RELATIONSHIP BETWEEN AUDITORY LONG LATENCY RESPONSE AND SPEECH IDENTIFICATION SCORES IN INDIVIDUALS WITH AUDITORY NEUROPATHY

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MAY 2009.

Dedicated to my Mom, Dad, Granny,

Sister, Brother-in-law and to

all others, who helped me in achieving this.

Certificate

This is to certify that this dissertation entitled ***"Relationship between Auditory Long Latency Response and Speech Identification Scores in Individuals with Auditory neuropathy"*** is a bonafide work in part of fulfillment for the degree of Master of Science (Audiology) of the student Registration no: 07AUD013.This has been carried under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any diploma or degree.

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Declaration

This is to certify that this master's dissertation entitled ***"Relationship between Auditory Long Latency Response and Speech Identification Scores in Individuals with Auditory neuropathy"*** is the result of my own study under the guidance of Dr. Animesh Barman, Lecturer, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier to any other university for the award of any degree or diploma.

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1. INTRODUCTION

Auditory neuropathy (AN), more recently referred to as auditory dys-synchrony (Berlin, Hood & Rose 2001), is one of the hearing disorders in which cochlear outer hair cell function is spared but neural transmission in afferent pathway is disrupted. The integrity of cochlear function in these individuals is indicated by the presence of evoked otoacoustic emissions and/or cochlear microphonics (CM). The abnormal neural transmission or dys-synchrony in the auditory nerve fibers is indicated by the absence of auditory brainstem responses and acoustic reflexes (Rance et al., 2002).

Audiological and electrophysiological test findings in auditory neuropathy are suggestive of a retro-cochlear pathology, but the exact site of pathology and pathophysiological mechanism leading to auditory neuropathy is not known. Two physiological explanations proposed for the neurophysiological manifestations observed include dys-synchronized spikes and/or reduced spike of the auditory nerves (Rance et al., 2002).

Some possible sites of lesion that could produce the audiometric and electrophysiological profile of AN include: inner hair cells, synaptic junction between inner hair cell and type I afferent nerve fibers, spiral ganglion cells, demyelinization of type I auditory nerve fibers and reduce number of type I auditory nerve fibers. Therefore, AN consists of many varieties, depending on the site of lesion (Starr, Picton, Sininger, Hood & Berlin, 1996).

Hearing sensitivity in individuals with auditory neuropathy may range from normal hearing to profound hearing impairment (Rance, Beer & Cone-Wesson, 1999; Starr, Sininger & Pratt, 2000). However, hearing loss is generally not uniform across frequencies. A majority of the individuals with auditory neuropathy have low frequency hearing loss with wide range of speech identification scores. These individuals typically have speech identification scores that are out of proportion to their degree of hearing impairment and do not benefit from conventional amplification. Poor speech perception abilities in these patients are attributed to abnormal temporal coding and asynchrony (Zeng, Oba, Sininger & Starr, 1999; Kraus et al., 2000; Rance, McKay & Grayden, 2004; Zeng, Kong, Michalewski & Starr, 2004).

Speech perception abilities in these patients appear to depend on the extent of supra-threshold temporal distortions of cues rather than access to speech spectrum, unlike the patients with sensory hearing loss. Zeng et al. (1999) observed abnormal results on two measures of temporal perception in their group of children with AN: (i) gap detection threshold (identification of silence embedded in within the bursts of noise) and (ii) temporal modulation transfer function (measure of sensitivity to slow and fast amplitude fluctuation). They also found a correlation between temporal modulation transfer function (TMTF) and speech perception abilities in their patients.

Rance et al. (2004) also reported poor performance on the task involving timing cues (TMTF, temporal aspects of frequency discrimination) in a group of children with AN. These temporal processing abnormalities had significant correlation with speech

perception abilities. They attributed the poor speech perception scores in relation to pure tone hearing loss to these supra-threshold temporal processing deficits.

Need for the study

In auditory neuropathy/dys-synchrony, auditory brainstem responses are severely disrupted. Hence, it might be expected that more central evoked responses such as the middle latency response and cortical auditory evoked potential (CAEP) would be similarly affected. However, CAEPs may be recordable in some cases of auditory neuropathy/dys-synchrony because these potentials are less dependent on synchronous firing of the auditory nerve than auditory brainstem responses. Many individuals with AD had normal CAEP latencies and amplitudes (Starr et al., 1996; Rance et al., 2002). Hence, the current study has been designed to record ALLR in individuals with normal hearing and also with AD.

Infants with auditory neuropathy and possible hearing impairment are being identified at very young ages through the implementation of hearing screening programs. The diagnosis is commonly based on evidence of normal cochlear function but abnormal brainstem function. This lack of normal brainstem function is highly problematic when prescribing amplification in young infants because prescriptive formulae require the input of hearing thresholds that are normally estimated from auditory brainstem responses to tonal stimuli. Without this information, there is great uncertainty in providing amplification. Cortical auditory evoked potentials may, however, still be evident and reliably recorded to speech stimuli presented at conversational levels. In these clinical

populations, it can also be used to evaluate the benefits with rehabilitative measures. Thus, click and speech is used as stimulus to record ALLR.

In individuals with AD, the audiometric configuration with a low-frequency emphasis (reverse slope) is a common finding (Rance, Beer & Cone-Wesson, 1999). Sininger and Oba (2001) and Starr, Sininger and Pratt (2000) showed similar findings, with rising audiograms reported in about 30% of ears in both the studies. The high-frequency hearing loss configuration which is most commonly seen with sensorineural hearing loss was only observed in approximately 10% of individuals with AD.

Hence, different speech sounds composed of different spectral energy composition would be preferable to obtain ALLR in individuals with AD. This might suggest the processing of different speech signal having different frequency energy concentration. There is a dearth of information in which they correlate whether speech evoked or click evoked ALLR parameters represents the speech perception ability in these individuals. Thus, the present study was under taken to record ALLR for three different speech stimulus having different spectral energy.

Martin and Stapells (2005) investigated the event related potentials (ERP) using low pass noise with different cutoff frequencies. The speech sounds (/ba/ & /da/) were presented at 65 and 80 dB SPL. The results indicated that as the cutoff frequency to low pass noise masker was raised, ERP latencies increased and amplitudes decreased. N1 showed a smaller decrease in amplitude and smaller increase in latency when compared to other parameters. These results suggest that decreased audibility caused by noise

affected N1 in a differential manner compared to later waves (N2 & P3). Thus, the present study also attempted to record ALLR in the presence of noise.

A very few investigations have been reported that AN individual having poor speech identification scores in quiet have abnormal or absent click evoked cortical potentials, suggesting that integrity of processing at cortical level is important for speech understanding (Rance et al., 2002). Investigations using speech stimuli for cortical potentials are even lesser. Numbers of subjects taken were very less; hence, such study should be done using more number of subjects.

Cunningham, Nicol, Zecker and Kraus (2001) found similar fundamental sensory representation of speech between normal children and learning problem populations when evoked responses were recorded in quiet, but learning problem children demonstrated neurophysiological abnormalities at both cortical and sub-cortical levels when the speech was presented in background noise. In that study, noise degraded sub-cortical encoding of both transient and periodic stimulus features in the auditory brainstem and frequency following responses in learning problem children. Additionally, cortical responses in noise showed a dramatic amplitude decrease in learning problem children with respect to normal children in the P2-N2 complex occurring between 150–250 ms post-stimulus onset.

Since individuals with auditory neuropathy also had difficulties in understanding speech in the presence of noise, evaluating these individuals using ALLR with and without noise helps us in understanding the processing of speech better. No studies have been done to correlate ALLR in noise and speech identification scores in noise. So

research is required in optimizing whether click evoked or speech evoked cortical potentials correlates better with speech identification scores.

Aims of the study

Thus the current study taken up with the aim to:

- know whether the ALLR vary for different speech sounds in quiet and with ipsilateral noise in normal hearing individuals and individuals with AN/AD.
- investigate the relationship between the click evoked ALLR and speech identification scores in quiet and noise in individuals with normal hearing and with AN/AD.
- investigate the relationship between the speech evoked ALLR and speech identification scores in quiet and noise in individuals with normal hearing and with AN/AD.
- know whether the non-speech stimulus or speech stimulus is better to elicit ALLR in individuals with AN/AD.
- know which speech sounds is more suitable to elicit ALLR in individuals with auditory dys-synchrony.

2. REVIEW OF LITERATURE

Auditory neuropathy (also referred to as *auditory dys-synchrony*) is a term used to describe a range of disorders characterized by absence of auditory brainstem responses (ABR) in the presence of normal otoacoustic emissions and/or cochlear microphonics (Starr, Picton, Sininger, Hood, & Berlin, 1996). However, if ABRs are present, then they are of poor morphology (Starr et al., 1996). That is, a person with auditory dys-synchrony presents evidence of normal outer hair cell functioning, but abnormal auditory nerve functioning.

Audiological findings like acoustic reflex, ABRs, masking level difference and efferent suppression of otoacoustic emissions are absent, as all these require intact auditory nerve or brainstem function, which are abnormal in these individuals. However, the extent of abnormality is disproportionate to the subject's audiometric thresholds for pure tones.

2.1 Pathophysiology of Auditory Dys-synchrony

Individuals with auditory neuropathy/ dys-synchrony (AD/AN) can present with a wide range of clinical symptoms, which can be due to differing degrees of the same pathology or may be the result of a range of distinct auditory pathway disorders. Some possible sites of lesion include the cochlear inner hair cells, the synapse between the inner hair cells and type 1 auditory nerve fibers, and the auditory nerve itself (Starr et al., 1996; Rance, Beer & Cone-Wesson, 1999; Amatuzzi, Northrop & Liberman, 2001).

2.1.1 Inner Hair Cell Loss

The pathological condition in auditory neuropathy/dys-synchrony pattern can be restricted to the inner hair cells only. An inner hair cell abnormality could result in the absence of the entire auditory brainstem response, including wave I, with the preservation of outer hair cell responses (Starr et al., 1996). These findings are consistent with AN, suggesting that the pathology in AD could be inner hair cell loss.

Amatuzzi, Northrop and Liberman (2001) identified 2 out of 15 non-survivors from NICU with loss of both inner and outer hair cells, 2 with loss of outer hair cells alone, and 3 babies with selective inner hair cell loss. These infants with specific inner hair cell loss were tested for auditory brainstem response and showed no response at screening levels (40 dB nHL), which could be due to the reduced number of inner hair cells rather than an insult to the neural elements. Similar results were found in animal studies, which suggest that certain types of cochlear insult like prolonged hypoxia can have a greater effect on inner hair cells rather than outer hair cell survival (Bohne, 1976; Shirane & Harrison, 1987; Billet, Thorne & Gavin, 1989).

Mc Mohan, Patuzzi, Gibson and Sanli (2008) conducted a study on 14 individuals with auditory neuropathy. They investigated the possible physiological mechanisms underlying this disorder, using frequency specific electrocochleography. They found two dominant patterns, one group having delayed summating potential with small or no dendritic potential indicating a pre-synaptic mechanism of AN and other group showed electrocochleography waveforms with normal latency of summating potential followed

by a broad negative dendritic potential waveform indicating a post-synaptic mechanism of AN.

2.1.2 The Synapse between the Inner Hair Cells and Auditory Nerve Terminals

A disorder at the synapse between the inner hair cells and type 1 auditory nerve fibers has also been proposed as a mechanism that could produce the auditory neuropathy/dys-synchrony (Starr et al., 1991). In response to sound stimulation, the neurotransmitters which are at the base of the inner hair cell acts upon the receptor sites and helps in generation of action potential. Disorders at this site may be presynaptic, involving the release of transmitters or postsynaptic, affecting the ability of the receptor sites on the auditory nerve dendrite to respond these substances (Starr, Sininger & Pratt, 2000).

The mechanisms by which synaptic disruption occurs in the auditory pathway in human subjects are yet to be determined. Genetic dysfunction involving disruption of the otoferlin (OTOF) protein which is present in inner hair cells affecting the transmitter release has been identified in subjects presenting with the auditory neuropathy/dys-synchrony (Varga, Kelley & Keats, 2003).

2.1.3 Auditory Nerve Abnormality

As the term auditory neuropathy suggests, the affected site in these patients can involve the auditory nerve itself. Starr et al. (1996) found that 8 of their 10 subjects participated in the study had evidence of other peripheral nerve abnormality in addition to hearing loss. This includes neuropathy of the other cranial nerves, weakness and muscle atrophy. Overall the generalized neuropathic disorders have been indicated in 30% to

40% of reported auditory neuropathy/dys-synchrony cases. About 80% of these patients had onset occurring after 15 years. The site of the disorder affecting the auditory nerve and auditory brainstem in these cases may be the myelin sheath or the neuron itself.

2.1.3.1 Myelin Disorder

The myelin sheath helps in transmission of action potentials across the neuron. Partial or complete loss of myelin results in an increase- in membrane capacitance and a decrease in membrane resistance, thereby it can have profound effects on the generation and propagation of action potentials within auditory nerve fibers. This leads to a delayed excitation, a reduction in the velocity of action potential propagation, and an increase in conduction vulnerability (McDonald & Sears, 1970; Rasminsky & Sears, 1972; Pender & Sears, 1984). Fibers that are demyelinated to differing degrees conduct neural signals at different speeds, and hence the synchrony of discharges will be affected (Starr, Picton & Kim, 2001).

The pathophysiological changes in neural conduction properties associated with demyelination are likely to have profound effects on the auditory brainstem response. ABR require precise synchronous response of a group of auditory nerve fibers to a transient acoustic stimulus. Reductions in the temporal synchrony of demyelinated VIII nerve fibers lead to significant reduction in the amplitude of the averaged evoked responses. Demyelinated fibers are sensitive to increase in temperature and may develop conduction block and can also display emphatic transmission (cross talk) between fibers with one active fiber setting of discharge in adjacent fibers (Starr et al., 1996).

2.1.3.2 Axonal Neuropathy

Axonal neuropathy is characterized by normal conduction velocity and reduced amplitude of compound action potentials. Axonal damage can occur in isolation as a result of specific disease processes or can occur in conjunction with or as a consequence of demyelinating conditions (Starr, Sininger & Pratt, 2000).

Axonal neuropathies reduce the number of neural elements but do not directly affect conduction speed. Therefore, a reduction in the amplitude of the whole nerve action potential and reduction of amplitude of auditory brainstem response rather than an increase in latency or a broadening of these potentials (as in the patients with myelin related disorders). Since the individuals with auditory dys-synchrony display the abnormal auditory brainstem responses which can be due to myelin or axonal neuropathies, these two are clinically undistinguishable (Starr, Sininger & Pratt, 2000).

2.2 Audiological profile of individuals with auditory dys-synchrony/ neuropathy

Audiological profile of persons with auditory dys-synchrony is highly variable, with a wide range of clinical symptoms, which can be due to differing degrees of the same pathology or may be the result of a range of distinct auditory pathway disorders. Some possible sites of lesion include the cochlear inner hair cells, the synapse between the inner hair cells and type 1 auditory nerve fibers, and the auditory nerve itself (Starr et al., 1996; Rance, Beer & Cone-Wesson, 1999; Amatuzzi, Northrop & Liberman, 2001).

2.2.1 Behavioural audiogram:

The degree of hearing loss in these individuals can range from normal hearing sensitivity to profound hearing loss. Majority of these patients show bilateral symmetrical

hearing loss (Rance, Beer & Cone-Wesson, 1999; Sininger & Oba, 2001). It is often difficult to determine the degree of hearing loss in persons with auditory dys-synchrony due to inconsistent responses and reverse sloping or peaked audiograms.

Starr, Sininger and Pratt (2000) found that the average hearing levels were less than 35 dBHL in 31% of ears, 39% of ears between 35 and 70 dB HL, and greater than 70 dBHL in 30% of the ears. Madden, Rutter and Hilbert (2002) had found an even spread of behavioural audiograms, with 6 (33%) in their group of 18 affected children presenting with audiograms in the normal-to-mild hearing loss range, 6 in the moderate-to-severe hearing loss range, and 6 having profound hearing loss range.

2.2.2 Hearing Loss Configuration

Audiograms with a low-frequency emphasis (reverse slope) are a reasonably common finding in both adults and children with auditory neuropathy/ dys-synchrony. Rance, Beer and Cone-Wesson (1999) noticed that subject's audiometric configuration varied with the degree of hearing loss. Ears with normal or near normal hearing acuity showed equal sensitivity at all the frequencies. 30% of subjects with mild to severe hearing loss had audiograms with poor hearing sensitivity in the low and mid frequencies, but better thresholds at high frequencies. Starr, Sininger and Pratt (2000), in a study of 67 patients with auditory dys-synchrony, reported flat audiogram in 41%, reverse sloping in 29%, an irregular saw-tooth pattern in 9%, a 'U' shaped audiogram in 5%, and a tent shaped audiogram with a peak usually at 2 kHz in 5% of the patients. However, 43% of the patients of Sininger and Oba (2001) showed flat audiometric shape, while 28% had reverse sloping configuration. The high-frequency hearing loss configuration most

commonly seen with sensorineural type hearing loss was observed only in approximately 10% of cases in these studies.

2.2.3 Threshold Stability

Patients with auditory neuropathy/dys-synchrony show fluctuation in both hearing level and perceptual ability (Kumar & Jayaram, 2005). Rance, Beer and Cone-Wesson (1999) observed significant hearing level fluctuations with threshold variances of approximately 20 dB on repeated measures.

Madden, Rutter and Hilbert (2002) found that 9 of the 22 auditory neuropathy/ dys-synchrony children showed spontaneous hearing recovery. In most of the subjects, the behavioural audiograms improved from profound to moderate-to-severe range, but in 4 subjects, hearing thresholds reportedly improved to normal or near-normal levels. Hearing recovery was more likely in this group amongst the subjects who had suffered neonatal hyperbilirubinemia, and in all cases recovery had occurred before the age of 25 months.

2.2.4 Acoustic Reflexes

In individuals with AN/AD abnormal middle-ear muscle reflexes are a consistently reported finding. Acoustic reflexes have been absent for both ipsilateral and contralateral acoustic stimulation irrespective of severity of hearing loss. Recent reports have shown that the non-acoustic middle-ear muscle reflexes can be elicited in auditory neuropathy patients by tactile stimulation to the face, suggesting that the efferent components of the reflex arc (facial nerve and stapedius muscle) are intact in many

clients with AD (Gorga, Stelmachowicz & Barlow, 1995; Starr, Sininger & Winter, 1998).

Konradsson (1996) found that in 4 children with unilateral auditory neuropathy/ dys-synchrony an acoustic reflex in the AN/AD ear could be elicited by contralateral stimulation but that neither ipsilateral nor contralateral responses were present when the stimulus was directed to the affected side. It is most likely that in these patients, the afferent pathway (auditory nerve) is not able to provide sufficient synchronized rates of discharge to activate the motor neurons of the stapedius muscle (Starr, Sininger & Winter 1998).

2.2.5 Auditory brainstem responses:

Auditory brainstem responses are absent (or grossly abnormal) at maximum stimulus presentation levels regardless of behavioural hearing level in auditory neuropathy/dys-synchrony individuals (Starr et al., 1996; Rance, Beer, & Cone-Wesson, 1999; Sininger & Oba, 2001). In such clients, disruption of the auditory brainstem response is thought to be the result of either a reduction in the number of neural elements available to contribute to the response, or a disruption in the temporal integrity of the neural signal.

The main positive peaks in the auditory brainstem responses are separated by only about 1 ms. Thus, successful recording of the averaged response requires that the timing of discharges within the auditory brainstem be almost identical after each test stimulus. Various authors have suggested that a dys-synchrony in the neural firing of the order of fractions of a millisecond (Starr et al., 1991; Sininger Hood & Starr, 1995; Kraus et al.,

2000) is sufficient to disrupt the response and render the averaged potentials unrecognizable.

2.2.6 Otoacoustic Emissions

Otoacoustic emissions (OAE's) are sounds that generate in the cochlea and propagate through the middle ear that is recordable in the ear canal (Kemp, 1978). OAE's are by-product of the active bioelectric process of outer hair cells (Davis, 1983).

OAE's helps in differentiating between ears with normal cochlear (outer hair cell) function and those with sensorineural hearing loss (Harris & Probst, 2002). Since the individuals with AN/AD have normal cochlear outer hair cell functioning, OAE's helps in differentiating AN/AD from pure sensorineural hearing loss. Starr, Sininger and Pratt (2000) conducted a study on adults and children with auditory neuropathy and found that in 19 of 63 ears (30%) TEOAEs could not be detected and no relation was observed between pure tone audiogram hearing level and presence/ absence of otoacoustic emissions in their subjects, a result consistent with the findings from Rance, Beer and Cone-Wesson (1999).

In some individuals with auditory neuropathy, OAE's may disappear over time and the mechanisms underlying the deterioration of OAE's are yet to be determined. Starr, Sininger and Pratt (2000) also found that otoacoustic emission responses in 9 subjects disappeared over time in the absence of middle ear disease and amplification. However, even when otoacoustic emissions are absent in individuals with auditory neuropathy, cochlear microphonics may be present (Rance et al., 2002).

2.2.7 Cochlear Microphonics:

The cochlear microphonic is an alternating receptor potential produced by the polarization and depolarization of the cochlear hair cells and it provides a bioelectric analog of the input (hence termed as *microphonic*). The response is preneural and it shows little or no latency delay from the onset of the stimulus. Starr, Sininger and Nguyen (2001) found that in normals the latency of initial peak in the cochlear microphonic waveform occurred around 0.42 msec.

The cochlear microphonics is sensitive to the phase of the eliciting stimulus and can be identified by using rarefaction and condensation clicks (Sohmer & Pratt, 1976; Berlin, Bordelon & John, 1998). In contrast, the polarity of neural responses is unaffected by the phase of the stimulus waveform, although variations in the latency of the compound action potential of Wave I in the auditory brainstem response with the stimulus phase can give the appearance of response phase changes (Stockard, Stockard & Wesmoreland, 1979).

The presence of cochlear microphonic reflects the integrity of cochlear hair cells and it helps in identifying ears with auditory neuropathy/dys-synchrony. An absence or severe abnormality of the auditory brainstem response at maximum presentation levels in ears with sensorineural hearing loss is consistent with significant cochlear damage. In such cases, the presence of cochlear microphonic is indicative of at least some degree of outer hair cell function and is therefore suggestive of neural transmission abnormality in ears with absent or disrupted brainstem potentials (Chisin, Pearman & Sohmer, 1979;

Starr, McPherson & Patterson, 1991; Berlin, Hood & Cecola, 1993; Starr et al., 1996; Berlin, Bordelon & St. John, 1998).

Rance, Beer, and Cone-Wesson (1999) carried out cochlear microphonics and TEOAE assessment in 33 of the affected ears. In 16 ears robust otoacoustic emissions are observed which are consistent with the presence of some degree of outer hair cell function. However, 17 ears showed no emission response despite the presence of clear cochlear microphonics.

Delentre, Mansbach and Bozet (1999) also reported a similar result where they described the findings of 2 children, who were identified with auditory neuropathy in infancy showing present otoacoustic emissions/ cochlear microphonic responses. Subsequently, they lost their OAE's but the cochlear microphonic responses in these children were relatively unchanged.

2.2.8 Psychophysical test results in individuals with Auditory Dys-synchrony

Several psychophysical tests were administered in individuals with auditory neuropathy to assess the processing deficit. The information regarding the psychophysical tests that were administered and results obtained are gathered below.

2.2.8.1 Intensity Processing:

Zeng, Oba and Garde (2001) analyzed the loudness growth function in one subject with auditory dys-synchrony and results indicated that the subject had a larger compressive loudness function than the normal control subject. In another study, Rance, McKay and Grayden (2004), demonstrated that persons with auditory dys-synchrony

show a slightly larger difference limen at low sensation levels than normal hearing individuals, but it approached normal values at high sensation levels similar to that seen in individuals with normal hearing. Zeng, Kong, Michalewski and Starr (2005) also observed the similar results.

2.2.8.2 Frequency Processing:

Frequency discrimination ability of patients with auditory dys-synchrony is significantly affected (Rance, McKay & Grayden, 2004; Starr et al., 1991; Starr et al., 1996; Zeng et al., 2005). These individuals have more problems in discriminating low frequency signals than high frequency signals (Rance, McKay & Grayden, 2004; Zeng et al., 2005). This is because, the discrimination of low frequency signals depends upon phase locking cues which are more affected, whereas discrimination of high frequency depends on spatial changes in the excitation pattern along the basilar membrane which is normal (Sek & Moore, 1995). Also, frequency discrimination abilities were strongly correlated with speech perception scores.

2.2.8.3 Temporal integration:

Temporal integration function in individuals with auditory dys-synchrony was not affected except for very short duration signals. However, the slope of the integration function was slightly elevated in individuals with auditory dys-synchrony than in normal hearing subjects (Zeng et al., 1999; Zeng et al., 2005).

2.2.8.4 Gap detection:

Individuals with auditory dys-synchrony show poor gap detection thresholds at higher sensation levels because of smeared temporal representation of the acoustic

stimulus. However, the detection threshold in these individuals was similar to normal hearing subjects at low sensation levels (Zeng et al., 2005).

2.2.8.5 Temporal Modulation detection Thresholds

Temporal modulation transfer function is a measure of sensitivity to amplitude fluctuation over a range of modulation frequencies. This ability is affected in individuals with auditory dys-synchrony, suggesting that the temporal processing is severely impaired (Rance, McKay & Grayden, 2004; Zeng et al., 1999; Zeng et al., 2005).

In most of the individual's gap detection and TMTF were severely impaired, suggesting that the temporal processing is affected, which is due to the fact that timing and synchronicity in the firing of neurons in the auditory nerve was affected leading to poor auditory perception. Patients with auditory dys-synchrony have difficulty in perceiving timing-related information, but not intensity or frequency related information.

2.2.9 Speech Identification Profile

Speech perception difficulties are a consistently reported consequence of hearing impairment. In post-lingual deafened adults with sensorineural loss, a reasonably strong relationship exists between the behavioural audiogram and open-set speech understanding. In contrast, speech perception ability in adults diagnosed with auditory neuropathy/dys-synchrony are disproportionate to their pure tone hearing loss (Starr, Sininger & Pratt, 2000; Zeng, Oba & Starr, 2001), and in most of the cases, speech identification scores has been significantly poorer than would have been expected for sensorineural losses of equivalent degree.

The speech perception abilities in these individuals is highly variable, with some patients performing at levels expected for cochlear hearing loss of the same degree, while some others show little or no measurable speech identification despite having adequate sound detection abilities. Furthermore, this discrepancy between sound detection and speech identification can be attributed to suprathreshold distortion of temporal cues rather than audibility (Rance, Mckay, & Grayden, 2004; Zeng et al., 1999; Zeng et al., 2005).

Zeng et al. (1999) investigated the temporal and speech processing deficits in 8 auditory neuropathy patients using gap detection, temporal integration and temporal modulation transfer function (TMTF). They found that the temporal integration function in most of the subjects showed normal or near normal function, suggesting that the poor speech recognition is not due to temporal integral function. But in most of the individuals gap detection and TMTF were severely impaired, suggesting that the temporal processing is affected. They concluded that asynchronous firing of the auditory nerve resulted in distorted temporal coding of speech which in turn resulted in poor speech recognition that was disproportionate to the degree of hearing loss.

Starr et al. (1996) investigated speech perception abilities in auditory neuropathy individuals using open set speech tests. 8 of the 10 subjects had word recognition scores ranged from 0% to 92% and were significantly lower in 12 of the 16 ears than predicted from the norms generated by Yellin, Jerger and Fifer (1989) for ears with sensorineural hearing loss. The other two subjects had speech identification scores proportionate to their hearing loss. Similarly, Sininger and Oba (2001) reported that the speech discrimination scores (CID W-22 lists) for 25 of 36 auditory neuropathy/ dys-synchrony

patients fell below the Yellin Jerger and Fifer (1989) normative range and 30% subjects showed scores within normal range for sensorineural of equivalent degree.

In general individuals with hearing impairment have problems in understanding speech in noisy situations. In particular auditory neuropathy/ dys-synchrony individuals have more problems in background noise suggesting that good speech understanding may be possible in ideal listening circumstances, even the least-impaired adult AN/AD subjects may struggle when redundancies in the speech signal are compromised (Kraus et al., 2000)

Shallop (2002) has presented a case of a woman diagnosed with hearing thresholds in the mild-to-moderate range, who had reported to have difficulties in understanding speech in noise throughout childhood. Hearing in Noise Test (HINT) using sentences in this case showed 100% perception in quiet listening conditions but extreme difficulty in noise. Word identification for this subject fell to 25% at a +15 dB signal-to-noise ratio and to 0% at +12 dB.

Zeng and Liu (2006) also studied the perception of 14 subjects and found consistent reductions in speech recognition ability, even at signal-to-noise ratios that show little or no effect on subjects with normal hearing. The mechanisms underlying these perceptual difficulties in noise are unclear.

Rance, Barker, Mok, Dowell, Rincon and Garratt (2007) investigated the effect of background noise on speech perception in a group of children with AN. They used closed, open speech tests (phomemically balanced CNC words) and adaptive spondees in

noise test (4 alternative forced choice spondee discrimination test) to assess speech perception. Results suggested that AN children had more difficulty than their normal hearing peer group. They attributed the poorer scores in clients with AD to deficits in temporal processing, central masking and forward and backward masking having minimal affects. However, the noise effects were not consistent across subjects and some children demonstrated reasonable perceptual ability at low signal to noise ratios (-2.5±4.7 SNR).

2.2.10 Evoked Potentials from the Central Auditory Pathways

Auditory neuropathy/dys-synchrony result profile includes the absence or severe disruption of the auditory brainstem response, it might be expected that more central evoked responses such as the middle latency and cortical auditory evoked potential (CAEP) would be similarly affected. And yet, many of the reported clients have shown clearly identifiable responses with reasonably normal morphology and response latency (Gorga, Stelmachowicz & Barlow 1995; Hood, 1999; Kraus et al., 2000; Rance et al., 2002; Zeng & Liu, 2006).

As the CAEPs are less dependent on synchronous neural firing than auditory brainstem responses these may be recordable in some clients with auditory neuropathy/dys-synchrony. The peaks in the normal auditory brainstem response waveform are biphasic and are usually only separated by approximately 1 ms. Small variations in the timing of responses to individual stimuli can thus lead to cancellation in the averaged signal. The component peaks in the CAEP waveforms, which are much

broader and are separated by 50 to 100 ms in adult subjects (and longer in children), are more resistant to subtle changes in the timing of individual responses (Starr et al., 1996).

Starr et al. (1991) manipulated the synchrony of auditory brainstem responses by systematically varying the timing of each stimulus relative to the start of the averaging window. This found that timing fluctuations of the order of tenths of a millisecond are sufficient to disrupt the averaged response for the cat auditory brainstem response. In contrast, studies considering the timing of responses from the auditory cortex have shown a much greater tolerance to temporal fluctuation.

Michalewski, Prasher and Starr (1986) determined the latency of N1 and P2 of cortical event related potentials in normal adult subjects for individual stimulus trials and showed peak latency standard deviations of about 17 ms and 22 ms respectively. These individual trials, when subjected to conventional signal averaging procedures, produced robust waveforms. However, if the standard deviation of normal temporal fluctuation in these potentials is around 20 ms, then the level of dys-synchrony required to affect the CAEP waveform is likely to be of the order of tens of milliseconds. This level is significantly higher than that is required to disrupt the auditory brainstem response and as such, the cortical event-related potentials can offer a gross measure of the effect of peripheral neural disruption in the signal reaching the auditory cortex.

ALR can be elicited by stimuli like clicks, tone bursts and speech stimuli. This objective measure provides a tool to investigate the neurophysiological processes that underlie individual's ability to perceive speech (Purdy, Katsch, Sharma, Dillon & Ching, 2001; Trembley, Friesen, Martin & Wright, 2003). 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 central auditory pathways (Eggermont & Ponton, 2003).

Tremblay et al. (2003), obtained P1-N1-P2 responses 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.

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.

Kraus et al. (2000) presented a case report where in a 24-year-old woman who had normal hearing thresholds. The electrophysiological data showed robust OAE's in both ears and cochlear microphonics was observed during ABR testing. Wave I was absent and wave III was observed inconsistently, when present wave III and V displayed poor morphology. She obtained a perfect word recognition score on a CUNY-Sentence in quiet, demonstrating that speech perception can be achieved despite measurable neural disruption in the auditory brainstem. However, assessment in noise showed abnormally depressed results. While the speech evoked cortical potential like LLR and MMN showed good wave morphology, latency and amplitude. Hence, it was concluded that optimal auditory nerve and auditory brainstem synchrony do not appear to be essential for understanding speech in quiet listening conditions. However, synchrony is critical for understanding speech in presence of noise.

Cunningham et al. (2001) found similar fundamental sensory representation of speech between normal children and learning problem populations when evoked responses were recorded in quiet, but learning problem children demonstrated neurophysiological abnormalities at both cortical and subcortical levels when the speech was presented in background noise. In their study, noise degraded subcortical encoding of both transient and periodic stimulus features in the auditory brainstem and frequency following responses in learning problem children. Additionally, cortical responses in noise showed a dramatic amplitude decrease in learning problem children with respect to normal children in the P2-N2 complex occurring between 150– 250 ms post-stimulus onset.

Rance et al. (2002) investigated the relationship between the event related potentials and speech perception ability in 18 children with auditory neuropathy. Unaided and aided speech identification scores were assessed using PBK words. Results indicated that 50% (7/15) of the children had no open set speech perception ability and 50% (8/15) showed performance levels equal to their sensorineural counterparts. A subgroup of children (approximately 50%) with AN, who had recordable cortical evoked potential performed well on open set speech perception task and derived significant benefit from amplification. In contrast, subjects who had no recordable cortical evoked potential, performed poorly on the same tasks. Therefore, they concluded that presence of cortical auditory evoked potential reflects some amount of preserved synchrony in central auditory system which contributes to better speech understanding despite the distortion that occurs at 8th nerve and auditory brainstem in these individuals.

Kumar and Jayaram (2005) studied the auditory evoked potentials and psychophysical abilities in 14 adults with auditory neuropathy using open set speech identification scores, just noticeable difference for transition duration of syllable /da/ and temporal modulation transfer function to characterize their perceptual capabilities. Auditory evoked potentials measures were, recorded for P1/N1, P2/N2 complex and mismatch negativity (MMN). Results revealed that there was significant correlation between temporal processing deficits and speech perception abilities. In majority of individuals with auditory neuropathy P1/N1, P2/N2 complex and mismatch negativity could be elicited with normal amplitude and latency and none of the measured evoked potential parameters correlated with the speech perception scores. Many of the subjects with auditory neuropathy showed normal MMN even though they could not discriminate

the stimulus contrast behaviourally. From this study they concluded that individuals with auditory neuropathy have severely affected temporal processing and the presence of MMN may not be directly linked to presence of behavioural discrimination and to speech perception capabilities at least in adults with auditory neuropathy.

Kumar and Vanaja (2008) investigated the relationship between speech identification scores in quiet and parameters of cortical potentials (latency of P1, N1, P2 & amplitude of N1-P2) in ten individuals with auditory neuropathy. Speech identification ability was assessed for bi-syllabic words and cortical potentials were recorded for click stimuli. Results revealed that individuals with auditory neuropathy had speech identification scores significantly poorer than that of individuals with normal hearing, which they attributed to disrupted neural synchrony. Auditory neuropathy individuals were further classified into two groups, good performers and poor performers based on their speech identification scores. It was observed that the mean amplitude of N1-P2 of poor performers was significantly lower than that of good performers and those with normal hearing. They attributed the reduction in amplitude to the severity of pathology. Speech identification scores showed a good correlation with the amplitude of cortical potentials (N1-P2 complex) but did not show a significant correlation with the latency of cortical potentials. Therefore, measuring the amplitude of the cortical potentials may offer a means for predicting perceptual skills in individuals with auditory neuropathy.

From the review of the literature, it is evident that the click evoked auditory late responses have applications in the electrophysiological assessment of the representation

of speech cues at the cortical level in the auditory nervous system. It's also evident that the amplitude of the click evoked auditory late potentials in auditory neuropathy correlated reasonably well with their speech perception ability.

It can be concluded that Rance et al. (2002); Kumar and Vanaja (2008) showed that in individuals with auditory dys-synchrony the presence of ALLR and amplitude of ALLR correlates with speech identification scores, suggesting that the brainstem synchrony may not be essential for speech in quiet situations. However, these individuals have difficulty in understanding speech in the presence of noise, suggesting that the synchrony is critical for understanding in the presence of noise, which has not been evaluated. The degree of dys-synchrony varies from individual to individual and with reference to frequency as well. So, evaluating patients with auditory neuropathy using different frequency stimuli helps in understanding them better and in rehabilitation as well. Although, Rance et al. (2007) reported that the perception of speech in the presence of noise in individuals with AN was assessed, the results were inconsistent. Electrophysiological assessment of speech perception in noise might help us in understanding the processing of speech at higher levels in adverse listening conditions.

It is also evident from the literature that there are no studies done in normal hearing individuals to find the correlation between speech identification scores and ALLR parameters. Since, individuals with auditory neuropathy had more problems in understanding speech in the presence of noise, assessing speech identification scores and ALLR in the presence of noise for normal hearing individuals, may help us in understanding the auditory neuropathy better. Hence, it is necessary to conduct a study

on normal hearing individuals and individuals with auditory dys-synchrony to find correlation between speech identification scores and ALLR with and without noise.

To date, only few studies have investigated the ALLRs using speech stimuli in auditory neuropathy individuals to predict speech identification abilities. However, these studies had a small number of subjects and reported conflicting results. Cortical auditory evoked potentials elicited using speech stimuli were not compared with the speech perception abilities to find which one correlates best, whether click or speech evoked cortical potentials. No studies have been done to correlate ALLR in noise and speech identification scores in noise. So research is required in optimizing whether click evoked or speech evoked cortical potentials correlates better with speech identification scores in noise in individuals with AD and normal hearing individuals.

3. METHOD

The main objective of the study was to find the correlation between speech identification scores and auditory long latency response with and without noise in individuals with auditory neuropathy and normal hearing. An attempt was also made to know how an ALLR differs for spectrally different speech sounds. Two groups of subjects were taken and the following method was adopted for the study.

3.1 Subjects

The subjects in the present study whose age was 12 years and above were considered and divided into two groups.

- Individuals with normal hearing (control group)
- Individuals with auditory neuropathy (clinical group)

3.1.1 *Control group*

A total of 15 ears from 15 subjects with normal hearing in the age range of 15 to 38 years were evaluated. The criteria considered for the selection of the subject was as follows:

Subject selection criteria:

- Pure tone threshold were within 15 dB HL at octave frequencies between 250 to 8000 Hz for air conduction and between 250 to 4000 Hz for bone conduction.
- All the subjects had ‘A’ type tympanogram with normal acoustic reflex thresholds.
- Speech identification scores were greater than 90%.

- Speech identification scores in the presence of noise at 0 dB SNR were assessed and all of them had scores above 60%.
- Good ABR waveform morphology was present for all the individuals at 80 dB nHL for both 11.1 or 90.1/sec repetition rate.
- TEOAE's were present in all the subjects for both the ears.
- No history of any otological or neurological problems was reported.

3.1.2 Clinical Group

For the clinical group, 25 ears from 16 subjects with auditory neuropathy in the age range of 13 to 40 years were evaluated. The following criteria were considered for the selection of subject:

- All the subjects had pure tone audiometry thresholds ranging from normal to moderate sensorineural hearing loss.
- Subjects had speech identification scores ranging from 0-100%.
- Speech identification scores in noise at 0 dB SNR were poor.
- All the ears tested had "A" type tympanograms with absent acoustic reflexes.
- TEOAE's or cochlear microphonics was present in all the ears tested.
- ABR was absent at 80 dB nHL for all the subjects even at 11.1/sec repetition rate.
- No history of any otological or neurological problems was reported.

3.2 Instrumentation

- A Calibrated double channel diagnostic audiometer (GSI-61) with TDH-39P ear phone and B-71 bone vibrator was used for pure tone audiometry and to assess speech identification scores with and without noise.
- A Calibrated immittance meter (GSI-Tympstar) was used to assess middle ear status.
- ILO V6 DP-echo port system was used to record Transient evoked oto-acoustic emissions (TEOAE).
- Intelligent Hearing Systems (IHS smart EP windows USB version 3.22) evoked potential system was used to record and analyze the ABR and ALLR. ER-3A insert phone was used to deliver the click and speech stimulus.

3.3 Stimulus generation

Syllables /ba/ /ga/ and /da/ were used to record ALLR. 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 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/, 146 ms for /ga/ and 150 ms for /da/.

3.4 Test environment

All the tests were carried out in a well illuminated and acoustically treated air conditioned rooms. The noise level was within permissible levels as recommended by ANSI (1991-S3.1).

3.5 Test procedure for subject selection

Several audiological tests were carried out prior to the selection of the subject for the experiment. The procedure adapted for each test is given below.

3.5.1 *Pure tone audiometry*

Air conduction and bone conduction thresholds for all the subjects were 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.

3.5.2 *Speech audiometry:*

Speech identification scores were assessed with and without noise using speech material developed by Vandana (1998). SPIN (speech perception in noise) scores were assessed at 0 dB SNR by using SPIN CD developed by Vargesh (2004). SIS and SPIN scores were established at 40 dB above the SRT (speech recognition threshold) level.

3.5.3 *Immittance*

The tympanometric measurements were assessed 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 middle ear pathology.

3.5.4 *Transient evoked oto-acoustic emissions (TEOAE)*

The transient evoked oto-acoustic emissions were recorded using nonlinear clicks presented at 85 dBpSPL. The responses of 260 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 a reproducibility of greater than or equal to 50% as described by Glattke, Pafitis, Cummiskey and Herrer, (1995).

3.5.5 *ABR recording*

Subjects were instructed to sit comfortably on a reclining chair and relax during the testing. They were instructed to close their eyes during the testing to avoid any artifacts.

3.5.5.1 *Preparation of the subjects and electrode placement*

Electrode sites were cleaned using NU prep cleaning gel to remove the dead cells and dirt. 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 k Ω and inter electrode impedance was within <2 k Ω. Impedance for each electrode was also checked during testing, to make sure that patient movement did not cause any variation in the impedance. The protocol used to acquire ABR is as follows:

Table 1: *Parameters used to record ABR*

Stimulus parameters		**Acquisition parameters**	
Transducer	Insert ear phones ER-3A	Amplification	100,000
Type of stimulus	Clicks	Analysis window	0 to15 ms
Intensity	80 dB nHL	Filters	100– 3000 Hz
Presentation ear	Monaural	Notch filter	On
Stimulus polarity	Rarefaction	Artifact rejection	40 µV
		Electrode montage:	
		Non-inverting	Vertex (Cz)
No of sweeps	1500	Inverting	Test ear mastoid (A1/A2)
Repetition rate	11.1/s, 90.1/s	Ground	Non test ear mastoid (A2/A1)

ABR was recorded to rule out space occupying lesions in individuals with normal hearing. A good wave morphology even at 90.1/sec and normal inter wave latencies elicited at 11.1/sec repetition rate was considered as having no RCP (retro cochlear pathology). Individuals with absent ABR and presence of OAE or cochlear microphonics were considered as having AD. Those who have fulfilled the criteria have undergone ALLR testing.

3.5.6 *Auditory Long Latency Responses (ALLRs) to reach out to objectives*

Subjects were instructed to sit comfortably on a reclining chair and relax during the testing and to stay awake during the testing. They were also instructed to ignore the stimulus and restrict the movement of head, neck and eye during testing. Preparation of

the subjects and electrode montage used to record ALLR was the same as used for ABR recording. The parameters used to record ALLR are given in Table 2.

Table 2: *Parameters used to record ALLR*

Stimulus parameters		**Acquisition parameters**	
Transducer	Insert ear phones ER-3A	Amplification	50,000
Type of stimulus	Clicks and speech stimuli /ba/, /ga/, and /da/.	Analysis window	-100 to 500 ms
Duration of the stimulus	Click- 100μsec /ba/- 147 ms, /ga/- 146 ms and /da/- 150 ms	Filters	1– 30 Hz
Intensity	80 dB SPL	Notch filter	None
Presentation ear	Monaural	Artifact rejection	100 μV
Stimulus polarity	Alternating	Electrode montage:	
No of sweeps	300	Non-inverting	Vertex (Cz)
Repetition rate	1.1/s	Inverting	Test ear mastoid (A1/A2)
Ipsilateral masking	Without noise With noise at 80 dB SPL (0 dB SNR)	Ground	Non test ear mastoid (A2/A1)

The recording was done twice at each presentation level to check for the reliability. The waveforms elicited in this manner were shown to three experienced

audiologists and they were asked to identify N1, P2 waves. They were not told about the condition and the stimulus for which the responses were obtained. The latencies and amplitudes identified in this way were compared across the judges and the waveforms in which the latencies and amplitude markings were similar by at least two judges were noted.

3.6 Analysis

The latency of N1, P2 and peak to peak amplitude of N1-P2 of ALLR were noted for different stimulus at different conditions. The Mean, standard deviation (SD) and range for N1 and P2 latencies and also N1-P2 amplitude were calculated for both the groups.

- A comparison between speech and click evoked ALLR parameters were made between the groups using Mann Whitney U test.
- To find the effect of speech stimuli and the effect of noise on the latency and amplitude of ALLR, two way repeated measure ANOVA was administered for control group and Wilcoxon's signed rank test was done in the clinical group.
- The parameters of ALLR elicited with and without noise were compared using paired t-test in the control group and Wilcoxon's signed rank test in the clinical group.
- Relationship between the speech identification scores and ALLR parameters was made, using Spearman's rank correlation coefficient for both the groups.

4. Results

To arrive at the objective of the study, the latencies of N1, P2 and peak to peak amplitude of N1-P2 complex were measured. The mean and standard deviation (SD) were calculated for the latencies of N1, P2 wave and the amplitude of N1-P2 for both the groups for all the stimuli (click and speech /ba/, /da/ & /ga/) with and without noise.

Comparison of latency and amplitude of the auditory long latency responses to speech and click, between the groups and within the groups were carried out. The following statistical analyses were administered separately for latencies of N1, P2 and peak to peak amplitude of N1-P2 complex.

- To compare the latencies of N1, P2 and peak to peak amplitude of the clinical and control group Mann Whitney U test was administered. The data available for all these parameters in the clinical group was less; hence ANOVA measures could not be carried out.
- To find the effect of speech stimuli and the effect of noise on the latency and amplitude of ALLR, two way repeated measure ANOVA was administered for control group and Wilcoxon's signed rank test was done in the clinical group. Two way repeated measure ANOVA was not done for the clinical group because of less number of data could be obtained for the various ALLR parameters.
- The latencies of N1, P2 and N1-P2 complex elicited in the presence of noise were compared with latencies of N1, P2 and N1-P2 complex elicited without noise using paired t-test for control group and Wilcoxon's sign rank test for the clinical group.
- Latencies, amplitude of ALLR waves were correlated with speech identification scores using Spearman's rank correlation coefficient for both the groups.

The result obtained from different statistical analysis is given below for both the control and clinical group:

4.1 N1 Latency

The N1 latency obtained for different stimuli from both the groups were analyzed and the results obtained are given below.

4.1.1 Control group

The mean, standard deviation and range for N1 latency obtained in both the conditions (with and without noise) for click and different speech stimulus in normal hearing individuals was calculated. The results are outlined in the Table 3.

Table-3: *Mean, SD, range for N1 latencies and t-values with significance level between with and without noise for different eliciting stimulus obtained in the control group.*

Parameter	Stimulus	Control group				
		Mean (N= 15)	Standard deviation	Range	t-value (df = 14)	Significance level
N1 latency (msec)	Click without noise	118.00	9.01	101-135	1.774	0.098
	Click with noise	121.46	6.86	109-134		
	/ba/ without noise	164.33	9.86	148-187	**3.288****	**0.005**
	/ba/ with noise	171.13	14.12	148-206		
	/ga/ without noise	162.93	10.88	142-176	1.845	0.086
	/ga/ with noise	170.46	14.42	156-208		
	/da/ without noise	158.73	9.67	148-173	**3.419****	**0.004**
	/da/ with noise	172.26	13.11	158-198		

***p< 0.01**

From the Table 3, it can be inferred that the mean latencies of N1 for the speech stimuli were greater than click evoked N1 latency. The latencies of N1 were shorter in without noise condition than in the presence of noise for all the eliciting stimuli. It can also be noted that the variation in N1 latency in the presence of noise was slightly greater than latencies obtained without noise for all the eliciting stimuli.

Two way repeated measure ANOVA (stimuli 4 × condition 2) was done for N1 latency to see the interaction between the variables. The results indicated a significant interaction between the stimuli [($F(3, 42) = 139.566$, $p < 0.05$], and conditions (in the presence of noise and without noise) [$F(1, 14) = 14.147$, $p < 0.01$]. There was no significant interaction between conditions and the stimuli [$F(3, 42) = 2.284$, $p > 0.05$].

As the ANOVA results indicated significant interaction between the stimuli, Bonferroni's test was done to see the significant difference between the N1 latency evoked by any two stimuli. The results revealed a statistically significant difference in latency between click and other speech stimuli. However, no such differences were noted between any two speech stimuli (Table 4).

Table 4: *Results of Bonferroni test for N1 latency in the control group.*

Stimulus	/ba/	/ga/	/da/
Click	Significant (p< 0.001)	Significant (p< 0.001)	Significant (p< 0.001)
/ba/		Not significant (p> 0.05)	Not significant p> 0.05)
/ga/			Not significant (p> 0.05)

There was no significant interaction for N1 latency between the conditions and eliciting stimuli, but significant interaction between conditions was observed in ANOVA results. Paired t-test was done to see significant difference for each stimulus between N1 latency evoked in two stimulus conditions. It was found that there was no significant difference between two conditions for click and /ga/ stimuli, but there was a significant difference for N1 latency elicited by /ba/ and /da/, which can be seen in the Table 3.

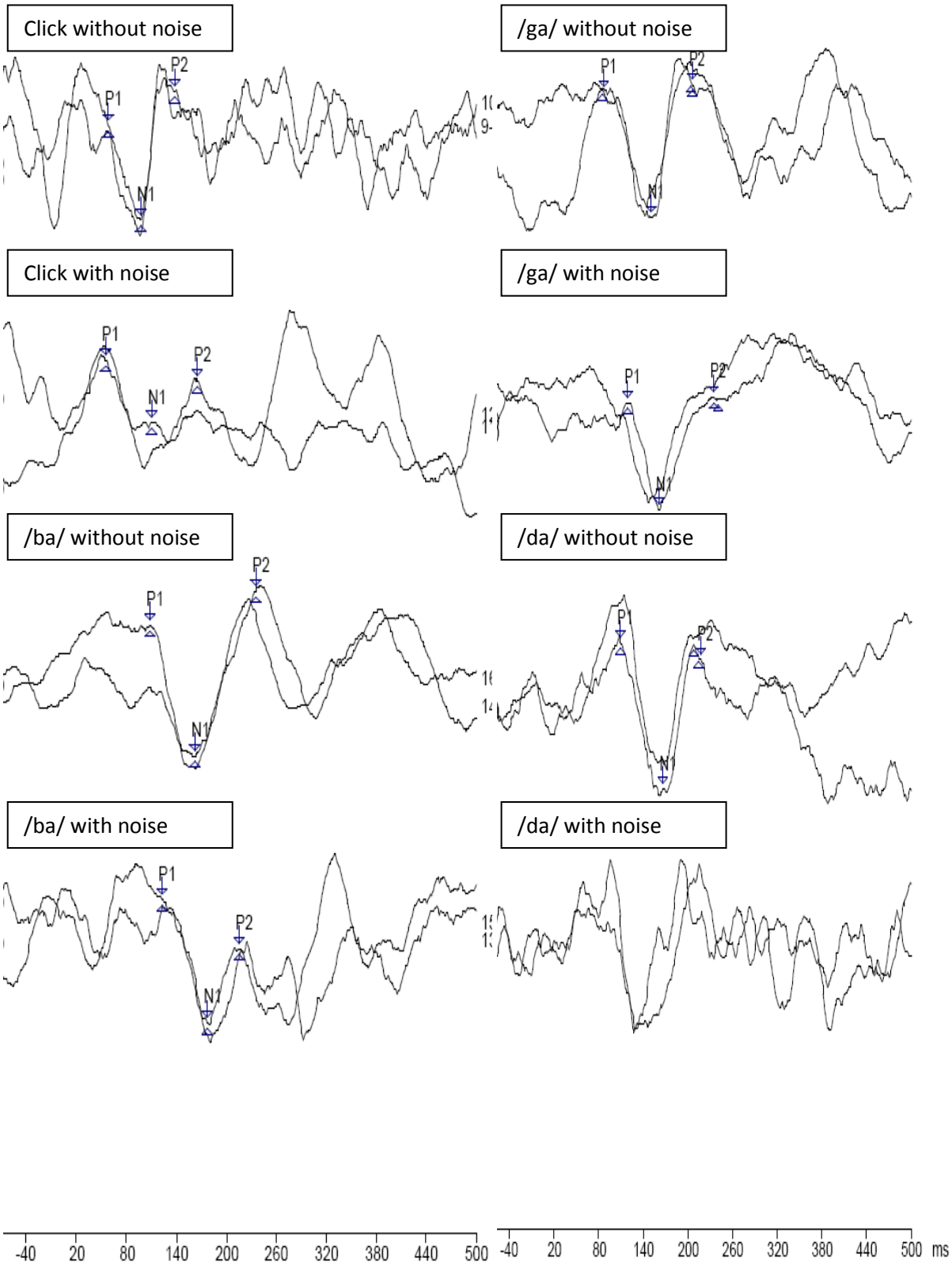

Figure 1: ALLR elicited in normal hearing individual for click and different speech stimulus in both the conditions.

4.1.1.1 Correlation between the speech identification scores and N1 latency in the control group

Speech identification scores elicited with and without noise were correlated with

- N1 latency with noise
- N1 latency without noise.

Table 5: *Correlation coefficient value along with significance level for N1 latency and SIS obtained without noise and at 0 dB SNR in the control group.*

Parameter	Stimulus and condition	SIS without noise		SIS with noise	
		Correlation Coefficient (N= 15)	Sig. (2-tailed)	Correlation Coefficient (N= 15)	Sig. (2-tailed)
N1 latency (msec)	Click without noise	-.007	.980	-.181	.519
	Click with noise	.141	.616	.247	.375
	/ba/ without noise	.078	.783	-.246	.377
	/ba/ with noise	.085	.764	**-.518***	**.048**
	/ga/ without noise	.367	.178	-.099	.726
	/ga/ with noise	.042	.881	-.205	.464
	/da/ without noise	.035	.901	.018	.948
	/da/ with noise	.113	.689	-.360	.187

***p< 0.05**

The Spearman's rank correlation coefficient was obtained to see any significant correlation between N1 latency and SIS. It is obvious from the Table-5, that N1 latency elicited by /ba/ at 0 dB SNR had significantly negative correlation with SIS obtained at 0

dB SNR, i.e., speech identification scores reduces with increase in latency. No significant correlation between N1 latency and SIS was obtained in the other conditions.

4.1.2 Clinical group (individuals with auditory dys-synchrony)

The mean, standard deviation and range were computed for N1 latency for all the four stimuli with and without noise. A look into the mean values, as depicted in the Table 6, indicates that the latencies of N1 for speech stimuli were greater than click stimulus. The latencies of N1 elicited for all the stimuli were shorter in without noise condition than at 0 dB SNR. However, N1 latency elicited by /ga/ stimulus in the presence of noise showed a different pattern which could be due to smaller sample size and the ALLR could not be elicited for click evoked N1 at 0 dB SNR in the clinical group.

Table 6: *Mean, SD and range for N1 latency elicited by click, /ba/, /da/ and /ga/ syllables with and without noise in the clinical group.*

Parameter	Stimuli	Clinical group		
		Mean	Standard deviation	Range
N1 latency (msec)	Click without noise	134.71 (N= 7)	19.81	100-149
	Click with noise	-	-	-
	/ba/ without noise	156.50 (N= 14)	15.38	142-190
	/ba/ with noise	208.66 (N= 3)	62.93	161-280
	/ga/ without noise	165.41 (N= 12)	13.30	140183
	/ga/ with noise	165.50 (N= 2)	7.77	160-171
	/da/ without noise	164.38 (N= 18)	18.92	141-216
	/da/ with noise	193.62 (N= 8)	44.00	148-284

It was further followed by the Wilcoxon signed rank test to check for any significant difference between and within the stimuli in both with and without noise conditions. The N1 latency evoked by /da/ stimulus was found to be statistically significant between the conditions (with and without noise) but not for /ba/ and /ga/ stimuli. Pair-wise analysis between the stimuli (click, /ba/, /da/ and /ga/) revealed no significant difference, which is evident from the Table 7. Click and /ga/ elicited in the presence of noise were not included in the comparison due to insufficient data that could be obtained in the clinical group.

Table 7: *Z- values with significant level for N1 elicited by click and speech stimuli at 0 dB SNR and without noise in the clinical group.*

Pair compared	Z - value
/ba/ with noise - /ba/ without noise	1.604
/da/ with noise - /da/ without noise	**2.380***
/ba/ without noise – click without noise	1.782
/da/ without noise – click without noise	1.782
/ga/ without noise – click without noise	1.753
/da/ without noise – /ba/ without noise	1.924
/ga/ without noise – /ba/ without noise	0.931
/ga/ without noise – /da/ without noise	0.311
/da/ with noise - /ba/ with noise	0.000

***p< 0.05**

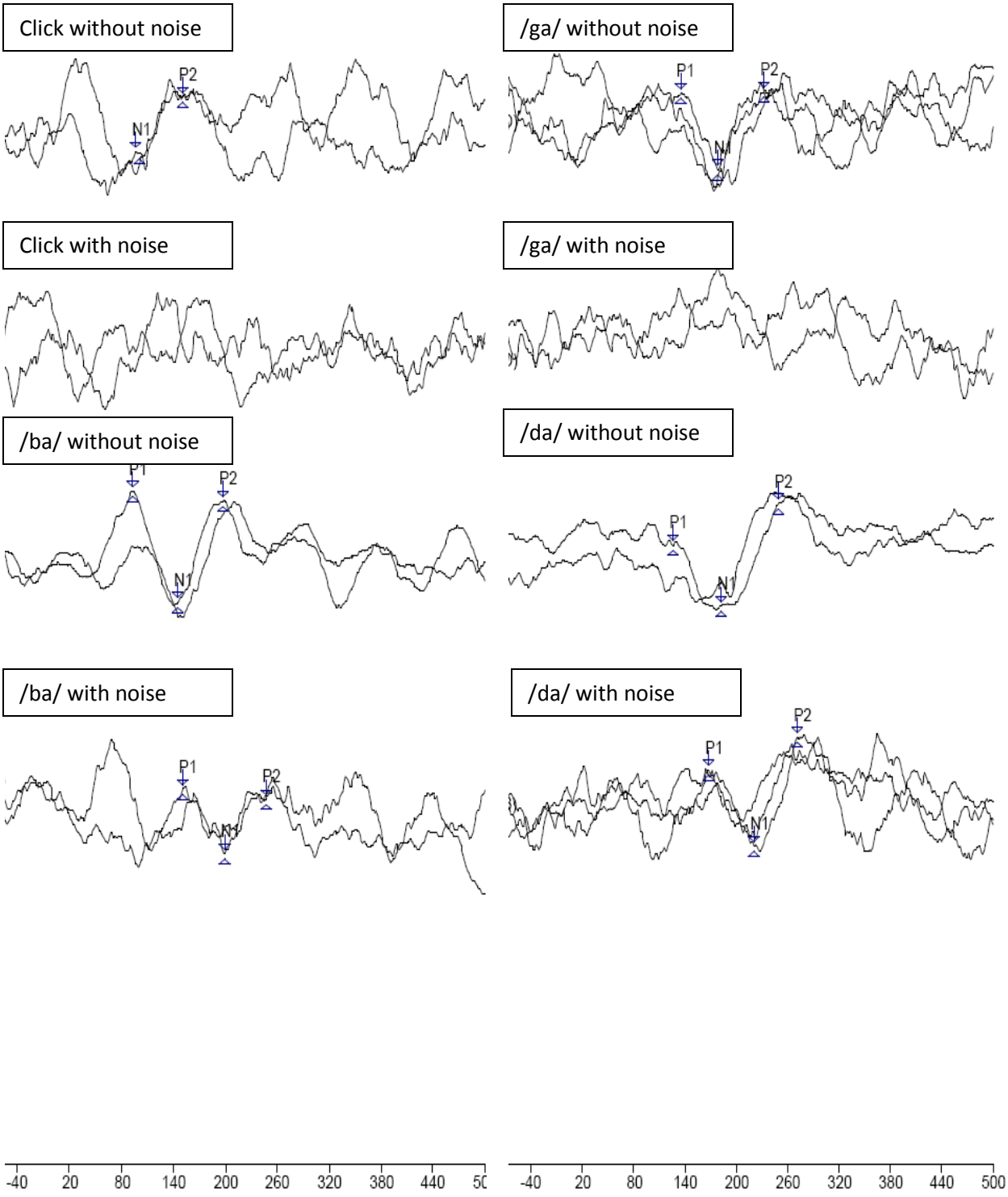

Figure 2: ALLR elicited in an individual with auditory neuropathy for click and speech stimulus in both the conditions.

4.1.2.1 Correlation between speech identification scores and N1 latency in the clinical group

Speech identification scores obtained with and without noise were correlated with

- N1 latency with noise
- N1 latency without noise.

Table 8: *Correlation coefficient value along with significance level for N1 latency and SIS obtained without noise and at 0 dB SNR in the clinical group.*

Parameter	Stimulus and condition	SIS without noise		SIS with noise	
		Correlation Coefficient	Sig. (2-tailed)	Correlation Coefficient	Sig. (2-tailed)
N1 latency (msec)	Click without noise	.150 (N= 7)	.749	.462	.434
	Click with noise	-	-	-	-
	/ba/ without noise	-.268 (N= 13)	.376	-.390	.339
	/ba/ with noise	.866 (N= 3)	.333	-.500	.667
	/ga/ without noise	-.253 (N= 12)	.428	-.073	.863
	/ga/ with noise	-	-	-	-
	/da/ without noise	-.466 (N= 17)	.059	-.586	.127
	/da/ with noise	-.135 (N= 8)	.750	.000	1.000

Spearman's rank correlation coefficient was carried out for the same. The values of which are outlined in Table 8. Form the table it can be noted that there is no significant correlation obtained for speech identification scores (SIS) and N1 latency in both the conditions. None of the subjects in the clinical group exhibited N1 latency evoked by

click and only two individuals had for /ga/ in the presence of noise; hence correlation could not be done.

4.1.3 Comparison between control and clinical group

The mean value obtained for click evoked N1 latency in quiet condition was shorter for normal hearing individuals than the clinical group as evident from the Table 9. No ALLR could be recorded using click at 0 dB SNR in the clinical group. Though there was difference between N1 latency obtained for different speech stimuli in both the conditions between the groups, no specific pattern could be observed. N1 latency shift occurred in the presence of noise in clinical group was more than that in normals for /ba/ and /da/ stimulus. For /ga/ stimulus the data obtained in the presence of noise was less due to which large shift in latency with noise was not seen.

Table 9: *Mean, SD for N1 latency and Z-values with significance level obtained for click and different speech stimulus in two conditions between both the groups*

Parameter	Control group		Clinical group		
N1 latency (msec)	Mean (N= 15)	Standard deviation	Mean	Standard deviation	/Z/
Click without noise	118.00	9.01	134.71 (N= 7)	19.81	1.872
Click with noise	121.46	6.86	-	-	-
/ba/ without noise	164.33	9.86	156.50 (N= 14)	15.38	**1.966***
/ba/ with noise	171.13	14.12	208.66 (N= 3)	62.93	1.245
/ga/ without noise	162.93	10.88	165.41 (N= 12)	13.30	0.782
/ga/ with noise	170.46	14.42	165.50 (N= 2)	7.77	-
/da/ without noise	158.73	9.67	164.38 (N= 18)	18.92	0.272
/da/ with noise	172.26	13.11	193.62 (N = 8)	44.00	1.164

***p< 0.05**

For comparison of N1 latency obtained between the groups for each stimulus, Mann Whitney U test was carried out. A statistically significant difference was obtained for N1 latency elicited in quiet only for /ba/ stimulus and not for the other three stimuli.

Both the groups did not show any significant correlation between speech identification scores obtained and N1 latency evoked by all the stimulus in both with and without noise conditions. Both the clinical and control group showed a significant reduction in speech identification score in the presence of noise when compared with SIS obtained without noise. However, SIS was poor in clinical group than in control group in both the conditions.

4.2 P2 latency

The P2 latency obtained for different stimuli from both the groups were analyzed. The results obtained are given below.

4.2.1 Control Group

The data obtained was analyzed to obtain the mean, standard deviation and range of P2 latency elicited by the four stimuli. The values are outlined in the Table 10. It can be noted from the table that the mean P2 latencies elicited for speech stimuli (/ba/, /da/ and /ga/) were greater than P2 latency evoked by click. The latencies were shorter in the absence of noise than in the presence of noise for all the stimuli.

Table 10: *Mean, SD, range of P2 latency and t-values with significant level for P2 latency elicited by different stimulus with and without noise in the control group*

Parameter	Stimulus	**Control group**				
		Mean (N= 15)	Standard deviation	range	t-value	Significance level
P2 latency (msec)	Click without noise	181.71	7.84	165-192	**2.981***	**0.011**
	Click with noise	186.78	8.65	165-198		
	/ba/ without noise	215.14	12.66	197-241	**7.483****	**0.000**
	/ba/ with noise	225.00	14.81	198-257		
	/ga/ without noise	217.78	4.47	208-224	**3.934****	**0.001**
	/ga/ with noise	226.64	9.21	206-238		
	/da/ without noise	211.35	11.41	196-229	**3.831****	**0.002**
	/da/ with noise	225.14	12.40	205-248		

***p< 0.05 and **p< 0.01**

To find the significant interaction between variables for P2 latency two way repeated measure ANOVA (stimuli 4 × condition 2) was done. The results showed that there was a significant interaction between the stimuli [$F (3, 39) = 61.710$, $p < 0.001$] and conditions [$F (1, 13) = 68.851$, $p < 0.001$]. But, there was no significant interaction between conditions and the stimuli [$F (3, 39) = 1.910$, $p > 0.05$]. Since ANOVA showed significant interaction between the stimuli, Bonferroni's test was done to check for significant difference between any two stimuli. The result is given in the Table 11.

Table 11: *Bonferroni test results for P2 latency in the control group.*

Stimulus	/ba/	/ga/	/da/
Click	Significant (p< 0.001)	Significant (p< 0.001)	Significant (p< 0.001)
/ba/		Not significant (p> 0.05)	Not significant (p> 0.05)
/ga/			Not significant (p> 0.05)

Paired t-test was done to see the significant difference between the two conditions (with and without noise) for each stimulus. It was found that there was a significant difference between the two conditions in P2 latency evoked by all the stimulus, which can be seen in the Table 10.

4.2.1.1 Correlation between speech identification scores and P2 latency in the control group:

Speech identification scores obtained with and without noise were correlated using the Spearman's rank correlation coefficient with

- P2 latency with noise
- P2 latency without noise.

A significant negative correlation was found between speech identification scores at 0 dB SNR and P2 latency elicited by click without noise and with noise i.e., as the latency increases speech identifications elicited was less. None of the other conditions showed any significant correlation which can be seen in the Table 12.

Table 12: *Correlation coefficient value along with significance level between P2 latency and SIS obtained without noise and at 0 dB SNR in the control group.*

Parameter	Stimulus and condition	SIS without noise		SIS with noise	
		Correlation Coefficient (N= 15)	Sig. (2-tailed)	Correlation Coefficient (N= 15)	Sig. (2-tailed)
P2 latency (msec)	Click without noise	-.221	.430	**-.520***	**.047**
	Click with noise	-.127	.666	**-.537***	**.048**
	/ba/ without noise	.169	.547	-.057	.840
	/ba/ with noise	.000	1.000	-.254	.361
	/ga/ without noise	.050	.860	-.414	.125
	/ga/ with noise	-.473	.075	.082	.771
	/da/ without noise	-.417	.122	-.127	.653
	/da/ with noise	.106	.707	-.262	.346

***p< 0.05**

4.2.2 Clinical group (individuals with auditory dys-synchrony)

The mean, standard deviation and range of P2 latency elicited for different stimuli were calculated and given in the Table 13. It is evident in the table that the mean P2 latency evoked by the three speech stimuli was longer than that evoked by the click stimuli without noise. P2 latency evoked in the presence of noise was longer than without noise for all the stimulus. ALLR could not be obtained for the subjects in the clinical group for click stimuli with noise.

Table 13: *Mean, SD and range for P2 latency elicited by different stimulus with and without noise in the clinical group*

Parameter	Stimulus	Clinical group		
		Mean	Standard deviation	range
P2 latency (msec)	Click without noise	184.85 (N= 7)	14.36	162-200
	Click with noise	-	-	-
	/ba/ without noise	219.42 (N= 14)	21.41	192-265
	/ba/ with noise	248.00 (N= 3)	31.04	218-280
	/ga/ without noise	228.16 (N= 12)	14.79	201-253
	/ga/ with noise	234.00 (N= 2)	9.89	227-241
	/da/ without noise	227.70 (N= 17)	17.02	203-265
	/da/ with noise	253.00 (N= 8)	26.81	223-284

To check whether there was any statistically significant difference in P2 latency within the stimuli in both the conditions and across the stimuli and conditions, Wilcoxon signed ranks test was done. Within the stimuli a significant difference was obtained only for /da/ and not for /ba/ and /ga/. The pair-wise analysis also revealed a statistically significant difference for P2 latency across click and speech stimuli (/ba/, /da/ & /ga/); and /da/ and /ba/ in the quiet condition. However, no difference was obtained across the stimuli in the presence of noise condition. The values are depicted in the Table 14. Click and /ga/ elicited in the presence of noise were not included in the comparison for P2 due to insufficient data that was recorded in the clinical group.

Table 14: *Z-values with significance level for P2 latency elicited by click and speech stimuli at 0 dB SNR and without noise in the clinical group*

Pair compared	Z - value
/ba/ with noise - /ba/ without noise	1.604
/da/ with noise - /da/ without noise	**2.521***
/ba/ without noise – click without noise	**2.201***
/da/ without noise – click without noise	**2.201***
/ga/ without noise – click without noise	**2.023***
/da/ without noise – /ba/ without noise	**2.002***
/ga/ without noise – /ba/ without noise	0.911
/ga/ without noise – /da/ without noise	1.126
/da/ with noise - /ba/ with noise	0.000

***p< 0.05**

4.2.2.1 Correlation between speech identification scores and P2 latency in the clinical group

Speech identification scores obtained with and without noise were correlated with

- P2 latency with noise
- P2 latency without noise.

The Spearman's rank correlation coefficient was done between the SIS and P2 latency. The results indicated a significant negative correlation between P2 latency evoked by da/ stimuli and speech identification scores in the quiet condition. However, a

significant correlation was not observed between SIS and P2 latency in any other combination. The details of the results can be seen in the Table 15.

Table 15: *Correlation coefficient value along with significance level for P2 latency and SIS obtained without noise and at 0 dB SNR in the clinical group.*

Parameter	Stimulus and condition	SIS without noise		SIS with noise	
		Correlation Coefficient	Sig. (2-tailed)	Correlation Coefficient	Sig. (2-tailed)
P2 latency (msec)	Click without noise	-.519 (N= 7)	.233	.500	.391
	Click with noise	-	-	-	-
	/ba/ without noise	-.216 (N= 13)	.478	-.344	.405
	/ba/ with noise	.866 (N= 3)	.333	-.500	.667
	/ga/ without noise	-.416 (N= 12)	.178	.122	.774
	/ga/ with noise	-	-	-	-
	/da/ without noise	**-.669*** (N= 17)	**.005**	-.467	.290
	/da/ with noise	-.293 (N= 8)	.482	.000	1.000

***p< 0.05**

4.2.3 Comparison between normal hearing individuals and individuals with auditory dys-synchrony

It can be inferred from the Table 16 that the mean P2 latencies elicited by different stimuli in different conditions were longer in clinical group compared to that of the controls. Though there was difference in P2 latency evoked for different speech

stimuli in both with and without noise conditions between the clinical and control groups, no specific trend was observed. P2 latency shift in the presence of noise in clinical group was more than that in normals for /ba/ and /da/ stimulus. The Mann Whitney U test was carried out for a comparison of P2 latency evoked by each of the 4 stimuli and between the groups. There was a significant difference obtained for P2 latency evoked by /da/ and /ga/ in quiet and for /da/ evoked P2 latency at 0 dB SNR. None of the other stimulus condition was significantly different.

Table 16: *Mean, SD and Z-values with significance level for P2 latency for click and different speech stimulus for control and clinical group in both the conditions*

Parameter	Control group		Clinical group		
P2 latency (msec)	Mean (N= 15)	Standard deviation	Mean	Standard deviation	Z - value
Click without noise	181.71	7.84	184.85 (N= 7)	14.36	1.097
Click with noise	186.78	8.65	-	-	-
/ba/ without noise	215.14	12.66	219.42 (N= 14)	21.41	0.000
/ba/ with noise	225.00	14.81	248.00 (N= 3)	31.04	1.365
/ga/ without noise	217.78	4.47	228.16 (N= 12)	14.79	**1.957***
/ga/ with noise	226.64	9.21	234.00 (N= 2)	9.89	-
/da/ without noise	211.35	11.41	227.70 (N= 17)	17.02	**2.554***
/da/ with noise	225.14	12.40	253.00 (N= 8)	26.81	**2.133***

***p< 0.05**

There was no significant correlation between speech identification scores and P2 latency for both the groups. SIS obtained at 0 dB SNR showed a significant reduction when compared with SIS obtained without noise in both the clinical and control group. However, the clinical group showed poor SIS than the control group.

4.3 N1-P2 amplitude

The N1-P2 amplitude elicited for different stimuli were analyzed for control and clinical. The results are given below.

4.3.1 Control group

The data of N1-P2 amplitude evoked by click and the three speech stimuli in both with and without noise condition was compiled and descriptive statistical analysis was carried out which yielded the mean values, standard deviation and range. The values are presented in the Table-17. It can be noticed that the mean N1-P2 amplitude values elicited by click were lesser than speech evoked N1-P2 amplitude in both the conditions. The amplitude of N1-P2 elicited in the presence of noise was considerably smaller than without noise for all the four stimuli.

To check for the interaction between the variables in N1-P2, two way repeated measure ANOVA (stimulus 4 × condition 2) was carried out. The results indicated that there was no significant interaction between stimuli [$F (3, 39) = 1.834$, $p > 0.05$] and between conditions and stimuli [$F (3, 39) = 0.297$, $p > 0.05$]. But there was a significant interaction between the conditions [$F (1, 13) = 53.346$, $p < 0.001$]. As there was no significant interaction between the condition and stimuli for N1-P2, but significant interaction between conditions, paired t- test was carried out, to check for the statistically

significant difference between the two conditions for each stimulus. For all the stimuli used to evoke N1-P2 there was significant difference in amplitudes between those two conditions which is seen in the Table 17.

Table 17: *Mean, SD, range for N1-P2 amplitude and t-value along with significance level elicited by click, /ba/, /da/ and /ga/ syllables with and without noise in the control group*

Parameter	Stimulus	**Control group**				
		Mean (N= 15)	Standard deviation	range	t-value (df= 14)	Significance level
N1-P2 Amplitude (μv)	Click without noise	3.04	0.66	1.97-4.57	**6.848****	**0.000**
	Click with noise	1.97	0.44	1.17-2.78		
	/ba/ without noise	3.50	1.42	2.24-8.09	**4.263****	**0.001**
	/ba/ with noise	2.32	0.84	1.08-3.98		
	/ga/ without noise	3.58	0.97	2.34-5.23	**5.407****	**0.000**
	/ga/ with noise	2.24	0.81	0.98-3.70		
	/da/ without noise	3.56	1.08	2.42-6.35	**5.881****	**0.000**
	/da/ with noise	2.41	1.18	1.24-5.92		

** **p< 0.01**

4.3.1.1 Correlation between speech identification scores and N1-P2 in control group

Speech identification scores obtained with and without noise were correlated with

- N1-P2 amplitude with noise
- N1-P2 amplitude without noise

Spearman's rank correlation coefficient was done to find the relationship between SIS and N1-P2 amplitude. It was observed that N1-P2 amplitude of click without noise was significantly correlated with speech identification elicited at 0 dB SNR and also between N1-P2 amplitude of /ba/ without noise and speech identification scores with noise. No other combination showed any significant correlation (Table 18).

Table 18: *Correlation coefficient value along with significance level for P2 latency and SIS obtained without noise and at 0 dB SNR in the control group.*

Parameter	Stimulus and condition	SIS without noise		SIS with noise	
		Correlation Coefficient	Sig. (2-tailed)	Correlation Coefficient	Sig. (2-tailed)
N1P2 Amplitude (μv)	Click without noise	.367	.179	**.573***	**.026**
	Click with noise	.415	.140	.350	.219
	/ba/ without noise	.465	.081	**.671****	**.006**
	/ba/ with noise	.212	.448	.448	.094
	/ga/ without noise	.437	.104	.409	.130
	/ga/ with noise	-.380	.162	.153	.586
	/da/ without noise	.268	.335	.144	.609
	/da/ with noise	-.113	.689	.302	.273

***p< 0.05 an **p< 0.01**

4.3.2 Clinical Group (individuals with auditory dys-synchrony)

The mean, SD and range for N1-P2 amplitudes of ALLR elicited by click and speech stimuli in both the conditions were calculated and results are given in the Table 19. The mean amplitude elicited by click stimulus was lesser than that evoked by all the speech stimuli. The N1-P2 amplitude of ALLR evoked at 0 dB SNR was lesser than N1-P2 elicited in the absence of noise for all the four stimuli. However, the amplitude of click evoked ALLR in the presence of noise could not be obtained due to absence of ALLR.

Table 19: *Mean, SD and range for N1-P2 amplitudes of ALLR elicited by click, /ba/, /da/ and /ga/ syllables with and without noise in the clinical group*

Parameter	Stimulus	Clinical group		
		Mean	SD	range
N1P2 Amplitude (μv)	Click without noise	1.76 (N= 7)	0.67	1.09-3.00
	Click with noise	-	-	-
	/ba/ without noise	2.71 (N= 14)	0.76	1.62-3.82
	/ba/ with noise	1.92 (N= 3)	1.26	1.09-3.37
	/ga/ without noise	2.71 (N=12)	1.24	0.92-5.11
	/ga/ with noise	1.45 (N= 2)	0.94	0.78-2.12
	/da/ without noise	2.55 (N= 17)	1.21	0.68-6.10
	/da/ with noise	2.12 (N= 8)	0.99	0.66-3.20

Wilcoxon signed ranks test was carried out which revealed a statistically significant difference between the N1-P2 amplitude evoked by /ba/ and click without noise. Whereas, other pair wise comparisons within and across the stimuli for with and without noise, no significant difference was noted. The comparison between click and /ga/ in the presence of noise was not compared with other stimulus and conditions due to absence of ALLR in the clinical group.

Table 20: *Z-values with significant level for N1-P2 elicited by click and speech stimuli in the presence of noise and without noise in the clinical group*

Pair compared	Z - value
/ba/ with noise - /ba/ without noise	1.604
/da/ with noise - /da/ without noise	1.472
/ba/ without noise – click without noise	**1.992***
/da/ without noise – click without noise	1.782
/ga/ without noise – click without noise	1.753
/da/ without noise – /ba/ without noise	1.177
/ga/ without noise – /ba/ without noise	0.980
/ga/ without noise – /da/ without noise	0.764
/da/ with noise - /ba/ with noise	0.535

***p< 0.05**

4.3.2.1 Correlation between speech identification scores and N1-P2 amplitude in the clinical group

Speech identification scores obtained with and witout noise were correlated with

- N1-P2 amplitude with noise
- N1-P2 amplitude without noise

Spearman's rank correlation coefficient was used to find the correlation. There was no significant correlation found between any two conditions. As none of the subjects exhibited ALLR elicited by click and two subjects had N1-P2 amplitude for /ga/ in the presence of noise, correlation couldn't be obtained. The detail results obtained is given in the Table 21.

Table 21: *Correlation coefficient value for N1-P2 amplitude and SIS elicited at 0 dB SNR and without noise in the clinical group*

Parameter	Stimulus and condition	SIS without noise		SIS with noise	
		Correlation Coefficient	Sig. (2-tailed)	Correlation Coefficient	Sig. (2-tailed)
N1P2 amplitude (μv)	Click without noise	-.408	.364	-.600	.285
	Click with noise	-	-	-	-
	/ba/ without noise	-.216	.478	.366	.373
	/ba/ with noise	-.866	.333	.500	.667
	/ga/ without noise	.000	1.000	-.049	.908
	/ga/ with noise	-	-	-	-
	/da/ without noise	.108	.691	-.037	.937
	/da/ with noise	.098	.817	-.200	.800

4.3.3 Comparison between normal hearing individuals and individuals with auditory dys-synchrony

The mean amplitudes obtained from the clinical group were comparatively lesser than the control group in both the conditions. However, the amplitudes elicited in the presence of noise were lesser than amplitudes elicited without noise in both the groups. In both the groups, the amplitudes elicited by speech stimuli were greater than the amplitude evoked by the click stimulus without noise. Comparison of N1-P2 amplitude between control and clinical group for each stimulus was done using Mann Whitney U test. A significant difference was noted between the groups for N1-P2 amplitude evoked by click, /ga/ and /da/ without noise, which can be seen in the Table 22.

Table 22: *Z-values with significance level along with mean, SD for N1-P2 amplitude elicited by click and speech stimulus in two conditions for both the groups*

	Control group		Clinical group		
	Mean (N= 15)	Standard deviation	Mean	Standard deviation	/Z/
Click without noise	3.04	0.66	1.76 (N= 7)	0.67	**3.034***
Click with noise	1.97	0.44	-	-	-
/ba/ without noise	3.50	1.42	2.71 (N= 14)	0.76	1.811
/ba/ with noise	2.32	0.84	1.92 (N= 3)	1.26	0.772
/ga/ without noise	3.58	0.97	2.71 (N=12)	1.24	**2.172***
/ga/ with noise	2.24	0.81	1.45 (N= 2)	0.94	-

/da/ without noise	3.56	1.08	2.55 (N= 17)	1.21	**2.834***
/da/ with noise	2.41	1.18	2.12 (N= 8)	0.99	0.097

***p< 0.05**

No correlation was found between SIS and N1-P2 amplitude evoked for all the stimulus. SIS obtained at 0 dB SNR was significantly reduced in both the groups when compared with SIS obtained without noise. SIS obtained in the clinical group were poor than SIS obtained in control group, in both the conditions.

It can be concluded from the results that a significant difference was obtained for N1 and P2 latency between the click versus other speech stimuli. But no significant difference for N1 and P2 latency obtained between any two speech stimuli. Amplitude of N1-P2 was greater for speech evoked than for click evoked ALLR. Presentation condition (with noise and without noise) showed a significant affect on N1, P2 latency as well as for N1-P2 amplitude for both the groups.

It can also be noted that there was no correlation between speech identification scores and parameters of ALLR in the clinical group. But in the control group, even though there was a significant correlation found in between SIS and parameters elicited by stimuli in few conditions, definite trend were not observed.

Overall there was greater shift in latencies of N1 and P2 in clinical group than in control group for ALLR elicited by /ba/ and /da/ in both the conditions. ALLR could not be recorded for click from the clinical group in the presence of noise and for stimulus /ga/ only two subjects exhibited ALLR due to which the latency variation was less. The N1-P2 amplitude for speech stimulus showed greater amplitude than click stimulus in both

the groups, as well as in both the conditions. The effect of noise on N1-P2 amplitude was greater for the clinical group than the control group.

Most importantly it could be observed from the data that /da/ stimulus could elicit ALLR from most of the individuals with AD in both the conditions. Click could elicit ALLR from a few individuals with AD in without noise, but failed to record ALLR in the presence of noise. Within the speech stimulus, /da/ elicited ALLR more than /ba/, than /ga/ in both the conditions in individuals with AD.

5. DISCUSSION

In the present study, three speech stimuli and click were used to elicit ALLR. The speech stimuli selected covered the entire speech frequency range, i.e. from low to high frequencies.

The ALLR data obtained from the individuals with normal hearing was statistically analyzed. The results obtained from the statistical analyses are discussed below.

5.1 Effect of type of stimulus on parameters of ALLR:

5.1.1 Latency:

It has been noticed in the current study that the latencies of N1 and P2 evoked by speech stimuli were longer than those elicited by click in normal hearing individuals. This difference in latencies between click and speech stimulus was statistically significant.

Similarly results were reported by Ceponiene et al., (2001). They have investigated the cortical auditory evoked potentials (CAEP's) in normal hearing individuals using speech (/baka/ & /baga/) and non-speech stimuli. The results indicated longer latencies for speech stimuli than non speech stimuli. Purdy, Katsch, Storey, Dillon, Agung and Sharma *(in press)* also investigated the ALLR using speech (/t/, /k/, /d/ & /g/) and non-speech stimuli in individuals with normal hearing. The results

indicated that the latencies elicited by speech stimulus were significantly longer than latencies evoked by non-speech stimulus.

The prolonged latencies obtained for speech evoked ALLR than click could be because a single mechanism in the auditory cortex might be involved in general temporal processing for speech and non-speech stimuli, but may underlie further processing of verbal stimuli (Liegeois-Chauvel, Graaf, & Laguitton 1999). Thus, the discrepancy in latencies between the speech and non-speech stimuli could be due to a difference in the mechanism of neurophysiological processing. Another reason could be due to the rise time of the stimulus i.e., click has steeper rise time than speech stimulus which can lead to shorter ALLR latencies (Onishi & Davis 1968).

Within the speech stimuli the N1 and P2 latencies of ALLR didn't vary significantly from one another. However, the latencies elicited by /da/ were shorter followed by /ga/ and /ba/.

The findings are in agreement with Liegeois-Chauvel, Graaf, and Laguitton (1999), who investigated cortical auditory responses evoked for voiced (/ba/, /ga/ and /da/) and voiceless (/pa/, /ka/ and /ta/) syllables, which are produced naturally. They found that there are no significant differences in latencies between the stimuli.

But the results of the current study conflicted with the findings of Purdy et al., *(in press)*. They recorded ALLR for different speech stimuli (/t/, /k/, /d/ & /g/). They found a significant latency differences across the speech stimuli.

The reason why ALLR didn't vary significantly in latency within speech stimuli could be due to similarities in characteristics of speech stimulus except for frequency dominance in spectral energy. However, ALLR evoked by /da/ stimulus had slightly shorter latency may be because its dominated high frequency spectral energy. Hence, it stimulated the base of the basilar membrane compared to /ga/ (mid frequency spectral energy) and followed by /ba/ (low frequency spectral energy). This could be the reason for shortest latency obtained for /da/ stimulus.

5.1.2 Amplitude:

The amplitude of ALLR elicited for all the speech stimuli was greater than click evoked ALLR in the control group. However, it was not statistically significant.

Similar results were obtained by Ceponiene et al., (2001). They investigated the cortical auditory evoked potentials using speech and non-speech stimulus. They showed that the amplitude of N1-P2 complex was larger for speech sounds than for non-speech, however, it was not statistically significant.

The results of the current study are in contrast with the findings of Tampas, Harkrider and Hedrick (2005). They investigated CAEPs using speech (/ba/ & /da/) and non-speech stimuli. The results indicated that non-speech stimuli elicited significantly larger amplitude than speech stimuli.

This amplitude of N1-P2 being greater for speech stimulus than click stimulus might be due to the duration of stimulus leading to temporal integration. The longer duration stimulus activated the neurons other than simply onset detectors in generation of

ALLR waves (Alain, Woods & Covarrubias 1997) and minimal duration required for the temporal integration to take place is ≥ 30 msec (Forss, Makela, McEvoy & Hari 1993). Hence, resulting in higher compound action potential. This might have lead to the greater N1-P2 amplitude for speech, though it has failed to reach significant level.

Another possible reason could be the optimum stimulus required to elicit ALLR. The stimulus should have 10 msec rise/fall time and 50 msec plateau to elicit an ALLR effectively (Hall 2007). The click had shorter rise time, resulting in reduced amplitude.

The amplitude of N1-P2 complex varied slightly within the speech stimulus. This is because all the three stimuli had similar characteristics except for energy concentration in terms of frequency.

5.2 Effect of the noise on parameters of ALLR:

5.2.1 Latency:

The mean latency values in the presence of noise were increased when compared to ALLR evoked without noise for all the stimuli in the control group. The shift in the latencies between conditions was statistically significant. The latency shift was more for P2 than for N1 peak.

These findings are in agreement with Martin and Stapells (2005) who investigated the effect of masking noise on CAEPs. They used speech sounds /ba/ and /da/ to elicit the response and they concluded that the latencies were prolonged significantly in the presence of noise than compared to without noise conditions.

Since N1 and P2 are obligatory potentials, the presence of noise at 0 dB SNR decreases the audibility of the stimulus. Hence, it leads to prolonged latencies in the presence of noise (Martin & Stapells, 2005). Another reason for prolonged latency could be due to the pronounced disruption of timing features in cortical processing, when extracting and encoding rapidly presented acoustic signals that have been masked by noise (Wible, Nicol & Kraus, 2005).

5.2.2 Amplitude:

The amplitude of N1-P2 complex also reduced at 0 dB SNR for all the stimuli when compared to without noise in individuals with normal hearing. This difference in N1-P2 amplitude elicited in both the conditions was statistically significant.

These results are in consonance with Martin and Stapells (2005). They investigated the effect of masking noise on CAEPs evoked by speech stimulus (/ba/ & /da/). Results indicated that the amplitude of N1-P2 reduced significantly in the presence of noise.

Since ALLR is an exogenous potential, the components of ALLR (N1, P2 & N1-P2) depends upon characteristics of the stimulus. Hence, the presence of noise reduces the audibility of the stimulus leading to reduction in amplitude of N1-P2 (Martin & Stapells, 2005).

5.3 Relationship between speech identification scores and ALLR parameters:

Both SIS and parameters of ALLR are affected in the presence of noise. However, there was no significant relationship observed between SIS and parameters of ALLR in both the condition in individuals with normal hearing.

The lack of correlation between speech identification scores and ALLR could be due to the wide variability in terms of latencies and amplitude of ALLR across the subjects. Moreover the parameters of ALLR are affected by a number of factors like background EEG, sleep or drowsiness etc, which might have lead to poor correlation.

The ALLR latency and amplitude obtained from the clinical group (Individuals with auditory neuropathy) were also statistically analyzed. The results obtained are discussed below.

5.4 Effect of type of stimulus on ALLR parameters:

5.4.1 Latency:

In individuals with auditory neuropathy, it was observed that the latencies for speech evoked ALLR were greater than click evoked ALLR. However, these differences are not statistically significant.

There are no studies available in the literature for the comparison of click and speech evoked ALLR in individuals with auditory dys-synchrony. The prolonged latencies that were observed for speech evoked ALLR than click could be due to the same reason which has lead to prolonged latency for normal hearing group.

Most of the individuals with AD had ALLR for speech stimuli than for click. This could be because the click is a short duration signal with steeper rise time and hence it

requires high synchronous firing. However, synchrony is affected in individuals with AD, leading to abnormal ALLR. One more reason could be due to impaired detection of short duration signals in individuals with AD (Zeng et al., 2005). As click is a short duration stimulus, ALLR responses might have been severely affected than for speech evoked ALLR.

Within the speech stimuli the latencies of N1 and P2 did not vary significantly. This could be because of the spectral properties of the stimulus. ALLR recorded for the speech stimulus in the increasing order was /ga/, /ba/ and /da/. The presence of ALLR for the speech stimulus dominated by different frequency spectral energy can be explained in terms of spectral and temporal theories. According to temporal theory, any stimulus having frequency of $\leq$ 1 kHz is coded based on the firing rate of the auditory nerve for each phase of the signal i.e., by phase locking. In individuals with auditory neuropathy, phase locking is affected leading to dys-synchrony in low frequency auditory nerve fibers (Rance, McKay & Grayden 2004; Zeng et al., 2005). Hence, ALLR elicited for /ba/ and /ga/ stimuli were more affected. According to spectral theories, the high frequencies are represented by the place of excitation on the basilar membrane because auditory nerve cannot fire at higher rates due to refractory period of nerve fibers (Starr, Picton & Kim, 2001). Hence, high frequency discrimination, which does not involve phase locking cues are relatively better compared to discrimination of low frequencies which depends on phase locking cues. As the energy concentration was greater in high frequency for /da/, most of the individuals with auditory dys-synchrony, responses could be recorded.

However, the absence of ALLR in remaining individuals can be explained in terms of severity of the problem and the site of lesion. Another possible explanation for the less number of individuals showing ALLR for /ba/ and /ga/ could be due to the pattern of hearing loss. Most of the individuals with auditory neuropathy in the present study had low frequency hearing loss and ALLR is majorly dominated by low frequencies. However, this unlikely the reason because individuals who have moderate hearing loss showed ALLR, whereas in individuals with minimal hearing loss, ALLRs were absent.

5.4.2 Amplitude:

In individuals with AD/AN, it was observed that amplitude of speech evoked ALLR was greater than click evoked ALLR. Nevertheless, it was not statistically significant.

The greater amplitude for speech stimulus could be due to the duration of stimulus leading to temporal integration. It is because neurons other than onset detectors are also stimulated (Alain, Woods & Covarrubias 1997) and temporal integration for ALLR occurs when the stimulus duration is ≥ 30 msec (Forss et al., 1993). It has been reported that the temporal integration for short duration stimulus was affected in individuals with AD (Zeng et al., 2005). As the click is a short duration signal, it might have failed to elicit ALLR response in AD. However, ALLR could be elicited with speech, because temporal integration function to long duration signal is normal in individuals with AD (Zeng et al., 2005). It can also be because of the optimum stimulus

duration required to elicit ALLR should be 50 msec duration with minimum of 10 msec rise and fall time and 50 msec plateau (Hall 2007).

Within speech stimuli amplitude of N1-P2 complex didn't vary much. This is because all the speech stimulus had approximately same duration (~150 msec) and similar characteristics except for the frequency content of spectral energy.

5.5 Effect of noise on the parameters of ALLR:

5.5.1 Latency:

It was observed that the latencies for click and speech stimuli increased in the presence of noise, which was not statistically significant. None of the individuals showed ALLR for click stimulus in the presence of noise. At 0 dB SNR, for the speech stimuli ALLR elicited in the increasing order was /ga/, /ba/ and /da/, but the number of individuals showing ALLR response at 0 dB SNR was considerably less.

The increase in latency at 0 dB SNR can be due to disruption in synchrony of the auditory nerve fibers caused by noise (Kraus et al., 2000). In most of the individuals with AD, both dys-synchronization and reduced number of fibers often coexists. This produces an average discharge pattern similar to background activity and exaggerates the masking affects seen in these individuals (Zeng et al., 2005). This over masking affect could have lead to absence of ALLR in the presence of noise along with dys-synchrony in most of the individuals with AD.

Even though the latency shift was not statistically significant, greater shift was observed for /ba/ and /da/ stimuli than compared to /ga/ evoked ALLR. This could be due to lesser number of individuals showing ALLR for /ga/ stimulus.

5.5.2 Amplitude:

The amplitude of N1-P2 was reduced in the presence of noise for all the stimuli, which was not statistically significant. The reduction in the amplitude was lesser for /da/ stimulus than /ba/ and /ga/ stimulus.

The reduction in the amplitude of ALLR could be due to disruption of synchrony being more in the presence of noise (Kraus et al., 2000). Another reason is low frequencies are coded based on the phase locking of auditory nerve fibers are affected in these individuals whereas high frequency coding which is based on the place is normal. Hence, the reduction in the amplitude was greater for /ba/ and /ga/ when compared to /da/ as phase locking ability is affected in individuals with AD (Zeng, Oba & Garde 2001). An additional reasoning that could explain the findings is that ALLR is an obligatory potential and the presence of noise affects audibility of the stimulus, thus leading to reduction in amplitude (Martin & Stapells, 2005).

5.6 Relationship between speech identification scores and ALLR:

There was no significant correlation obtained between speech identification scores and parameters of ALLR. This result is same for both the conditions.

Similar results were obtained by Kumar and Jayaram (2005). In majority of individuals with auditory neuropathy P1/N1, P2/N2 complex and mismatch negativity

could be elicited with normal amplitude and latency and none of the measured evoked potential parameters correlated with the speech perception scores.

These results of the present study are in contradiction with Kumar and Vanaja (2008). They investigated the cortical auditory evoked potentials and speech identification scores in individuals with AD. They observed a good correlation between speech identification scores and amplitude of cortical potentials (N1-P2 complex) but did not show a significant correlation with the latency of cortical potentials. The absence of correlation could be due wide range of pathologies in individuals with auditory dys-synchrony.

However, the presence of ALLR did correlate with speech identification scores. Individuals who had greater than 60% of speech identification scores showed ALLR for all the stimuli. Individuals having SIS around 40% showed ALLR for any two of the speech stimuli. Only one individual showed 78% of SIS without noise and 48% of speech identification scores with noise and this individual showed ALLR for all the stimuli without noise and at 0 dB SNR only for /ba/ and /da/ stimuli.

Similar results were also obtained by Rance et al. (2002). They investigated open set speech perception ability and cortical auditory potentials in individuals with AD. They found the children who had recordable cortical evoked potential performed well on open set speech perception task and in contrast, children who had no recordable cortical evoked potential, performed poorly on the same tasks.

The reason for correlation between the presence of ALLR and speech identification scores is that the presence of cortical auditory evoked potential reflects some amount of preserved synchrony in central auditory system which contributes to better speech understanding despite the distortion that occurs at 8th nerve and auditory brainstem in these individuals (Kraus et al., 2000 & Rance et al., 2002).

5.7 Comparison between control group and clinical group:

5.7.1 Latency:

The mean latencies of ALLR elicited for all the stimuli in both the conditions were greater for the clinical group than in control group; even though it was not statistically significant. It was also observed that some individuals with auditory neuropathy had normal latencies, whereas some had greater latencies. Large variation in latency was seen in individuals with auditory neuropathy.

The variability in latency across the individuals may be due to degree of dys-synchrony and underlining patho-physiology. In individuals with AN, one of the possible site of lesion is demyelination of auditory nerve fibers. Demyelination results in an increase in membrane capacitance and a decrease in membrane resistance. Thus, it leads to a delay excitation, reduction in the velocity of action potential propagation and an increase in conduction vulnerability (McDonald and Sears, 1970; Rasminsky and Sears, 1972). The repetitive activation of demyelinated fibers results in a progressive increase in

conduction time of action potential and may lead to intermittent or total in their propagation (Rasminsky & Sears 1972). Therefore, the latencies of the evoked potentials will be prolonged. Another possible site of lesion in these individuals is axonal neuropathy. This axonal neuropathy reduces the number of neural elements but doesn't directly affect the conduction speed. The refractory periods of these fibers also tend to be normal and are capable of firing at higher rates. Therefore the classic signs of axonal neuropathy are reduction in whole nerve action potential rather than an increase in latency or broadening of potentials (Kuwabara, Nakajima & Hattori 1999). This might have lead to the latency variations observed in the clinical group.

5.7.2 Amplitude:

The amplitude of ALLR elicited was greater for control group than clinical group in both with and without noise conditions, however it was not significant. In clinical group, some individuals had normal N1-P2, whereas some had abnormal amplitude.

The reduction in amplitude in the clinical group can be due to the site of the lesion and severity of the pathology (Kumar & Vanaja 2008).

None of the group showed significant correlation between SIS and parameters of ALLR in both the conditions. The lack of correlation between speech identification scores and ALLR could be due to the wide variability in ALLR parameters recorded from both the groups especially in individuals with AD. Another reason could be, ALLR is affected by large number of factors like sleep or drowsiness, background EEG etc.

6. SUMMARY AND CONCLUSION

Auditory neuropathy is one of the hearing disorders, in which cochlear outer hair cell function is normal, but the afferent neural transmission is affected. The integrity of cochlear function is indicated by the presence of evoked otoacoustic emissions and/or cochlear microphonics (CM). The abnormal neural transmission or dys-synchrony is indicated by the absence of auditory brainstem responses and middle ear muscle reflexes (Rance et al., 2002).

In these individuals, PTA (pure tone average) may range from normal to profound hearing impairment. A majority of the individuals with auditory neuropathy have low frequency hearing loss with speech identification scores disproportionate to their pure-tone hearing loss. Even though some individuals show normal speech identification scores in quiet, speech identification scores in the presence of noise were affected to greater extent. Poor speech perception abilities in these patients are attributed to

abnormal temporal coding and asynchrony. Thus, use of different speech stimulus having different spectral composition could help us to understand underlying pathophysiology of AD, and also, if they are recorded in the presence of noise it might give more information about the speech processing ability of the individual with AD in the presence of noise.

Evoked potentials require synchronous firing of nerve fibers and currently are the only way to evaluate neural synchrony. These potentials reflect the response patterns of neurons responsible for encoding the acoustic complexities of speech in the normal auditory system. In individuals with auditory neuropathy/dys-synchrony, auditory brainstem responses are absent or severe disrupted. It might be expected that the middle latency and cortical auditory evoked potential (CAEP) would also be similarly affected. However, some of these individuals show CAEPs, because these potentials are less dependent on synchronous neural firing than auditory brainstem responses (Starr et al., 1996 & Rance et al., 2002).

Thus, studying the processing of auditory signals in the presence of noise using evoked potentials helps us in understanding the processing difficulties of individuals with auditory neuropathy. It also might help us in understanding the poor speech identification scores obtained in the presence of noise, in these individuals. Hence, the present study was aimed to:

- know whether the ALLR vary for different speech sounds in quiet and with ipsilateral noise in normal hearing individuals and individuals with AN/AD.

- investigate the relationship between the click evoked ALLR and speech identification scores in quiet and noise in individuals with normal hearing and individuals with AN/AD.
- investigate the relationship between the speech evoked ALLR and speech identification scores in quiet and noise in normal hearing individuals and individuals with AN/AD.
- know whether the non-speech stimulus or speech stimulus is better to elicit ALLR in individuals with AN/AD.
- know which speech sounds is more suitable to elicit ALLR in individuals with auditory dys-synchrony.

To arrive at the objectives, 15 normal hearing individuals (control) and 16 individuals with auditory dys-synchrony (clinical group) in the age range of 12-39 years were taken. ALLRs were evoked for click and speech stimuli (/ba/, /ga/ & /da/) from all the participants in the control group in one ear and in the clinical group one or both ears. Different speech stimuli were used because /ba/ is dominated by low frequency spectral energy, /ga/ is dominated by mid frequency spectral energy and /da/ by high frequency spectral energy. ALLRs were elicited for all the participants without noise and at 0 dB SNR using all the stimuli.

The ALLRs obtained from both the groups were analyzed by three experienced judges for N1, P2 latencies and N1-P2 amplitude. From the data obtained the mean, standard deviation and range were calculated and following statistical analysis were done.

- To compare between the parameters of ALLR between groups Mann Whitney U test was administered.
- To find the effect of stimuli and effect of noise on latency and amplitude, two way repeated measure ANOVA was done in control group and Wilcoxon's sign rank test in clinical group.
- The parameters of ALLR elicited in the noise and without noise were compared using paired t-test in control group and Wilcoxon sign rank test in clinical group.
- Correlation between speech identification scores and ALLR parameters was done using Spearman's rank correlation coefficient for both the groups.

The data obtained without noise condition was analyzed for both the clinical and control group. The results obtained are as follows:

Control group:

- There was a significant difference in N1 and P2 latency obtained between the click and speech stimuli, which can be due to the type of the stimulus and difference in rise time of the stimulus between click and speech.
- But no significant difference for N1 and P2 latency obtained between any two speech stimuli.
- Amplitude of N1-P2 was greater for speech evoked ALLR than for click evoked ALLR, which was not significant. This could be due to duration of the stimulus and temporal integration.

Clinical group:

- Even though the latencies of speech evoked ALLR are prolonged compared to click, significant difference was not seen. The possible explanation could be due to the type of the stimulus and steeper rise time of the click than speech stimulus.
- Amplitude of N1-P2 is greater for speech stimulus than for click, which was not statistically significant. The reason can be the duration of the stimulus and temporal integration.
- Within the speech stimuli /da/ elicited more number of responses followed by /ba/ and then /ga/, which could be due to the spectral content of the stimulus. Since /da/ is dominated by high frequencies, it doesn't depend upon phase locking cues which are affected in individuals with AD. Hence, /da/ elicited more responses than the other stimuli.

Comparison between control and clinical group:

- The latencies elicited in individuals with auditory dys-synchrony were prolonged for all the stimuli compared to control group. However, it was not statistically significant. The reason for greater latencies in individuals with AD, which could be due to degree of dys-synchrony.
- Even though the amplitude of N1-P2 is greater for all the stimulus in control group than in clinical group, it was not significant. The reduction in the amplitude could be due to dys-synchrony or axonal loss.

The data obtained at 0 dB SNR was analyzed in both the groups. The results obtained are given below:

Control group:

- Presentation condition had (with noise and without noise) showed a significant effect on N1, P2 latency as well as for N1-P2 amplitude. Since ALLR is an exogenous potential, it depends on stimulus characteristics. Hence, the presence of noise affects the audibility of the stimulus, thereby affecting the parameters of ALLR.

Clinical group:

- Latencies of N1 and P2 elicited in the presence of noise were longer compared to without noise, which is not statistically significant. Reason for prolonged latencies could be due to reduced audibility and greater masking affects seen in these individuals.
- N1-P2 amplitude is reduced at 0 dB SNR when compared to without noise for all the stimuli. This could be due to the dys-synchrony of auditory fibers in the presence of noise and reduced audibility.
- /da/ elicited ALLR response in more number of individuals with AD. As /da/ is dominated by high frequencies, which are coded based on place of excitation on basilar membrane they are less affected in these individuals.
- Click evoked ALLR was absent in all the individuals with AD at 0 dB SNR, because click evoked responses depends on the high synchronous firing. As the synchronous is affected more in the presence of noise in individuals with AD, click evoked ALLR is absent.

Comparison between control and clinical group:

- Even though, latency shift was observed in the presence of noise for both the groups, shift was greater in clinical than in control group. This latency shift was not statistically significant. Greater latency shift in the clinical group could be due to dys-synchronous firing and reduction in the number of neural elements responding to the stimulus.
- Amplitude of N1-P2 at 0 dB SNR was also affected in both the groups, but to greater extent in clinical group than in control group. The reason for greater reduction in amplitude in clinical group could be due to disruption in synchrony and greater masking affects seen in these individuals.
- Most of the individuals with auditory neuropathy showed no ALLR in the presence of noise for click, /ba/, /ga/ and only about 35% of ears showed ALLR for /da/ stimulus. It could be because of the frequency content of the spectral energy.

Speech identification scores and ALLR:

Control group:

- No significant correlation was obtained between speech identification scores and parameters of ALLR. This can be accredited to the wide variability in parameters of ALLR.

Clinical group:

- There was no significant difference between the speech identification scores and ALLR parameters in both the conditions, which can be attributed to the wide range of variability in ALLR parameters.

Conclusion:

It can be concluded from the above results that the speech elicits better ALLR than click. Hence, speech evoked ALLR can be recommended in clinical use for both normal hearing individuals and in clinical population (individuals with auditory dys-synchrony). /da/ stimulus could elicit ALLR from more number of individuals with AD in both the conditions. Hence, it could be a useful stimulus to elicit ALLR in individuals with AD. There was no significant relationship between speech identification scores obtained and parameters of ALLR in both the conditions for both the groups. But there was a good relation between the presence of ALLR for different stimuli and speech identification scores obtained in both the conditions in individuals with AD. It can also be concluded that optimal auditory nerve and auditory brainstem synchrony do not appear to be essential for understanding speech in quiet listening conditions. However, synchrony is critical for understanding speech in the presence of noise.

Clinical implication of the present study:

The study can have the following implications:

- It can be used as an electrophysiological tool to evaluate the processing of speech sounds in normal population as well as in the impaired population.
- The present study also suggests the usage of speech stimulus for eliciting ALLR in individuals with auditory neuropathy.
- It also suggests the usage of /da/ stimulus to elicit the ALLR response in individuals with AD.

- ALLR can be used to assess the hearing ability in individuals with auditory neuropathy from whom behavioral thresholds cannot be obtained.
- Using different stimuli dominated by different spectral energy helps us in estimating the severity of pathology across speech spectrum.

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