by 10 dB.

They conclude if CAEP is present ($p<0.05$) in response to sound at conversational level, that means sound is stimulating the auditory cortex at that level (> 10 dB SL). If CAEP is absent ($p<0.05$), i.e. below 0 to 10 dB SL, statistical detection criterion will show no response is detected 95% of the time. If true sensation level were to be within the range of 0 to 10 dB SL, then the probability of detection of responses is intermediate. The trend of increasing CAEP amplitudes and decreasing latencies with increasing sensation level, has been confirmed by other studies (Suzuki, Taguchi & Yoda, 1979) with hearing impaired infants.

Dun, Carter & Dillion (2012) evaluated the amount of audibility in 18 young infants at 55, 65 and 75 dB SPL. Results showed higher sensation levels led to a greater number of present CAEP responses being detected. More CAEP waveforms were detected in the aided condition than in the unaided condition.

There is some evidence that CAEPs in infants evoked by different speech phonemes differ in latency and morphology (e.g., Kurtzberg, 1989). However, no significant differences in the group averages for amplitude and latency were found between the different speech sounds (/m/, /g/, and /t/) as reported by Dun et al (2012).

Polen (1984) studied normal hearing and sensory-neural hearing impaired subjects with phonemic stimuli and noted a trend toward reduced amplitude for all late components in the hearing impaired group and reasoned that the late components of the auditory ERP might be altered in the presence of sensory neural hearing impairment because sensory neural hearing impairment at high frequencies affects individual's ability to discriminate phonemes.

Waves N1 and P2 were not significantly reduced in amplitude. From all the components, P2 was most drastically reduced in amplitude due to sensory neural hearing impairment. Finally Polen, (1984) reported that, for all components, peak latencies were prolonged for the sensory neural group. However, the drawback of the study was that they made no attempt to match the subjects for the degree of hearing impairment.

Oates, Kurtzberg and Stapells (2002) studied the effects of sensorineural hearing loss on cortical event-related potential from 20 normal hearing adults and 20 adults who were hearing impaired (mild to severe- profound) using /ba/ and /da/ stimuli at 65 and 80 dB SPL. Results showed that scores were lower for listeners with

sensorineural hearing loss than for those with normal hearing. However, these differences in response strength were evident only for those listeners whose average hearing loss at 1000 to 2000 Hz exceeded 60 dB HL for the lower intensity stimuli and exceeded 75 dB HL for the higher intensity stimuli. The results indicate that latency measures are more sensitive indicators of the early effects of decreased audibility. Sensorineural hearing loss has a greater impact on higher level or "nonsensory" cortical processing in comparison with lower level or "sensory" cortical processing.

Purdy, Dillion, Katsch Storey & Sharma. (2002) evaluated hearing aids in 20 infants (Age range: 3 to 7 months) using cortical evoked potentials of stimuli /tae/, /gae/ and /mae/, tone bursts at 500 Hz and 2000 Hz presented via loudspeaker at 65 dB SPL. Results found by averaging across the subjects that different stimuli gave rise to cortical resonses with different peak amplitudes and/or latencies.

They also studied the cortical response change shape as the change– frequency response varies. 5 boys aged 6 – 9 years with bilateral moderate to severe hearing loss were the subjects for the study (With similar stimuli and method as the above 2 studies). Results indicated that for every one of the five subjects, an ANOVA showed that the shape of the cortical response depended on the stimulus and the filter setting. The P1 and the following negative (N2) appeared at 150 and 250 ms respectively which is slightly delayed in comparison to children of this age with normal hearing.

Sharma et al. (2005) studied the influence of a sensitive period on central auditory development in children with unilateral and bilateral cochlear implants. They examined the longitudinal development of the cortical auditory evoked potential (CAEP) in 21 children who were fitted with unilateral cochlear implants and in two

children who were fitted with bilateral cochlear implants either before age 3.5 years or after age 7 years.

Their results showed a fundamentally different pattern of development of CAEP morphology and P1 cortical response latency for early- and late-implanted children. Early-implanted children and one child who received bilateral implants by age 3.5 years showed rapid development in CAEP waveform morphology and P1 latency. Late-implanted children showed aberrant waveform morphology and significantly slower decreases in P1 latency post implantation. In the case of a child who received his first implant by age 3.5 years and his second implant after age 7 years, CAEP responses elicited by the second implant were similar to late-implanted children. The results are consistent with animal models of central auditory development after implantation and confirm the presence of a relatively brief sensitive period for central auditory development in young children.

Korczak, Kurtzberg & Stapells (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 dB SPL from 20 normal-hearing adults and 14 adults with moderate sensorineural hearing losses (50-74 dB HL) to severe-profound losses (75-120 dB).

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

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 aided RT latencies at both stimulus intensities and N2b latencies at the higher stimulus intensities. They concluded those hearing-impaired individuals' brains process speech stimuli with greater accuracy and in a more effective manner when these individuals use their personal hearing aids.

Billings, Tremblay, Souza and Binns, (2007) studied the Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. They examined the effects of stimulus presentation level on the physiological detection of sound in unaided and aided conditions.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 the importance of controlling for stimulus intensity when evoking responses in aided conditions, and the need to better understand the interaction between the hearing aid and the CAS.

Henkin, Paul, Kileny, Hildesheimer & Rabin (2008) investigated the effect of increasing acoustic-phonetic difficulty in children with cochlear implants (CI) by means of auditory event-related potentials (AERPs). AERPs were recorded from a group of ten 9- to 14-year-old prelingually deafened children who exhibited open-set speech recognition, using the Nucleus 22 CI for at least 5 years. AERPs were recorded in sound field while children were performing oddball discrimination tasks with increasing acoustic-phonetic demand. The tasks consisted pairs of naturally produced stimuli that differed by one phonetic feature: vowel place (/ki/ versus /ku/), vowel height (/ki/ versus /ke/), voicing (/ka/ versus /ga/), and place of articulation (/ka/ versus /ta/). Using a repeated measure design, the effect of increasing acoustic-phonetic difficulty on P3 latency, amplitude, and scalp distribution as well as on the simultaneously obtained behavioural measures, performance accuracy, and reaction time was evaluated.

Results indicated that AERPs elicited in the range of 350 msec post stimulus onset were contaminated by the CI stimulus artifact, thus enabling reliable identification of the P3 component only. Increasing acoustic-phonetic difficulty was manifested in all measures in a hierarchical manner: P3 latency and reaction time increased, whereas P3 amplitude and performance accuracy decreased. The correlations, however, between behavioural and electrophysiological measures were not significant. Further support for P3 sensitivity to increasing acoustic-phonetic demand was its absence in four of the 10 children, but only in the most difficult place of articulation task. P3 amplitude was maximal at the midline parietal cite, with equal amplitudes over the right and left scalp regardless of side of implant.

They concluded based on the results that the results underscore the significant value of the P3 potential as a sensitive neural index of speech-sound processing in

children with CI. The similar hierarchy of acoustic-phonetic demand manifested in both behavioural and electrophysiological measures suggests that speech perception performance relates to neurophysiologic responses at cortical levels of the auditory system. Thus, recording the P3 potential to distinct phonetic contrasts may be useful for studying accessibility and neural encoding at the cortical level in CI recipients.

In general, a child who receives stimulation via a cochlear implant within the first 3.5 years of life will have a P1 latency that enters the normal range within the first 6–8 months after implant activation (Sharma, Dorman & Spahr, 2002; Dorman, Sharma, Gilley, Martin & Roland, 2007). If the auditory system does not receive adequate stimulation within approximately 7 years after birth, it is likely that the higher order auditory cortex gets re-organized, CAEP latencies generally remain abnormal, and the overall chances for normal speech and language while using a cochlear implant decrease significantly (Sharma et al., 2002; Sharma et al., 2005; Kral et al, 2007).

Shruti and Vanaja (2007) studied the usefulness of cortical potential responses obtained for speech stimuli /i/, /m/. /sh/ at 65 dB SPL in the validation of appropriate hearing aid in 10 children with hearing impairment (5-7 years) and if the speech sounds produced significant differences in the CAEP peaks in 15 normal hearing children (5-7 years). The unaided responses were absent because of severe to profound hearing loss. In aided condition it was observed that /i/ had shortest latency followed by /m/ and /sh/ and there was significant difference between /i/ and /sh/ and /i/ and /m/ for rank I hearing aid, but for rank II hearing aid there was no stimuli effect. They observed no significant difference for CAEP waves between normal and hearing impaired using hearing aid as children were aided for 2 years and were receiving auditory training regularly for both the hearing aid.

Apeksha and Devi (2010) studied how ALLR differed for different speech stimuli sounds (/ba/, /da/, /ga/) in 12 normal hearing individuals (20-50 years) 25 hearing impaired individuals (moderate, moderately-severe to severe hearing loss, 20-50 years). In aided condition /ga/ stimuli showed shortest latency followed by /ba/ and /da. There was significant difference for CAEP between normal, and hearing impaired groups, high significance for /ba/ and /da/ than for /ga/. They concluded that in spite of individuals wearing hearing aid according to the degree of hearing loss, the responses obtained were different from that of normal hearing, the hearing aid helps to compensate for hearing loss by amplifying sound but effectiveness depends on the central auditory system ability to integrate the spectral and temporal information by hearing aid (tremblay, 2006)

Hassaan (2011) studied aided cortical potentials in 10 young children with the history of regular hearing aid using for the correction of mild to moderately severe sensorineural hearing loss. Aided AECP consisting of P1, N1, P2 and N2 waves was measured for them using 500 and 4000 Hz tone bursts speech syllable /ga/ and /ba/. Results showed that the potential was traced in all subjects with different degrees of reproducibility of waves. It paralleled the speech recognition ability with reasonable correction values to the aided behavioural thresholds. They concluded the tracing of aided evoked cortical potential constituted a valuable tool for assessment of hearing aid benefit.

Koravand, Jutras, Lassonde (2011) examined the patterns of neural activity in the central auditory system in children with hearing loss (9-10 years). Results indicated a trend toward larger P1 amplitude, a significant reduction in amplitude, and latency of N2 in children with hearing loss compared to control. They concluded that

there is a maturational delays and/or deficits in central auditory processing in children with hearing loss, as indicated by the neurophysiological markers P1 and N2.

Chang, Dillon, Carter, Dun & Young, (2012) studied the relationship between cortical auditory evoked potential (CAEP) detection and estimated audibility in 18 infants with sensorineural hearing loss. They recorded CAEP to speech-based stimuli at three presentation levels (55, 65, or 75 dB SPL) under aided and unaided conditions. They later compared the results to the behavioural audiometric responses. The results indicated that the higher sensation levels led to a greater number of present CAEP responses being detected. More CAEP waveforms were detected in the aided condition than in the unaided condition. They concluded that the presence/absence of CAEP responses defined by the automatic statistical criterion was effective in showing whether increased sensation levels provided by amplification were sufficient to reach the cortex. This was clearly apparent from the significant increase in cortical detections when comparing unaided with aided testing.

Carter et al (2013) studied the Cortical Auditory-Evoked Potentials in adults in response to filtered speech stimuli to systematically examine the relationship between CAEP detection and the audibility of speech sounds (as measured behaviourally), when the listener is wearing a hearing aid fitted based on prescriptive targets. Aided thresholds were measured, and cortical responses evoked for 6 females and 4 males who had sensorineural hearing loss ranging from mild to severe-profound in degree. Participants' own hearing aids were replaced with a test hearing aid, with linear processing, during assessments. Three speech stimuli, (/m/, /t/, and /g/) were presented aided (monaurally, nontest ear occluded), free field, under three conditions at levels of 40, 50, and 60 dB SPL (measured for the unfiltered condition).

They obtained statistically significant CAEPs (p <05) for virtually every presentation where the behavioural sensation level was above10 dB, and for only 5% of occasions when the sensation level was negative. They concluded that CAEPs are a sensitive tool for directly evaluating the audibility of speech sounds. CAEP evaluation was found to be more accurate than audibility predictions, based on threshold and hearing aid response measures.

Reliability of Cortical Evoked Potentials

Tremblay et al. (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/). 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

The following method was adopted to meet the aim of the study.

Participants

Two groups i.e. control and clinical groups of participants were included in the study. Control group consisted of 13 children (20 ears) with normal hearing in the age range 3 to 6 years and Clinical group consisted of 13 children (20 ears) with severe to profound sensorineural hearing loss in the age range of 3 to 6 years.

Participant selection criteria for control group

Participants with hearing sensitivity within 15 dB HL for octaves frequencies between 250 to 8000 Hz for air conduction and from 250 Hz to 4000 Hz for bone conduction were taken. They had normal middle ear functioning as indicated by immittance evaluation without any history of otologic and neurologic problems. They were having no illness at the day of testing. Participants from the control group were excluded from the study who had clinically abnormal click-evoked ABR findings and abnormal middle ear functioning.

Participant selection criteria for Clinical group

Participants in the clinical group were Severe to profound sensorineural hearing loss children diagnosed based on ABR/ Conditioned Audiometry (table 1). They had normal middle ear functioning based on immittance evaluation without any history of any neurologic problems. Their aided audiogram was within the speech spectrum atleast up to 2 kHz. Retro cochlear pathology (auditory neuropathy) was ruled out based on ABR and TEOAE findings. Participant from the clinical group were

excluded from the study who had middle ear pathology and aided audiogram not within the speech spectrum at least up to 2 kHz.

Testing environment

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

Instrumentation

The below mentioned instruments were used to carry out the study

Calibrated double channel clinical audiometer (Orbitor-922) was used for Conditioned audiometry, unaided and aided audiogram. Calibrated GSI-Tympstar Immittance meter was used for tympanometry and reflexometry. Biologic Navigator Pro EP (version 7) was used for ABR threshold estimation and HEARLab system (Frye Electronics Tigard, USA, version 1.0) was used for recording CAEPs with and without hearing aids.

Procedure

The test was carried out in two phases for both clinical and control group.

Phase 1: Hearing evaluation and aided audiogram

Conditioned audiometry/visual reinforcement audiometry/ PTA was carried out at octaves 250 to 8 kHz for air conduction and between 250 Hz to 4000 Hz for bone conduction through modified Hughson Westlake procedure (Carhart & Jerger, 1959) for threshold estimation. Immittance audiometry was carried out with a probe frequency of 226 Hz. Ipsilateral and contralateral acoustic reflex thresholds was measured at 500, 1000, 2000, and 4000 Hz.

The electrophysiological testing included click-evoked ABR testing to verify normal hearing thresholds in control group and severe to profound sensorineural hearing loss in clinical group and also to rule out retro cochlear pathology. The client was made to sit 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. Subjects were instructed to relax and refrain from extraneous body movements to minimize artifacts. The testing was done monaurally.

Aided audiogram using conditioned audiometry or visual reinforcement audiometry from 250 Hz to 4 kHz was done for clinical group to verify that the aided thresholds are within speech spectrum at least up to 2 kHz. All the participants were using their own digital behind the ear hearing aids prescribed by Audiologist and their hearing aids were programmed with appropriate gain. Electroacoustic measurement was carried out to check the performance of the hearing aid before recording of cortical potential.

Phase 2: Cortical Auditory Evoked Potentials

CAEPs were recorded for control (unaided) and clinical group (aided) for all the participants who fulfilled the above criteria mentioned for control and clinical group. The client was made to sit at the test position with his/her head 1 meter from the loudspeaker positioned at 0 degree azimuth. The children were 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 lesser than 5 kΩ. Disposable self-adhesive button electrodes, placed at Fz (active), mastoid (reference) and Fpz (ground) were used.

The HEARLab system uses an automatic statistical detection procedure which does not require a subjective response interpretation from the operator. The system generated p-value will determine presence or absence of a response. Testing at a given intensity level was concluded immediately if HEARLab indicates that the p-value for stimulus being tested at that level is $p \leq 0.001$, provided that at least 30 accepted epochs are collected. Otherwise testing was concluded after acquisition of 200 epochs. Speech Stimulus was presented at different intensity levels (55 dB SPL, 65 dB SPL and 75 dB SPL). The protocols for aided cortical potentials were used, as mentioned in table 1.

Table 1: *Test Protocol for Click- Evoked ABR and ACA (aided cortical assessment)*

Parameters	Click evoked ABR	ACA
Stimulus	Click (100 µs duration)	/m/ (30 ms), /g/ (30 ms) and /t/ (30 ms)
Transducer	Insert ear phones	Loudspeaker at 0 degree azimuth
Electrode Placement	Active- Fz Reference- M1 Ground- M2	Active- Fz Reference- M1 Ground- M2
Intensity	80 dB SPL	55 dB SPL, 65 dB SPL, 75 dB SPL
Polarity	Alternating	Alternating
Filter setting	100 - 3000 Hz.	1- 30 Hz
Repetition rate	30.1/sec	1.1/sec
Number of sweeps	2000	200
Impedance	< 5k Ω	< 5k Ω
No. of Channels	One	One
Analysis Time	10 ms	500 ms
Amplification	100,000	50,000

Statistical Analysis

Statistical analysis was done using SPSS (Version 18). The collected data were tabulated in SPSS and tests performed were parametric and non-parametric tests. The Parametric test includes repeated measure ANOVA, one way ANOVA, Bonferroni pair wise comparison test and independent t-test. The non parametric test include Kruskal Wallis test for smaller sample size.

RESULTS AND DISCUSSION

The aim of the study was to investigate the effect of different speech sounds and intensity on the aided CAEP and comparison with the control group. Data of 20 severe to profound ears (12 left ears & 8 right ears in 5 males & 8 females) in aided condition and 20 normal hearing participants (10 each for left & right ear from 15 males & 5 females) were recorded. A total of 180 aided waveforms and 180 unaided (control group) waveforms were recorded.

The speech evoked CAEPs latencies and amplitudes of P1 and N1 were measured. The mean and standard deviation (SD) were calculated for control group and clinical group for P1 and N1 for /m/, /t/ and /g/ at 75 dB SPL, 65 dB SPL and 55 dB SPL. In the clinical group, ear effect, effect of intensity and effect of speech sounds was seen. Comparison within clinical group for different parameters as well as between two groups was also carried out. A sample waveform from both control and clinical group individuals are mentioned as figure 1 and 2 respectively.

The above parameters were measured using descriptive statistics, parametric tests like repeated measure analysis of variance (ANOVA), one way ANOVA, and independent t-test as well as non-parametric tests like Kruskal-Wallis test was used for responses obtained at 55 dB SPL since there were several missing data at this intensity level.

Latency and Amplitude measures in children with normal hearing (control group)

Descriptive statistics included mean and standard deviation for latency and amplitude measures from both ears for wave P1 and N1 across different speech stimuli (/m/, /t/, and /g/) and intensities (75 dB SPL, 65 dB SPL & 55 dB SPL) for

both clinical and control group. Table 1 shows mean and standard deviation for P1 and N1 for different speech stimuli at 75 dB SPL. From table 2, 3, and 4, it is observed that the standard deviation is little higher for N1 peak in comparison to P1 peak for latency measure. However, amplitude measure did not such differences with reference to P1 and N1 peaks. The higher SD probably reflects variability at different speech stimuli elicited at different intensities for control group (Table 2, 3 & 4).

Table 2: *Mean and standard deviation (SD) of latencies at 75 dB SPL for different speech sounds (/m/, /t/, /g/) in control group.*

stimuli	**Peaks**	**Ear**	**N**	**Latency (ms)**		**Amplitude (µv)**	
				Mean	**SD**	**mean**	**SD**
/m/	**P1**	LE	11	92.72	16.33	5.95	4.08
		RE	9	101.88	9.42	6.55	3.91
	N1	LE	11	195.72	32.59	-1.13	6.50
		RE	9	201.88	28.031	-5.59	5.01
/t/	**P1**	LE	11	84.18	15.72	7.18	6.28
		RE	9	88.77	19.77	3.64	4.37
	N1	LE	11	197.00	35.53	-2.57	5.44
		RE	9	202.88	24.42	-7.38	5.88
/g/	**P1**	LE	11	97.72	16.38	5.59	2.97
		RE	9	88.44	14.21	7.21	5.68
	N1	LE	11	196.36	36.52	-4.84	5.92
		RE	9	214.77	31.05	-4.48	4.48

SD: standard deviation; LE: left ear; RE: right ear; N: number of ears

Table 3: *The group means and SD of latencies at 65 dB SPL for different speech sounds (/m/, /t/, /g/) in control group.*

stimuli	Peaks	Ear	N	Latency (ms)		Amplitude (µv)	
				Mean	SD	mean	SD
/m/	P1	LE	11	98.18	18.23	6.10	3.51
		RE	7	94.42	13.85	4.03	3.91
	N1	LE	11	185.45	52.77	-6.35	7.18
		RE	7	180.42	43.86	-6.27	9.37
/t/	P1	LE	11	87.00	11.85	3.76	2.14
		RE	7	86.00	14.41	4.09	2.18
	N1	LE	11	181.54	41.57	-5.56	5.26
		RE	7	191.28	26.78	-6.02	6.67
/g/	P1	LE	11	85.18	14.95	3.07	3.51
		RE	7	83.14	16.43	5.64	3.42
	N1	LE	11	171.27	39.44	-5.35	7.16
		RE	7	172.71	35.67	-2.75	4.00

SD: standard deviation; LE: left ear; RE: right ear; N: number of ears

Table 4: Mean and standard deviation (SD) of latencies at 55 dB SPL for different speech sounds (/m/, /t/, /g/) in control group.

Stimuli	Peaks	Ear	N	Latency (ms)		Amplitude (µv)	
				Mean	SD	mean	SD
/m/	P1	LE	10	91.40	17.63	6.36	3.62
		RE	7	92.42	9.58	5.15	6.32
	N1	LE	10	194.90	39.11	1.81	5.93
		RE	7	183.14	40.42	-3.69	5.89
/t/	P1	LE	10	92.00	17.75	2.20	3.60
		RE	7	65.85	30.52	4.28	5.37
	N1	LE	10	193.40	39.12	-1.08	7.24
		RE	7	170.85	35.67	-4.96	7.19
/g/	P1	LE	10	90.40	17.99	7.04	3.10
		RE	7	83.28	17.93	3.22	4.13
	N1	LE	10	189.30	39.30	-.57	5.72
		RE	7	177.14	41.47	-.62	4.69

SD: standard deviation; LE: left ear; RE: right ear; N: number of ears

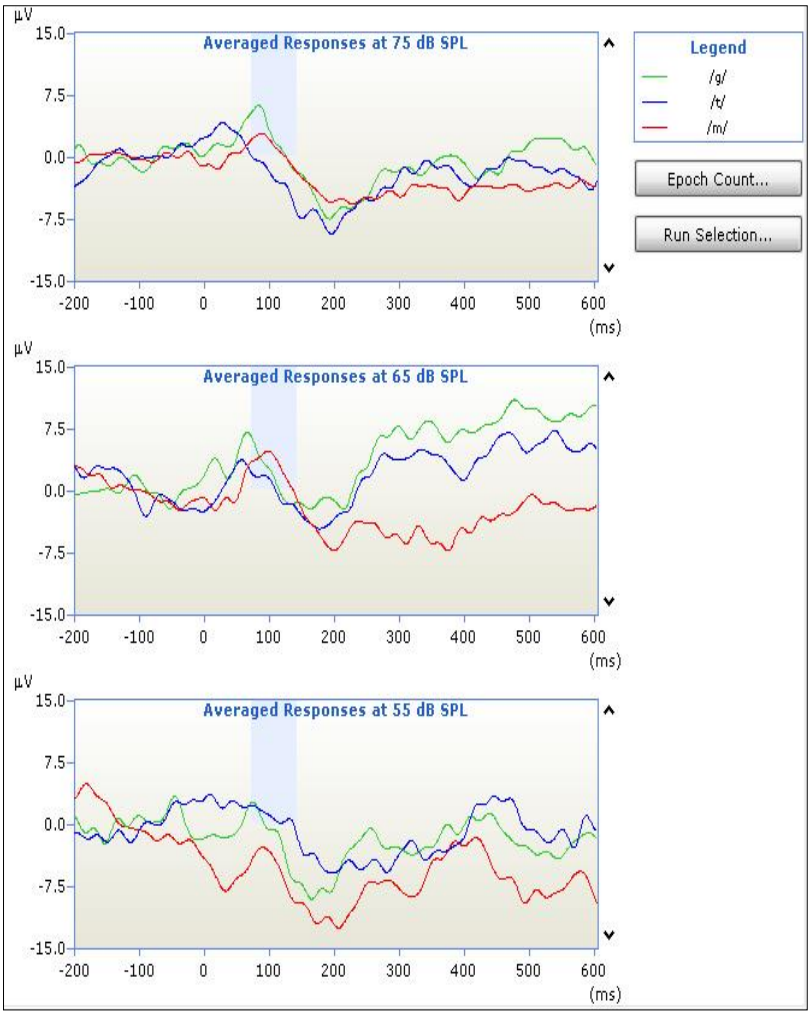

Figure 1: *A sample CAEP waveform at 75, 65 and 55 dB SPL for different speech stimuli in children with normal hearing.*

Latency and Amplitude measures in children with hearing impairment (clinical group)

Descriptive statistics were measured as mean and standard deviation (SD) at different intensities in children with hearing impairment using different speech stimuli. They were using hearing aids on regular basis fitted based on their degree of hearing impairment. The speech evoked cortical potential was measured as wave P1 and N1 for each ear at different intensities. From table 5, and 6, it is observed that the

standard deviations for latency measures in both ears are higher for wave N1 in comparison to wave P1. However, similar observation is not seen for amplitude measure in clinical group. Further, at 55 dB because of missing data, both ears were combined and descriptive statistics was obtained as wave P1 and N1 (Table 7).

Table 5: *Mean and standard deviation (SD) of latencies and amplitudes at 75 dB SPL for different speech sounds (/m/, /t/, /g/) in clinical group.*

Stimuli	Peaks	Ear	N	Latency (ms)		Amplitude (µv)	
				Mean	SD	Mean	SD
/m/	P1	LE	10	117.30	14.23	4.32	4.80
		RE	9	108.44	17.50	2.80	2.79
	N1	LE	10	219.40	63.36	-5.29	5.38
		RE	9	182.44	31.97	-5.36	3.33
/t/	P1	LE	10	108.90	8.82	2.08	2.55
		RE	9	107.66	15.69	3.43	3.43
	N1	LE	10	211.40	34.39	-4.79	4.26
		RE	9	200.88	27.08	-2.26	2.06
/g/	P1	LE	10	105.40	31.29	2.63	3.02
		RE	9	113.88	21.59	2.58	1.82
	N1	LE	10	170.50	55.42	-5.35	4.52
		RE	9	203.22	38.21	-4.30	3.01

SD: standard deviation; LE: left ear; RE: right ear; N: number of ears

Table 6: *Mean and standard deviation (SD) of latencies and amplitudes at 65 dB SPL for different speech sounds (/m/. /t/, /g/) in hearing impaired group*

Stimuli	Peaks	Ear	N	Latency (ms)		Amplitude (µv)	
				Mean	SD	Mean	SD
/m/	P1	LE	11	143.00	36.57	2.86	2.99
		RE	7	118.85	18.06	2.71	.96
	N1	LE	11	188.09	47.56	-6.36	3.13
		RE	7	203.85	39.25	-4.53	2.35
/t/	P1	LE	11	114.54	16.30	2.26	2.36
		RE	7	151.28	16.41	3.06	3.00
	N1	LE	11	219.09	32.10	-4.20	4.22
		RE	7	197.14	29.51	-3.61	2.33
/g/	P1	LE	11	103.27	33.34	3.76	3.80
		RE	7	122.42	26.44	2.01	3.11
	N1	LE	11	180.72	48.27	-4.60	3.90
		RE	7	207.57	48.00	-5.82	3.15

SD: standard deviation; LE: left ear; RE: right ear; N: number of ears

Table 7: *Kruskal Wallis outcome as mean and SD of latency at 55 dB SPL for different speech sounds in hearing impaired group.*

Stimuli	Peaks	N	Latency (ms)		amplitude(µv)	
			Mean	SD	Mean	SD
/m/	**P1**	14	160.14	43.82	0.83	3.89
	N1	14	235.00	30.08	-5.52	4.12
/t/	**P1**	13	124.30	36.85	-0.58	3.92
	N1	12	202.50	57.18	-4.93	4.71
/g/	**P1**	15	120.06	40.66	1.54	2.64
	N1	16	183.56	49.20	-4.61	3.81

SD: standard deviation; N: number of ears

The above outcomes of the study mentioned in table 5, 6 and 7 shows almost similar latencies and amplitudes in children with hearing impairment using hearing aids. Similar observation in terms of latencies and amplitudes were seen for different speech stimuli for both ears. The different speech stimuli were basically represents different frequency region such as low, mid, and high frequencies.

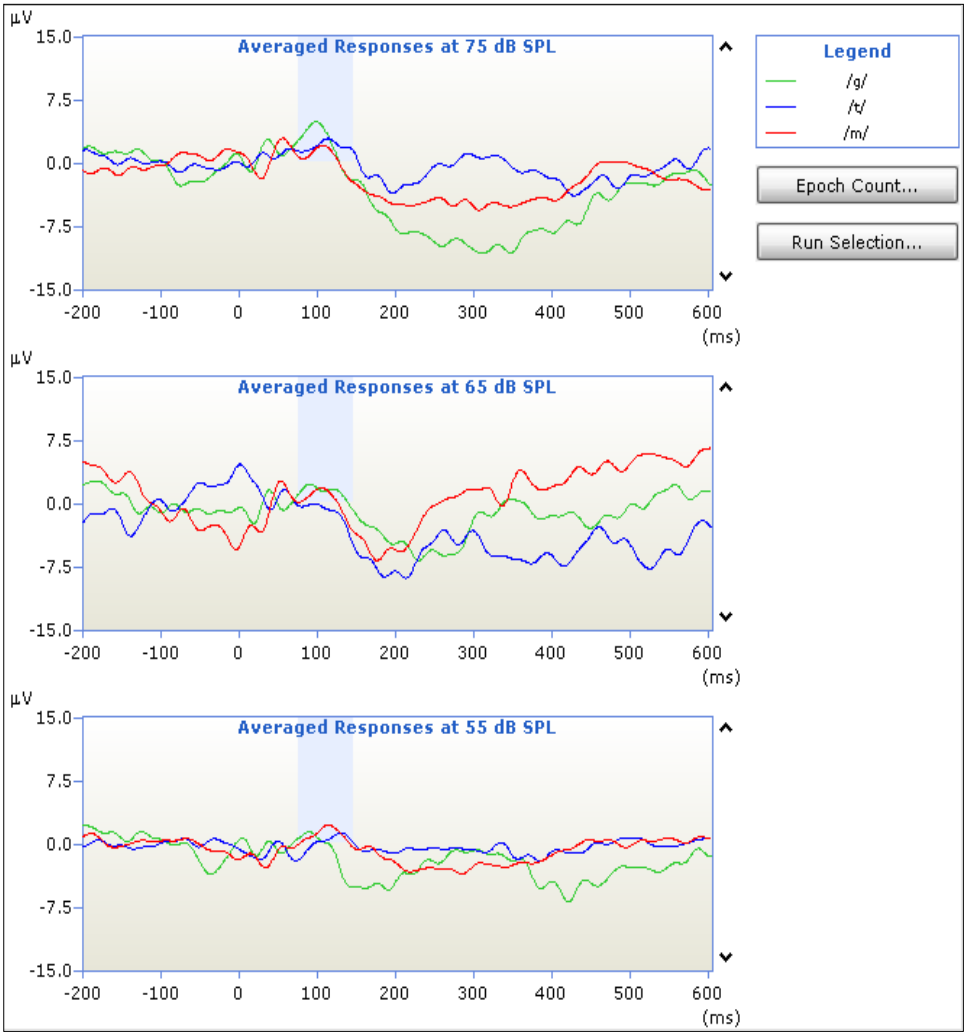

Figure 2: A *sample CAEP waveform at 75, 65 and 55 dB SPL for a severe-profound hearing impaired ear*

Table 8: *F values for latency and amplitude measures of* wave *P1 and N1 at 75 dB SPL and 65 dB SPL for clinical group*

	Intensity	Peaks	F-Value	p- value
Latency (ms)	**75 dB**	**P1**	F(2,34)=0.29	0.74
		N1	F(2,34)=0.75	0.48
	65 dB	**P1**	F(2,32)=3.89	**0.03***
		N1	F(2,34)=1.001	0.37
Amplitude (µv)	**75 dB**	**P1**	F(2,34)=0.48	0.60
		N1	F(2,34)=2.34	0.11
	65 dB	**P1**	F(2,32)=0.04	0.96
		N1	F(2,34)=2.19	0.12

** P<0.05*

Table 9: Kruskal Wallis outcomes for latency and amplitude measures of wave P1 and N1 *at 55 dB for different speech stimuli*

	Peaks	Stimuli	$\chi 2$	p-value
Latency (ms)	P1	/m/	1.62	0.20
		/t/	0.59	0.44
		/g/	1.39	0.23
	N1	/m/	1.13	0.28
		/t/	0.42	0.51
		/g/	0.13	0.71
Amplitude (µv)	P1	/m/	1.28	0.25
		/t/	0.85	0.25
		/g/	1.39	0.11
	N1	/m/	2.0	0.15
		/t/	0.41	0.51
		/g/	1.75	0.18

*$p<0.05$ significant

From the above tables (Table 8 & 9), it was observed that there were no significant differences between the right and left ears for all the intensities. However, only at 65 dB for wave P1, a significant response observed for latency. This may be due to a chance factor.

Effect of Intensity on Latency and amplitude measures

Repeated measure ANOVA was done to check the effect of intensity on latency and amplitude measures for different speech stimuli. The results revealed that, there were statistically significant differences for wave P1 latency only across

different intensity levels. Further, for amplitude measures wave N1 showed significant differences across different intensity levels (Table 10). Since, wave P1 is more prominent in early age even in individuals with hearing impairment, could be the reason to observe difference at different intensity levels. Further, present study included children with severe to profound hearing impairment using high gain hearing aids, where there is a possibility that at higher intensity levels responses were easier to identify in comparison to lowest level (55 dB SPL).

Further, Bonferroni pair wise comparison was done to check the differences if any observed at each intensity level. The results revealed that at 75 dB SPL, there were significant differences observed for P1 latency measures for only speech stimuli pair of /m/ versus /g/ sounds (p= 0.006) levels. However, different combination of speech stimuli pairs like /m/ versus /t/ and /t/ versus /g/ stimuli did not show significant differences. Even P1 amplitude measure at 75 dB SPL also did not show significant differences between different pairs of speech stimuli. Further, at 65 dB SPL, Boneferroni pair wise comparison did not show any significant differences between different speech stimuli pairs for latency as well as amplitude measures of wave P1 and N1 at 65 dB SPL.

Table 10: F values for latency and amplitude measures for wave P1 and N1

	Peaks	F-Value	p- value
Latency (ms)	**P1**	F(2,90)=6.92	**0.002***
	N1	F(2,88)=2.07	0.13
Amplitude (µv)	**P1**	F(2,90)=1.51	0.22
	N1	F(2,88)=3.22	**0.04***

***p<0.05 significant**

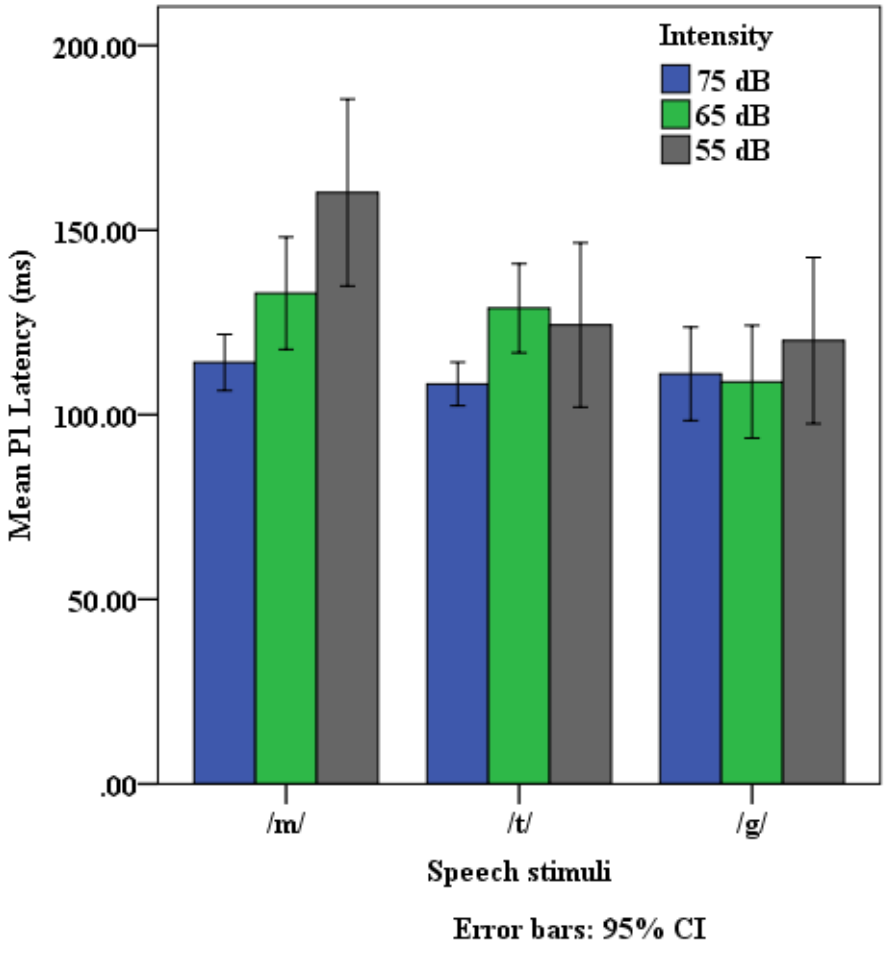

Figure 3: Mean and 95% confidence interval (CI) for wave P1 latency (ms) at different intensities for different speech sounds

From Figure 3, it can be inferred very clearly that for /m/, /g/ and /t/ sounds which represents low, mid and high frequency signals, there is a prolongation in wave P1 latency at 55 dB intensity level in comparison to responses observed at 75 dB SPL. However, at 65 dB SPL similar trends did not observed in these children with severe to profound hearing impairment.

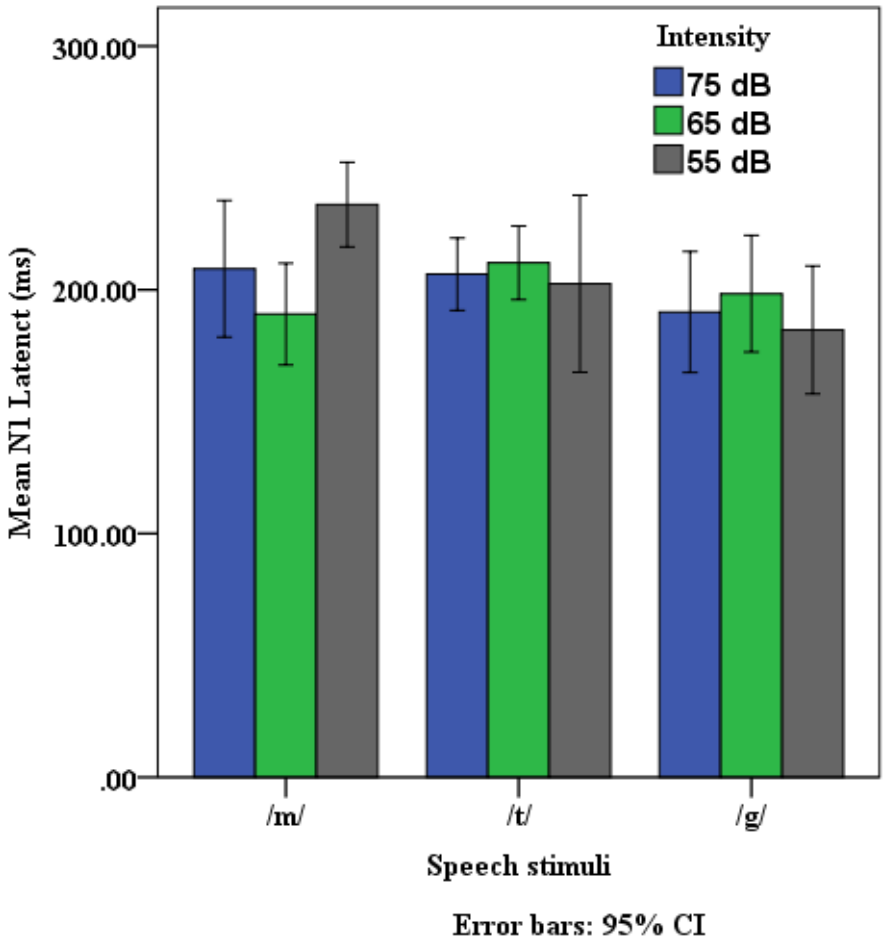

Figure 4: Mean and 95% confidence interval (CI) for wave N1 latency (ms) at different intensities for different speech sounds

For wave N1 latency, effect of intensity only observed for /m/ sounds which are low frequency stimuli. However, /t/ and /g/ sounds did not show change in latency at different intensity levels, it could be because wave N1 is not well represented in

younger ages as well as it could be difficult to show representation at mid and higher frequencies at cortical levels in children with severe to profound hearing impairment (figure 4).

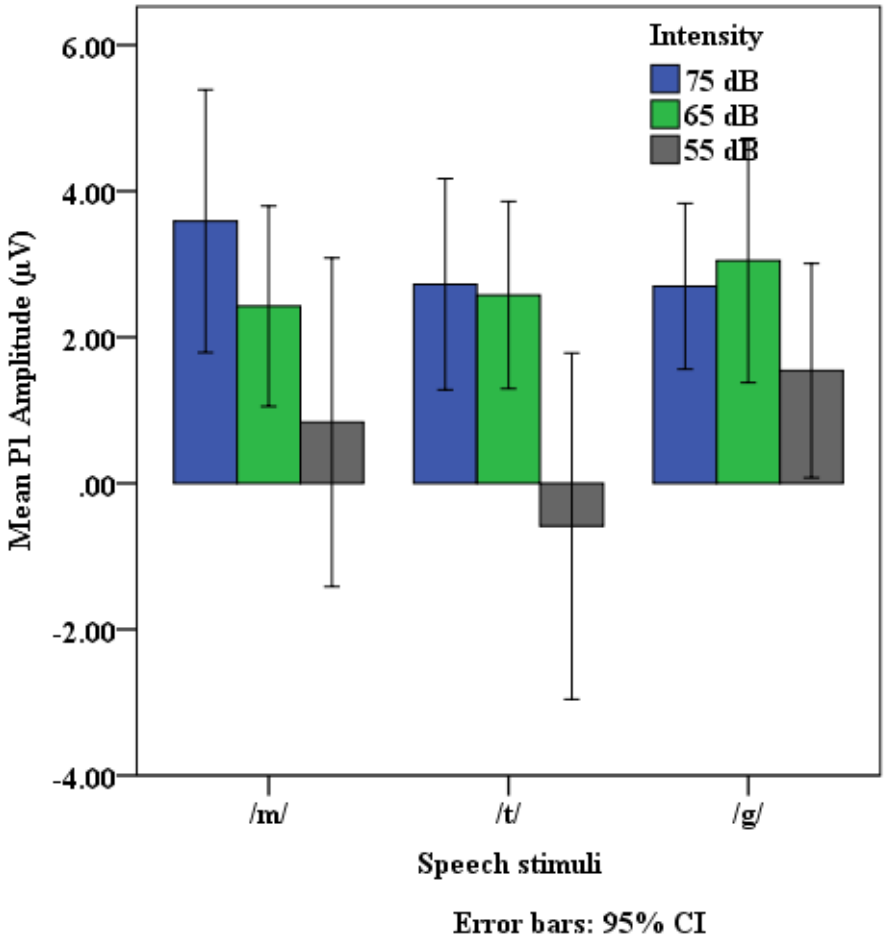

Figure 5: Mean and 95% confidence interval (CI) for wave P1 Amplitude (µV) at different intensities for different speech sounds

Figure 5 shows amplitude measures for wave P1 at different intensity, which represents higher amplitude for higher intensity for all the three speech stimuli in comparison to lowest intensity levels. However, it is also observed that there is higher

SD at lowest intensity levels (55 dB SPL), it could be because at threshold levels cortical potential responses may not be easier to identify.

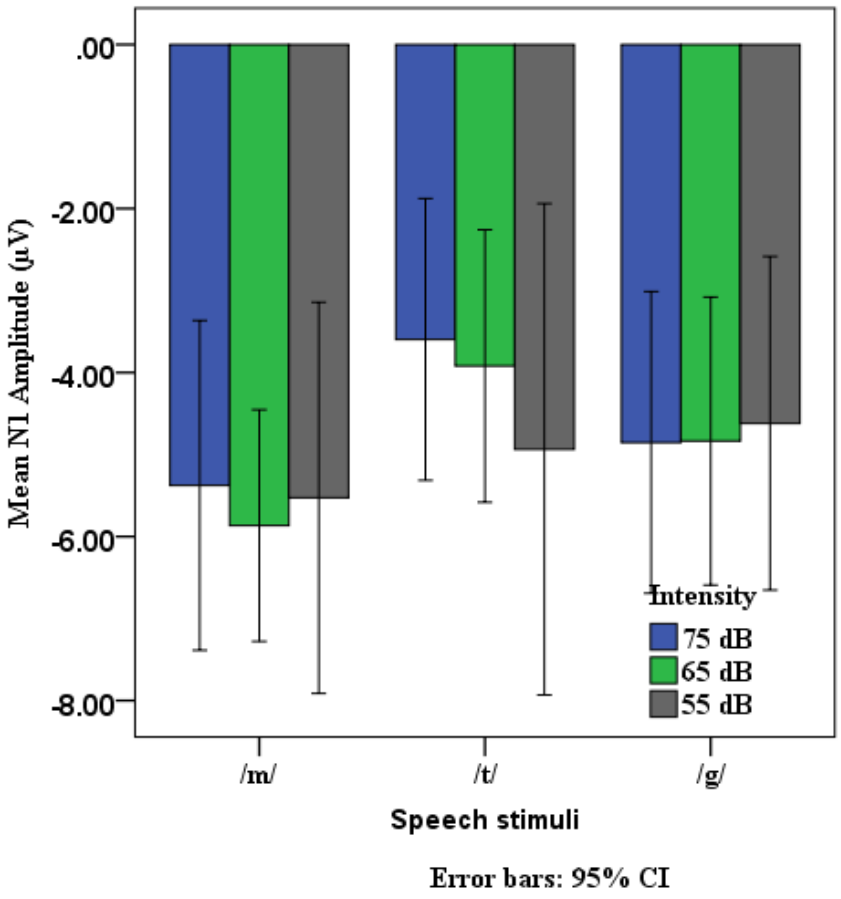

Figure 6: Mean and 95% confidence interval (CI) for wave N1 Amplitude (µV) at different intensities for different speech sounds.

Figure 6 shows that at higher intensity levels, wave N1 amplitude is lesser for /m/ and /t/ sounds in comparison to the /g/ sound in comparison to responses observed at 55 dB SPL. In spite of differences seen in graphical representation, it was not statistically significant at different speech stimuli.

Overall From figure 3, 4, 5 & 6 which represents latency and amplitude measures for wave P1 and N1 did show statistically significant differences for few

speech stimuli across different intensity levels. However, in terms of waveform morphology at different intensity levels did show better morphology at higher intensity levels in comparison to lower intensity levels. The above findings are partially in agreement with the previous studies (Suzuki et al., 1979, Oates et al, 2002; Dun et al, 2012, Carter et al, 2013,).

Dun et al (2012) recorded CAEPs at sensation levels (SL) above 0, 10, and 20 dB and observed detection sensitivities equal to 72±10, 75±10, and 78±12%. They conclude if CAEP is present ($p<0.05$) in response to sound at conversational level, that means sound is stimulating the auditory cortex at that level (> 10 dB SL). If CAEP is absent ($p<0.05$), that is below 0 to 10 dB SL, statistical detection criterion will show no response is detected 95% of the time. If true sensation level is within the range of 0 to 10 dB SL, then the probability of detection of responses is intermediate.

Chang et al (2012) recorded CAEP to speech-based stimuli at three presentation levels (55, 65, or 75 dB SPL) under aided and unaided conditions. The results indicated that the higher sensation levels led to a greater number of present CAEP responses being detected. In another study done by Carter et al (2013) reported a strong level effect, as amplitude increases with sensation level as low as 5 dB, responses were traceable. There are studies done even in adults with normal hearing where they observed similar trends (Antinoro et al, 1969; Davis, Bowers, Hirsh, 1969; Ross et al, 1999). Oates et al (2002) found that the amplitude of the N1 and P300 was larger and the latency shorter at 80 dB SPL compared to 65 dB SPL in adults with hearing loss. In the contrary Koravand et al (2011) did not find any such pattern while studying CAEP using /ba/ and /da/ stimuli from intensity 80 dB HL to 105 dB HL. They reported the P1 amplitude was larger and N2 latency was shorter in children with hearing loss comparatively with normal hearing children.

On the contrary to the present study finding, Korczak et al (2005) reported no effect of intensity on CAEP when using 65 dB SPL and 80 dB SPL for presentation of /ba/ and /da/ stimuli in 20 normal-hearing adults and 14 adults with sensorineural hearing losses ranged from moderate losses (50-74 dB HL) to severe-profound losses (75-120 dB HL).

Effect of speech stimuli on latency and amplitude measures

One way ANOVA was done to check at each intensity level whether there is effect of different speech stimuli on latency and amplitude measures while recording cortical potential in children with severe to profound hearing impairment using hearing aids. The results revealed there were no statistically significant differences observed for latency measures for wave P1 and N1 at 0.05 levels. Similar finding was observed even for amplitude measure of wave P1 and N1 at each intensity level at 0.05 levels (Table 11). Further, Bonferroni pair wise comparison was done for wave P1 and N1 latency measure, which showed no statistical significant differences between different speech stimuli pairs at 75 dB SPL as well as at 65 dB SPL.

Table 11: F Values for latencies and amplitude measures at different intensities for wave P1 and N1

	Intensity	Peaks	F-Value	p- value
Latency (ms)	75 dB	P1	F(2,56)=0.43	0.65
		N1	F(2,56)=0.75	0.48
	65 dB	P1	F(2,53)=3.59	0.06
		N1	F(2,56)=1.163	0.32
	55 dB	P1	F(2,39)=3.01	0.06
		N1	F(2,39)=2.63	0.08
Amplitude (µv)	75 dB	P1	F(2,56)=0.51	0.79
		N1	F(2, 55)=1.06	0.20
	65 dB	P1	F(2,53)=0.22	0.96
		N1	F(2,55)=1.62	0.12
	55 dB	P1	F(2,39)=0.63	0.53
		N1	F(2,39)=0.38	0.68

The above finding is similar to several researchers finding (Shruti & Vanaja, 2007; Dun et al., 2012; Carter et al, 2013). Dun et al, 2012 found that there were no significant differences in the group averages for amplitude and latency between the different speech sounds (/m/, /g/, & /t/). This finding is in contrast with Golding et al (2006) who reported that /t/ sound evoked CAEPs larger in amplitude and earlier in latency than the other two sounds. This they explained it could be because of differences in methodology in terms of younger age group, and by their normal hearing status. Further, Golding et al (2007) used only one intensity (65 dB SPL) level

but in this study different stimuli level were used which led to spread of amplitude and latency as reported by Dun et al, (2012).

Shruti and Vanaja (2007) studied cortical potentials using different speech stimuli (/i/, /m/, & /ʃ/) in hearing aid users. They observed in differences of waveform morphology for different speech stimuli in terms of latency and amplitude measures. However, no stimuli effect was observed for one among different categories of hearing aids. They further observed differences in cortical potential responses based on different hearing aid features.

Carter et al (2013) reported that the CAEP responses depend on prescriptive formula as they observed that when NAL-NP was used, which gives less low frequency real ear insertion gain. Cortical evoked potentials for /m/ stimulus was not detected even at the highest presentation level, and when they gave a low frequency boost, the response for /m/ was present at 60 dB SPL. They recommended NAL NL1 for adults but the evidence for its use in children is uncertain. Further, they used filtered speech for evoking CAEPs and found that CAEP are not sensitive to very minute changes in hearing aid characteristics to guide in fine tuning. Measurement of hearing aid gain for speech stimuli is less accurate when hearing aid with complex signal processing is fitted especially in young children. But can be used as an objective measure to prevent over amplification resulting from subjective measures.

Comparison between control and clinical groups for latency and amplitude measures

Independent t-test was done to compare the performance between control group and hearing impaired group. The results revealed that there were statistically significant differences for latency and amplitude measures for wave P1 and N1 between two groups. For latency measure, it was observed from table 12 that, wave

P1 and N1 responses elicited from /m/, /t/, and /g/ stimuli shows statistically significant differences at 0.05 levels. In addition to that, it also inferred that in spite of suitable hearing aids used by children with severe to profound hearing impairment, there cortical responses at different frequency regions showed prolonged latency in comparison to children with normal hearing. Similar observation noticed even for amplitude measures for different speech stimuli for P1 and N1 waves except N1 wave response for /g/ stimuli (Table 12).

Table 12: *Independent t-test outcomes for different speech stimuli for latency and amplitude measures of wave P1 and N1*

	stimuli	Peaks	t-Value	p- value
Latency (ms)	**/m/**	P1	6.71	0.00**
		N1	16.6	0.00**
	/t/	P1	-11.62	0.00**
		N1	2.70	0.008**
	/g/	P1	25.38	0.00**
		N1	29.23	0.00**
Amplitude (µv)	**/m/**	P1	-21.22	0.00**
		N1	-37.40	0.00**
	/t/	P1	-5.56	0.00**
		N1	-10.67	0.00**
	/g/	P1	7.43	0.00**
		N1	-1.62	0.10#

**P<0.05; **p<0.001; #p>0.05*

The above finding is in agreement with previous studies (Oates et al, 2002; Korczak et al., 2005; shruti & Vanaja, 2007). Korczak et al (2005) reported that the CAEPs responses are depended upon the degree of sensorineural hearing loss, and intensity of stimuli. Further, they also suggested that despite the benefits provided by

the hearing aid the brain may not be processing the speech stimuli with the same degree of effectiveness and accuracy compared to individuals with normal hearing sensitivity at the same intensities. The above information is further supplemented by Apeksha and Devi (2010) and Tremblay et al (2006) in individual with normal hearing as well as hearing impaired group.

There are cortical potential recorded in individuals with hearing impairment using different speech stimuli. Apeksha and Devi (2010) reported that cortical responses observed for individuals with hearing impairment was statistically different in comparison to normal hearing individuals for different speech stimuli (/ba/, /da/ & /ga/). Further, both researchers concluded that in spite of individuals wearing hearing aid according to the degree of hearing loss, the hearing aid helps to compensate for hearing loss by amplifying sound but effectiveness depends on the central auditory system ability to integrate the spectral and temporal information by hearing aid.

The use of hearing aids improves the detectability of wave P1-N1 for individuals with severe to profound hearing loss but the latencies are usually prolonged and amplitude decreased especially at low intensity levels. The result of the present study with the previous findings report that presence of CAEPs responses in children with hearing impairment provides indication that auditory cortex is not only responding to speech but also detects acoustical changes present in the stimuli even in severe to profound hearing loss (Oates, 2002). According to Koravand et al (2011), wave P1-N1 responses of cortical potentials is the biomarker for presence of audibility of speech stimuli in individuals with hearing impairment with larger P1 amplitude. These peaks can be recorded to measure improvement in audibility with hearing aids in children.

The findings of present study should be considered with caution since there are several parameters which might be responsible for differences in responses observed. These are stimulus parameters like type of stimulus, intensity levels, and different stimuli used to elicit the responses. Further, results may vary with the type of hearing aid used, the prescriptive formula for gain used in the present study as study by Carter et al (2013), the signal processing features of the hearing aid used were disabled resulting in no dynamic range compressions.

Another difference while recording cortical potentials in present study for hearing aid users were signal processing features were not disabled. Further, the limitation with HEARLab system is there is no feature to replicate the waveform, to check reliability of the responses present or absent. However, the presence and absence of responses in present equipment is based on Hotelling T2 statistical tools which detects responses based on probability curve. In addition to that, this equipment is single channel instrument where eye blink artefacts cannot be removed by using ocular electrodes.

The response also depends on state of arousal, which is difficult to sustain in young children. Suzuki et al (1969) reported the responses obtained from sleeping children (1-4 years) are unreliable as brain produces theta wave at 4-7 Hz which may change the morphology of waveform. On the contrary Taguchi et al (1999) reported large amplitude responses in sleeping children with tone burst stimuli. It would be beneficial if alternating stimuli with more number of presentations could be used for recording (Dun, 2012). But collecting artefact free 200 epochs for every young child at different presentation level, for a range of stimuli is clinically not feasible (Carter et al, 2010). If all these parameters are controlled then CAEPs is a good objective tool to

measure the neural representation of different speech stimuli at cortical levels in different group of population.

SUMMARY AND CONCLUSION

The aim of the study was to obtain the aided CAEPs in children with severe to profound hearing impairment in the age range of 3 to 6 years. There were two groups of participants included in the study. Control group consisted of 13 children (20 ears) with normal hearing in the age range 3 to 6 years and Clinical group consisted of 13 children (20 ears) with severe to profound sensorineural hearing loss.

The collected data were tabulated and analyzed further using both Parametric and non parametric tests. Parametric tests includes repeated measure ANOVA, one way ANOVA, Bonferroni pair wise comparison test and independent t-test. The non parametric test include Kruskal Wallis test for smaller sample size. Results show different mean and SD values of latencies and amplitudes of P1 and N1 wave at 75 dB, 65 dB, and 55 dB SPL in children with severe to profound hearing impairment using hearing aids. There was no effect of different speech stimuli on latency and amplitude measures. Further there were statistically significant differences for wave P1 and N1 latency and amplitude measures across different intensity levels for children with hearing impairment. In addition, there were also statistically significant differences observed between control and clinical groups in terms of latency and amplitude measures for wave P1 and N1.

To conclude, the results of the present study shows that CAEPs are a good objective tool to measure the neural representation of different speech stimuli at cortical levels in different group of population. However, it may depend on several factors like degree of hearing impairment and type of stimuli used. Prolonged CAEPs may show despite the benefits provided by the hearing aid fitted according to the degree of hearing loss, the brain may not be processing the speech stimuli with the

same degree of effectiveness and accuracy compared to individuals with normal hearing sensitivity at the same intensities. It may depend on the degree of hearing loss, age of hearing aid fitted, duration of hearing aid usage and auditory training

Further research may throw light on the effect of changing different parameters of hearing aid like disabling the signal processing features of hearing aid, digital versus analog hearing aid, and effect of different prescriptive formula for hearing aid gain on CAEP in different degrees of hearing impairment. Further, finding the benefit of using combined effect of subjective measure of wave analysis of CAEP and objective measure based on automated statistical detection for CAEP wave detection in HEARLab system can be beneficial.

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