THE CORTICAL NEURAL PROCESSING FOR SPECTRALLY DIFFERENT SPEECH SOUNDS IN INDIVIDUALS WITH COCHLEAR HEARING LOSS

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ALL INDIA INSTITUTE OF SPEECH AND HEARING
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This work is dedicated to four people who mean the world to me...

Papa, Amma,

Putti

&

Anand

With out whom my very existence is meaningless…

CERTIFICATE

This is to certify that this dissertation entitled ***"The Cortical Neural Processing for Spectrally Different Speech Sounds in Individuals with Cochlear Hearing Loss"*** is the bonafide work submitted in part fulfillment for the degree of Master of Science (Audiology) of the student (Registration N0.O6AUDOI6). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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CERTIFICATE

This is to certify that the dissertation entitled **"The Cortical Neural Processing for Spectrally Different Speech Sounds in Individuals with Cochlear Hearing Loss"** has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any Diploma or Degree.

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DECLARATION

This is to certify that this dissertation entitled **"The Cortical Neural Processing for Spectrally Different Speech Sounds in Individuals with Cochlear Hearing Loss"** is the result of my own study under the guidance of Mr. Animesh Barman, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted in any other university for the award of any diploma or degree.

Mysore

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1. INTRODUCTION

The cortical auditory evoked potentials are scalp recorded evoked potentials that occur in response to variety of stimuli (Näätänen & Picton, 1987). Cortical auditory evoked potentials can be classified into 'obligatory' and 'discriminative' potentials. Discriminative potentials are evoked by a change from frequent 'standard' stimulus to an infrequent 'deviant' stimulus. The discriminative potentials consist of mismatch negativity (MMN) and P300. The 'obligatory' ALR are classified in terms of their latencies or the time of occurrence after presentation of a stimulus (Hall, 1992). The obligatory ALR is also called auditory late latency responses (ALR). These responses are reported to test the integrity of the auditory system (Hall, 2007).

The auditory late latency responses have four major components. The first positive voltage component, P1 occurs in the 50 to 80 ms region. It is followed by a negative component, N1 between 100 and 150 ms, P2 between 150 – 200 ms and N2 between 180 to 250 ms. Early positive component in the region of 40 to 50 ms (P1) occurs less consistently than N1 and P2. The amplitude of long latency auditory evoked potentials is around 2 – 7 micro volts (Hall, 2007).

The ALRs can be used as an electrophysiological method for estimation of hearing sensitivity in infants and young children. It can be used to document high level central auditory dysfunction in patients with abnormal ABR findings. It has been used in the evaluation of auditory processing disorders in learning disabilities and auditory neuropathy (Hall, 2007). ALRs have been recently used to determine the effect of phonologic and acoustic features (Crottaz-Herbette & Ragot, 2000) and to identify the cortical areas activated by these features (Makela, Alku & Tiitinen,

2003). This objective measure provides a tool to investigate the neurophysiological processes that underlie our ability to perceive speech (Purdy, Katsch & Sharma, & Dillon, 2001; Trembley, Friesen, Martin & Wright, 2003). Furthermore, it has been used to index changes in neural processing with hearing loss and aural rehabilitation (Martin, Trembley, & Stapells, 2007).

The auditory late responses elicited by speech stimuli can be applied in the electrophysiological assessment to assess the representation of speech in the central auditory nervous system. Furthermore, it can be used to understand the neural encoding of speech in individuals with impaired auditory pathways (Eggermont & Ponton, 2003).

Earlier investigations in this direction have reported a good correlation of morphology of ALR with acoustic features of speech. Sharma and Dorman (1999) used /da/ and /ta/ syllables to record ALR in 16 normal hearing subjects. They varied the VOTs of the syllables from 0 to 80 ms. The results showed two distinct onset responses (N1 and N1ˈ) that behaved differently in response to VOT. This finding suggested that the presence of double peaked N1 component can be considered as a correlate of the categorical perception.

Agung, Purdy, McMahon, and Newall (2006) recorded ALR for, /a, u, i, s, sh, m and כ / which covered a broad range of frequencies across the speech spectrum. The objective of the study was to investigate whether the response latency and amplitude measures can differentiate each speech sound from the rest. P1 and P2 elicited by longer duration vowels /u/, /a/,כ //i / decreased in latency in the order as written above.

Hence, it was concluded that ALR wave components may provide an objective indication about the neurophysiological process of speech processing. Spectrally different speech sounds might be encoded differently at the cortical level. However, the ALR recording using different speech sounds may not be sufficient to measure the discrimination ability of an individual.

Need for the study

The P1-N1-P2 complex signals the arrival of stimulus information to the auditory cortex and the initiation of cortical sound processing (Hillyard & Kutas, 1983). As reported by Novak et al. (1989), Trembley et al. (2003) cortical potentials reflect the functional integrity of the auditory pathways involved in processing of complex speech stimuli. Cortical potentials can be used to understand the neurophysiological basis for speech perception, which would give information of speech processing abilities of the individuals. In general, majority of the studies have focused on recording of ALRs to click stimulus or more frequency specific tone bursts. But recoding of ALRs using tone burst doesn't give much information about the processing or perception of the speech. Hence, speech sounds were selected for the study. Based on the cortical potentials recorded using speech sounds it can be possible to predict the communication abilities of an individual, and also can be used as a tool to evaluate the improvement due to treatment. ALR changes have been shown to occur prior to improvement seen in behavioral perception of speech sounds; physiological recordings may be helpful to predict the prognosis (Trembley, Kraus, & McGee,1998). Audiologists could monitor such changes in the neural detection of sound during auditory rehabilitation to predict the prognosis too.

Recording of ALRs using speech sounds can probe how the brain processes that underlie auditory detection and discrimination is altered in the individuals with cochlear hearing loss. To date, there are only few studies which have investigated the effects of cochlear hearing loss on the ALRs to the speech stimuli. Hence, there was a need to study the speech processing abilities in individuals with cochlear hearing loss. As there are no normative to compare, and no such data is available in the Indian population, the study also evaluated individuals with normal hearing as a control group.

Speech does not consist of a single frequency component; the speech sounds cover a wide frequency region. It is important for any listener to listen to all the speech sounds which encompasses the speech spectrum. It is not sufficient to study only the processing of single frequency stimuli. Hence, there was a need to study the ALRs which is evoked by speech stimuli which largely encompasses the speech spectrum. Hence, the three different speech stimuli /ba/ which has spectral energy concentration in low frequency, /ga/ syllable dominated by mid frequency spectral energy and /da/ syllable dominated by high frequency spectral energy was taken up for the study.

There was a need to study the processing of the different speech sounds which lie in the different areas in the speech spectrum. Speech perception of individuals with cochlear hearing loss is poorer relative to normal hearing individuals in spite of presenting stimuli at most comfortable levels. This is because spectral and temporal cues of speech get distorted at the peripheral level before reaching the higher structures. Hence, it was hypothesized that cortical processing may be abnormal in individuals with cochlear hearing loss as cortical structures receive abnormal inputs

from the lower auditory structures. Because dynamic cues like speech burst and transition are more susceptible to show abnormality.

Aims of the study

The aims of the present study were to determine:

- Whether the auditory late latency responses recorded for spectrally different syllables differ significantly in normal hearing adults.
- Whether the auditory late latency responses recorded from spectrally different syllables differ significantly in hearing impaired adults.
- Whether the ALRs from two groups differ significantly.
- To investigate the difference in speech evoked ALR between the normal hearing and cochlear hearing loss individuals when the signal reaching at the same level or at the different intensity level.

2. REVIEW OF LITRATURE

The long latency auditory evoked potentials are the low voltage (microvolt) discrete electrical potentials generated in the brainstem or cortical regions and are time locked to the auditory stimulus. These potentials occur between 50 to 250 ms after the auditory stimulation and are considered exogenous, referring to the characteristic of the response being related more to extrinsic or stimulus factors (Hall, 2007).

The ALRs have four major components. The first positive voltage component, wave P1 is a vertex positive voltage deflection that often occurs approximately 50 ms after the sound onset. The amplitude of P1 is usually small in adults (typically <2 μV) but it is large in young children and can dominate their response (Naatanen & Picton, 1987; Sharma, Kraus, McGee & Nicol, 1997). Knight, Scabini, Woods and Clayworth (1988) reported that the late thalamic projections into the auditory cortex and/or early auditory cortex and the specific sensory system are the generators for the P1 potential.

Wave N1 appears as a negative peak that occurs approximately 100 ms after sound onset. Compared to P1, amplitude of N1 is relatively large in adults, as reported by Ceponiene, Cheour and Naatanen (1998). The earlier positive components in the region of 40 to 50 ms (P1) occur less consistently than wave N1 and wave P2 (Hall, 2007). Knight et al., (1988) stated that the supra temporal auditory cortex and non specific polysensory system are the generator sites for the N1 potential.

Wave P2 is a positive voltage deflection that occurs at approximately 180 ms. The amplitude of wave P2 in adults is 2-5 μv or more. The P2 responses may be

absent in young children as reported by Martin, Tremblay and Stapells, (2007). It has generators in multiple auditory areas, including the primary auditory cortex (Scherg, Vajsar & Picton, 1989; Makela & Hari, 1990), the secondary auditory cortex (Hari, Pelizzone & Makela, 1987), and the mesencephalic reticular activating system (Woods, Knight & Scabini, 1993; Naatanen & Picton, 1987).

Baumann, Rogers, Papanicollaou and Syadjari (1990) reported that lateral-frontal supra temporal auditory cortex and the non specific polysensory system as being the generators of P2 potential. P2 potentials are consistently reported to be delayed in older adults (Trembley, Piskosz & Souza, 2003; Trembley, Billings & Rohilla, 2004).

Wave N2 is a negative voltage which occurs between 180 and 250 ms. The N2 is not invariable and may or may not be present in normal subjects (Hall, 2007). The wave N2 gets generated from the supra temporal auditory cortex and the non specific polysensory system (Makela & Hari, 1990).

Factors affecting ALR

Some of the major factors that can affect ALR are discussed here.

Subject Factors

Subject state

Unlike the auditory brainstem response, the P1-N1-P2 complex can be affected by subject state. P1 doesn't get affected by either attention or awakefulness and the sleep state, while N1 is reported to increase in amplitude by upto 0.61 μV

when the stimulus in attended (Picton & Hillyard, 1974). James, Gordon, Kraiuhin, Howson and Meares (1989) found amplitude of the N1 to be larger in the attending compared to that is non attending condition. Similarly, the P2 also increases in amplitude by about 0.70 μV when stimulus is attended (Freeze, 1990). Wave N2 shows a slight increase in latency, increase in amplitude and a biphasic peaks in attentive condition (Ford, Roth & Kopell, 1976).

Sleep has pronounced effect on ALR. There are significant but differential changes in the major ALR waves as the person becomes drowsy and falls asleep. Amplitude of N1 is reported to progressively diminish from wake to sleep state (Campbell & Colrain, 2002). They found that, during the transition to deep sleep, P2 amplitude increases. But the overall amplitude of N1 and P2 may remain reasonably stable across sleep stages (De Lugt, Loewy & Campbell, 1996). These sleep-related changes in morphology can significantly increase the variability of the response. Hence, P1-N1-P2 should be typically recorded while subjects are awake.

Maturation and aging

The morphology of the P1-N1-P2 complex is affected by maturation. The complex changes dramatically over the first 2 years of life as reported by Kurtzberg (1989) and Kurtzberg, Hilpert, Kreuzer and Vaughan (1984). The complex begins as a large P1 wave is followed by a broad, slow negativity occurring near 200 to 250 ms after the onset of the sound. The P1-N1-P2 complex that is similar to that of adults is not seen upto 10 years of age unless stimuli are presented at a very slow rate (Ponton et al. 2000). The cortical potentials are generated by multiple brain regions including the primary auditory cortex, auditory association areas, frontal cortex and sub cortical regions (Stapells, 2002). These areas mature at different rates and hence there are

complex changes in morphology, scalp distribution, amplitude and latency of peaks with maturation (Cunningham, Nicol, Zecker & Kraus, 2000; Ponton et al., 2000). These potentials continue to mature until the second decade of life and then change again during old age.

Games (1997) examined maturational changes in spectro-temporal features of central and lateral N1 components of the auditory evoked potential to tone stimuli presented with a long stimulus onset asynchrony. He reported that the peak latencies of both the components decrease with age. Peak amplitude also decreased with age consequently, the difference between the lateral N1 and the central N1 amplitude also decreased with age.

Latency prolongation and amplitude decrease of N1 & P2 have been reported in aging adults. Latency of ALR decreases and amplitude increases as a function of age during childhood, until about 10 years of age (Weitzman, Fishbein & Grabiani, 1965). The increase in latency and decrease in amplitude are reported in advanced age also (Callaway & Halliday, 1973).

Gender

There is some evidence that N1 latencies are shorter and amplitudes larger in women than in men (Altman & Vaitulevich, 1990). Onishi and Davis (1968) found that ALR amplitude tended to be larger and the amplitude versus intensity function steeper for females than the males. However, gender difference has not been observed in 6 to 10 months old infants (McIsaac & Polich, 1992).

Stimulus Factors

Stimulus intensity

Intensity of the stimulus is an important parameter while studying speech processing. Beagley and Knight (1967) and Picton, Woods, Baribeau-Braun, and Healey, (1977) reported increase in the amplitude of P1-N1-P2 with stimulus intensity in an essentially linear manner, though the amplitude-intensity function may saturate at intensities exceeding at around 70 dBnHL. This is true particularly when short inter stimulus interval is used (Picton, 1970). The maximum response is obtained for moderate intensity stimuli, 50-60 dBHL as reported by Hall (2007) and 60-70 dBHL by Hyde (1997). Amplitude of P2 may saturate at higher stimulus intensities than N1 (Adler & Adler, 1989). In general, latencies decrease as stimulus intensity increases. At low intensities, latency of P2 increases more than that of N1 (Adler & Adler, 1989).

Stimulus frequency

Alain, Woods and Covarrubias (1997) reported that the amplitude of the N1 as well of P2 components of the ALR is larger and the latencies longer for low frequency tonal signals in comparison to the higher frequency signals. As stimulus frequency increases, amplitude of the complex decreases (Antinoro, Skinner & Jones, 1969), even when loudness is controlled (Picton, Woods & Proulx, 1978). Latencies increase as frequency decreases particularly when high stimulus intensities are used (Jacobson, Lombardi, Gibbens, Ahmed & Newman, 1992)

Stimulus rate

Amplitude of P1-N1-P2 increases as the rate of stimulus presentation decreases until the inter stimulus interval (ISI) is approximately 10 seconds (Davis, Mast, Yoshie & Zerlin, 1968 ; Hari, Kaila, Katila, Tuomisto & Varpula, 1982). However, it is also influenced by the stimulus intensity. Picton, Goodman and Bryce (1970) reported that, at low stimulus intensities, amplitudes asymptote or level off at shorter ISIs. Similar results were reported by Nelson and Lassman (1973). Whereas at high stimulus intensities, amplitude keep increasing even beyond ISIs of 10 seconds (Hari et al. 1982). The most pronounced effect of longer ISI is within 1 to 6 seconds. There is little change in latency with the stimulus rate (Davis, Mast, Yoshie & Zerlin, 1968; Hari et al. 1982). Stimulus rates of 1/s or less are appropriate as recommended by Hall (2007). A slow repetition rate of about 1/s is also necessary to avoid neural refractory effects and to get better amplitude (Budd, Barry, Gordon, Rennie & Michie, 1998).

Stimulus duration

Amplitude increases as stimulus duration increases up to approximately 30 to 50 ms, but the amplitude decreases when rise and fall times exceed 50 ms as reported by Onishi and Davis (1968). The stimulus should be relatively longer in duration (>10 ms). A tone burst should have rise time of 5 ms, plateaus of 25 ms and fall time of 10 ms, as reported by McPherson (1996). Rise times of 10 ms or more and total durations of 30-75 ms are recommended by Hyde (1997).

Stimulus onset polarity should be alternated to minimize stimulus artifact effects on the recording.

Type of stimulus

The long latency auditory evoked potentials can be evoked by a wide variety of transient sounds such as clicks, tone bursts, noise bursts and different types of speech signals. Speech signals that are used in the earlier investigation include natural or synthetic vowels, syllables, and words (Ceponiene et al. 2001; Kurtzberg, 1989; Martin & Boothroyd, 1999; Naatanen & Picton, 1987). It can also be evoked by "non-stimuli" such as, gaps in a tone or noise (Simson, Varghan & Ritter, 1976).

(i) Tonal stimulus

Typically tonal stimulus has been used to elicit ALR (Davis, Bowers & Hirsh, 1968). Optimal tone bursts that are used to elicit ALR have rise/fall times and plateau times of greater than 10 ms (Onishi & Davis, 1968; Rothman, Davis & Hay, 1970; Ruhm & Jansen, 1969; Skinner & Jones, 1968). Rise/fall times of over 20 ms and durations of hundreds of milliseconds are even effective in eliciting ALR.

(ii) Speech stimulus

The tonal stimuli give very limited information about the processing and in turn about perception of speech. Speech stimulus also has been used to elicit ALR. Different types of speech signals, including natural and synthetic vowels, syllables and words have been reported in the literature (Martin & Boothroyd, 1999; Ostroff, Sharma, Marsh & Dorman, 2000). In general, amplitude of the N1 to P2 complex is larger for speech stimuli than for single frequency tonal stimuli, but latency values for the N1 and P2 are usually earlier for tonal stimuli compared to that of speech stimuli (Ceponiene et al. 2001; Tiitinen, Sivonen, Alku, Virtanen & Naatanen, 1999).

Applications of ALR

ALRs can be used as an electrophysiological method for estimation of hearing sensitivity in infants and young children (Hall, 2007). It can be used to document high level central auditory dysfunction in patients with abnormal ABR findings. It has been used in evaluating auditory processing disorders in learning disabilities and auditory neuropathy (Hall, 2007). ALRs has been recently used to study the processing of phonologic and acoustic features of speech (Crottaz-Herbette & Ragot, 2000) and to identify the cortical areas activated by these features (Makela, Alku & Tiitinen, 2003). It has also been used as an objective measure to investigate the neurophysiological processes that underlie our ability to perceive speech (Purdy, Katsch & Sharma, 2001; Trembley, Friesen, Martin & Wright, 2003). Furthermore, it has been used to index changes in neural processing with hearing loss and aural rehabilitation (Trembley, 2007).

Estimation of Hearing Threshold

The P1-N1-P2 complex is highly sensitive to hearing loss. P1-N1-P2 responses and behavioral thresholds typically fall within approximately 10 dB of each other as reported by several investigators (Davis, 1976; Stapells, 2002). However, there are also certain studies where larger discrepancies are reported (Rapin, 1964; Prevec, Cernelc & Ribaric, 1976).

Hyde (1997) used the N1 for the assessment of threshold in adult compensation cases and medico legal patients. He has reported that the P1-N1-P2

complex is reliable tool for estimating hearing thresholds in cooperative and awake patients.

ALR has been used to examine changes in the neural processing of speech in simulated and actual hearing loss. Martin and colleagues (1999) examined N1, MMN (along with P3), and behavioral measures in response to the stimuli, /ba/ and /da/ in normally hearing listeners when audibility was reduced using high-pass, low-pass, or broadband noise masking. This was done to simulate the effects of high-frequency, low frequency, and flat hearing loss respectively. In general, N1 amplitude decreased and latency increased systematically as audibility was reduced. This finding is consistent with the role of N1 in the cortical detection of sound and supports the use of N1 for hearing sensitivity estimation.

Indexing changes in neural processing with hearing loss and aural rehabilitation

One of the recent applications of ALR is monitoring experience-related changes in neural activity. Because the central auditory system is plastic, that is, capable of reorganization as a function of deprivation and stimulation, ALR have been used to monitor changes in the neural processing of speech in patients with hearing loss. Improvement with various forms of auditory rehabilitation, such as use of hearing aids, cochlear implants, and/or auditory training using ALR has been monitored (Trembley, Martin & Stapells, 2007).

Evaluation of benefit from the hearing aids

Auditory late responses can be reliably recorded in individuals, even when the sound is processed through a hearing aid. There are number of studies where in they have used ALRs for prescribing hearing aids. Rapin and Grazianni (1967) found a

majority of 5 to 24 months infants with severe to profound sensory neural hearing loss had ALR thresholds at 20 dB lower in aided conditions compared to the unaided thresholds for click and tonal stimuli.

Trembley et al. (2006) recorded ALRs for amplified speech sounds /Si/ and /Si/ in 7 adults with mild to severe sensorineural hearing loss and in 7 normal hearing adults. The results revealed that the speech evoked ALR can be used reliably both in aided and unaided conditions. Similar results are reported by Korezak, Kurzberg and Stapells (2005), in individuals with severe to profound hearing loss. Most of the subjects with hearing loss showed increased amplitudes, decreased latencies, and improved waveform morphology in the aided conditions. Furthermore, most subjects with hearing loss tested by Korezak and colleagues (2005) showed longer peak latencies and reduced amplitudes than a normally hearing group. The amount of response change is quite variable across individuals as reported by Trembley et al. (2006).

Cochlear Implants

Long latency responses can be recorded from individuals with cochlear implants (Friesen & Tremblay, 2006). ALRs can be recorded in implant users in response to sound presented either electrically (directly to the speech processor) or acoustically (presented via loud-speaker to the implant microphone). However, stimulus-related cochlear implant artifact can sometimes interfere (Friesen & Tremblay, 2006). Groenen, Beynon, Snik and Broek (2001) used four contrasts (500-1000 Hz, /ba –da/, /ba- pa/ & /i- a/). N1/P2 was elicited in post-lingually deaf cochlear implant users. N1 and P2 were present in all subjects for all conditions. Prolonged N1 and P2 latencies were found in the cochlear implant group compared to a control

group of subjects with normal hearing. Cochlear implant users showed smaller amplitudes of N1 for all the speech signals as well as smaller amplitudes of P2 for the consonants compared to the controls. The results suggest that cortical responses can be useful and can have additional value in the evaluation of speech recognition evaluations in cochlear implant users.

Auditory Training

Once the subject is prescribed amplification device, it is necessary to provide auditory training. The goal of the auditory training would be to improve the perception of acoustic contrasts. In other words, patients are taught to make new perceptual distinctions. ALRs have been used to examine the brain and behavior changes associated with auditory training.

Trembley and Kraus (2002) reported that when individuals were trained to perceive different sounds, changes in the N1-P2 complex were observed. As perception improves, N1-P2 peak-to-peak amplitudes increase. Similar results are reported by Trembley, Kraus, and Mc Gee, (1998). Because ALR changes have been shown to occur prior to improvement in behavioral perception of speech sounds, physiological recordings may be helpful to predict the prognosis (Trembley, 1998).

Tecchio and colleagues (2000) found that the latencies were prolonged and the magnetic N1 responses to tones showed enlarged cortical representation after surgery. These plastic changes were reported to have occurred within few weeks of surgery. Rapid changes in the N1 component of the P1-N1-P2 response, secondary to cortical reorganization that followed sudden unilateral hearing loss are also reported (Tecchio

et al. 2000). N1 response has also been reported to be delayed in children with congenital unilateral hearing loss.

In addition to hearing loss, auditory late responses are being used to explore the biological processes underlying impaired speech understanding in response to various types of sound and in individuals with various communication disorders. Abnormal neural response patterns have been recorded in children with various types of learning problems (Hayes, Warrier & Nicol, 2003). ALRs are now being used to examine children with learning problems undergoing speech sound training and other forms of learning, such as speech sound segregation and music training (Warrier, Johnson, Hayes, Nicol, & Kraus, 2004).

The ALRs elicited by speech stimuli can be of immense value in assessing the representation of speech in the central auditory nervous system. It can be used to investigate the neurophysiological processes that underlie the ability to perceive speech (Purdy et al. 2001; Trembley et al. 2003). Furthermore, it can be used to understand the neural encoding of speech in individuals with impaired auditory pathways (Eggermont & Ponton, 2003).

The reliability of ALRs elicited by naturally produced speech sounds was evaluated by Tremblay et al. (2003). P1-N1-P2 responses were obtained from 7 normal hearing young adults in response to four naturally produced speech tokens (/bi/, /pi/, /shi/ & /si/). The subjects were tested and retested within an eight day period. The results of the study revealed that the P1-N1-P2 responses were reliably elicited using naturally produced speech sounds. These speech sounds, which represented different acoustic cues, evoked distinct neural response patterns. It was

suggested that these responses can be applied to study the neural processing of speech in individuals with communication disorders. It can also be used to study changes over time during various types of rehabilitation.

Earlier investigations have reported a good correlation of morphology of ALR with acoustic features of speech. Rance, Cone-Wesson, Wunderlich and Dowell (2002) found that the development of reasonable speech perception performance in children with auditory neuropathy was correlated with ALRs of normal latency, amplitude and morphology whereas, the absence of ALR was associated with poor speech recognition scores. Thus, ALRs are thought to reflect the functional integrity of the auditory pathway involved in processing of complex speech stimuli (Trembley et al. 2003).

Sharma and Dorman (1999) used /da/ and /ta/ syllables to record ALR in 16 normal hearing subjects. They varied the voice onset time (VOT) of the syllables from 0 to 80 ms. The results showed two distinct onset responses (N1 and N1') that behaved differently in response to VOT. This finding suggested that the presence of double peaked N1 component can be considered as a correlate of the categorical perception.

However, similar study done by Sharma, Catherine, and Micheal, (2000), contradicted the earlier reports by Sharma and Dorman (1999). Using ALR they investigated the electrophysiological correlates of the neural representation of speech stimuli. The purpose of the study was to determine the (VOT) related change in the ALR. The subjects were 5 normal hearing males and 5 normal hearing females in the

age range of 20-30 years. Two sets of continua were used, /ga – ka/ continuum that varied in VOT from 0 – 70 ms and /ba – pa/ continuum that varied from 0 – 60 ms. Behavioral identification scores were obtained for the same stimuli continua. The results of this study showed that N1 component was seen for stimuli with VOTs of 0 – 30 ms and, two components (N1 and N1') were seen for stimuli with 40 – 70 ms VOT for both continua. The change in N1 morphology from single to double peaks was consistent with the change in perception from voiced to voiceless for /ba – pa/ continuum, but not for /ga – ka/ continuum. It was concluded that N1 morphology does not reliably predict phonetic identification of stimuli varying in VOT. They further concluded that the previously reported appearance of double peak does not indicate a cortical correlate of the perception of voicing.

Ostroff, Martin and Boothroyd (1998) recorded ALR using three naturally produced speech stimuli; 1) the syllable /sei/, 2) the sibilant /s/, extracted from the syllable and 3) the vowel /ei/ extracted from the syllable. The results showed that response amplitudes to the /ei/ stimulus showed largest amplitude followed by /sei/ and then /s/. The response to both /s/ and /ei/ followed the classic N1-P2 pattern for stimulus onset. The response to /ei/ also contains a clear P1 component. It was also noted that, N1 in response to /ei/ is offset from N1 in the response to /s/ by approximately 130 ms which roughly corresponds to the onset delay to the stimulus /ei/ relative to that of /s/. P2 in response to /ei/ is similarly offset from P2 in response to /s/ by approximately 120 ms. The authors concluded that the complete response to the entire CV syllable /sei/ is combination of the response to the two constituent phonemes /s/ and /ei/, but it is not the sum of the responses of the two. The change in morphology and latency was accounted to the acoustic change occurring at the CV

transition. This change occurring during an acoustic stimulus was called acoustic change complex.

Agung et al. (2006) recorded ALR for, /a/, /u/, /i/, /s/, /sh/, /m/ and /ɔ / which covered a broad range of frequencies across the speech spectrum. The objective of the study was to investigate whether the response latency and amplitude measures can differentiate each speech sound from the rest. The responses were recorded from 10 normal hearing adults in the age range of 20 to 29 years. Presentation of the stimuli was through a loud speaker at 65 dBSPL. N1-P2 response amplitudes elicited by higher frequency speech stimuli /s/ and /S/ produced significantly smaller amplitudes compared to stimuli that had dominant spectral energies in low frequencies /m/, /a/, /u/ and / i/. Latency of N1 decreased systematically when elicited by /u/, /ɔ/, /a/ and /i/. Similarly, P1 and P2 elicited by longer duration vowels /u/, /a/, / ɔ / and /I /, decreased in latency in the respective order. Hence, it was concluded that ALR latencies and amplitudes may provide an objective indication that spectrally different speech sounds are encoded differently at the cortical level. However, the parameters of ALR cannot be distinguishing measures across speech sounds, as the differences when elicited by different speech sounds are not significant.

Trembley et al. (2002) examined the neural representation and perception of VOT, a temporal cue that distinguishes /b/ from /p/ in young and older adults. They found that older adults had more difficulty than younger listeners in discriminating voice-onset contrasts behaviorally. In addition, these same speech stimuli evoked abnormal neural responses in older adults. Latencies of N1 and P2 were prolonged for older adults than for the younger adults. This suggests that age related delays in

synchronous firing among neural population can be successfully detected using N1 and P2 responses.

Shruti (2007) recorded ALRs using /i/, /m/ and /s/ in 10 hearing impaired (sensorineural in type) children in the age range of 5 to 7 years. The ALRs were recorded in the unaided and aided conditions. The responses were compared with that of 10 age matched controls. The ALRs were reliably elicited in all the participants of normal hearing group and in aided condition in the hearing impaired group for all the stimuli. The responses obtained for the three stimuli resulted in distinct responses indicating that the stimuli are coded differently in the auditory system. Stimuli /i/ resulted in better morphology, shorter latency and higher amplitude than /m/ and / s/ stimuli, indicating that the vowels are better coded than the consonants. It was also found that the N1 response is a critical potential in determining the usefulness of speech evoked ALRs in clinical as well as in normal hearing population.

Anirban (2007) used an extracted transition portion and an extracted burst portion of naturally produced syllables /pa/, /ta/ and /ka/ to evoke cortical responses. The correlation of speech identification scores with the cortical responses was investigated. The subjects were 10 cochlear hearing loss adults in age range of 18-50 years. The responses were compared with that of 12 age matched controls. It was found that the latencies of ALR in hearing impaired population were not significantly different from the normal hearing individuals. He reported that ALR may not be a reliable measure for assessing the impaired processing of short duration cues of speech signal in cochlear hearing loss individuals.

From the review of the literature, it is evident that the speech evoked auditory late responses have applications in the electrophysiological assessment of the representation of speech cues at the cortical level of auditory neural system. This is true in normal as well as in clinical population. In clinical population, it can be used to evaluate the benefits with rehabilitative measures. To date, only few studies have investigated the effects of peripheral hearing loss on ALRs to speech stimuli. However, these studies had a small number of subjects and reported conflicting results. Polen (1984) found that moderate to severe sensorineural hearing loss resulted in a prolongation of N1 and N2 latencies and reduction in N2 amplitude in comparison with results from normal hearing individuals. Wall and colleagues (1991) reported that there were no significant differences in the latencies of waves of ALR for mild to moderate sensorineural loss when compared with the normal hearing group.

The majority of studies have compared the hearing impaired group with the normal hearing group at equal presentation level. The differences thus noticed can be because of difference in sensation levels. However, there is a need to compare the cortical processing of the speech sounds across the normal hearing subjects and hearing impaired subjects at the equal sensation levels. There are several behavioral measures to evaluate the speech perception in the cochlear hearing loss subjects and it is found that the speech perception is effected in these subjects. There is dearth of studies investigating the processing of speech at the cortical level in the cochlear hearing loss individuals. The results of this study can aid the audiologist in deciding the gain for different frequency regions using the speech sounds of different frequency region.

3. METHOD

The main objective of the study was to know, how ALLR differs for spectrally different speech sounds in individuals with normal hearing and also in individuals with cochlear hearing loss. To accomplish the aim two groups of subjects were taken for the study. An attempt was also made to know whether the auditory late latency responses recorded from individuals with cochlear hearing loss are affected, compared to normal hearing individuals. The study also investigated the difference between the normal hearing and cochlear hearing loss individuals when the signal reaching was at same level and when at different level. To arrive at the aim the following method was adapted.

Subjects

A total of 55 ears from 28 subjects with normal hearing and cochlear hearing loss in the age range of 18 to 55 years participated in the study. The subjects were classified into 2 groups: control group and clinical group.

Control group

Thirty two ears from 16 subjects with normal hearing in the age range of 18 to 55 were evaluated. The following criteria were considered for the selection of subject.

- Pure tone threshold were within 15 dB HL at octave frequencies between 250 to 8000 Hz for air conducted and between 250 to 4000 Hz for bone conducted.
- All the subjects had 'A' type tympanogram with normal acoustic reflex thresholds.

- Speech identification scores were > 90%.
- No history of acute or any chronic ear infection, ear ache, tinnitus, vertigo or any other otological problems were reported.
- No relevant history of any medical and neurological impairment was reported.

Clinical Group

Twenty three ears from 12 subjects with cochlear hearing loss in the age range of 18 to 55 were evaluated. The following criteria were considered for the selection of subject.

- All of them were diagnosed as having cochlear hearing loss by an experienced audiologist.
- Air bone gap was within 10 dB HL.
- Pure tone average (PTA) ranged from 26 dB HL to 55 dB HL.
- All the ears tested had 'A' type tympanogram with elevated or absent acoustic reflex.
- Speech identification scores were proportionate to their pure tone average.
- No history of acute or any chronic middle ear infection, ear ache, tinnitus, vertigo or any other otological problems.

- No retro cochlear pathways component was noted, which was ruled out based on Auditory Brainstem Responses (ABR) and oto acoustic emission (OAEs) and also based on neurological assessment.

Instrumentation

- A Calibrated double channel diagnostic audiometer orbitter 922 with TDH-39P ear phone and B-71 bone vibrator was used for pure tone audiometry.

- A Calibrated immittance meter (GSI tymp star) was used to assess middle ear status.

- ILO 292 DPecho port system was used to record Transient evoked oto-acoustic emissions (TEOAE).

- Intelligent Hearing Systems (IHS smart EP windows USB version 3.91) evoked potential system was used to record and analyze the ABR and ALR. TDH 49-P headphone was used to deliver the stimulus.

Stimulus generation

Syllables /ba/ /da/ and /ga/ were used to record LLR. These stimulus were selected as /ba/ is dominated by low frequency spectral energy, /ga/ is dominated by mid frequency spectral energy and /da/ is dominated by high frequency spectral energy. These syllables were spoken by a male speaker and digitally recorded into a computer with the PRAAT software version 4.2.01 with a sampling frequency of 44,000 Hz and a 16 bit resolution. Each recorded syllable was then edited. The voice

onset time, burst portion and a little portion of the vowel was retained to make the syllable duration approximately 150 ms. The stimuli durations were 147 ms for /ba/, 150 ms for /da/ and 146 ms for /ga/.

Test environment

All the tests were carried out in a well illuminated air conditioned rooms which were acoustically treated. The noise levels were within permissible levels as recommended by ANSI (1991).

Test procedure

Pure tone audiometry

Pure tone air conduction and bone conducted thresholds for each individual was established using Modified Hughson Westlake method (Carhart & Jerger, 1959). Air conduction thresholds were obtained in octave frequencies from 250 to 8000 Hz. Bone conduction thresholds were established for 250 Hz to 4000 Hz in octave frequencies.

Immittance

The tympanometric measurements were done using 226 Hz probe tone at 85 dB SPL. For reflex measurements, the reflex eliciting tone of 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz were presented ipsilaterally and contralaterally to find out the presence or absence of reflexes. A significant change of admittance value of 0.03ml was considered as a presence of reflex. This was done to rule out any middle ear pathology.

Transient otoacoustic emissions (TEOAE)

The transient Otoacoustic emissions were recorded for nonlinear clicks presented at 85 dBpeSPL. The responses of 256 sweeps were averaged to obtain the TEOAE responses. The amplitude of TEOAE and noise levels was measured and the amplitude to noise ratio of 6 dB SPL or more was considered as the presence of TEOAE with the reproducibility of greater than or equal to 50% as described by Glattke, Pafitis, Cummiskey and Herrer, (1995). The absence of TEOAEs in the presence of hearing loss was considered as an indicator of cochlear hearing loss.

ABR recording

ABR testing was done to rule out retro cochlear pathology. Subjects were instructed to sit comfortably on a reclining chair and relax. They were instructed to close their eyes during the testing to avoid any artifacts. The recording was done at 90 dBnHL at 11.1 and 90.1 repetition rates. If the difference in wave V latency between the two repetition rates was less than 0.8ms, it was considered as absence of retro cochlear component.

Preparation of the subjects and electrode placement

Electrode sites were cleaned using NU prep cleaning gel and conductive paste was used to place the electrode. A surgical tape was used to hold the electrode in place firmly. It was made sure that each electrode impedance was within <5 kOhms and inter electrode impedance within <3 kOhms.

Electrode montage: Vertical electrode placement was used.

Non inverting: vertex (Cz)

Inverting: test ear mastoid (M1/M2)

Ground: non test ear mastoid (M2/M1)

The acquisition and stimulus parameters used to record ABR is given in Table.1.

Table 1: *Parameters used for ABR recording*

Acquisition parameters	
Amplification	100,000
Analysis window	0 to15 ms
Filters	100– 3000 Hz
Notch filter	On
Artifact rejection	40 µV
Stimulus parameters	
Transducer	TDH 49-P head phone
Type of stimulus	Clicks
Intensity	90 dBnHL
Presentation ear	Monaural
Stimulus polarity	Rarefaction
No of averages	1500
Rate	11.1/s 90.1/s

Auditory Long Latency Responses (ALRs):

Subjects were instructed to sit comfortably on a reclining chair and relax, restrict movement of head, neck and eye during the testing and to stay awake during the testing. No voluntary responses were required. Preparation of the subjects and electrode montage used to record ALR was the same as used for ABR recording. The parameters used to record ALR are given in Table 2.

Table 2: *Parameters used to record ALR*

Acquisition parameters	
Amplification	75,000
Analysis window	-100 to500 ms
Filters	1– 30 Hz
Notch filter	On
Artifact rejection	100 µV
Stimulus parameters	
Transducer	TDH-49 head phone
Type of stimulus	/ba/ /ga/ /da/
Duration	/ba/- 147, /da/-150 ms, /ga/-146 ms
Intensity	70 dBnHL 40 dB SL
Presentation ear	Monaural
Stimulus polarity	Alternating
No of averages	300
Rate	1.1/s

The recording was done twice at each presentation level. It was done for each syllable to check for the replicability. The ALR peaks P1, N1 and P2 were identified by 2 experienced judges other than the investigator. The latency of P1, N1 and P2 and peak to peak amplitude of P1-N1, N1-P2 were noted for /ba/, /ga/ and /da/ eliciting stimuli recorded at 70 dBnHL and at 40 dB SL. As N2 wave is not invariable and may or may not be present in normal subjects, N2 was not noted and was not taken for analysis.

Analysis

The latencies of P1, N1 and P2 were measured and peak to peak amplitude of N1-P2 peak was noted. The Mean, standard deviation (SD) and range were calculated

for both the groups, elicited using 3 syllables at each of the presentation levels for P1, N1, P2 latency and for the amplitude of N1-P2 complex.

- Latencies and amplitude obtained from both the group were compared across three speech stimuli elicited at 40 dB SL and 70 dBnHL separately.

- Comparison across the presentation level (40 dB SL and 70 dBnHL) was done for both the groups separately for ALR components elicited by three speech sounds.

- Comparison of the ALR components across the groups was done for each syllable and presentation level separately.

4. RESULTS

The aim of the present study was to investigate study the effects of spectrally different speech syllables on the auditory long latency responses in individuals with normal hearing and cochlear hearing loss. Attempt was also made to study the effects of the presentation level (equal SL and equal dBnHL) on the auditory long latency responses in normal hearing individuals and individuals with cochlear hearing loss.

The latencies of P1, N1, P2 and peak to peak amplitude of N1-P2 complex peaks were measured. The Mean and standard deviation (SD) were calculated for 2 groups for 3 syllables at each of the presentation levels for latencies of P1, N1 and P2 and for the amplitude of N1-P2.

Comparison of latency and amplitude of the Long latency responses to speech, between the groups and within the groups were carried out. The following statistical analyses were administered to attain the aim of the study:

- To compare the latency and the amplitude of ALR parameters between the groups elicited by the three spectrally different speech syllables at two different presentation level independent t-test was administered.
- To find out the effect of speech stimuli and the effect of presentation level on the latency and amplitude of ALR within the groups, two way repeated measures ANOVA was administered. This test was done separately for both the control and the clinical group.
- To find out the significance differences in the latencies of P1, N1 and P2 peaks and the amplitude of N1-P2 across three different speech stimuli within the group, one-way ANOVA was administered. This test was carried out for two presentation level and two groups separately.

- To evaluate the effect of the presentation level on each of the parameters of ALR elicited by three different speech stimuli, paired t-test was carried out for each group independently.

Table 3: *Mean, SD and range for P1, N, P2 latencies and amplitude of N1-P2 elicited by /ba/, /da/ and /ga/ syllables at 40 dB SL and 70 dBnHL in control and clinical group*

Parameter			Control group			Clinical group		
	Level	Syllables	Mean	SD	Range	Mean	SD	Range
P1	40 dB SL	/ba/	100.00	19.65	50-133	75.95	15.44	55-112
		/da/	110.81	16.88	75-136	78.21	19.58	50-118
		/ga/	104.37	19.95	63-150	69.43	13.50	48-117
	70 dBnHL	/ba/	74.25	21.31	41-111	87.52	12.92	66-117
		/da/	83.72	17.53	60-123	97.17	18.27	60-126
		/ga/	78.72	15.19	47-104	83.17	17.17	60-137
N1	40 dB SL	/ba/	152.65	18.53	105-196	131.78	18.78	107-17
		/da/	162.25	20.761	108-194	142.21	18.64	110-175
		/ga/	155.15	22.84	101-194	127.17	22.33	99-171
	70 dBnHL	/ba/	124.87	27.15	76-169	144.52	16.37	125-201
		/da/	137.65	21.69	90-165	160.65	18.72	121-199
		/ga/	129.09	22.58	80-181	142.73	22.38	109-183
P2	40 dB SL	/ba/	213.93	25.59	162-268	197.391	24.45	152-267
		/da/	222.56	43.90	240-270	205.13	24.73	139-258
		/ga/	225.90	16.81	184-267	202.30	35.11	132-277
	70 dBnHL	/ba/	183.80	42.76	164-260	210.43	22.85	171-275
		/da/	205.25	23.30	152-239	227.6	16.14	199-274
		/ga/	191.78	28.53	144-233	219.1	24.62	185-273
N1-P2	40 dB SL	/ba/	3.91	2.04	0.77-9.55	5.33	4.40	0.99-22.27
		/da/	3.61	2.14	0.64-9.22	4.67	2.00	0.62-8.25
		/ga/	3.28	1.34	1.22-8.12	4.75	2.48	1.09-11.66
	70 dBnHL	/ba/	3.88	2.10	0.04-7.86	5.23	2.75	2.20-12.54
		/da/	3.83	2.66	0.45-13.03	5.45	3.13	1.11-10.80
		/ga/	3.20	1.83	0.51-8.27	4.48	2.29	1.32-8.79

It can be inferred from the Table 3, that the mean latency values for the control group were shorter for all the speech sounds elicited at 70 dBnHL, compared to the clinical group at the same presentation level. This trend was not seen at the 40 dB SL

level. The control group was having a mean latency values which were longer than the latency values obtained from the clinical group. The amplitude elicited was larger in the clinical group for both at 40 dB SL and 70 dBnHL.

Comparison between the groups

P1 latency

The mean latency values of P1 for control group were longer than the clinical group at 40 dB SL, this can be observed from the Figure 1 (a). The mean latency values for P1 was prolonged for /ba/, /da/ and /ga/ in the clinical group compared to control group at 70 dBnHL.

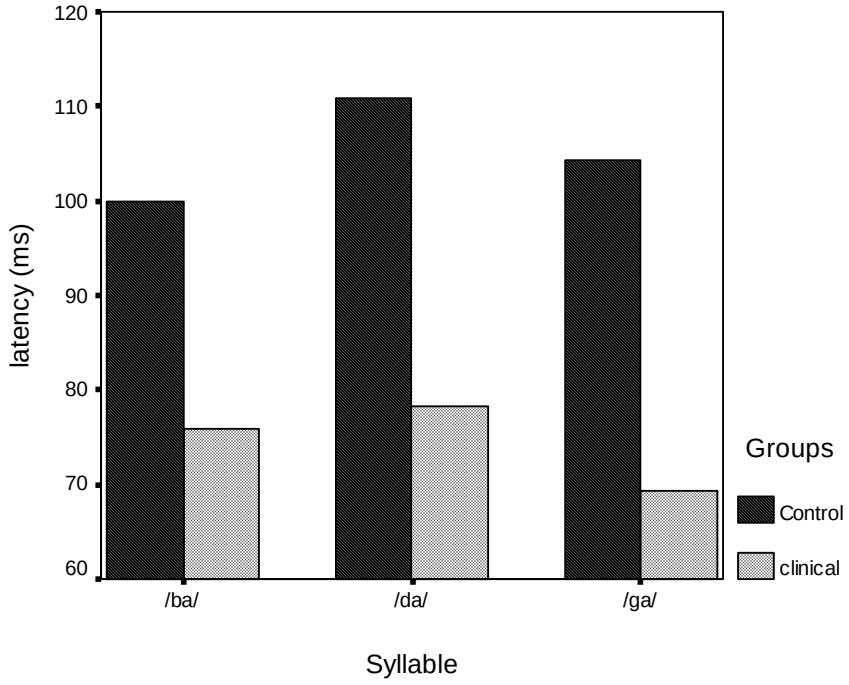

Figure 1 (a): Mean P1 latency values for /ba/, /da/ and /ga/ for control and clinical group at 40 dB SL.

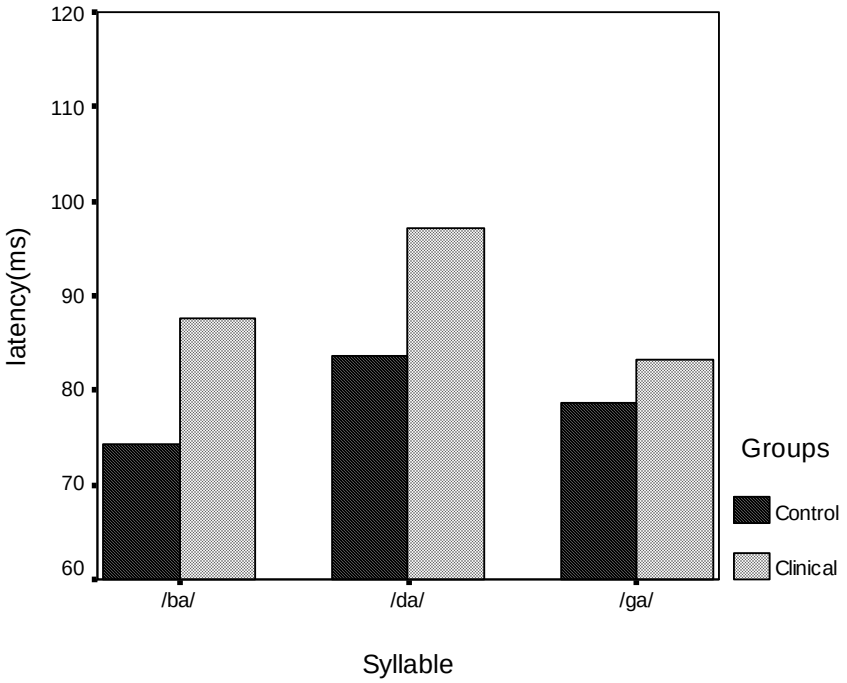

Figure 1 (b): Mean P1 latency values for /ba/, /da/ and /ga/ for control and clinical group at 70 dBnHL.

To compare the latency of the P1 elicited by the three spectrally different speech syllables at two different presentation levels between the groups independent-t test was administered. The results of the independent t-test are given in Table 4.

Table 4*: t-values with significance level for P1 latencies elicited by three speech sounds at 40 dB SL and 70 dBnHL between the groups*

Parameter	Level	Syllable	*t*-value
P1 latency	40 dB SL	/ba/	4.87**
		/da/	6.60**
		/ga/	7.27**
	70 dBnHL	/ba/	2.65**
		/da/	2.75**
		/ga/	1.01

** $p < 0.01$

It can be observed in the Table 4, that there was a statistically significant difference in P1 latency between the control group and the clinical group for all the speech sounds at 40 dB SL. At 70 dBnHL a statistically significant difference between the two groups for syllable /ba/ and /da/, was obtained but no significant difference was noticed for the syllable /ga/.

N1 latency

It is evident from the Figure 2 (a), that the mean latency values of N1 for control group was longer than the clinical group at 40 dB SL. The mean latency values for N1 wave was prolonged for /ba/, /da/ and /ga/ in the clinical group compared to control group elicited at 70 dBnHL. It can be observed in Figure 2(b).

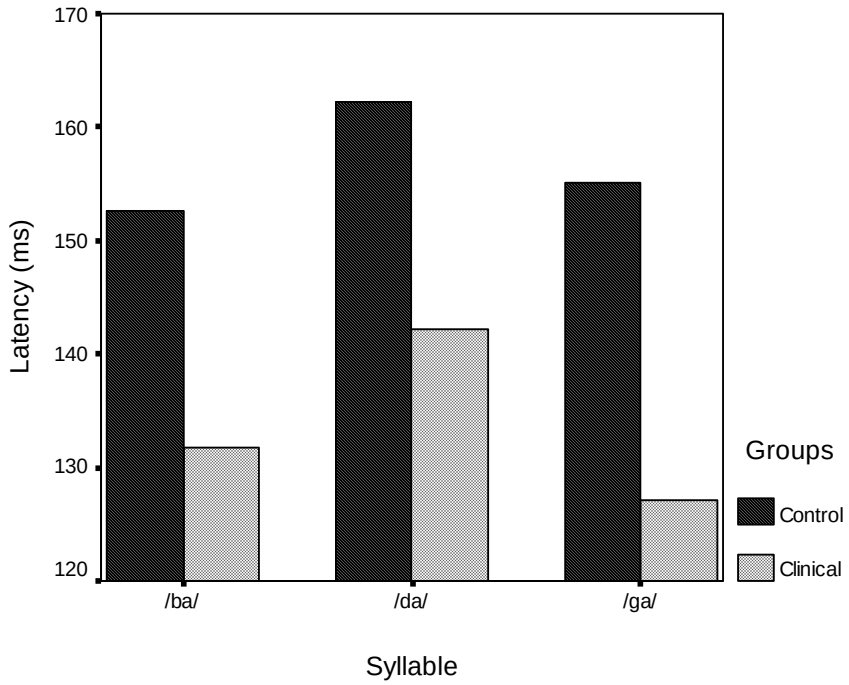

Figure 2 (a): Mean N1 latency values for /ba/, /da/ and /ga/ for control and clinical group at 40 dB SL.

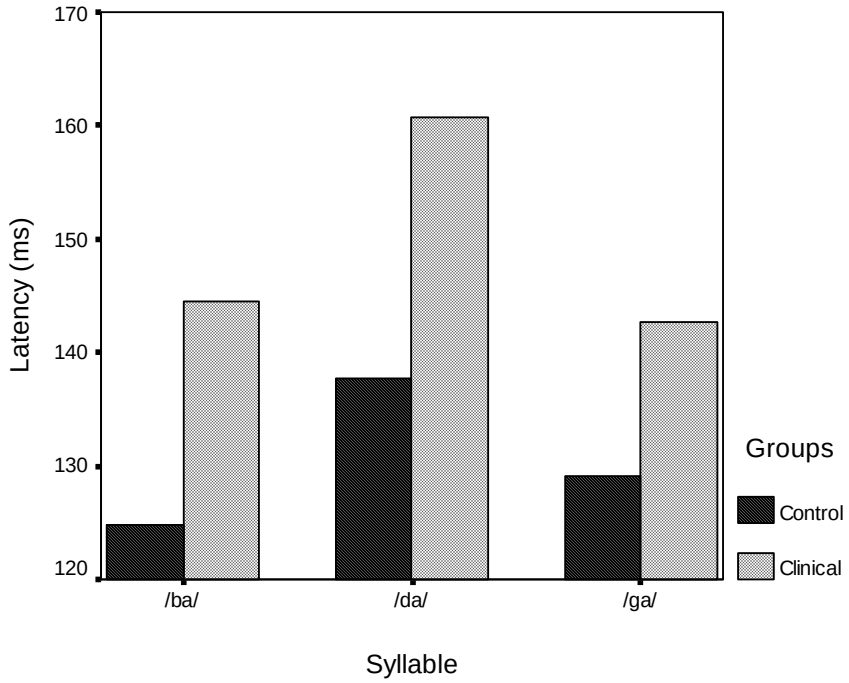

Figure 2 (b): Mean N1 latency values for /ba/, /da/ and /ga/ for control and clinical group at 70 dBnHL.

To compare the clinical and the control group for the N1 latency elicited by three spectrally different speech syllables at two different presentation level, independent t-test was administered. The t-values are shown in the Table 5.

Table 5*: t-values with significance level for N1 latencies elicited by three speech sounds at 40 dB SL and 70 dBnHL between the groups*

Parameter	Level	Syllable	*t*-value
N1 Latency	40 dB SL	/ba/	4.096 **
		/da/	3.680**
		/ga/	4.522**
	70 dBnHL	/ba/	3.085**
		/da/	4.100**
		/ga/	2.218*

** $p < 0.05$, ** $p < 0.01$*

P2 latency

The mean latency values of P2 for control group was longer than the clinical group at 40 dB SL, this can be noticed in the Figure 3(a). It can be noted from the Figure 3 (b), that the mean latency values for P2 wave was prolonged for /ba/, /da/ and /ga/ in the clinical group compared to control group at 70 dBnHL.

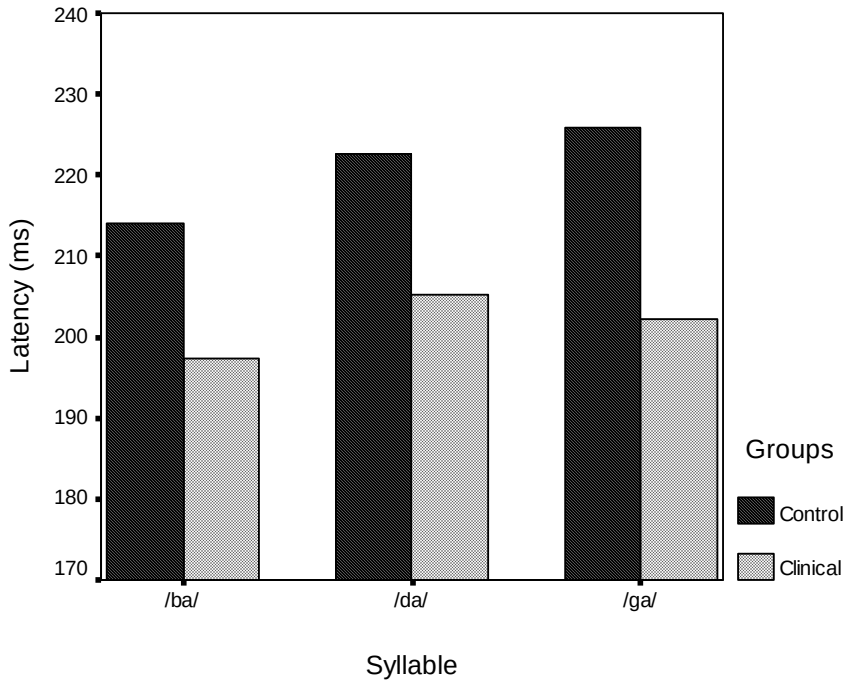

Figure 3 (a): Mean P2 latency values for /ba/, /da/ and /ga/ for control and clinical group at 40 dB SL.

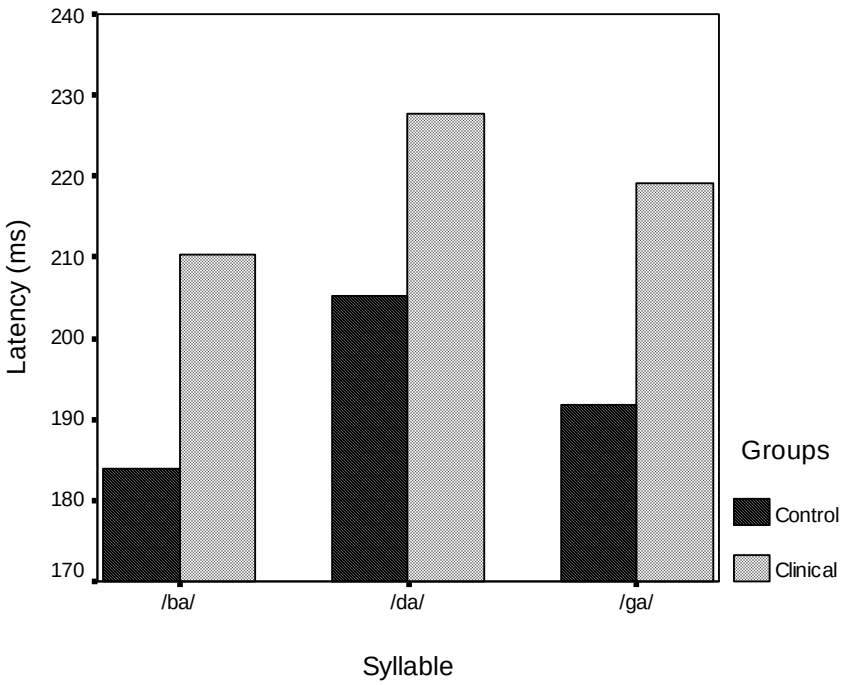

Figure 3 (b): Mean P2 latency values for /ba/, /da/ and /ga/ for control and clinical group at 70 dBnHL.

To compare P2 latency differences between clinical and control group evoked by the three spectrally different speech syllables at two different presentation levels, independent t-test was administered. The details of the independent t-test results are given in the Table 6.

Table 6*: t-values with significance level for P2 latencies elicited by three speech sounds at 40 dB SL and 70 dBnHL between the groups*

Parameter	Level	Syllable	*t*-value
P2	40 dB SL	/ba/	2.409*
		/da/	1.716
		/ga/	3.318**
	70 dBnHL	/ba/	2.716**
		/da/	3.979**
		/ga/	3.714**

** p < 0.05, ** p < 0.01*

Table 6 reveals that, there was a statistically significant difference in P2 latency between the control and the clinical group for /ba/ and /ga/ speech sounds at 40 dB SL. It can also be observed that the latency was significantly different for /ba/, /da/ and /ga/ at 70 dBnHL.

N1-P2 amplitude

The mean amplitude values obtained for different speech sounds are displayed in Figure 4(a) and (b) for both the groups at both 40 dB SL and 70 dBnHL. The mean amplitude values of N1-P2 in clinical group were larger than the clinical group at both 40 dB SL and 70 dBnHL. This trend was noticed for all the three stimuli.

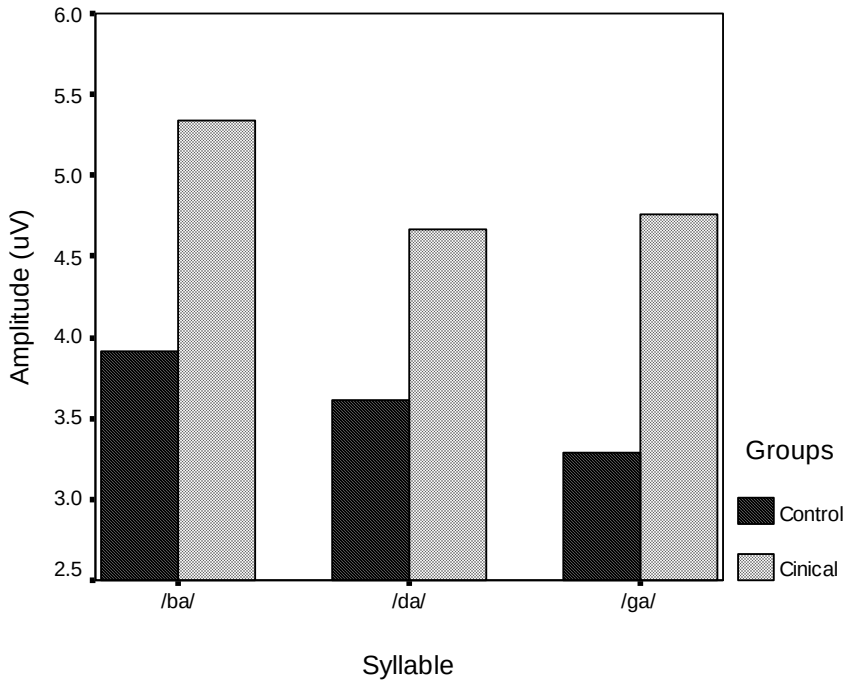

Figure 4 (a): Mean N1-P2 amplitude values for /ba/, /da/ and /ga/ for control and clinical group at 40 dB SL.

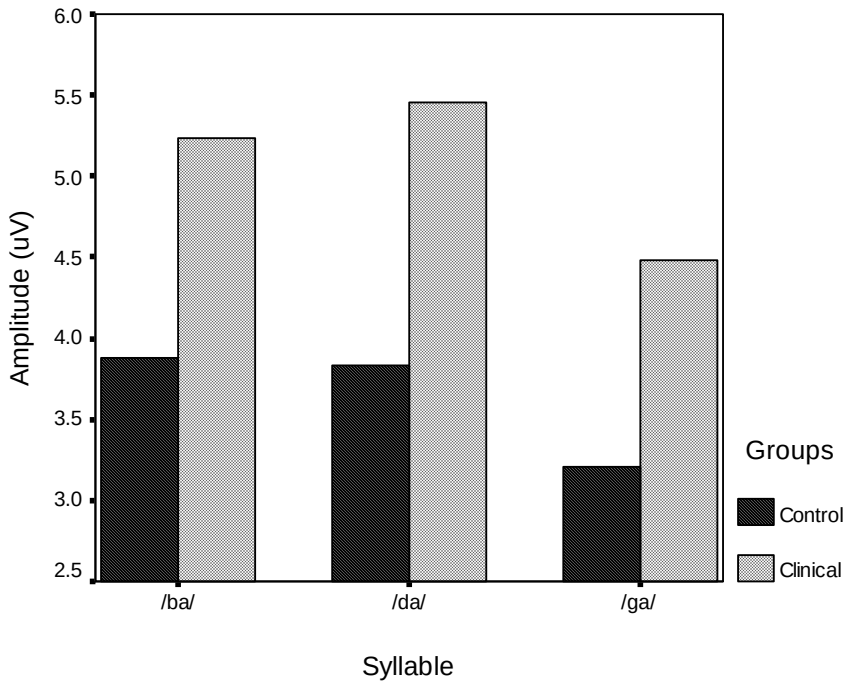

Figure 4 (b): Mean N1-P2 amplitude values for /ba/, /da/ and /ga/ for control and clinical group at 70 dBnHL.

To compare the amplitude of N1-P2 obtained in control and clinical group using three spectrally different speech syllables at two different speech presentation levels, independent t-test was administered. The results of the independent t-test are given in the Table 7.

Table 7*: t-values with significance level for N1-P2 amplitude at 40 dB SL and 70 dBnHL between the groups*

Parameter	Level	Syllable	*t*-value
N1-P2	40 dB SL	/ba/	1.609
		/da/	1.849
		/ga/	2.826**
	70 dBnHL	/ba/	2.068*
		/da/	2.062*
		/ga/	2.397*

** p < 0.05, ** p < 0.01*

It can be seen in the Table 7, that there was a statistically significant difference in N1-P2 amplitude between the control group and the clinical group for all speech stimuli at 70 dBnHL. Whereas, significant difference was obtained for /ga/ at 40 dB SL and no difference obtained for /ba/ and /da/ at 40 dB SL for the N1-P2 amplitude between the groups, though the amplitude was more in the clinical group.

Within group comparison

The data obtained for each groups for different parameters of ALR were analyzed independently. A Two-way repeated measure (3 speech sounds×2 levels) ANOVA were used to check for the effect of the speech stimuli and the level on the latency and the amplitude of ALR parameters within the group. This was done separately for the control group and for the clinical group. Bonferroni post hoc test was administered to see the pair wise comparison, when there was significant difference observed.

Control group

A two-way repeated measure ANOVA results indicated a significant effect of presentation level for latency of three ALR waves at 0.01 levels. The amplitude of N1-P2 did not show any significant effect due to the presentation level, whereas, ALR eliciting syllable had significant effect only for latency of P1 and N1 component. Levels and syllables did not have significant interaction affect for any of the ALR component. The results are displayed in Table 8.

Table 8: *F-values with significance level for P1, N1 and P2 latency and N1-P2 amplitude for /ba/, /da/ and /ga/ in control group*

Parameters	Presentation level	Syllable	Level and syllable
P1	(1,31)=256.56**	(2,62)=3.87*	(2,62)=0.07
N1	(1,31)=218.97**	(2,62)=3.57*	(2,62)=0.11
P2	(1,31)=62.72**	(2,62)=2.38	(2,62)=2.13
N1-P2	(1,31)=0.015	(2,62)=2.355	(2,62)=0.160

** p < 0.05, ** p < 0.01*

Table 9*: Result of Bonferroni post hoc test for effect of syllable for P1 and N1 latency in control group*

(a) *(b)*

Peak	Syllable	/da/	/ga/		Peak	Syllable	/da/	/ga/
P1	/ba/	10.14**	4.42		N1	/ba/	11.18*	3.35
	/da/		5.71			/da/		7.82

** p < 0.05, ** p < 0.01*

It can be noticed from the Table 9(a) and (b), that there was a significant difference between the syllables /ba/ and /da/. The /da/ latency was prolonged when compared to the /ba/ syllable. This is true for both P1 and N1 latencies (Table 3 and Figure 1 & 2).

Clinical group

A two-way repeated measure ANOVA results indicated a significant effect of presentation level for latency of three ALR waves. The amplitude of N1-P2 did not show any significant effect due to the presentation level, whereas, ALR eliciting syllable had significant effect only for latency of P1 and N1 component but not for the P2 latency and for amplitude parameters. Levels and syllables had a significant interaction effect only for P1 latency, but did not have significant affect for any other ALR component. These results are shown in the Table 10.

Table 10*: F-values with significance level for P1, N1, P2 latency and N1-P2 amplitude for /ba/, /da/ and /ga/ in the clinical group*

Parameters	Presentation level	Syllable	Level and syllable
P1	(1,22)=44.70**	(2,44)=5.72**	(2,44)=3.22*
N1	(1,22)=54.69**	(2,44)=10.02**	(2,44)=1.65
P2	(1,22)=33.76**	(2,44)=2.74	(2,44)=2.09
N1-P2	(1,22)=.23	(2,44)=.87	(2,44)=.91

*$p < 0.05$, ** $p < 0.01$*

Table 11*: Result of Bonferroni post hoc test for effect of syllable on P1 and N1 latency in the clinical group*

(a) *(b)*

Peak	Syllable	/da/	/ga/		Peak	Syllable	/da/	/ga/
P1	/ba/	5.95	5.43		N1	/ba/	13.28**	3.19
	/da/		11.39**			/da/		16.47**

** p < 0.05, ** p < 0.01*

Table 11(a), indicates that there was a significant difference between the syllables /da/ and /ga/ for the P1 latency. It can be noted from the Table 3, that the mean P1 value for the /da/ is longer compared to the /ga/ syllable, which could have led to this result. In Table 11(b), it can be noticed that there is statistically significant difference for N1 latency between the /ba/ and /da/ syllable and also between /da and /ga/ syllable. It can also be observed from the Table 3, that the /da/ latency was prolonged when compared to the /ba/ and /ga/ syllable. The /ga/ syllable had the least N1 latency values.

Across syllable

Control group

To find out the significant differences in the latencies of P1, N1 and P2 peaks and the amplitude of N1-P2 across three different speech stimuli within the normal hearing group, one-way ANOVA was administered. This test was carried out for two presentation level and for two groups separately. A Bonferroni post hoc test was done when there was a significant difference.

Table 12*: F- values with significance level for P1, N1, P2 latency and N1-P2 amplitude at 40 dB SL and 70 dBnHL for the control group*

Parameters	F values	
	40 dB SL	70 dBnHL
P1	(2,62)=3.24*	(2,62)=2.58
N1	(2,62)=2.19	(2,62)=2.96
P2	(2,62)=1.15	(2,62)=3.44*
N1-P2	(2,62)=1.30	(2,62)=1.37

** p < 0.05*

Table 12 shows that a significant difference was obtained for P1 latency at 40 dB SL and significant difference for P2 latency at 70 dBnHL. None of the other parameter showed significant effect across the stimuli. The results of the Bonferroni post hoc test are shown in the Table 13.

Table 13*: Result of Bonferroni post hoc test for effect of syllable on P1 and P2 latency in the control group*

(a) *(b)*

Peak	Syllable	/da/	/ga/		Peak	Syllable	/da/	/ga/
P1 at 40 dB SL	/ba/	10.81*	6.43		P2 *at 70 dBnHL*	/ba/	21.44*	7.98
	/da/		6.43			/da/		13.46

** p < 0.05*

It can be observed from the Table 13 (a) that there was a significant difference between the P1 latency elicited by /ba/ and /da/ at 40 dB SL. It can be seen from Table 3 that the mean P1 value for the /da/ stimulus was prolonged compared to the

/ba/ syllable in normal hearing individuals. A statistically significant difference was also obtained between the /ba/ and /da/ syllable for the P2 latency at 70 dBnHL which can be seen in Table 13(b). It is evident in Table 3 that the mean P2 latency values for the syllable /da/ were longer than the /ba/ stimulus. This can also be noticed from the Figure 1 and 3. This would have resulted in significant difference between the two speech sounds for P2 latency.

Clinical group

The results of the one-way repeated measure are shown in the Table 14. It can be noted from the Table that, at 40 dB SL there was a significant effect of syllables only for N1 latency. No significant effect on P1, P2 and N1-P2 parameters observed at 40 dB SL. At 70 dBnHL, there was a significant effect of syllables on the P1, N1 and P2 latencies at a 0.01 level, but there was no effect observed for N1-P2 amplitudes.

Table 14*:F- values with significance level for P1, N1, P2 latency and N1-P2 amplitude at 40 dB SL and 70 dBnHL for clinical group*

Parameters	F values	
	40 dB SL	70 dBnHL
P1	(2,44)=3.09	(2,44)=7.49**
N1	(2,44)=9.02**	(2,44)=8.76**
P2	(2,44)=.743	(2,44)=5.56**
N1-P2	(2,44)=.44	(2,44)=1.79

** $p < 0.01$

Table 15 shows the result of the Bonferroni's post hoc test. It is evident from the Table 15 that there was a significant difference between the N1 elicited by ba/-/da/ and /da/-/ga/ syllable at 40 dB SL. The mean values for the /da/ syllable was prolonged compared to the /ba/ and /ga/ syllable, this resulted in the significant difference.

Table 15: *Result of Bonferroni post hoc test for effect of syllable on N1 latency in the clinical group at 40 dB SL*

Peak	Syllable	/da/	/ga/
N1	/ba/	10.43*	4.60
	/da/		15.04**

* $p < 0.05$, ** $p < 0.01$

In Table 16(a), it can be noticed that there is statistically significant difference between the /da/ and /ga/ syllable for P1 latency at 70 dBnHL. And also from the Table 3, it is clear that the mean P1 latency values for the syllable /da/ are longer than the /ga/ stimulus in the clinical group. This can also be noticed from the Figure 1, 2 and 3. In the Table 16(b), there is significant difference between /ba/-/da/ and /da/-/ga/ syllables for N1 latency. It can be noted in table 3 that /da/ had longer latency when compared to /ga/ and /ba/ syllable. /ga/ had the least latency values. In Table 16(c), it is shown that there was a significant difference in the P2 latencies across /ba/ and /da/. /da/ had prolonged P2 latency when compared to /ba/ syllable.

Table 16: *Result of Bonferroni post hoc test for effect of syllable on P1, N1 and P2 latency in the clinical group at 70 dBnHL*

(a) *(b)*

Peak	Syllable	/da/	/ga/		Peak	Syllable	/da/	/ga/
P1	/ba/	9.65	4.34		N1	/ba/	16.13**	1.78
	/da/		14.0**			/da/		17.91**

(c)

Peak	Syllable	/da/	/ga/
P2	/ba/	17.26**	8.73
	/da/		8.52

** $p < 0.01$

Effect of the presentation level:

To evaluate the effect of the presentation level on each of the parameters of ALR elicited by three different speech stimuli, paired t-test was carried out for each group independently. The results obtained for each presentation level can be seen in Table 17.

Table 17*: t-values along with significance level for control group and clinical group*

Parameter	Syllable	Control group		Clinical group	
		df	t	df	t
P1 Latency	/ba/	31	8.94**	22	4.95**
	/da/	31	9.37**	22	5.31**
	/ga/	31	8.47**	22	5.91**
N1 Latency	/ba/	31	7.88**	22	5.83**
	/da/	31	6.73**	22	5.79**
	/ga/	31	7.41**	22	5.42**
P2 Latency	/ba/	31	5.33**	22	4.92**
	/da/	31	2.17**	22	4.65**
	/ga/	31	9.61**	22	3.91**
N1-P2 Amplitude	/ba/	31	.070	22	.15
	/da/	31	.42	22	1.29
	/ga/	31	.22	22	.63

** $p < 0.01$

Table 17 reveals that, both the control group and the clinical group had a significant effect of the presentation level across the speech sounds on the P1, N1 and P2 latencies, whereas there was no significant effect observed for the amplitude of N1-P2 in both the groups. It can also be noted from the Table 3 that the mean latency values for P1, N1 and P2 for the control group is shorter for all the speech stimuli at 70 dBnHL. However, the latency values were shorter in the clinical group when presented at 40 dB SL. These differences lead to the significant difference in latencies of different ALR waves between the two presentation levels for both the groups.

5. DISCUSSION

Effect of speech stimuli

The speech stimulus in the present study was selected in such a way that it covered the low frequency, mid frequency and high frequency region. The stimuli varied only in the spectral content. All the stimuli selected for the study was voiced CV syllable, the vowel /a/ was kept constant. /ba/ which had a spectral energy concentration majorly in low frequency was selected as low frequency stimuli, /ga/ was selected as mid frequency stimuli and /da/ as high frequency stimuli. The duration of the three stimuli was approximately 150 ms (/ba/-147 ms, /ga/- 146 ms and /da/-150ms).

It has been noticed that latency obtained for /da/ stimulus was longer in both normal and cochlear hearing loss group. This was noticed at both 40 dB SL and 70 dBnHL. However, significant difference was there for P1 latency at 40 dB SL and P2 at 70 dBnHL in control group. The speech stimuli /ba/ elicited a shorter latency in control group both at 40 dB SL and 70 dBnHL. There was significant difference for N1 latency at 40 dB SL in clinical group. The P1, N1 and P2 latency was significantly longer for /da/ at 70 dB SL in individuals with Sensory-Neural hearing loss. The speech stimuli /ga/ elicited a shorter latency in clinical group both at 40 dB SL and 70 dBnHL. Amplitude did not show significant difference across the three speech sounds in both groups at 40 dB SL and 70 dBnHL.

These findings are consistent with the findings of Shruti (2007), she used /i/, /m/ and /s/, and found that the latency of the high frequency content speech stimuli

had a prolonged latency than the other. This can also be supported by results of Agung et al., (2006), they used the speech stimuli /a/, /u/, /i/, /s/, /sh/, /m/ and / כ / which covered a broad range of frequencies across the speech spectrum. They found that the latencies of speech stimuli with high frequency content /s/ and /sh/ had significantly prolonged latencies than the other stimuli. The present results are in agreement with the results of these studies.

This can be attributed to the fact that the high frequency has a less speech energy concentration when compared to the low or the mid frequency syllable. This would have resulted in latencies for /da/.

Another reason could be the duration of the stimulus. The duration of /da/ (150 ms) stimulus was longer than the /ba/(147 ms) and /ga/(146 ms), this difference in the latency for /da / can also be attributed to the duration difference. However, the slight variability in stimulus duration may not cause significant difference in latency difference.

The another physiological reasons for difference in ALR responses for low and high frequency stimuli was investigated using fMRI studies by Yeltin, Ronald, Chriestensen and Purdy, (2004). These investigators reported that the cortical areas that respond to the low frequency auditory information are located more superficially (ie. closer to the surface of the scalp) than the deep layer of the cortical regions for high frequency. Hence, the low frequency stimuli may activate more superficial cortical areas and produce smaller latency of ALR component than the high frequency speech sounds, when surface scalp electrodes are used.

Effect of presentation level

At equal dB SL:

All the speech sounds elicited a shorter latency in the clinical group at 40 dB SL. All most all the peak latencies differed between the groups was statistically significant at 40 dB SL. Amplitude obtained in clinical group was significantly more only for /ga/ stimuli.

When the presentation level of the stimulus was 40 dB SL, it was much higher for clinical group when compared to the control group. Higher the intensity reaching the ear, broader will be the excitation of the basilar membrane which leads to excitation of more number of auditory nerve. Hence this could have resulted in faster transmission and shorter latency and more amplitude of ALR components in clinical group.

Another reason for decrease in the latency with an increase in the stimulus intensity could be due to the progressively faster rising generator potential within the cochlea and similarly faster development of excitatory post synaptic potential (Moller, 1981). Picton and Hillyard, (1974) reported that the latency of the compound action potential directly depends on how quickly the generator potential and the excitatory post synaptic potential reach the threshold for firing. Hence, this would lead to shorter latency in cochlear hearing loss group when presented at 40 dB SL as the presentation level was much higher in this group than the control group.

Increase in the amplitude parameters with the increase in the stimulus intensity may be because of the increase in the audibility of the stimulus. This is supported by

Hall (1992). He said that the AEPs amplitude increases with the increase in the intensity. The amplitude of an AER is decided by the number of neurons firing for particular stimulus intensity. At higher intensities, the number of neuron beginning to fire will be more and amplitude of the compound action potential thus generated will be high. This would have result in the high amplitude evoked responses in cochlear hearing loss group.

In control group the presentation level would have been approximately 40 to 55 dBnHL, which was much lesser than clinical group. In normal hearing individuals the active mechanism was dominated at this intensity level, leading to sharp tuning of the basilar membrane. Thus resulted in excitation of less number of auditory nerve, and less volume conduction, which leads to slow transmission. This might have leads to longer latency and lesser amplitude of response in the control group.

At equal dBnHL

At 70 dBnHL latency of all the ALR waves were shorter for control group. All most all the peak latencies differed between the groups was statistically significant at 70 dBnHL. Amplitude obtained in the clinical group for all speech sounds were more for all speech sounds at 70 dBnHL.

The latencies were shorter in the control group and prolonged in the clinical group. This can be supported by the fact that at 70 dBnHL both the passive as well as the active mechanism are responsible were equally important in normal hearing individuals, which leads to faster transmission and shorter latency. In the clinical group the energy reaching to the cochlea was less as they had hearing loss. The level

would have reduced with the increase in severity of hearing loss. Hence, less compound action potential would have generated which would have lead to slower transmission and thus led to longer latency.

To conclude, the speech stimuli dominated by high frequency energy elicited a latency which was longer than the other sounds; this was true for both control as well as clinical group. These findings can be supported by Agung et al.,(2006) and Shruthi (2007). ALRs recorded for three stimuli at each presentation level differ significantly in control and clinical group. This suggests that the speech processing is altered in clinical group which leads to reduced speech perception abilities in clinical group.

The comparison across groups at equal hearing level were done in order to see the difficulties that the hearing impaired individuals will face in day to day situation. As we know that in day to day situation both normal and hearing impaired individuals would be exposed to sounds at equal hearing levels.

At equal presentation level the transmission of signal could be slower due to reduced energy at the cochlea. This suggests that in individual with cochlear hearing loss temporal processing may be affected if the signal is low.

At 40 dB SL the transmission of information is faster in clinical group, but still the processing is affected in clinical group. This can be due to the degraded frequency resolution due to broadening of the basilar membrane excitation. In sensorineural hearing loss group, speech perception ability is correlated with the pure tone threshold. The cochlear distortion effects, increases with the increase in the

degree of hearing loss, which results in loss of cochlear amplifier leading to poor speech perception abilities (Moore, Poston, Eggermont & Huang, 1996).

The results of the present study and the earlier reports indicate that the latencies probably depend on the stimulus used for evoking the responses and the latency probably depend on the spectral content of the stimuli used. This is true in normal hearing as well as in individuals with cochlear hearing loss.

6. SUMMARY AND CONCLUSION

The neural encoding of sound stimulus begins at the auditory nerve and continues till the cortex. The perception skills can be evaluated by using auditory late latency responses. It can be used to document high level central auditory dysfunction in patients with abnormal ABR findings. It can be used in evaluating auditory processing disorders in learning disabilities and auditory neuropathy (Hall, 2007). Auditory late latency responses have been recently used to determine the effect of phonologic and acoustic features (Crottaz-Herbette & Ragot, 2000) and to identify the cortical areas activated by these features (Makela, Alku & Tiitinen, 2003).

Studying the neural encoding of speech sounds provides insight into some of the auditory processes involved in normal communication. The speech processing abilities can be studied in the normal hearing individuals as well as in the hearing impaired population. Taking all these into consideration the present study was carried out to evaluate the neurophysiological processing of three spectrally different speech sounds in normal hearing and cochlear hearing loss individuals using ALLR.

Speech perception of individuals with cochlear hearing loss is poorer relative to normal hearing individuals in spite of presenting stimuli at most comfortable levels. This is because spectral and temporal cues of speech get distorted at the peripheral level before reaching to the higher structures. Hence, it was hypothesized that cortical processing may be abnormal in individuals with cochlear hearing loss as cortical structures receive abnormal inputs from the lower auditory structures. Thus the present study aimed at:

- Whether the auditory late latency responses recorded for spectrally different syllables differ significantly in normal hearing adults.
- Whether the auditory late latency responses recorded from spectrally different syllables differ significantly in hearing impaired adults.
- Whether the ALRs from two groups differ significantly.
- To investigated the difference in ALR between the normal hearing and cochlear hearing loss individuals when the signal reaching was at same level and at different level.

The study consisted of a control group and clinical group. Control group consisted of 32 ears and clinical group consisted of 23 ears in the age range of 18 to 55 years. ALRs were evoked from all the participants by three different speech stimuli; /ba/ dominated by low frequency spectral energy, /ga/ dominated by mid frequency spectral energy and /da/ dominated by high frequency spectral energy. The ALRs were evoked at two presentation levels, 40 dB SL and 70 dBnHL.

ALRs were obtained for all the participants for all three stimuli. The data obtained from the participants of the study was analyzed by two experienced judges and the latencies of P1, N1 and P2 were marked and the amplitudes of N1-P2 were noted. The data was subjected to statistical analyses by using SPSS version 15.0 for windows. The mean, SD and range were calculated for both the groups. Latencies and amplitude obtained from both the group were compared across three speech stimuli elicited at 40 dB SL and 70 dBnHL separately. Comparison across the presentation level (40 dB SL and 70 dBnHL) was done for both the groups for ALR components

elicited by three speech sounds. Comparison of the ALR components across the groups was done for each syllable and presentation level.

Effect of stimuli

The different speech stimuli elicited different responses in both the groups at both levels. The latency obtained for /da/ stimulus was longer in both normal and cochlear hearing loss group both at 40 dB SL and 70 dBnHL. These findings are consistent with the findings of Agung et al., (2006) and Shruti (2007). They found that the latencies of speech stimuli with high frequency content had significantly prolonged latencies than the other stimuli. It was concluded that since high frequency stimulus has a less speech energy concentration, it evoked longer latency.

A statistical significance was also noticed for each ALR parameters between the groups for each speech sound at each presentation level. This suggests that cortical processing of speech sounds are altered in cochlear hearing loss individuals.

Effect of presentation level

At equal dB SL

All the speech sounds elicited a shorter latency in the clinical group at 40 dB SL. Amplitude obtained in clinical group was significantly more only for /ga/ stimuli. It was concluded that the presentation level to the clinical group was higher when compared to the control group. Higher the presentation level, broader will be the excitation of the basilar membrane which leads to excitation of more number of auditory nerve. Hence, this resulted in faster transmission and shorter latency and

more amplitude of ALR components in clinical group. Similar observation was also made by Moller, 1981.

Increase in the amplitude parameters with the increase in the stimulus intensity may be because of the increase in the audibility of the stimulus. Similar observation was made by Hall (1992). At higher intensities, the number of neuron beginning to fire will be more and amplitude of the compound action potential generated will be high. This would have resulted in the high amplitude evoked responses in cochlear hearing loss group.

In control group the intensity at which stimulus presented was less compared to clinical group. In normal hearing individuals the active mechanism would be predominant, leading to sharp tuning of the basilar membrane, thus resulting in excitation of less number of auditory nerve, less volume conduction, which leads to slow transmission. This might have lead to longer latency and lesser amplitude of response in the control group.

At equal dBnHL

At 70 dBnHL latency of all the ALR waves were shorter for control group. Amplitude obtained in the clinical group for all speech sounds were more for all speech sounds at 70 dBnHL. This can be supported by the fact that at 70 dBnHL both the passive as well as the active mechanism are responsible in normal hearing individuals, which leads to faster transmission and shorter latency. In the clinical group the energy reaching to cochlea was less due to hearing impairment. Hence, less

compound action potential was generated, which would have lead to slower transmission. This in turn led to longer latency.

The results indicate that the speech processing is effected in individuals with cochlear hearing loss which can observed from the results of ALR, which can also be seen behaviorally. At the cortical level, the responses for the three different stimuli were significantly different from the normal hearing individuals, indicating that the processing of the signal in the hearing impaired population is altered.

Conclusion

It can be concluded that the ALR recorded by spectrally different speech sounds are different in both normal hearing and cochlear hearing loss individuals. This suggests that neurophysiological processes are different for different speech sounds. Longer latency for /da/ suggests that latency of the processing at the cortical center is also different depending on the frequency composition of the signal. A significant difference between the groups for all the parameters for all the speech sounds at each presentation level suggests that speech processing is altered in individuals with cochlear hearing loss.

Clinical implication of the present study

The study can have the following implications:

- ALR elicited by different speech stimuli can be used as a electrophysiological tool to evaluate the processing of speech sounds in normal population as well as in the impaired population.

- The data obtained in normal hearing individuals for three different speech sounds can be considered as reference to assess processing deficit in clinical population.
- It can be used in assessment of neurophysiological development of early age.
- ALR evoked by speech stimuli can be used to assess the benefit provided by the hearing aid.
- To evaluate benefit from the cochlear implant.
- To monitor prognosis of rehabilitation program and post therapy improvement.

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