SPEECH EVOKED CORTICAL POTENTIAL IN CHILDREN WITH MODERATE- MODERATELY SEVERE SENSORINEURAL HEARING LOSS (6-9 YEARS)

Tejaswini. G

Register No. 12AUD032

A Dissertation Submitted in Part Fulfilment For Degree Of

Master Of Science (Audiology),

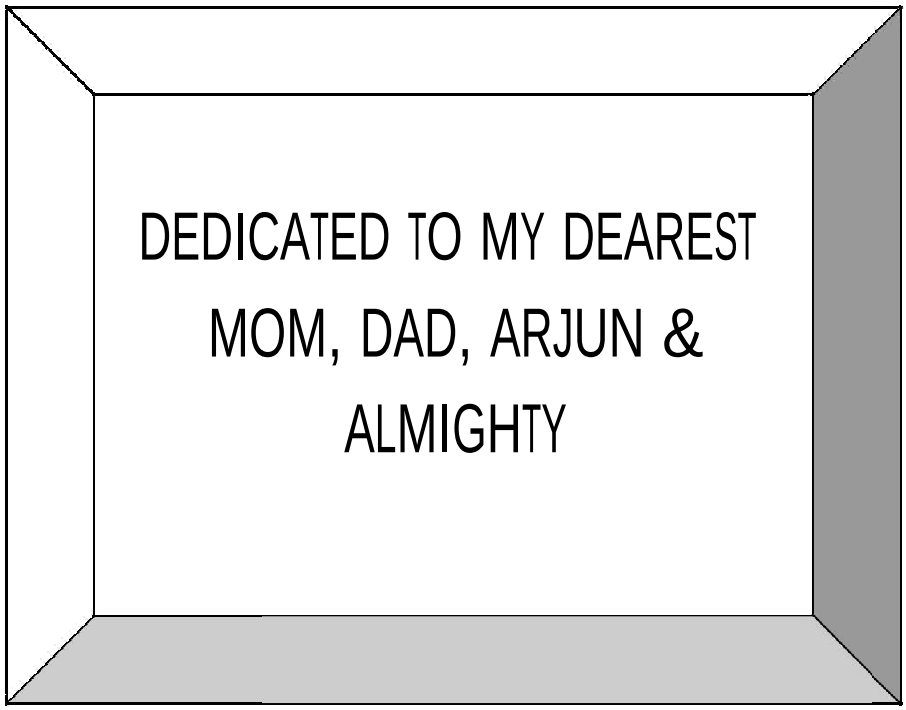
University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MANASAGANGOTHRI,

MYSORE- 570006

May, 2014


DEDICATED TO MY DEAREST
MOM, DAD, ARJUN &
ALMIGHTY

CERTIFICATE

This is to certify that this dissertation entitled "**Speech evoked cortical potential in children with moderate to moderately severe sensorineural hearing loss (6-9 years)**" is the bonafide work submitted in part fulfilment for the degree of Master of Science (Audiology) of the student with Registration No. 12AUD032. 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.

Prof. S.R. Savithri,

Director,

All India Institute of Speech and Hearing

Manasagangothri, Mysore -570006.

CERTIFICATE

This is to certify that this dissertation entitled "**Speech evoked cortical potential in children with moderate to moderately severe sensorineural hearing loss (6-9 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.

Dr. Prawin Kumar

Guide

Lecturer in Audiology,

Department of Audiology

All India Institute of Speech and Hearing

Manasagangothri, Mysore -570006

Mysore

May, 2014

DECLARATION

This dissertation entitled "**Speech evoked cortical potential in children with moderate to moderately severe sensorineural hearing loss (6-9 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

May, 2014 Register No. 12AUD032

ACKNOWLEDGEMENT

*I express my deepest gratitude and heartfelt thanks to **Dr. Prawin Kumar,** Lecturer in Audiology, AIISH, Mysore, for all his inspiration, encouragement, guidance, comments, and patient listening. It's my proud privilege to express my utmost thanks and indebtedness to him for offering me all the support that went into the preparation of this dissertation.*

*My sincere thanks to **Prof. S.R. Savithri,** Director, AIISH, Mysore, for permitting me to carry out this dissertation.*

*I thank **Dr. Ajith Kumar U,** HOD, Department of Audiology, AIISH, Mysore, for having allowing me to utilize instruments as and when I needed.*

*Dear **Shruti maa'm,** thanking you might not just be enough. You are my greatest inspiration. The interest towards audiology, in me, developed only after your interactive and informative lecturing. Words are scarce in offering my thanks to you maa'm. You are the best!!*

*I express my heartfelt thanks to **Sandeep sir, Vijay Narne sir, Ajith sir, Animesh Sir, Neeraj sir, Sreeraj sir, Majula maa'm and Nike sir** for their informative lecturing. Getting the opportunity to study under you has been one of the greatest privileges I've had. Thank u.*

I express my sincere gratitude to all my teachers who have imparted knowledge, moral values and taught the right path in my life.

This acknowledgement would be incomplete without thanking all the special people in my life.

*Heartfelt thanks to my beloved parents **"□□□□ □□□□□□□□".***

*□□□□**….** The most beautiful woman. **All I am I owe to my mother.** I attribute all my success in life to the moral, intellect, disciplines & values I received from you. □□□□□□□.. From tolerating my rants to keeping up with my antics, I want to say thank you to a dad who is just fantastic. I love when you make fun of things. You make me so happy. I'm Daddy's little girl and our relationship is priceless….! Thank you both for your unconditional love, trust, & constant enticement. Your confidence in me pushed me ahead. It's my fortune that I am blessed with the best parents in the world. You mean everything to me. And I am really sorry for throwing my tantrums at you when I was frustrated with my works. Love you both from the bottom of my heart.*

***Dodamma, Dodappaji, Rekha aunty, Bhimu uncle, Mama, Mami, Manju mama, Pri's mom and dad…** Thank u all for showering enormous blessings cuz of which I'm here today.*

*My notorious **bro,** you have always been great. Thanks for fighting with me each and every minute and acted like you hated me the most in this world (I know u don't). Wish you will achieve all you ever wanted.*

Guru anna, Suggu anna, Puttu anna.. *Blessed to have u people as my elder brothers. Thanks for always protecting your little sister and for all the moral support. You guys are simply awesome!!*

Sweet heart ***Priyanka,*** *Love you for the subtle way in which you show your care. I wonder how would I have been living in this world without your charisma in my life.. Thanks for your immense love, prop up and back-up which helped me to stay focused and balanced during my tough days. In those days I missed u a lot.*

My dear ***Gutti,*** *thanks for being such an amazing friend. I can never wipe out those sweet memories of our namsu hostel with all the namsu people around. All the giggling, gossips, and your great motivational speech which you would think will enthuse me when I felt low (most of them actually did). Thanks for always being there. Love you gutts.. (and I really hate Teni)..you are the best!!*

Chintu.. *I'll always cherish the insanely fun times and watching all those epic movies together (U know wat I mean)!!. I am sanctified to have you as my cousin and thank you for being my best friend (your secrets are safe with me. ☺).*

Thanks to my truly awesome cousins, ***Sushil, Sushila, Shantaraj, Chandu, Darshu.*** *No matter what I say and what I do, you guys are constantly there to pull my legs and bring a bracing smile on my face. Life would be so incomplete without you guys. Love u loads.*

My best friends, ***"my Cuties",*** *Ashu, Firu, Shar and Anu who have always been there for me. Thank you all. People have one or two best friends but I feel blessed.*

My dearest friends, ***Caren, Supriya & Kanss****… I couldn't have asked for better friends than you guys. You are some of the rare auspicious people with whom I used to share my "Sorrow & Joy". Miss u guys a lot..*

Junu, Sumpi, Revati di, & Radika.. *I'll always relish those non-stop laughing sessions. You guys have given me such mammoth memories to treasure in four years. Thanks for the care and love. Love u all .*

Somanna, *My bro, thank you for the love & support and for being so sweet and advising me to choose the right thing in life whenever I was in a great dilemma.*

My ***Train Nungshibis..*** *Cheers to all the sweet memories we had together. Thanks for always being there and for boosting up my energy, Eche Ibe. Love u all lasing mathums..*

Roja and Roji, *my darling friends, I have enjoyed the times with you incalculably. Both of you will always remain a prominent part of my "sweet memories of AIISH". Thanks for your love and care that never made me feel lonely. The moments spent with you guys will be treasured forever. Wish you all the best.*

Two most craziest people I have known. ***Astha & Juhi,*** *my partners in crime, I still can't believe we did this and can't forget all those lazy hard working Saturdays and Sundays. Thanks for all the love, concern, extreme help and shoutings…!! Really enjoyed being with you both.*

*My **CRAZY CLASSMATES (Suhani, Suman, Jebu, Rajan, Sabruu, Gatla, Souji, Praggi, Shilpa, Swati, Merin, Nigila, Ramize mone)** for having been a great company. We were a great batch (terms and conditions apply), will miss all the fun we had. Keep in touch. Best wishes to all of you for a bright future.*

***Deepika and Lakshmi,** thanks for clearing all our queries and for helping through out the data collection. You guys have been a great support.*

***Ishu.. (Chomu),** Thanks for always baring my non-stop complaints and for making me smile everytime I felt low. Jiddan, Haste raho aur aise hi hasate raho. Paglu.. Bhago ab!*

***Kiran, Vineetha, Shrilekha..** Thanks for the love and support. You guys are awesome.*

***Jithu..** Thanks a lot for taking care of me and accompanying me most of the times I travelled alone.*

*I would like to thank **Sharath sir and Baba sir** for providing us the materials required for the dissertation..*

*Special thanks to **Ganapathy sir, Hemanth sir, Arun Raj sir, Geetha mam, Kishore sir & Ramya mam** for opening the department whenever requested. Thanks for your timely help.*

*Special thanks to **Sachidanand sir,** for staying back with us in the evenings and helping so much in the crucial time. Wish u all the best sir.*

*Heartfelt thanks to all the **sweet little angels** who participated in the study and special thanks to their **parents** for being so patient and supportive, without your help the dissertation would be impossible.*

*Thanks to the **pre-school staff,** who co-operated and permitted us to take the children for testing during dissertation hours.*

*My sweet juniors, **Aishwarya, Arpitha and Sudeeksha**, thanks for getting children for the data collection. And the sweetest juniors, **Rashmi, Durga, Theerta, Neha, Vishali and Pooja** for helping in the work whenever required. A lot of love and wishes for you all.*

*Last but not the least, I thank the **Lord of Heavens, Balaji,** for giving me strength and courage, to overcome all my difficulties and showering immense blessings on me.*

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Sensory stimulation triggers the firing of neurons which results in small but measurable electrical potentials. The specific neural activity arising from acoustic stimulation, a pattern of voltage fluctuations lasting about one half second, is an auditory evoked potential (AEP). AEPs are now an integral part of the current audiologic test battery. With this technique, synchronous neural activity from the peripheral end organ of hearing up to the cortical structures responsible for audition can be examined. As such, AEP measurements allows the clinician to have a unique glimpse of the auditory system. With enough repetitions of an acoustic stimulus, signal averaging permits AEPs to emerge from the background spontaneous neural firing (and other non-neural interferences such as muscle activity and external electromagnetic generators), and they may be visualized in a time-voltage waveform (Ruth & Lambert, 1991).

In far field recordings from humans, the three typically used response classifications, based on response latency, are early (the first 10 ms), middle (10–80 ms) and late (80 ms to 500 ms) latency response. In terms of generators, these classes correspond roughly to brainstem, thalamus/cortex and cortex respectively (Kraus & Nicol, 2009).

Cortical Auditory evoked potentials (CAEPs) are brain responses that are evoked by the presentation of auditory stimuli and processed in or near the auditory cortex, and therefore they are referred to as cortical auditory evoked potentials. Such measures reflect the sum of synchronous, time-locked neural activity which are detected at the level of the central auditory nervous system, related to the strength (amplitude) and timing (latency) of a response. For these reasons, CAEPs have been recommended for clinical use in monitoring changes in neural activity related with auditory rehabilitation (e.g., hearing aids). CAEPs historically the first discovered

cortical potential in origin and are much larger and lower in frequency than early and middle-latency potentials. CAEPs are highly dependent upon stimulus type, recording location, recording technique, patient age and state. Hence it may differ dramatically in morphology and timing and may overlap one another (Kraus & Nicol, 2009).

In adults, the CAEP waveform consists of a series of peak or troughs (P1, N1, P2 & N2) that occur at about 50-250 ms. In infants and young children, the CAEP waveform has a different morphology and is dominated by a large positivity (P1) at about 100-250 ms followed by a late negativity at about 250-400 ms (Gilley, Sharma, Dorman, & Martin, 2005). The peaks of the P1-N1-P2 complex are thought to reflect neural activation of the central auditory system in response to the spectral and temporal properties of a given stimulus (Glista, Easwar, Purcell & Scollie, 2012).

The clinical applications of CAEPs range from their use as an indicator of auditory sensitivity in difficult to test population in the diagnosis and monitoring of various otologic and neurologic disorders. (Ruth & Lambert, 1991). The CAEPs responses are passively elicited in which participant is not required to perform a task and is asked simply to remain alert. Since these responses are not influenced by behavioral and performance related demands, it provides a reliable objective measure of cortical auditory function in children.

CAEPs can be elicited by a wide range of transient stimuli i.e. clicks, tone burts, noise bursts, and speech sounds (Naatanen & Picton, 1987). Auditory cortical evoked potentials elicited by naturally produced speech sounds have been reliably recorded in normal hearing and hearing impaired individuals. Also, naturally produced speech tokens representing different acoustic cues have evoked distinct neural response patterns. (Tremblay, Friesen, Martin & Wright, 2003)

Speech-evoked CAEPs have been used to objectively determine whether a child with hearing impairment is detecting speech sounds and processing them at the level of the auditory cortex. It is obligatory to know what the hearing aid is doing to the signal when sound is processed through a hearing aid. For this reason, current research in this area has focused on amplification-related modifications to the CAEP stimulus, related to factors such as poor signal-to-noise ratio (SNR) and/or rise time, both of which may interact with the input stimulus level used in the fitting procedure.

Further, it is essential to hypothesize how the hearing aid signal processing alters the acoustic content of the stimulus and, in turn, affects the evoked responses. Hearing aid signal processing causes many acoustic modifications to a stimulus that are likely to affect CAEPs. However, it remains uncertain if and how these acoustic modifications affect the aided CAEP. Further, Aided CAEPs or evoked potentials recorded from those wearing their hearing aids may be of use to evaluate hearing aid fittings as well as experience-related plasticity associated with amplification.

NEED FOR THE STUDY

In recent years, there has been an increasing interest in the use of cortical potentials to assess speech perception capacity in clinical population. There is a need to explore a specific electrophysiological response in terms of its ability to demonstrate peripheral discrimination ability. Such tests would contribute to the objective evaluation of subjects, who for reasons of age, hearing loss, lack of auditory, linguistic and/or cognitive pre-requisites for behavioral speech perception tests (Tyler, 1993; Boothroyd, 1991).

Various studies have examined the relationship between CAEPs and auditory perception (Gravel, Kurtzberg, Stapells, Vaughan & Wallace, 1989; Kraus et. al, 1993; Purdy et al, 2005; Tremblay, Billings, Frieson & Souza, 2006). Research has shown that CAEPs correlate well with pure tone audiometric thresholds (Maanen & Stappells, 2005). The presence of speech evoked CAEPs indicates that speech stimuli have been detected (Hyde, 1997). Study has shown that CAEP waveform is affected by changes in speech stimulus parameters such as place of articulation and Voice onset time (Tremblay et al., 2003). While there has been substantial progress in our understanding of CAEP development there remain several areas which require investigation (Wunderlich & Cone-Wesson, 2006).

Studies have reported differences in the CAEP waveforms in adults with different speech stimuli suggesting that underlying neural representation of the stimuli differs (Agung, Purdy, McMohan & Newall, 2006; Tremblay et.al, 2006). Research at National Acoustic Laboratory (NAL) has focused on speech sounds /m/, /t/ and /g/, selected for their spectral emphasis in the low, mid and high frequency regions respectively. Measurements on the cortical responses of babies with normal hearing has shown that cortical responses to these sounds can always be

detected, provided the babies are awake, alert and not too physically active (Golding, Dillon, Seymour & Carter, 2008). Further, the characteristics of the CAEP in the children are largely uncharted and those in the early years of childhood require further systematic examination.

In the present study, CAEPs elicited by consonant-vowel syllables (/m/, /g/ & /t/) are considered to examine normal maturation of the central auditory system. Understanding the normal patterns of maturation of AEPs evoked by speech sounds may aid in the development of electrophysiological techniques for diagnosing deviated central auditory maturation coincident with speech, language and learning impairments. Since the processing of all the speech sounds which encompasses the speech spectrum is important, study of processing of a single frequency may not prove to be sufficient. Hence, the effect on CAEPs is explored in the present study.

Polen (1984) found that moderate-to-severe sensory neural hearing loss resulted in prolongation of N1-N2 and P300 latencies and a reduction in N2 amplitude in comparison with results from normal hearing participants. In contrast studies have reported there were no significant differences in the latencies of waves N1, P2 or P3 for the normal versus hearing impaired individuals (Wall, Balebont, Davidson & Fox, 1991). Very few studies have been reported on the speech evoked CAEPs obtained in normal hearing versus hearing impaired children.

Hence, there is a need to study the CAEPs which is evoked by speech stimuli largely encompass the speech spectrum in children with hearing impairment. Therefore aided CAEPs to three different speech sounds /m/ which is a low frequency sound, /g/ which is a mid-frequency sound and /t/ which is a high frequency sound will be investigated in the present study.

AIM OF THE STUDY

To evaluate the speech evoked cortical potentials obtained for speech tokens such as /m/, /g/ and /t/ in children with moderate to moderately severe hearing impairment using hearing aids.

OBJECTIVE OF THE STUDY

1. To determine the effect of intensity on speech evoked cortical potentials in children with hearing impairment.
2. To determine the effect of different speech sounds on speech evoked cortical potentials in children with hearing impairment.
3. Comparison of aided speech evoked cortical potentials in children with hearing impairment and unaided speech evoked cortical potentials in children with normal hearing.

REVIEW OF LITERATURE

The cortical auditory evoked potentials (CAEPs) represent summed neural activity in the auditory cortex in response to sounds (Zhang et al., 2012; Agung et al., 2006). These potentials are elicited using click/tonal stimuli; although speech stimuli have been utilized recently in individuals with normal hearing and hearing impairment (Agung et al., 2006; Tremblay et al., 2006; Tremblay et al., 2003) and in other amplification device users (Groenen, Beynon, Snik, & Van den Broek, 2001; Kelly, Purdy & Thorne, 2005; Micco et al., 1995). 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. A number of studies have compared cortical responses between hearing aid and cochlear implant users and individuals with normal hearing using speech stimulus (Sharma, Dorman, Spahr, 2002; Eggermont & Ponton, 2003).

There are three major cortical AEPs classified by latency from the P1-N1-P2 complex with latencies between 80 ms and 200 ms to the late potentials such as the Mismatch Negativity (MMN) and P300 with latencies between 150 ms and 1000 ms (Oates, Kurtzberg, & Stapells, 2002). The P1-N1-P2 response is an obligatory cortical AEP. This response is always present in a healthy auditory system when subjects are awake. It can be elicited by the onset of a sound such as a click or a tone, or it can be elicited by a change in a stimulus.

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. The P1 wave latency reflects the sum of synaptic transmission delays throughout the central auditory pathways (Eggermont, Ponton, Don, Waring & Kwong 1997). Sharma and colleagues have described the developmental trajectory of the P1 response throughout infancy and childhood.

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 et al., 2002; Moor & Linthicum 2007). The infant CAEP component latencies were prolonged by 100-150 and the latency-intensity input output functions were steeper in infants compared to adults as reported by Cone and Whitaker, 2013.

Koravand, Jutras & Lassonde (2011) studied the patterns of neural activity in the central auditory system in children with hearing impairment in the age range of 9 to 10 years. The study showed a trend toward larger P1 amplitude, a significant reduction in amplitude and latency of N2 in children with hearing loss compared to control group. They concluded that maturational delays could be because of deficits in central auditory processing in children with hearing impairment, as indicated by the neurophysiological markers P1 and N2.

Further, the cortical responses are one of the idyllic objective tools for aided hearing instrument evaluation. Consistently present in young infants and adults, it correlates well with the perception and seems to be receptive to differences between speech stimuli like voice onset time, place of articulation (Tremblay et al., 2003). CAEPs reflect the functional integrity of the auditory pathway involved in the processing of complex speech stimuli measurements. CAEPs have been used previously for the evaluation of effectiveness of hearing aids for infants and young children (Tremblay et al., 2003). In children, latency changes in the cortical auditory evoked potentials have been used to document auditory system plasticity and recovery from auditory deprivation (Sharma, Gilley, Dorman & Baldman, 2007; Sharma, Nash & Dorman, 2009).

CAEPs are also used to monitor changes in the neural plasticity and the neural depiction of acoustic speech cues after auditory training in individuals using cochlear implantation or hearing aid amplification (Tremblay et al., 2003). The occurrence of CAEPs appears to associate well with speech recognition ability in children with auditory neuropathy (Sharma, Cardon, Henion, & Roland, 2011). The foremost applicability of CAEPs evolves from the verity that it can be recorded from premature and full term newborns and from older children. Conflicting to maturation effect seen in early childhood there is an increase in latency and decrease in amplitude with the advancing age (Wunderlich & Cone-Wesson, 2006).

Stimulus related factors on cortical auditory evoked potentials

Effects of stimulus frequency and presentation level on CAEPs should be considered when using CAEPs for hearing aid or cochlear implant evaluation in clinical population. 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 (Purdy, Sharma, Munro & Morgan, 2013).

Effect of intensity/ stimulus level on CAEPs

Billings, Tremblay, Souzaa and Binns in 2007 studied the effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. Waves 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 latencies decreased with increasing intensity for unaided and aided conditions. However, there was no significant effect of amplification on latencies or amplitudes. 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. They also report the importance of controlling for stimulus intensity when evoking responses in aided conditions.

Munro, Purdy, Ahmed, Begum and Dillion in 2011 investigated detection and differentiation of obligatory cortical auditory evoked potentials in normal hearing listeners with and without a simulated conductive hearing impairment using the HEARLab. CAEPs were obtained from 24 normal hearing adults, with and without earplugs using three natural speech sounds (/m/, /g/, & /t/) presented at 55, 65, and 75 dB SPL. They reported the response detection was good except for the lowest presentation level. However, differentiation of waveforms was generally poor for individual listeners. They concluded that waveform differentiation was relatively poor, especially at low presentation levels (55 dB SPL) using the HEARLab's stimulus and analysis protocol.

Purdy, Sharma, Munro and Morgan in 2013 studied the effects of stimulus level on speech-evoked obligatory cortical auditory evoked potentials in infants with normal hearing for a low (/m/) and high (/t/) frequency speech sound. CAEPs were recorded for two natural speech tokens, /m/ and /t/. The experiment was done on 16 infants aged 3-8 months with no risk factors for hearing impairment, normal tympanogram and otoacoustic emissions. Infants were either tested at levels of 30, 50, and 70 dB SPL or at 40, 60, and 80 dB SPL, in counterbalanced order. The study showed input-output functions having diverse effects of increasing sound level amid stimuli. There were minimal changes in latency with increase in level for /t/. For /m/, there were approximately 50-60 ms latency increases at soft compared to loud levels. Amplitudes saturated at moderate-high levels (60-80 dB SPL) for both stimuli. They concluded that infant's CAEP

input-output functions differ for /t/ versus /m/ and differ from those previously reported for adults for other stimuli.

In 2005, Korczak, Kurtzberg and Stapells studied the effects of sensorineural hearing loss and personal hearing aids on cortical event related potential and behavioral measures of speech sound processing. Participants were 20 normal-hearing adults and 14 adults with moderate to profound sensorineural hearing losses. They recorded cortical event related potentials (ERPs) for /ba/ and /da/ speech stimuli presented at 65 and 80 dB SPL for both the groups. Results indicated that the use of personal hearing aids substantially improved the detectability of all the cortical ERPs and behavioral d-prime performance scores at both stimulus intensities. 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 latencies at both stimulus intensities and N2 latencies at the higher stimulus intensities. This study infers that hearing-impaired individuals' 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.

Chang, Dillon, Carter, Dun and Young (2012) studied the relationship between cortical auditory evoked potential detection and estimated audibility in 18 infants (2.7 to 10.5 months) with confirmed bilateral sensorineural hearing impairment. CAEP to speech-based stimuli (/m/, /g/ & /t/) at three presentation levels (55, 65, & 75 dB SPL) under aided and unaided conditions were recorded. The results of cortical potentials were compared with the behavioral audiometric responses. They reported that higher sensation levels resulted to a greater number of present

CAEP responses. Further, more CAEP waveforms were detected in the aided condition than in the unaided condition. They concluded that the presence/absence of CAEP responses obtained by the automatic statistical criterion was effective in showing whether increased sensation levels provided by amplification were sufficient to reach the auditory cortex. This was clearly apparent from the significant increase in cortical detections when comparing unaided with aided testing.

Dun, Carter and Dillion in 2012 determined the relationship between cortical auditory evoked potential detection and audibility behaviorally in infants with sensorineural hearing loss. Twenty-five sensorineural hearing impaired infants with an age range of 8 to 30 months were tested in which 18 aided and 7 unaided conditions. First, behavioral 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 SPL, and CAEP recordings were made. All 22 children in this study showed CAEPs for at least one sound for one presentation level. However, based on sensitivities reported, as many as 25% of speech stimuli exceeding 10 dB SL did not evoke a CAEP responses.

Effect of stimulus frequency on CAEPs

Cortical auditory evoked potential have been recorded with wide range of stimuli including tones, clicks and speech stimuli. Several studies have shown differences in CAEPs latencies for different stimulus frequencies recording using conventional evoked potentials. (Roberts & Poepple, 1996; Crottaz, Herbette & Ragot, 2000). Studies have shown that it is possible to differentiate cortical responses based on different speech stimuli (Ostroff, Martin & Boothroyd, 1998).

Various attempts have been made to investigate the effectiveness of CAEPs. Rapin and Grazianni (1967) were the first to study the effect of sensorineural hearing loss and personal hearing aids on CAEPs. They found that a majority of their participants (5 out of 8 individuals) had aided cortical responses better than unaided cortical responses by at least 20 dB when click and tonal stimuli were used. However, two participants did not show any changes in cortical responses for aided versus unaided condition.

Since early 1960's Davis and Colleagues, have typically been using tonal stimuli to elicit the auditory evoked late latency responses (Davis, Bowers & Harish, 1968). As an imperative, amplitude for the N1 and P2 components of the late latency responses are larger and latencies were longer, for low frequency tonal signals in comparison to higher frequency signals (Antinoro et al., 1969; Jacobson, Lombardi, Gibbens, Ahmad & Newman, 1992). The tonal stimuli give very limited information about the perception of speech, which is not the ultimate aim of the hearing aids. Hence tonal stimuli are not preferred to evaluate benefit of a hearing aid. Speech stimuli have better validity and preferred for evaluating hearing aid benefit.

A majority of the later researchers concentrated on evaluating CAEPs using speech stimuli. Gravel et al. (1989) compared the aided versus unaided CAEPs in four children with moderate to profound sensorineural hearing impairment. They reported a clear P1 response followed by a prominent negativity only in aided CAEPs in three out of four children. In contrast, one child with progressive hearing loss initially showed a response when the loss was in range of severe degree but as the hearing loss increased no response was traceable using CAEPs.

As reported by Polen (1984) the late components of the auditory event related potentials might be altered in the presence of sensory neural hearing impairment. This is because sensory neural hearing impairment at high frequencies affects individual's ability to discriminate

phonemes; loss of frequency resolving power and increased difficulty in discrimination for any task is known to increase the latency of P3. Further, normal hearing and sensory neural hearing impaired subjects with phonemic stimuli was studied. A trend toward reduced amplitude for all late components in the hearing impaired group was noted but N1 and P2 were not significantly reduced in amplitude. P2 was most drastically reduced in amplitude due to sensory neural hearing impairment. Thus peak latencies were prolonged for the sensory neural group.

Purdy et al, in 2002 determined differences between infant and adult cortical auditory evoked potentials for a range of speech and tonal stimuli. CAEPs were recorded at C3, Cz, and C4 electrode locations from 14 adults and 20 infants (aged 3-7 months) with normal hearing. Adults were tested using 500, 1k, 2k, and 4k Hz tone bursts and /t/, /k/, /d/, /g/ speech sounds presented at 65 dB SPL. Infants were tested with a subset of these stimuli (500 Hz, 2 kHz, /g/, /t/) plus an additional sound /m/ that was included to achieve greater spectral contrast across speech stimuli. CAEPs were reliably present for all participants at all stimuli. There were substantial differences in the morphology of CAEPs between adults and infants. 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. Infant waveforms showed a broad positivity "P1" at 202 ms following by a late negativity "N_{late}" at 367 ms, on average. Few infants had a double-peaked P1 and responses with this morphology varied across stimuli. The most robust stimulus differences were seen in infants for /t/ versus /m/, two stimuli with very different spectral and temporal characteristics. The finding of significant CAEPs differences between speech stimuli is consistent with earlier studies indicating that cortical potentials provide objective evidence of the neural encoding of speech characteristics.

Purdy et al. (2005) recorded CAEP responses in 42 normal hearing infants and 38 infants and children with hearing impairment. Several speech stimuli (e.g. /mae/, /tae/ etc) were presented at normal conversational level (65dB SPL). They reported that aided CAEPs can be successfully recorded at this level in individuals with mild to severe hearing losses. However the detectability declined for the individuals with profound hearing impairment. For many children with mild to severe hearing impairment, aided responses were sensitive to the frequency content of the stimuli suggesting that this response can be used for fine tuning of the hearing aids.

Trembley et al. in 2006 recorded CAEPs in 7 adult hearing aid users with mild to severe sensory neural hearing loss using acoustic change complex. They used naturally produced speech tokens /si/ and /shi/ from nonsense syllable test. Test was carried out in aided and unaided conditions, for aided two preselected hearing aids common for all the clients were used. They concluded that consonant vowel boundary if preserved by the hearing aid can be detected for each speech sound. However when unaided and aided was compared, only subtle changes in amplitude were seen for/si/ whereas no amplitude changes seen for /shi/ which they attributed to the less amount of gain provided by the hearing aid.

Study done by Kumar, Bhatt, Udupa and Costa in 2011 to check the effect of click stimuli and speech burst on cortical processing in 12 individuals with normal hearing sensitivity. Auditory late latency responses were recorded using click and speech stimuli (/pa/, /ta/, & /ka/). All the components of late latency responses were elicited in all the participants using click stimuli. Among speech stimuli, /ta/ stimulus showed better responses than /pa/ and /ka/ stimuli in eliciting all the components of late potentials. This difference was attributed to the differences in spectral energy present in different stimuli. The difference in latencies when speech stimuli were used can be attributed to spectral differences, duration of stimulus and bandwidth. They

concluded that the speech stimuli can be used to elicit cortical potentials but responses are more accurate with click stimuli. Late latency responses may be used to objectively measure differences in neural encoding and perception of spectrally different stimuli.

Dun, Carter and Dillion in 2012 reported no significant differences in the group averages for amplitude and latency found between the different speech sounds /m/, /g/, and /t/. This is in contrast with Golding et al. (2006) who reported that the /t/ sound evoked cortical responses were significantly larger in amplitude and earlier in latency than for the other two sounds. This might be explained by a younger age group, by their normally hearing status, or by the greater spread in the amplitude and latency distribution due to different stimulus levels in this study when comparing with Golding et al. (2006) who only use one intensity (65 dB SPL).

Effect of stimulus duration on CAEPs

Studies have reported clear effect of duration on N1 and P2 latency and amplitude, with shorter duration stimuli eliciting responses that were significantly larger in amplitude and earlier in latency and also increasing stimulus duration up to approximately 30- 40 ms typically leads to increased N1 amplitude with longer duration producing small changes in CAEP amplitude. (Alain et al, 1997; Eddins & Peterson, 1999).

Onishi and Davis in 1968 studied tone burst of 1000 Hz with linear on and off ramps and plateaus of various durations to evoke cortical (vertex) potentials in five adult subjects. With 30 msec rise time, the amplitude, from N_1 peak to P_2 trough, and the latency to either the N_1 peak or P_2 trough were all independent of duration of the plateau from 0 to 300 msec. With 3 msec rise time, the amplitudes were progressively reduced when the plateau was shortened from 30 msec to 10, 3, or 0 msec. With a long plateau, the amplitudes were nearly constant for rise times of 50

msec or less. For evoked-response audiometry, the rise time of the test signal will not be critical if it is 30 msec or less and the plateau is at least 30 msec long, and any plateau of 30 msec or longer will be acceptable.

Kodera, Hink, Yamada and Suzuki (1979) studied effect of linear rise times (5, 10, 20 ms) with 1000 Hz tone burst at 60 dB SPL on 8 normal hearing adults between 24 to 32 years. Results indicated that longer rise times were associated with longer latencies and smaller amplitudes. CAEP studies show variability in stimulus duration when naturally produced speech stimuli were used, with the duration ranging from 300 ms (Ostroff, McDonald, Schneider & Alain, 2003) to 756 ms (Tremblay et al., 2003).

Effect of inter stimulus interval on CAEPs

CAEPs are highly dependent on inter stimulus interval (ISI) as reported by several researchers (Davis et al., 1966; Hari et al., 1982; Rothman, Davis & Hay, 1970). The duration of the stimuli used in eliciting CAEPs is about 50-60 ms or even longer. Consequently total accumulated duration constitutes a considerable portion of the analysis time. Also for evoked potentials the recovery time is longer (Hall, 1992). Though latency doesn't change markedly, the amplitude increases as ISI is lengthened, and concomitantly the stimulus rate is decreased. (Davis et al., 1966; Hari et al., 1982; Rothman et al, 1970). They also reported that more increase in amplitude occurs for ISI lengthened up to 8 seconds and at higher intensity levels. For ISI values greater than 4 seconds yielded larger amplitude (Hari et al., 1982).

Tremblay, Billings and Rohila in 2004 examined the effects of stimulus complexity and stimulus presentation rate in 10 younger and older normal-hearing adults. They used 1 kHz tone burst as well as a speech syllable to elicit the N1-P2 complex. Three different inter stimulus

intervals were used (510, 910, and 1510 msec). When stimuli were presented at the medium presentation rate, N1 and P2 latencies were prolonged for older listeners in response to the speech stimulus. These age effects were absent when stimuli were presented at a slower rate. Results from this study suggest that rapidly occurring stimulus onsets, either within a stimulus or between stimuli, result in prolonged N1 and P2 responses in older adults. This is especially true when processing complex stimuli such as speech. One potential explanation for this age effect might be age related refractory differences in younger and older auditory systems. Refractory issues might in turn affect synchronized neural activity underlying the perception of critical time varying speech cues and may partially explain some of the difficulties older people experience understanding speech.

METHOD

The objective of the study was to investigate the effect of intensity and the effect of different speech stimuli (/m/, /t/ & /g/) on hearing aid users using speech evoked cortical potential. To reach this objective the participants were divided into two groups.

Participants

A total of 23 children (40 ears) in the age range of 6 to 9 years were participated in the study. The participants were divided into following groups.

- Control group consisted of 10 children (20 ears) with normal hearing.
- Clinical group consisted of 13 (20 ears) children with moderate to moderately severe sensorineural hearing impairment.

Participant's selection criteria

The participants were selected for control group on the following criteria.

- Hearing sensitivity was within normal limits i.e, < 15 dB HL for octave frequencies between 250 to 8000 Hz for air conduction, from 250 Hz to 4000 Hz for bone conduction. Immittance evaluation showed normal middle ear functioning with 'A' type tympanogram and presence of acoustic reflexes at 500, 1000, 2000 and 4000 Hz. Normal auditory brainstem response and TEOAE were present in all the participants.
- They had no relevant otologic or neurologic history and no illness at the day of testing.
- Participants were excluded from the study who had clinically abnormal click-evoked ABR findings and abnormal middle ear functioning.

The participants were selected for clinical group on the following criteria.

- Participants in this group included moderate to moderately severe sensorineural hearing loss children who were diagnosed based on either threshold estimation using auditory brainstem responses or conditioned pure tone audiometry. Their pure tone thresholds were in between 41 dB HL to 70 dB HL with air bone gap less than 10 dB. They had normal middle ear functioning based on immittance evaluation. Retrocochlear pathology was ruled out based auditory brainstem responses and transient evoked otoacoustic emission tests.
- Aided audiogram was done for each participant and their responses was well within the speech spectrum at least up to 2 kHz.
- Participant from the clinical group were excluded from the study if they had middle ear pathology and aided audiogram not within the speech spectrum at least up to 2 kHz.
- They had no relevant otologic or neurologic history and no illness at the day of testing.

Testing environment

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

Instrumentation

The below mentioned instruments were used to carry out the study:

- A double channel clinical audiometer (Orbitor-922) was used for Conditioned audiometry, unaided and aided audiogram.

- A Grason Stadler (GSI) Tympstar Immittance meter was used for tympanometry and reflexometry in order to rule out the middle ear pathology.
- An evoked potential system, Bio-logic Navigator Pro (version 7.0) was used for ABR threshold estimation to check the integrity of neural pathway at the level of brainstem.
- HEARLab system (Frye Electronics Tigard, USA, version 1.0) was used for recording CAEPs with hearing aids and unaided in normal hearing children.
- All the above mentioned instruments were calibrated prior to use according to manufacturer's recommendations.

Procedure

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

Phase 1: Hearing evaluation and aided audiogram

Before Audiological evaluation, a detailed case history was obtained and otoscopy was completed to rule out active pathological conditions. The participant's ear canal was free of excessive cerumen and proceeded with following evaluations.

- Conditioned pure tone audiometry 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. It was carried out in double-room situation. The children were instructed to indicate whenever the tone is heard, no matter how faint it may be by raising and lowering the finger, hand/arm or to put rings on a peg according to how well they understood the instructions.

- Speech identification scores were obtained at 40 dB above the Speech recognition threshold (SRT) using speech stimuli developed by Vandana (1998). The normal hearing participants were expected to repeat the words as they were presented by the clinician.
- 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. Appropriate probe tips were used to obtain hermatic seal.
- TEOAE was measured using the default setting in the instrument (ILO- V6) using non-linear click trains of 260 sweeps. Foam tip was properly positioned in the external auditory canal to get a flat frequency spectrum across the frequency. The overall TEOAEs amplitude of 6 dB SPL above the noise floor with the reproducibility 80% was considered as presence of TEOAEs (Dijk & Wit, 1987).
- The electrophysiological testing included click-evoked ABR testing to verify normal hearing thresholds in control group and moderate to moderately severe sensorineural hearing loss in clinical group and 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 gel, 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 for click evoked ABR, and CAEPs.
- Aided audiogram using conditioned 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 on regular basis and their hearing aids were programmed with appropriate gain.
- Electroacoustic measurement was carried out to check the performance of the hearing aid used before CAEPs recording.

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 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 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 protocol for aided cortical potentials were used, as mentioned in table 1.

Statistical Analysis

Statistical analysis was done using SPSS (Version 18), which includes descriptive statistics, repeated measure ANOVA, one way ANOVA, independent t-test, and Bonferroni pair wise comparison tests.

Table 1: *Test Protocol for Click- Evoked ABR for threshold estimation, neurological evaluation and aided cortical auditory evoked potentials*

Parameters	Click evoked ABR for threshold estimation	Click evoked ABR for neurological evaluation	Aided cortical Auditory evoked potential
Stimulus	Click (100 µs duration)	Click (100 µs duration)	/m/ (30 ms), /g/ (30 ms) and /t/ (30 ms)
Transducer	Insert ear phones	Insert ear phones	Loudspeaker at 0^0 azimuth
Electrode Placement	Active- Fz Reference- M1 Ground- M2	$C_z - A_1$ and $C_z - A_2$	Active- Fz Reference- M1 Ground- M2
Intensity	80 dB nHL	90 dB nHL	55 dB SPL, 65 dB SPL, 75 dB SPL
Polarity	Alternating	Alternating	Alternating
Filter setting	100 - 3000 Hz.	100-3000Hz	2-30 Hz
Repetition rate	30.1/sec	11.1/sec and 90.1/sec	1.1/sec
Number of sweeps	2000	1500-2000	200
Impedance	< 5k Ω	< 5k Ω	< 5k Ω
No. of Channels	One	two	One
Analysis Time	10 ms	10ms	500 ms
Amplification	100,000	100,000	50,000

RESULTS AND DISCUSSION

Data of twenty-three children, which includes 10 children with normal hearing (20 ears served as control group) and 13 children with moderate to moderately severe hearing impairment (20 ears served as clinical group) were recorded. The speech evoked cortical potentials were successfully recorded in all tested children. A total of 360 recordings (180 aided recording from children with hearing impairment & 180 unaided recording from normal hearing children) were completed using three different speech stimuli at three different intensity levels.

The targeted cortical potential consisted of four components i.e. P1, N1, P2 and N2 waves. However, it was not obligatory to find all waves in the complex potential such as speech evoked cortical potential in younger children. Further, wave P1 and N1 wave was the early wave observed in children without hearing impairment in all tested ears. In present study using HEARLab system, only wave P1 and N1 could be identified using different speech stimuli at different presentation levels in children with hearing impairment in the age range of 6-9 years. In few children with normal hearing and hearing impairment, other two waves P2 and N2 was traceable but not mentioned in the present study because of inconsistency in identification of these two peaks. Hence, further only wave P1 and N1 is described in details. An example of the waveforms and data analysis for one child with hearing impairment is shown in the figure 1A &1B.

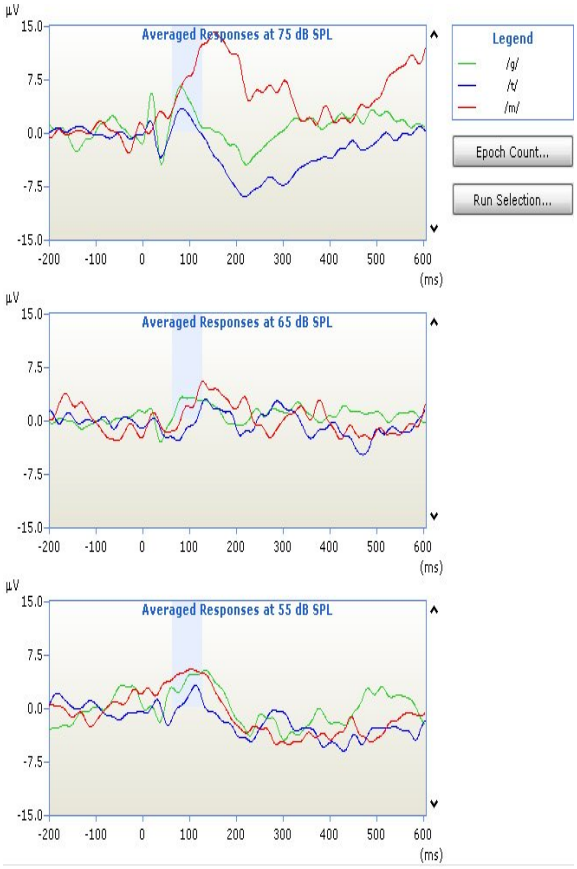


Figure 1A

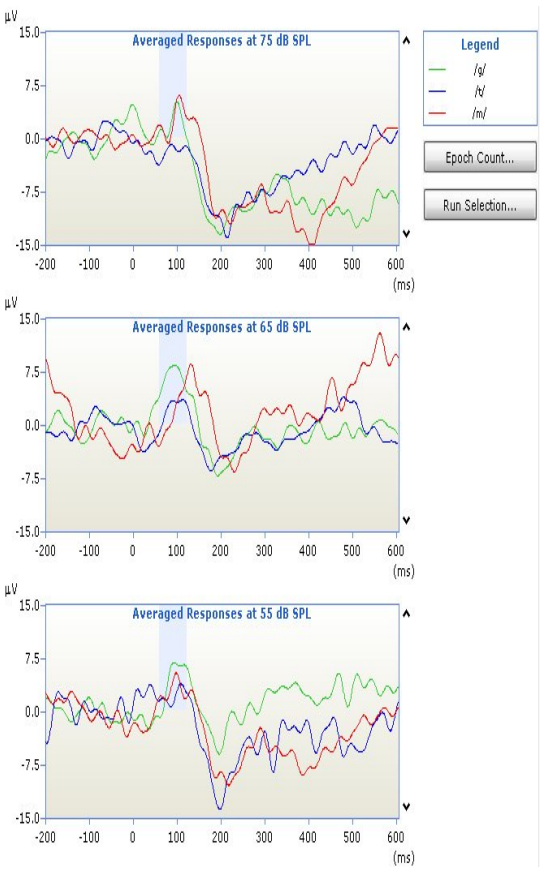


Figure 1B

Figure 1A & 1B: A sample waveform of the speech evoked cortical potential from a typical child with hearing impairment using hearing aids and child with normal hearing respectively (/m/ -red color; /t/-blue color; /g/-green color).

Latencies and amplitudes of wave P1 and N1 in children with normal hearing

Speech evoked cortical potential were recorded from children with normal hearing using three different speech stimuli (/m/, /t/, & /g/) at three different intensities (75 dB, 65 dB, & 55 dB). Descriptive statistics was done to find out mean and standard deviation (SD) for latency and amplitude measures for wave P1 and N1 at different intensity. The table 2 shows mean and standard deviation for P1 and N1 wave for different speech stimuli at 75 dB SPL, 65 dB SPL and 55 dB SPL respectively for both ears. From table 2, it is evident that there is higher standard deviation for both latency and amplitude measures of wave N1, which indicate could be because of higher variability observed in identification of wave N1 at different intensity levels (table 2).

Latencies and amplitudes of wave P1 and N1 in children with hearing impairment

Speech evoked cortical potentials were recorded from children with hearing impairment using three different speech stimuli (/m/, /t/, & /g/) at three different intensities (75 dB SPL, 65 dB SPL, & 55 dB SPL). Descriptive statistics were used to find out mean and standard deviation (SD) for latency and amplitude measures for wave P1 and N1 in children with hearing impairment. The table 3 shows mean and standard deviation for P1 and N1 wave for different speech stimuli at 75 dB SPL, 65 dB SPL and 55 dB SPL respectively for both ears. From table 3, it is evident that there is higher standard deviation for both latency and amplitude measures of wave N1, which could be because of higher variability observed in identification of wave N1 at different intensity levels (table 3).

Table 2: *Mean and standard deviation (SD) of children with normal hearing sensitivity for P1 and N1 latencies and amplitudes at 75 dB SPL, 65 dB SPL and 55 dB SPL for the three speech stimuli.*

Stimuli	Peaks	Ear	Latency		Amplitude		Latency		Amplitude		Latency		Amplitude	
			Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
/m/	P1	LE	79.66	9.86	2.25	2.15	84.72	13.02	4.42	3.75	80.12	6.85	5.67	3.18
		RE	82.00	19.04	2.95	2.95	80.57	14.69	4.39	3.01	81.80	16.72	4.01	3.43
	N1	LE	150.25	52.65	-2.40	8.35	165.54	52.50	-2.54	7.06	153.50	46.75	-1.50	3.79
		RE	149.50	49.74	1.00	7.33	143.28	32.95	-1.40	4.68	154.20	50.31	-1.45	3.49
/t/	P1	LE	81.58	17.41	3.08	3.13	80.09	19.28	3.87	2.50	84.12	18.04	2.86	3.21
		RE	82.50	15.50	2.80	1.64	74.14	8.57	3.43	1.56	71.80	15.95	2.07	1.72
	N1	LE	157.50	50.85	-4.20	6.44	151.36	39.54	-4.46	5.97	166.37	48.04	-2.73	4.31
		RE	151.33	36.46	0.43	4.37	145.00	40.34	1.56	4.17	133.20	33.44	1.19	4.70
/g/	P1	LE	74.91	12.19	3.34	1.91	83.54	17.57	4.15	3.02	85.00	20.44	3.84	4.14
		RE	75.50	11.53	3.62	1.68	77.85	7.69	4.65	2.79	80.20	19.03	2.24	4.85
	N1	LE	151.91	48.72	-3.94	4.50	160.72	51.03	-1.01	7.70	156.37	53.28	-1.59	4.69
		RE	149.55	42.20	-2.99	4.56	143.00	37.56	0.73	3.19	160.80	68.30	-1.82	3.15

LE= Left ear; RE = Right ear; SD= standard deviation

Table 3: *Mean and standard deviation (SD) of children with hearing impairment for P1 and N1 latencies and amplitudes at 75 dB SPL, 65 dB SPL and 55 dB SPL for the three speech stimuli.*

Stimuli	Peaks	Ear	Latency		Amplitude		Latency		Amplitude		Latency		Amplitude	
			Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
/m/	P1	LE	128.08	41.02	2.32	2.31	127.67	36.11	2.22	5.62	131.90	35.88	2.71	2.54
		RE	123.57	35.88	2.94	2.72	143.62	42.98	3.18	3.27	122.42	15.30	3.50	2.60
	N1	LE	215.91	56.28	-6.09	3.18	217.33	50.29	-6.94	4.92	228.10	31.72	-4.27	3.42
		RE	215.57	46.38	-7.27	6.02	224.50	25.76	-4.62	2.63	235.00	26.08	-5.33	2.88
/t/	P1	LE	119.16	40.31	3.53	3.39	118.91	34.44	3.15	4.30	112.80	36.52	3.08	3.10
		RE	118.14	27.44	3.57	3.08	131.87	18.79	3.74	1.91	124.57	22.86	0.88	0.43
	N1	LE	198.58	61.54	-3.33	4.20	212.33	42.13	-4.10	3.45	207.30	50.85	-4.17	4.69
		RE	210.10	24.72	-6.34	2.53	209.62	17.66	-3.00	2.69	219.47	37.52	-5.91	3.59
/g/	P1	LE	132.58	24.24	0.82	2.81	118.25	30.23	1.30	4.05	112.90	35.91	1.88	1.77
		RE	119.00	38.12	4.43	2.89	119.25	32.91	1.94	6.43	152.00	16.15	-2.68	0.55
	N1	LE	223.83	27.10	-6.26	3.10	204.41	44.01	-6.60	3.93	204.80	47.21	-5.18	3.58
		RE	216.00	18.04	-4.68	3.64	201.12	17.84	-5.13	4.44	224.00	22.21	-3.04	6.31

LE= Left ear; RE = Right ear; SD= standard deviation

Effect of intensity on latency and amplitude measures for children with hearing impairment

Repeated measure ANOVA was carried out to find the effect of different intensities on latency and amplitude measures for wave P1 and N1.The details of these measures are mentioned under individual headings.

Wave P1 latency

Repeated measures ANOVA shows no significant main effect across intensities for wave P1 latency [F (2,112) = 1.28, *p>0.05*] of speech evoked cortical potentials in children with hearing impairment. Further, there was no interaction observed for different speech stimuli with respect to different intensity [F (2,112) = 0.35, p>0.05]. Figure 2 shows the mean and 95% confidence interval (CI) for wave P1 latency for different intensity levels.

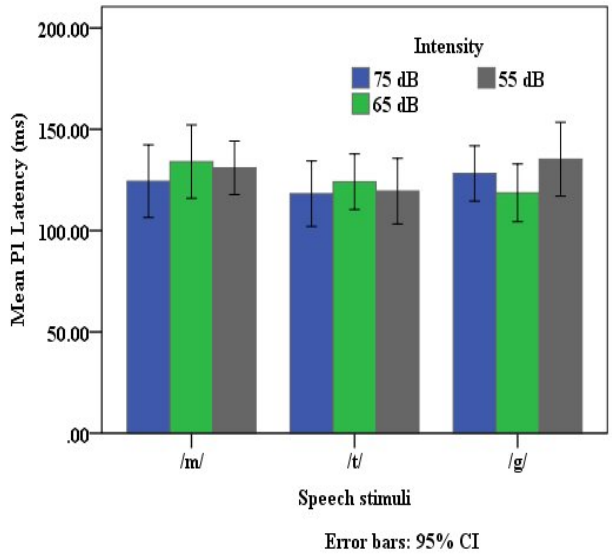


Figure 2: *Mean and 95% confidence Interval (CI) for P1 latency (ms) at different intensities for different speech stimuli*.

From figure 2 it is clear that there is shorter latency measure for P1 wave at 75 dB SPL in comparison to other two intensity levels for different speech stimuli. Further, for /m/ and /t/ sounds, the longer latency observed at 65 dB SPL. However, for /g/ sounds, longer latency was noticed at 55 dB SPL. Though the differences were observed at each speech stimuli, it was not statistically significant at different intensity levels.

Wave P1 amplitude

Repeated measures ANOVA shows no significant main effect across intensities for wave P1 amplitude [F (2,112) = 2.50, p>*0.05*] of speech evoked cortical potentials in children with hearing impairment. Further, there was no interaction observed for different speech stimuli with respect to different intensity [F (2,112) = 0.68, *p>0.05*]. Figure 3 shows the mean and 95% confidence interval (CI) for wave P1 amplitude for different intensity levels

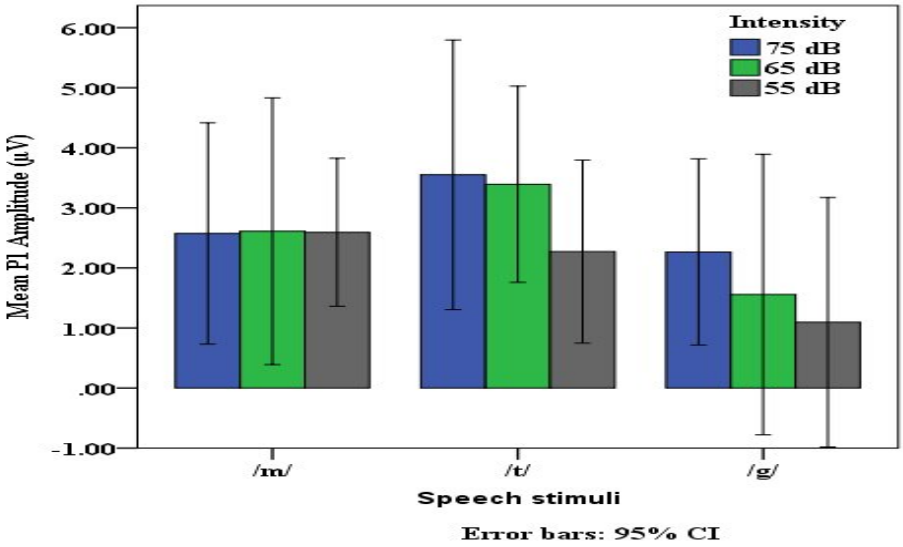


Figure 3: *Mean and 95% confidence Interval (CI) for wave P1 amplitude (µV) at different intensities for different speech stimuli.*

From figure 3, we can infer that amplitude measures for wave P1 at different intensity, which represents higher amplitude at 75 dB SPL for /t/ and /g/ sounds in comparison to lowest intensity levels, though it was not statistically significant. Further, it is also observed that there is higher SD at lowest intensity levels (55 dB SPL), which could be because at threshold levels cortical responses are not easy to identify.

Wave N1 latency

Repeated measures ANOVA shows no significant main effect across intensities for wave N1 latency [F (2,108) = 2.69, *p>0.05*] of speech evoked cortical potentials in children with hearing impairment. Further, there was no interaction observed for different speech stimuli with respect to different intensity [F (2,108) = 0.11, *p>0.05*]. Figure 4 shows the mean and 95% confidence interval (CI) for wave N1 latency for different intensity levels.

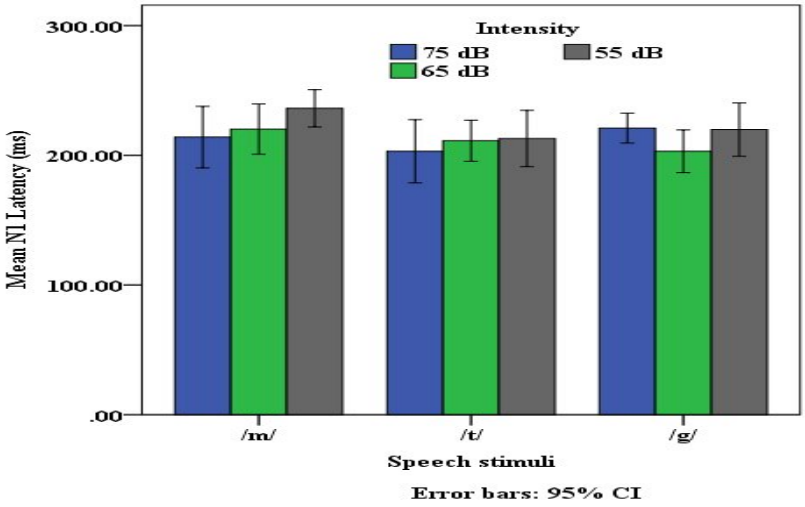


Figure 4: *Mean and 95% confidence Interval (CI) for N1 latency (ms) at different intensities for different speech stimuli.*

From figure 4, it is clear that there is shorter latency measure for N1 wave at 75 dB SPL in comparison to other two intensity levels for different speech stimuli. Further, for /m/ sound, the longer latency was observed at 55 dB SPL. However, for /t/ and /g/ sounds, there is prolongation of latency as intensity reduces though it was not statistically significant for different speech stimuli. Though the differences were observed at each speech stimuli, it was not statistically significant at different intensity levels.

Wave N1 amplitude

Repeated measures ANOVA shows no significant main effect across intensities for wave N1 amplitude [F (2,110) = 2.08, p>*0.05*] of speech evoked cortical potentials in children with hearing impairment. Further, there was no interaction observed for different speech stimuli with respect to different intensity [F (2,110) = 2.42, *p>0.05*]. Figure 5 shows the mean and 95% confidence interval (CI) for wave N1 amplitude for different intensity levels.

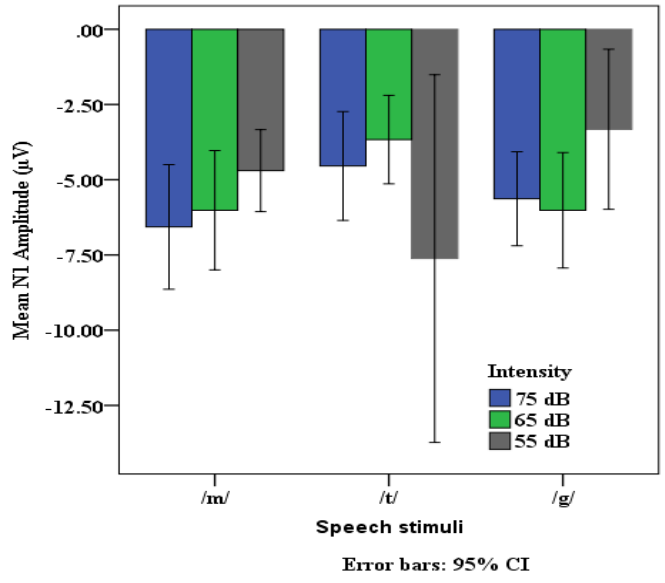


Figure 5: *Mean and 95% confidence Interval (CI) for N1 amplitude (µV) at different intensities for different speech stimuli.*

From figure 5 we can infer that amplitude measures for wave N1 at different intensity, represents higher amplitude at 55 dB SPL for /t/ stimuli and there is no statistical difference in N1 amplitude across intensities for /m/, and /g/ stimuli. Further, it is also observed that there is higher SD at lowest intensity levels (55 dB SPL), which could be because at threshold levels cortical responses are not simple to identify.

Further, Bonferroni pair wise comparison was done to check the differences observed at each intensity level for different combination of speech sounds. No significant differences were observed between pairs of different speech stimuli for latency as well as amplitude measures of wave P1 and N1.

As expected, the detection of cortical responses in hearing aid users reduced (lower) with presentation level, especially at the lowest presentation level where the signal is close to threshold. The 55 dB SPL presentation level corresponds to a sensation level of approximately 30 to 40 dB in normal-hearing listeners (Golding et al., 2009). Present study included only those children who were having aided responses well within speech spectrum at least up to 2 kHz. Probably that could be the reason to show responses even at threshold levels.

Further, Garinis and Cone-Wesson (2007) have also reported decrease in CAEP amplitudes for tonal and speech stimuli with decrease in stimulus level from supra threshold levels (70 or 50 dB SL) down to levels at or close to behavioral threshold (30 or 0 dB SL). However, Morita et al. (2007) observed that detection of cortical responses easier for stimuli at the same sensation level for listeners with sensorineural hearing loss (SNHL) because of the potential impact of loudness recruitment, which is likely to enhance CAEP amplitudes at low sensation levels.

Even in normal-hearing adults, it is observed that as intensity increases, latencies of different peaks P1, N1, P2, and N2 decreases and their amplitudes increases (Koravand et al., 2012). However, the results of the current study were partly in agreement with above finding. It could be because of differences in methodology as well as different factors responsible for variability in younger population where cortical structures are still under maturation. In contrast

Koravand et al (2012) have reported that the level of presentation could affect differently on latency and amplitude in children with hearing impairment.

Effect of different speech stimuli on latency and amplitude measures in children with hearing impairment

One-way ANOVA was carried out to see the effect of different speech stimuli on the latency and amplitude measures. The speech stimuli include /m/, /t/ and /g/ sounds which represents low, high and mid frequencies. The outcomes of one way ANOVA are mentioned in Table 9.

Table 4: O*ne-way ANOVA outcomes for latency and amplitude measures at different intensities*

Stimulus intensity	**Peaks**	**Latency**		**Amplitude**	
		F value	**P-value**	**F value**	**P value**
75 dB	P1	F(2,57) = 0.43	0.64	F(2,57) = 0.54	0.58
	N1	F(2,55) = 0.80	0.45	F(2,57) = 1.35	0.26
65 dB	P1	F(2,57) = 1.11	0.33	F(2,57) = 0.85	0.43
	N1	F(2,57) = 1.06	0.35	F(2,57) = 2.47	0.09
55 dB	P1	F(2,55) = 1.13	0.32	F(2,55) = 1.01	0.36
	N1	F(2,54) = 1.78	0.17	F(2,55) = 1.46	0.24

The results revealed no significant differences between different speech stimuli for amplitudes and latencies measures for wave P1 and N1 at 75 dB, 65 dB and 55 dB. The above finding is in agreement with the results obtained by different researchers (Dunn, Carter & Dillion, 2012; Hassan, 2011; Chang et al., 2012; Koravand, 2012). Hassan (2011) who reported no

significant difference between the latencies with different stimuli (/ga/ & /wa/ syllable), despite the /ga/ stimuli consistently produced waves with shorter latencies.

In contrast, studies have shown that it is possible to observed differences in response based on uses of different speech stimuli (Ostroff et al., 1998; Golding et al., 2006; Tremblay et al., 2003; Shruti & Vanaja, 2007). Shruti and Vanaja (2007) using /i/, /m/ and /□/ stimuli reported longer latencies in high frequency stimuli compared to low frequency stimuli and all the three stimuli are significantly different from one another for the wave P1 and N2 responses in hearing impaired children. Similar finding was also observed in the study done by Apeksha and Devi (2008) in individuals with hearing impairment in which they used /ba/, /da/ and /ga/ stimuli and reported prolonged latencies at high frequency stimuli.

Munro et al (2012) found that out of 24 individuals it was possible to differentiate between /m/ and /t/ stimuli only in 35% of cases where responses could be detected. Further, for /m/ and /g/ as well as /t/ and /g/ were detected as responses approximately 60% and 50% respectively in simulated hearing loss condition. However, the plosive speech contrasts /t/ and /g/ used in their study (which are relatively similar in spectral content and amplitude-time envelopes) did not reliably result in different CAEP waveforms. They concluded differentiation of CAEP waveforms between speech stimuli in individual participants, using the HEARLab's stimulus and analysis protocol, is not a reliable method for objectively demonstrating that the brain has discriminated between these speech stimuli which agrees with the results of the present study. Probably for this reason, CAEP waveform differentiation between speech stimuli available in the prototype software has been removed before commercial release of the HEARLab evoked potential system (Dillon, Personal communication).

Garinis and Cone-Wesson (2007) reported similar amplitude and latency differences between CAEP waveforms for the speech stimuli /sa/ and /da/ for presentation levels of 0 to 40 dB SL. Based on above studies, it is likely that the CAEP waveforms were similarly differentiated across different presentation levels in the present study, but the reduction in signal to noise ratio at low intensity levels could have affected the ability to show statistically different waveforms in individuals. Korczak et al. (2005) reported that the cortical responses of adults with hearing impairment to /ba/ and /da/ speech stimuli showed shorter latencies, larger amplitudes, better CAEP waveform morphologies, improved CAEP detectability, and increased behavioral performance in aided condition in comparison to unaided condition. However, the amount of improvement between unaided and aided was variable among individual.

With normal hearing individuals, CAEP waveforms were possible to be differentiated in many individual listeners if not in all the individuals, particularly for the two most acoustically disparate stimuli. As it has been reported elsewhere, it is possible to differentiate obligatory CAEP waveforms for different stimuli in group data (Beukes et al., 2009), but this is not sufficiently reliable on consideration of individual data, at least for adult listeners, when using the HEARLab's stimulus and analysis protocol. Further, this may also reflect the usual problem of far-field recordings using scalp electrodes. Although neural activity within CAEP generators should differ between stimuli, especially for stimuli that are temporally and spectrally very different, the electrical potential recorded at the surface of the scalp after transmission through the brain and the bony skull will have reduced amplitude and blurred spatial distribution compared with the potential at the source.

Comparison between two groups for latency and amplitude measures

Independent t-test was done to compare the performance between control 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 10 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 moderate to moderately severe 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 P1 wave response for /t/ stimuli (Table 10).

Table 5: *Independent t-test outcomes for latency and amplitude measures*

	Speech stimuli	Peaks	t-Value	p- value
Latency	**/m/**	P1	9.76	0.00**
		N1	23.68	0.00**
	/t/	P1	-4.58	0.00**
		N1	5.94	0.00**
	/g/	P1	27.59	0.00**
		N1	44.82	0.00**
Amplitude	**/m/**	P1	-40.12	0.00**
		N1	-26.77	0.00**
	/t/	P1	-1.05	0.29#
		N1	-7.82	0.00**
	/g/	P1	3.79	0.00**
		N1	-3.29	0.00**

***P<0.05, #P>0.05*

The above findings are in agreement with previous researchers (Koravand, 2012; Thabet & Said, 2012; Apeksha & Devi, 2010; Golding et al, 2009). Golding et al. (2009) recorded CAEPs which were reliably present at 30 dB SL, but not at lower sensation levels in normal-hearing listeners. There are several factors which could be an account for differences in findings such as participants age, onset of hearing loss, type and/or degree of hearing loss, level of stimulus presentation, and type of stimuli used.

In addition to that, Koravand (2012) recorded wave P1, N1, P2, and N2 clearly in children with normal hearing with the three different speech stimuli in 9 to 12 year olds. In contrast, the P2 and N2 components were not well defined in few children with hearing loss. Therefore, only the P1 and N1 components, which were clearly identified in all participants, were analyzed. If the

hearing system is stimulated in early stages, the P1 morphology and latency observed to reach age related normal values within 3 to 6 months following the beginning of auditory stimulation. In 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. The P1 amplitude observed in children with hearing loss could be the reflection of limited plasticity. However, the amplification provided by the hearing aids could have certainly contributed to get under way the maturational processes but it was not probably sufficient to supply entirely the effect of the auditory deprivation.

The different components of cortical auditory evoked potentials showed differences in emerging with reference to ages. The less prominent components of CAEPs (P2 and N2) which were less defined in children with hearing loss compared to their peers with normal hearing. These two components do not emerge constantly until the age of 8 to 11 years in children with normal hearing (Purdy, 2003; Ponton, 1984). The nonappearance of these peaks or their affected morphology in children with hearing impairment even at later stages could be manifestation of a delayed maturation of the central auditory nervous system.

Based on this study, the reduction in amplitude and latency of P1 and N1 in children with hearing impairment in the present study could be explained by a delay in maturation of the central auditory nervous system (Koravand et al., 2012). Taking into account the abnormal morphology of N1 and P2, the N2 changes would rather militate in support of delayed maturation in children with hearing loss.

With amplification, a neural response pattern tends to show a typical representation as observed in normal-hearing children that is for wave P1 response, followed by a prominent negativity. However, results have not always been consistent with above finding (Souza &

Tremblay, 2006). Further, they also observed that in normal hearing individuals, increase in intensity level result in significant waveform changes (Souza & Tremblay, 2006). Therefore, with increased in stimulus intensity levels (provided by the hearing aid), we expected aided evoked neural responses to be larger in amplitude and shorter in latency than the unaided recordings. The fact that we did not scrutinize these likely patterns of change suggests that long-standing principles underlying CAEPs recordings, when sound is processed and delivered to the auditory system through a hearing aid. Hence, there is a need to be careful while interpretation made for latency and amplitude patterns recorded in aided and unaided conditions reflect similar neural properties.

Most probably, some sort of interface occurs between the way sound is processed by the hearing aid and encoded in the auditory system. It is necessary to understand this interaction before aided evoked recordings can be interpreted. Further, when evoked potentials are a recorded using speech stimulus that is delivered through a hearing aid, the effects of hearing aid processing on the physical characteristics of the sound likely affect the evoked neural response pattern at cortical levels.

SUMMARY AND CONCLUSION

To aim of the present study was to measures speech evoked cortical potentials using speech tokens such as /m/, /g/ and /t/ in children with moderate to moderately severe hearing impairment. There were total of 23 children (40 ears) in which 10 normal hearing children and 13 children with hearing impairment in the age range of 6 to 9 years participated in the study.

From the present study we conclude that, the presence or absence of CAEPs can provide some indication of the audibility of a speech sound for individuals with sensorineural hearing loss. It was expected that the CAEP responses would vary from subject to subject. Response differentiation was relatively poor, especially at low presentation levels, using the HEARLab's stimulus and analysis protocol. Although we obtained CAEP responses in a limited number of children and in a restricted age range, these preliminary findings indicate that reduced auditory input early in life has an impact on the development of central auditory functions reflected by the specific patterns of CAEPs. Measuring P1 and N1, as the neurophysiological markers in children with hearing loss, can provide an objective assessment of the maturation of their central auditory system.

1) There is higher standard deviation for both latency and amplitude measures of wave N1 in normal hearing and hearing impaired children, which could be because of higher variability observed in identification of wave N1 at different intensity levels.
2) Though the differences were observed at each speech stimuli, it was not statistically significant at different intensity levels.
3) There is shorter latency measure for P1 wave at 75 dB SPL in comparison to other two intensity levels for different speech stimuli.

4) Amplitude measures for wave P1 at different intensity, which represents higher amplitude at 75 dB SPL for /t/ and /g/ sounds in comparison to lowest intensity levels, though it was not statistically significant.
5) There is shorter latency measure for N1 wave at 75 dB SPL in comparison to other two intensity levels for different speech stimuli.
6) Amplitude measures for wave N1 at different intensity, represents higher amplitude at 55 dB SPL for /t/ stimuli and there is no statistical difference in N1 amplitude across intensities for /m/, and /g/ stimuli.
7) No significant differences between different speech stimuli for amplitudes and latencies measures for wave P1 and N1 at 75 dB, 65 dB and 55 dB.
8) There is significant difference between Latency and amplitude of hearing impaired and normal hearing children.

REFERENCES

Agung, K., Purdy, S. C., McMahon, C. M., & Newall, P. (2006). The use of cortical auditory evoked potentials to evaluate neural encoding of speech sounds in adults. *Journal of the American Academy of Audiology, 17(8),* 559-572.

American National Standards Institute. (1991). Maximum permissible ambient noise levels for audiometric test rooms (ANSI S3.1-1991). New York: Acoustical Society of America.

Antinoro, F., Skinner, P. H., Jones, J. J. (1969). Relation between sound intensity and amplitude of the AER at different stimulus frequencies. *Journal of the Acoustical Society of America, 46,* 1433–6

Beukes, E. W., Munro, K. J., & Purdy, S. C. (2009). Duration-sensitive neurons in the auditory cortex. *Neuroreport*, *20*, 1129–1133.

Billings, C., Tremblay, K., Souza, P., & Binns, M. (2007). Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. *Audiology and Neurotology*, *12*, 234-46.

Sörnmo. L., & Laguna, P. (2005). Bioelectrical Signal Processing in Cardiac and Neurological Applications. Academic Press (Elsevier).

Boothroyd, A. (1991). Assessment of speech perception capacity in profoundly deaf children. *American Journal of Otology, 12,* 67-72.

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

Carter, L., Dillon, H., Seymour, J., Seeto, M., & Dun, B.M. (2013). Cortical auditory-evoked potentials (CAEPs) in adults in response to filtered speech stimuli. *Journal of American academy of audiology, 24(9),* 807-22.

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

Chang, H. W., Dillon, H., Carter, L., Dun, B. V., & Young, S. T. (2012). The relationship between cortical auditory evoked potential (CAEP) detection and estimated audibility in infants with sensorineural hearing loss. *International Journal of Audiology, 51,* 663–670.

Cone, B., & Whitaker, R. (2013). Dynamics of infant cortical auditory evoked potentials (CAEPs) for tone and speech tokens. *International Journal of Pediatric Otorhinolaryngology*, *77(7),* 1162-1173.

Crottaz-Herbette, S., & Ragot, R. (2000). Perception of complex sounds: N1 latency codes pitch and topography codes spectra. *Clinical Neurophysiology, 111,* 1759-1766.

Davis, H. (1965). Slow cortical responses evoked by acoustic stimuli. *Acta Otolaryngologica*, *59,* 179-185.

Davis, H., Mast, T., Yoshie, N., & Zerlin, S. (1966). The slow response of the human cortex to auditory stimuli: Recovery process. *Electroencephalography and Clinical Neurophysiology, 21(2),* 105-113.

Davis, H., Bower, C & Harish, S. K. (1968). Relation of human vertex potentials to acoustic input loudness and masking. *Journal of Acoustic Society of America, 43,* 431-438.

Dijk, P. V., & Wit, H. P. (1987). The occurrence of click-evoked otoacoustic emissions ("kemp echoes") in normal-hearing ears. *Scandinavian audiology, 16,* 62-4.

Dillon, H. (2005). So, baby, how does it sound? Cortical assessment of infants with hearing aids. *Hearing Journal*, *58*, 10-7.

Dimitrijevic, A., John, M. S., Roon, P. V & Picton, T. W (2001). Human auditory steady state responses to tones independently modulated in both frequency and amplitude. *Ear and Hearing, 22 (2),* 100-111

Dorman, M.F., Sharma, A., Gilley, P., Martin, K., & Roland, P. (2007). Central auditory development: Evidence from CAEP measurements in children fit with cochlear implants. *Journal of Communication Disorders*. *40(4),* 284-294.

Dun, B. V., Carter, L., & Dillon, H. (2010). The relationship between cortical auditory evoked potential (CAEP) detection and audibility assessed behaviorally in infants with sensorineural hearing loss. Conducted as part of the hearing crc.

Dun, B.V., Carter, L., & Dillon, H. (2012). Sensitivity of cortical auditory evoked potential detection for hearing-impaired infants in response to short speech sounds. *Audiology Research*, *2*, e13.

Easwar, V., Glista, D., Purcell, D. W., Scollie, S. D. (2012). The effect of stimulus choice on cortical auditory evoked potentials (CAEP): Consideration of speech segment positioning within naturally produced speech. *International Journal of Audiology, 51*, 926–931.

Eddins, A. C., & Peterson, J. R. (1999). Time-intensity trading in the late auditory evoked potential. *Journal of Speech Language Hearing Research, 42,* 516-25.

Eggermont, J. J. (1989). The onset and development of auditory function: contributions of evoked potential studies. *Journal of Speech Language Pathology and Audiology, 13(1),* 5–16.

Eggermont, J. J., Ponton, C. W., Don, M., Waring, M. D., & Kwong, B. (1997). Maturational delays in cortical evoked potentials in cochlear implant users. *Acta Oto-Laryngologica*, *117(2),* 161–163.

Eggermont, J. J., & Ponton, C.W. (2003). Auditory-evoked potential studies of cortical maturation in normal hearing and implanted children: Correlations with changes in structure and speech perception. *Acta Oto-Laryngologica*, *123(2),* 249–252

Friesen, L. M., & Picton, T. W. (2010). A method of removing cochlear implant artifact. *Hearing Research*, *259(1-2),* 95-106.

Frye Electronics Inc, HEARLab System. Operator's Manual, 2010.

Garinis, A. C., & Cone-Wesson, B. K. (2007). Effects of stimulus level on cortical auditory event-related potentials evoked by speech. *Journal of the American Academy of Audiology, 18*, 107–116.

Gilley, P.M., Sharma, A., Dorman, M, & Martin, K. (2005). Developmental changes in refractoriness of the cortical auditory evoked potential. Clinical Neurophysiology. *116(3),* 648–657.

Glista, D., Easwar, V., Purcell, D. W & Scollie, S. (2012). A Pilot Study on Cortical Auditory Evoked Potentials in Children: Aided CAEPs Reflect Improved High-Frequency Audibility with Frequency Compression Hearing Aid Technology. *International Journal of Otolaryngology, 2012,* 1-12.

Golding, M., Purdy, S., Sharma, M., & Dillon, H. (2006). The effect of stimulus duration and inter-stimulus interval on cortical responses in infants. *Australian* and *New Zealand Journal of Audiology*, *28,* 122-36.

Golding, M., Pearce, W., Seymour, J., Cooper, A., Ching, T., & Dillon, H. (2007). The relationship between obligatory cortical auditory evoked potentials (CAEPs) and functional measures in young infants. *Journal of American Academy of Audiology*, *18*, 117-25.

Golding, M., Dillon H., Seymour, J., & Carter, L. (2008). The application of cortical auditory evoked potential (CAEP) recordings in infant hearing aid fitting. Proceedings of the 53rd International Congress of Hearing Aid Acousticians - Leipzig Hearing EUHA 08.

Golding, M., Dillon, H., Seymour, J., & Carter, L. (2009). The detection of adult cortical auditory evoked potentials (CAEPs) using an automated statistic and visual detection. *International Journal Audiology*, *48(12),* 833-842.

Gravel, J. S., Kurtzberg, D., Stapells, D. R., Vaughan, H, G., & Wallace, I. F. (1989). *Case studies. Seminars in Hearing, 10,* 272-287.

Groenen, P. A., Beynon, A.J., Snik, A.F., & van den Broek, P. (2001). Speech-evoked cortical potentials and speech recognition in cochlear implant users. *Scandinavian audiology, 30(1),* 31-40.

Hall, J. W. (1992). Handbook of auditory evoked responses, Massachusetts: Allyn and Bacon.

Hari, R., Kaila, K., Katila, T., Tuomisto, T., & Varpule, T. (1982). Interstimulus interval dependence of the auditory vertex response and its magnetic counterparts: Implications for neural generation. *Electroenchephalography and Clinical Neurophysiology, 54*, 561- 569.

Hassaan, M. R. (2011). Aided evoked cortical potential: An objective validation tool for hearing aid benefit. *Egyptian Journal of Ear, Nose, Throat and Allied Sciences, 12,* 155–161.

Hyde, M., Alberti, P.W., Matsumoto, N. & Yao-Li Li. (1986). Auditory evoked potentials in audiometric assessment of compensation and medicolegal patients. *Annals of Otology, Rhinology and Laryngology, 95,* 514-519.

Hyde, M. (1997). The N1 response and its applications. *Audiology and Neuro-otology, 2,* 281-307.

Jacobson, G. P., Lombardi, D. M., Gibbens, N. D., Ahmad, B. K., & Newman, C. W. (1992). The effects of stimulus frequency and recording site on the amplitude and latency of multichannel cortical auditory evoked potential (CAEP) component N1. Ear and Hearing, *13,* 300–6.

Kelly, A. S., Purdy, S. C., & Thorne, P. R. (2005). Electrophysiological and speech perception measures of auditory processing in experienced adult cochlear implant users. *Clinical Neurophysiology, 116(6),* 1235-1246.

Kodera, K., Hink. R. F., Yamda, O., & Suzuki, J. I. (1979). Effects of rise time on simultaneously recorded auditory-evoked potentials from the early, middle and late ranges. *Audiology, 18(5),* 395-402.

Koravand, A., **Jutras, B.** & Lassonde, M. (2012). Cortical auditory evoked potentials in children with a hearing loss: a pilot study. *International Journal of Pediatrics, 2012*, 250-254.

Korczak, P. A., Kurtzberg, D., & Stapells, D. R. (2005). Effects of sensorineural hearing loss and personal hearing AIDS on cortical event-related potential and behavioral measures of speech-sound processing. *Ear and Hearing, 26(2),* 165-85.

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

Kraus, N., & Nicol, T. (2009). Auditory evoked potentials. In Encyclopedia of Neuroscience, Binder MD, Hirokawa N, Windhorst U, eds., Springer: Berlin, pp 214-218.

Kumar, K., Bhat, J. S., Udupa, P. S., & D'Costa, P. E. (2011). Effect of click stimuli and speech bursts on cortical processing. *International Journal of Medical Engineering and Informatics, 3,* 122-129.

Kumari, A., & Devi. (2010). Effect of sensorineural hearing loss and digital hearing aids on speech evoked auditory late latency responses. Unpublished Master's dissertation, University of Mysore, Mysore.

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

Maanen, A. V., & Stappells, D. R. (2005). Comparision of multiple auditory steady-state responses (80 versus 40 Hz) and slow cortical potentials for threshold estimation in hearing impaired adults. *International Journal of Audiology, 44,* 613-624.

Micco, A. G., Kraus, N., Koch, D. B., McGee, T. J., Carrell, T. D., Sharma, A., et al. (1995). Speech-evoked cognitive P300 potentials in cochlear implant recipients. *American Journal of Otology, 16(4),* 514-520.

Moore, J. K., & Linthicum, F. H. (2007). The human auditory system: A timeline of development. *International Journal of Audiology, 46,* 460–478.

Morita, T., Hiraumi, H., Fujiki, N., Naito, Y., Nagamine, T., Fukuyama, H., & Ito, J. (2007). A recovery from enhancement of activation in auditory cortex of patients with idiopathic sudden sensorineural hearing loss. *Neuroscience Research*, *58(1)*, 6–11.

Munro, K. J., Purdy, S. C., Ahmed, S., Begum, R., & Dillon, H. (2011). Obligatory Cortical Auditory Evoked Potential Waveform Detection and Differentiation Using a Commercially Available Clinical System: HEARLab™. *Ear and Hearing, 32,* 782–786.

Naatanen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology, 24,* 375–425.

Oates, P.A., Kurtzberg, D., & Stapells, D.R., (2002). Effects of sensorineural hearing loss on cortical event-related potential and behavioural measures of speech-sound processing. *Ear and Hearing, 23(5)*, 399–415.

Onishi, S.,& Davis, H.(1968). Effects of duration and rise time of tone bursts on evoked V potentials. *Journal of the Acoustical Society of America, 44*, 582–591.

Ostroff, J. M., Martin, B. A., & Boothroyd, A. (1998). Cortical evoked response to acoustic change within a syllable. *Ear and Hearing*, *19*, 290–297.

Ostroff, J. M., McDonald, K. L., Schneider, B. A., & Alain, C. (2003). Aging and the processing of sound duration in human auditory cortex. *Hearing Research. 181,* 1-7

Polen, S. B. (1984). Auditory event related potentials. *Seminars In Hearing, 5,* 127-141.

Purdy S. C., Dillion, H., Katsch, R., Storey, L., & Sharma, M. (2002). Using CAEPs to evaluate hearing aids in infants. Poster presentated at international hearing aid research conference IHCON. Lake Tahoe, USA. 21-25

Purdy, S. C., Katsch, R., Sharma, M., Dillon, H., Storey, L., & Ching, T. Y. (2002). Hearing aid evaluation in infants and young children using cortical auditory evoked potentials. *Paper presented at XXVI International Congress of Audiology, Melbourne, 17-21*.

Purdy, S.C., Katsch, R., Dillon, H., Storey, L., Sharma, M., & Agung, K., (2005). Aided cortical auditory evoked potentials for hearing instrument evaluation in infants. Proceedings of the Third International Conference, Stafa. Switzerland: Phonak AG. 115-128.

Purdy, S.C., Sharma, M., Munro, K.J., & Morgan, C. L. A. (2013). Stimulus level effects on speech-evoked obligatory cortical auditory evoked potentials in infants with normal hearing. *Clinical Neurophysiology*, 3, 474-480.

Rapin, I. & Granziani, L. J. (1967). Auditory-evoked responses in normal, brain-damaged, and deaf infants. *Neurology, 17,* 881-894

Roberts, T. P., & Poeppel, D. (1996). Latency of auditory evoked M100 as a function of tone frequency. *Neuroreport, 7,* 1138-1140.

Rothman, H. H., Davis, H., & Hay, I. S. (1970). Slow evoked cortical potentials and temporal features of stimulation. *Electroencephalography and Clinical Neurophysiology, 29,* 225-232.

Ruth, R. A., & Lambert, P. R. (1991). Auditory evoked potentials. *Otolaryngologic* Clinics of *North America*, *24(2),* 349-70.

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

Sharma, A., Dorman, M. F., & Spahr, A. J. (2002). A Sensitive Period for the Development of the Central Auditory System in Children with Cochlear Implants: Implications for Age of Implantation. *Ear & Hearing, 23,* 532–539.

Sharma, A., Gilley, P. M., Dorman, M. F., & Baldwin, R. (2007). Deprivation-induced cortical reorganization in children with cochlear implants. *International Journal* of *Audiology*, *46(9),* 494–499.

Sharma, A., Nash, A. A., & Dorman, M. (2009). Cortical development, plasticity and re organization in children with cochlear implants. *Journal of Communication Disorders, 42(4),* 272–279.

Sharma, A., Cardon, G., Henion, K., Roland. P. (2011). Cortical maturation and behavioral outcomes in children with auditory neuropathy spectrum disorder. *International Journal of Audiology, 50,* 98-106

Shruthi, K., & Vanaja, C. S. (2007). Speech evoked auditory late latency response (ALLR) in hearing aid selection. Unpublished master's dissertation, University of Mysore, Mysore.

Souza, P., & Tremblay, K. (2006). New perspectives on assessing amplification effects. *Trends in amplification, 10*, 119-143.

Thabet, M. T., & Said, M. N. (2012). Cortical auditory evoked potential (P1): A potential objective indicator for auditory rehabilitation outcome. *International Journal of Pediatric Otorhinolaryngology, 76,* 1712–1718.

Tremblay, K.L., Friesen, L., Martin, B.A., & Wright, R. (2003). Test-retest reliability of cortical evoked potentials using naturally produced speech sounds. *Ear and Hearing*, *24(3),* 225-32.

Tremblay. K. L., Billings, C., & Rohilla, N. (2004). Speech evoked cortical potentials: effect of age stimulus presentation rate. *Journal of American Academy of Audiology* , *15 (3),* 226-37.

Tremblay & Kelly, L. (2006). Hearing aids and the brain: What's the connection?. *Hearing Journal, 59 (8),* 10.

Tremblay. K. L., Billings, C. J, Friesen, L, M., & Souza, P.E, (2006). Neural representation of amplified speech sounds. *Ear and Hearing, 27,* 93-103.

Tyler, R.S (1993). Speech perception by children. In R.S. Tyler (Ed.), Cochlear implants Audiological foundations. San Diego; Singular publishing group.

Vandana. (1998). Speech identification test in Kannada. Unpublished independent project, University of Mysore, Mysore, India.

Wall, L. G., Dalebout, S. D., Davidson, S. A., & Fox, R. A. (1991). Effect of hearing impairment on event-related potentials for tone and speech distinctions. *Folia Phoniatrica*, *43,* 265–274.

Wunderlich, J. L., Cone-Wesson, B. K., & Shepherd, R. (2006). Maturation of the cortical auditory evoked potential in infants and young children. *Hearing Research*, *212*, 185–202.

Zhang, V. W., Ching,T. Y. C., Hou, S., Wong, A., Burns L., & Buynde, P. V. (2012). Effects of Frequency Compression on Aided Cortical Evoked Potentials (CAEPs) of Children with Hearing Loss. Proceedings of the 14th Australian International Conference on Speech Science and Technology, Sydney.