"Effect of spectral enhancement on speech identification scores and late latency response measures in subjects with auditory dys-synchrony".

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APRIL-2008

Dedicated

To

My Grand Parents,

My teachers & AIISH.

CERTIFICATE

This is to certify that this dissertation entitled is *"Effect of spectrial enhancement on speech identification scores and late latency responses in subjects with* auditory dis-synchrony" is a bonofide work in part of fulfillment for the Master of Science (Audiology) of thestudent (Registration No.06AUD019).

Mysore

April, 2008

Dr. Vijayalakshmi Basavaraj
Director
All India Institute of Speech and Hearing
Mysore-570 006.

CERTIFICATE

This is to certify that this dissertation entitled is *"Effect of spectrial enhancement on speech identification scores and late latency responses in subjects with* auditory dis-synchrony" has been prepared under my suprevision and guidence. It is also also certified that this has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysore

April, 2008

Dr. Vijayalakshmi Basavaraj
Director
All India Institute of Speech and Hearing
Naimisham Campus
Manasagangothri
Mysore-570 006.

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INTRODUCTION

Auditory neuropathy/auditory dys-synchrony(AN/AD) is a disorder characterized by the impairment of the peripheral auditory function with the preservation of the outer hair cell (OHC) integrity (Berlin et al., 1998; Berlin, 1999; Butinar et al., 1999; Starr, A., Sininger, Y., Pratt, H., 2000). The peripheral lesion could be localized at the level of the inner hair cells (IHCs), auditory nerve fibers or the synapse in between (Starr, A., Picton, T.W., Sininger, Y., Hood, L.J., Berlin, C.I., 1996, 2000; Berlin et al., 1998; Butinar et al., 1999).

It is now well established that speech identification abilities of individuals with auditory dys-synchrony are disproportionate to the degree of their hearing loss (Li, et al., 2005; Starr, et al., 1996). In fact, it can be said that disproportionately poor speech identification scores to pure tone thresholds is the cardinal characteristic of persons with auditory dys-synchrony. Speech perception abilities in these patients appear to depend on the extent of distortion of temporal cues at suprathreshold levels rather than access to speech spectrum (related to audibility), unlike in patients with cochlear hearing loss (Rance et al., 2004; Zeng et al., 1999)

Physiological tests generally used in diagnosing auditory dys-synchrony are auditory brainstem response and otoacoustic emissions. By clinical definition, a subject with auditory neuropathy/dys-synchrony will have abnormal or absent auditory brainstem response with presence of otoacoustic emissions. Because normal auditory brainstem response can be recorded only when multiple neurons fire synchronously at onset, even minor variation in the timing of neural discharge after each stimulus can make the auditory brainstem responses unrecognizable (Kraus et al., 2000).

Another physiologic test which has been studied widely in individuals with AN/AD is the auditory late latency responses (LLR). The peaks in LLR reflect the summation of excitatory post synaptic potentials i.e. it reflects slow dendritic event. Also the peak for LLR is on the order of tens of hertz (Moller, 1994). Further for LLR the waves are so slow that contribution separated by several milli seconds contributes to these later waves. The synchrony required for LLR is on the order of several milliseconds that's why the LLR is expected to be present in the individuals with AN/AD (Kraus et al.2000). However there are equivocal findings regarding presence/absence of LLR in individuals with AN/AD (Starr et al 1991., Starr et al., 1996)

Pyschoacoustical studies have demonstrated that individuals with AN/AD are impaired in temporal resolution and frequency discrimination whereas performance on other psycho-acoustical abilities such as difference limen of intensity and difference limen of frequency resolution is within normal limits. Zeng et al (1999) have clearly demonstrated that impaired ability to process the amplitude variation of the continuous signal in speech perception, leads to difficulties encountered by individuals with AN/AD.

As AN /AD adversely affects speech comprehension, appropriate management should be considered. Communication difficulties in individuals with auditory dys-synchrony, even in those with the mild hearing loss, are much more severe compared to those individuals with cochlear hearing loss of 60 dB HL or more. Conventional amplification through hearing aids does not seem to be beneficial as this does not address the problem of neural dyssynchrony (Rance et al., 2002). Cochlear implantation is of benefit to some patients with auditory dys-syn-chrony (Sininger et al., 1999). However, the usefulness of cochlear implantation seems to depend on the site of lesion and not all cases of AN /AD are suitable for a cochlear implant (Simmons, & Beauchaine, 2000). Cases with lesions at the inner hair cell level or at the synapse with the

auditory nerve, which are bypassed by the implant, may achieve greater benefit (Simmons & Beauchaine, 2000).

Thus, it is important to explore alternative strategies that are much less invasive than cochlear implants which may benefit individuals with AN, particularly for those who have relatively mild AN. One effective means of improving speech intelligibility is to speak clearly (Picheny, Durlach, & Braida, 1985, 1986, 1989). When talkers are instructed to speak clearly, they usually produce more intelligible speech than they would when interacting in casual conversation. The higher intelligibility in clear speech than in conversational speech is likely a result of acoustic and phonetic differences between these two styles of speech. These differences include reduced speaking rate, increased energy in the 1000–3000 Hz range, enhanced temporal modulations, expanded voice pitch range and vowel space (Ferguson & Kewley-Port, 2002; Krause & Braida, 2002, 2004; Liu, Del Rio, Bradlow, & Zeng, 2004; Payton, Uchanski, &Braida, 1994).

Another option which can be opted for individuals with the AN/AD is the envelope enhancement of speech. A number of investigators have studied the importance of envelope enhancement on speech perception in noise for subjects with normal hearing, cochlear hearing loss and learning disability (Tallal et al., 1996; Larenzi, C., Berthommier, F., Apoux, F., & Bacri, N. 1999; Apoux , F., Tribut, N., Dehruille, X., & Lorenize, C.2004). They have shown improvement with envelope enhancement for cochlear hearing loss and other group of individuals, but improvement observed was lesser in these groups. The rationale behind employing envelope enhancement in noise is that the noise reduces the ability to process amplitude variation in the speech signal, so enhancing amplitude variations improves speech

perception. Since AN/AD subjects have impairment in processing amplitude variation of speech signal, enhancing the modulations might improve speech perception.

Zeng and Liu, (2005) have demonstrated that clear speech improved speech perception in individuals with AN/AD. The improvement observed for clear speech has been attributed to enhanced envelope in clear speech. Clear speech has certain properties ; type of speaking style to facilitate better communication in adverse listening conditions, roughly 17 % more intelligible than normal conversational speech for mild to moderate hearing impaired individuals (Pichney 1985; Payton 1994).

Need of the study:

There is no consensus over the management issues of AN/AD subjects. Studies dealing with envelope enhancement have shown improvement in speech perception in persons with cochlear loss. However, there is a dearth of information regarding the usefulness of envelope enhancement of speech in improving speech perception in individuals with AN/AD.AN/AD group have been reported to have temporal deficits, and therefore difficulty in recognizing short signals effectively. Hence there is a need to study whether spectral enhancement of signal improves speech recognition in such subjects or not. It will be interesting and relevant to study the effect both through objective as well as subjective measures. Hence this study was convinced and conducted to examine the effect of envelope enhancement on speech perception in subjects with AN/AD.

Aims of the study: To compare the effect of envelope enhancement on speech perception and late latency response in subjects with auditory dys-synchrony/auditory neuropathy with those obtained in subjects with normal hearing.

Objectives:.

1) To compare LLR amplitudes for /da/ syllable in non enhanced and enhanced condition in subjects with AN/AD
2) To compare LLR latencies for /da/ syllable in non enhanced and enhanced condition in subjects with AN/AD
3) To compare LLR amplitude for /da/ syllable in non enhanced and enhanced condition in subjects with normal hearing.
4) To compare LLR latencies for/da/ syllable in non enhanced and enhanced condition in subjects with normal hearing.
5) To compare LLR amplitude for /da/ in non enhanced and enhanced condition between normal and AN/AD subjects.
6) To compare LLR latencies for /da/ syllable in non enhanced and enhanced condition between subjects with normal hearing and subjects with AN/AD.
7) To compare the morphology of LLR for /da/ syllable in non enhanced and enhanced condition for subjects with normal hearing subjects and subjects with AN/AD on 3 point rating scale.
8) To compare speech perception results obtained with non enhanced and enhanced signals in AN/AD subjects in: (i) Quite and (ii) 10 dB SNR condition

9) To compare speech perception results obtained with non enhanced and enhanced signals in normal subjects in: (i) Quite and (ii) 10 dB SNR condition

Research Design:

Mixed group pretest posttest design wherein independent variables are the speech stimuli in non enhanced and enhanced condition, age and sex of the subjects in both groups and the dependent variables are the latencies , amplitude , morphology obtained for each peak as well as the responses given by the subjects for perceptual measure.

Hypothesis:

1) There is no difference in LLR amplitudes in subjects with AN/AD for syllable /da/ in the non enhanced and enhanced condition.
2) There is no difference in LLR latencies in subject with AN/AD for syllable /da/ in the non enhanced and enhanced condition.
3) There is no difference in LLR amplitudes in subjects with normal hearing for syllable /da/ in the non enhanced and enhanced condition.
4) There is no difference in LLR latencies in subjects with normal hearing for syllable /da/ in non enhanced and enhanced condition.
5) There is no difference between subjects with normal hearing and AN/AD in LLR amplitude in non enhanced and enhanced condition.
6) There is no difference between subjects with normal hearing and AN/AD in LLR latencies in non enhanced and enhanced condition.

7) There is no change in morphology in non enhanced and enhanced condition for subjects with normal hearing and AN/AD on 3 point rating scale.
8) There is no difference in speech perception results obtained for non enhanced and enhanced condition in subjects AN/AD/.
9) There is no difference in speech perception results obtained for non enhanced and enhanced condition in subjects with normal hearing.

REVIEW OF LITERATURE

Of the all senses in man, hearing sense has its own importance. It not only enables us to communicate effectively but is also essential for localizing sound, to balance and for learning. From the peripheral organ of hearing the sound energy are transmitted to higher centers in the brain where it gets decoded. Any problem in the transmission can hamper this message. This problem could be either in the conductive system or in the sensory system. In the sensori system, the problem could be either in the organ of corti or in the neural pathway.

1.1) Auditory dys-dysnchrony/ Auditory neuropathy

Auditory dys-synchrony (AD) which was earlier referred to as auditory neuropathy (AN), is a recently described hearing disorder that has unique pathologies and perceptual consequences (Starr et al. 1991; Starr, Picton, Sininger, Hood & Berlin 1996). It is a form of hearing disorder in which the outer hair cell function is normal but afferent neural conduction pathway in the auditory system is disordered (Starr et al. 1996). The clinical findings that define auditory neuropathy / dys-synchrony are the demonstration of outer hair cell integrity in evoked otoacoustic emissions (OAE) and / or cochlear microphonic (CM) recordings in conjunction with the inability to record evoked neural activity at the level of 8^{th} cranial nerve (Starr, et al., 1996).

In the study of Starr et al. (1991), an "11 year old girl was described who had an absence of sensory components of auditory evoked potentials (brainstem, middle and long latency potentials) to click and tone burst stimulus that otherwise she could hear clearly. Upon psychoacoustic testing it was revealed that there is marked improvement on auditory perception dependent on temporal cues; (lateralization of binaural clicks, change of binaural masked threshold with changes in signal phase, binaural beats, detection of paired monaural clicks, and monaural detection of a silent gap in sound, monaural threshold elevation of short duration tones). In contrast, auditory functions reflecting intensity or frequency discriminations (difference limens) were only minimally impaired. Pure tone audiometry showed a moderate (50 dB) bilateral hearing loss with a

disproportionate severe loss of word intelligibility. Cochlear microphonic, cortical sustained potentials and long latency cognitive potentials (P300) were present". Finally, they attributed changes in temporal encoding of acoustic signals at synapse between hair cell and VIIIth nerve dendrites for deficit in perceptual measure and evoked potential.

Starr et al (1996), studied 10 subjects with auditory neuropathy/dys-synchrony, who had preserved OAEs and absent auditory brainstem responses (ABR). Both ipsilateral and contralateral acoustic reflexes along with contralateral suppression of OAEs were absent in all the tested patients. Behaviorally nine had mild to moderate hearing loss on audiometry. The shape of pure tone loss varied, being predominantly low frequencies in five, flat in three and high in two patients. Speech intelligibility was poor ,out of proportion to pure tone average in six of eight patients tested. All patients were neurologically normal.

Auditory dys-synchrony is a hearing disorder in which peripheral hearing appears normal, but the eighth nerve fiber and brainstem functioning are abnormal (Kraus, Ozdamar, Stein & Reed, 1984; Starr et al. 1991). Kraus et al (1984) had collected data over 3 years. Out of 543 children who had no clinical evidence of brainstem damage were evaluated for suspected hearing loss and 99 % had absent ABRs most of them had severe to profound hearing loss, seven had moderate hearing loss.

1.2. Causes of auditory neuropathy/dys-synchrony

Currently the specific risk factors for auditory neuropathy /dys-synchrony are not clearly understood. Some individuals have risk factors related to hearing loss in their history, however, a significant amount of patients have no risk factors (Hood, Berlin, Morlet, Brashears, Rose, & Tedesco, 2002). A number of infants diagnosed with auditory neuropathy/dys-synchrony have history of major neonatal illness including pre-maturity, low-birth weight, anoxia and hyperbilurubinemia (Sininger, 2002). Cochlear hypoxia has also been suggested as a possible cause for auditory neuropathy / dys-synchrony (Harrison, 1998). Genetics also plays an important role in the etiology of auditory

neuropathy/dys-synchrony. Families have been identified with siblings with auditory neuropathy/dys-synchrony. In addition, there are also parents with auditory dys-synchrony who have children with this disorder. Therefore, it is hypothesized that auditory dys-synchrony appears to follow both recessive and dominant inheritance patterns (Hood, et al., 2002).

1.2) Pathophysiology of the disorder

The site lession AN/AD is not completely understood. "Many patients with this hearing disorder have concomitant peripheral neuropathy, which makes the auditory nerves logical site of lesion" (Abdala et al, 2000). However, the characteristics of AN/AD most likely reflect more than a single etiology; thus the disorders(s) may more accurately be described as auditory neuropathies (Hood, 1998). There could be pathology at inner hair cells (IHC), or IHC synapse with the auditory nerve, spiral ganglion cell disorder, demyelinization of auditory nerve (Rance et al, 2004). Still the confusion prevails regarding the exact site of lesion as there is no test to assess the integrity of IHC or synapse between IHC and auditory nerve independently.

Disruption of Synchronization in the Auditory Nerve Fibers

According to Tlumak (2002) "when myelin, which insulates and aids neurons in the conduction of electrical impulses degenerate the properties of intermodal membranes changes, impairing their ability to transmit trains of impulse". The electrical impulse travelling along the demyleinated nerves are distorted, stopped, or slowed due to the temporal spread of electrical activity (Rapin & Gravel, 2003). Hence there is poor ABR, also acoustic reflex and contralateral suppression of OAEs are affected. Asynchronization not only affects ABRs, but also influences auditory perception dependent on temporal cues (Starr et al., 1991; Starr et al. 1996; Zeng, Oba, Garde, Sininger, & Starr, 1999, Kraus et al., 2000; Rance, Mckay, & Grayden, 2004; Zeng, Kong, Michalewski, & Starr, 2005).

Pathology restricted to IHCs or their Synapse with Type I Auditory Nerve Fibers

At present there is no test to assess IHC status, however Harrison (1998), suggested that AN/AD could be due to scattered loss of IHC and their central cochlear afferent connections (Harrison 1998). Also he said this could be related to extended period of hypoxia, which is quite prevalent in high risk birth (Harrison 1998). In a histopathological study by Starr et al (2003) everything was found to be normal throughout the cochlea except at apical turn where 30 % loss of outer hair cell was observed. Inner hair cell was normal throughout the length of the cochlea. However there was a profound loss of ganglion cells. The auditory nerve adjacent to the cochlear nucleus showed marked decrease in the auditory fibers.

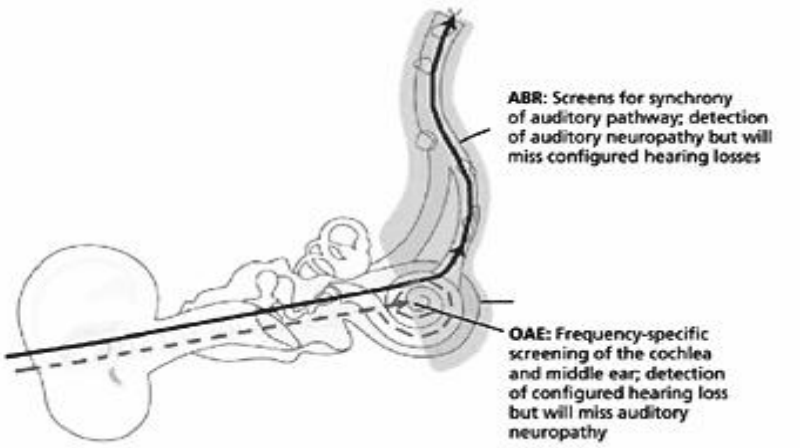


Figure 1 : The parts of the auditory system assessed by OAE and ABR screening. OAE assess the OHC functioning, while ABR assess the neural synchrony. (Willam F. Dolphin, 2004)

Interruption in the Afferent-to- Efferent Loop

Myelinated afferent fibers (type I) originate at the base of IHCs and terminate in the cochlear nuclei with connections to the ipsilateral and contralateral superior olivary complex. Efferent fibers originate from the olivocochlear bundle, in which its

contralateral medial system synapses with the OHCs of the organ of corti. It is well known that there is contralateral suppression of DPOAE, TEOAE and SOAE (Puel 1990; Mott et al 1989; Collet et al 1990). However, Contralateral suppression of OAE is absent in AN/AD, suggesting that there is interruption in the afferent- to- efferent loop.

1.3) Audiological characteristics of auditory –dys-synchrony

The main complaint of this group of subjects is that they can hear but can't understand the speech, and the poor perception of speech goes out of proportion when compared to cochlear hearing loss subjects (Li, Wang, Chen & Liang, 2005; Starr et al., 1996). The problem gets worse in noisy situation like in reverberated room or on road etc. The neural events depend upon the neural synchrony. AD, a disorder of stimulus timing related to neural synchrony, provides a model for studying the role of synchrony in auditory perception (Kraus et al., 2006). The clinical diagnosis of AD has been typically characterized by the presence of oto-acoustic emission and/or cochlear microphonic and the concurrent absence of the averaged auditory brain stem responses, acoustic reflexes both ipsilateral and contralateral.

Behavioral tests results in Auditory Dys-synchrony:

The behavioral test results are of utmost importance in routing testing, and no test can replace the originality of these tests. The hearing thresholds in individuals with auditory dys-synchrony (AD) can range from normal hearing to profound loss (Starr et al., 1996). The degree of hearing loss is not always static, out of ten patients, 9 had thresholds ranging between mild to moderate and one had profound hearing loss. The configuration for five was low frequency loss, flat for three and high frequency for two.

Starr et al. (2000), report in their study of 67 subjects with AD, 41 % had flat audiogram, 29 % had reverse sloping pattern, 9 % had irregular, 5 % had "U" shaped audiogram, tent shape at 2 kHz in 5 % and only 11 % had high frequency sloping audiogram. However, Sininger and Oba (2001) reported 43% of individuals with flat

audiometric shape, and 28% with a reverse sloping hearing loss with higher thresholds for low-frequency stimuli than for higher frequencies.

Speech identification abilities of the subjects with auditory neuropathy are disproportionate with degree of hearing loss (Li et al. 2005; Starr et al. 1996; Zeng et al., 1999). Zeng et al (1999) studied 8 AD subjects, 3 subjects had 0 % speech identification scores (SIS), for 2 subjects testing could not be done and rest had below chance level SIS. They attributed the abnormal measures are due to distorted temporal coding in the auditory nerve. Rance et al (2004) concluded that the disproportionate scores in the speech perception task are due to poor temporal processing. Psycho-acoustical studies have revealed that subjects with auditory neuropathy are poor in temporal resolution and frequency discrimination where as performance on other pychoacoustical abilities such as DLI and frequency resolution is within normal limits. Zeng et al., (1999; 2005) have clearly demonstrated that impaired ability to process the amplitude variation of the continuous signal in speech perception, lead to difficulties encountered by individuals with auditory neuropathy.

Shivaprakash & Manjula (2003) studied temporal resolution using gap detection test in two subjects with auditory dys-synchrony. Their results showed that there is variation in the temporal resolution of subject with auditory dys-synchrony.

Starr et al., (1996) reported a marked impairment of those auditory perceptions that are dependent on temporal cues in their study of ten subjects. These include lateralization, masking level difference, monaural detection of a silent gap in a sound and monaural threshold elevation for short duration tones. In contrast, auditory function reflecting intensity or frequency discriminations (difference limens) were only minimally impaired.

Electrophysiological test results in AD.

To study objectively, the processing of sound evoked potentials have been used. Auditory brain stem responses are usually absent or abnormal in cases with

auditory neuropathy/dys-synchrony. Kraus et al., (1984) reported 4 subjects with audiometric findings of normal to moderate hearing loss with absence of ABR. Results of ABR was disproportionate as per the loss on the audiogram. One subject showed normal Middle Latency Response (MLR), while for the rest it was absent.

In another study Starr et al., (1996) did behavioral and electrophysiological tests on ten individuals with hearing impairment. The test was as per the disorder of the auditory portion of the VIIIth cranial nerve. OAE and preserved Cochlear Microphonic revealed normal cochlear outer hair cell function whereas abnormal auditory pathway was revealed by abnormal ABR or absence of ABR. MLR was found in one of the five subjects with absent ABR, and long latency potential components (N100, P200) were recorded in three out of four subjects with absent ABR. Cognitive potential (P300) evoked in an auditory discrimination target detection task, were also present in the two patients tested.

Kraus et al., 2000 studied speech perception skills ranging from pure tone to sentence levels in an individual with auditory neuropathy having normal limits of hearing. Findings were viewed in the context of the sound structure of the signals and the physiological activity along the auditory pathway. It was observed that speech elicited cortical potential (LLR and MMN) were present .

Rance, Con-Wersson, Wunderlich and Dowell (2002) studied cortical event related potential in 18 children with auditory dys-synchrony. Results showed that approximately 50% of children had event related potential of normal latency, amplitude and morphology. Govil, (2001) recorded auditory evoked potentials (ABR, MLR, LLR and MMN for intensity deviance) in 7 subjects with auditory-dys-synchrony, and found that in a majority of the subjects LLR and MMN were present though there was absence of MLR.1 individual had all the potentials absent.

Rance et al., (1999) studied ASSR for high modulation rate in 20 infants and children with auditory dys-synchrony and results revealed weak co-relation between behavioral threshold and ASSR threshold. Recording of potentials were present only at high sensation levels.

1.4) Relationship between the Speech Identification Score and other test results.

Speech identification scores and psychophysical tests

There is distortion of the suprathreshold cues. Rosen (cited in Kraus et al., 2000) reported that suprasegmental components of speech typically are expressed over hundreds or thousands of milliseconds, and fine structure of speech, characteristic of many consonants, occur in the tenth-of-millisecond range.

Marked impairment of monaural timing is consistent with poor speech and word comprehension that was out of proportion to the pure tone loss (Starr et al., 1991). Individuals with auditory dys-synchrony had difficulty distinguishing words differing in their vowels but could distinguish words bound on their high frequency consonants. From the study it was concluded that disorder of the peripheral part of the auditory system at the eighth cranial nerve principally affecting the temporal precision of the neural coding and or its transmission centrally.

Kraus et al., (2000) in their study, reported 24 year old women having auditory dys-synchrony where speech perception was good in quiet for sentences, words, but were markedly impaired in noise. This says that neural synchrony is critical for understanding speech in the presence of noise. In the same study it was also reported that she had difficulty in response to sound containing critical acoustic information at stimulus onset, rather than to stimuli requiring discrimination of durational cues within a syllable. The subject had good discrimination for speech sounds along /ba-wa/ continuum but poor discrimination for /da-ga/ continuum, because the difference between /da/ and /ga/ occurs at stimulus onset while for /ba/ and /wa/ occurs in the syllable. So, due to deficits in the representation of transient stimulus there is poor performance in /da-ga/ whereas as longer duration harmonic aspects of voicing appeared to be preserved giving better score in /ba-wa/.

Rance, Mckay, and Grayden (2004) reported, in a group of 14 subjects having AN/AD the degree of temporal disruption were strongly correlated with speech perception ability. They showed that inability of some subjects to perceive amplitude modulation fluctuation at modulation rate as low as 10 Hz suggests a degree of temporal processing abnormality which is more than SNHL. It was also reported that individuals with good perception had normal DLF where as subjects with severely depressed DLF showed poor speech perception.

Speech Identification scores and electrophysiological tests

Hood , (1999) reported Late Latency response to be better with regard to auditory perception than compound action potential (CAP) and ABR, which are highly dependent on neural synchrony. According to Speckmann and Walden cited in Rance et al., (2002), cortical related potentials reflect the postsynaptic potentials in the dendritic zone of neurons within the auditory cortex, whereas ABR reflect the volume conducted action potentials of axons. Hence there could be presence of cortical potentials even when ABR is absent.

Rance et al., (2002) studied 15 children with auditory dys-synchrony and recorded event related potentials. It was reported that good speech perception ability with normal latencies had recordable potentials, and was absent in the individual where there was poor speech perception ability.

Vanaja and Manjula (2002) studied the usefulness of cortical evoked potential in predicting benefit derived from amplification in individuals with auditory dys-synchrony. Individuals who had absent cortical evoked potential had lesser benefit with hearing aids than who had better waveforms.

1.5) Management options for individuals with Auditory neuropathy/dys-synchrony

At present, there is no consensus on a specific therapeutic approach, but some of the viable options suggested are the use of hearing aids, some type of communication method

like sign language and cued speech, frequency modulated (FM) auditory trainers, or cochlear implants.

1.5 .a. Hearing aids/ FM system

In a sensory loss, when OHCs are damaged, hearing thresholds are elevated and hearing aids and/or FM systems are used to provide amplification, that is, to restore sensitivity for low-level sounds or increase the signal-to-noise ratio. Most researchers argue that the presence of OAEs in AN/AD patients indicate functional OHCs; therefore, no further amplification of sound is necessary. In addition, the use of amplification may cause damage to the existing OHCs, causing hearing loss that was not present in AN/AD patients (Starr et al, 1996).

There is report that some patients with AN/AD may experience poorer speech perception in background noise (Kraus et al, 2000). Therefore, to answer this, the clinician may consider the use of directional microphones or personal FM systems to improve the signal-to-noise ratio (Stredler-Brown, 2002). There is a major disadvantage of using FM systems as a primary means of managing AN/AD. For example, a FM system is more feasible in a classroom setting than it would be on a playground for a child. Therefore, even if FM systems do improve the signal-to-noise ratio, it is not an appropriate management strategy for everyday normal situations outside of the school environment.

Rance et al, (1999) showed that approximately 50% of affected children (total 20 children were taken for study) benefited from amplification similar to that expected in children with a comparable degree of sensorineural hearing loss, and that promoted the hearing aid trials for AN/AD patients (Rance et al, 1999). If hearing aids are used, there are some precautions to be considered. According to Hood, (1998), the aids should be fit conservatively, with a low maximum power output. This is important so that the outer hair cells are not exposed to loud noise that could damage them. The fitting is often done by trial and error (Stredler-Brown, 2002). Therefore, an audiologist should monitor cochlear function using OAEs while using hearing aids.

Zeng et al, (1999) in their study of 8 AN subjects, suggested that a special type of speech processing hearing aid is needed for them. In addition to amplifying and making sounds audible, they propose that a hearing aid should compensate for the impaired temporal processing at suprathreshold levels (Zeng et al, 1999). Until the speech processing devices predicted by Zeng et al, (1999) become available, it is recommended that infants or children with AN/AD who have abnormal hearing thresholds (>25 dB HL) be provided with amplification in the context of a comprehensive habilitation program and constantly monitored (Zeng et al, 1999).

1.5. b. Cochlear implants

Experience is growing with cochlear implantation for children with AN/AD, and the outcomes appear to be positive (Shallop et al, 2001; Trautwein et al, 2000; Berlin 2001). Shallop et at, 2001 followed five children with AN/AD who received implants (Shallop et al, 2001). All five children had severe to profound bilateral hearing impairment. None of the children benefited from acoustic amplification used prior to implantation. All of the children in their study demonstrated significant improvements is sound detection, and communication skills after cochlear implantation (Shallop et al, 2001). Three of the five children were reported as being able to use the telephone, which suggests good speech perception abilities without the use of visual cues (Shallop et al, 2001). Shallop et al, (2001) also demonstrated synchronous electrical auditory brainstem responses (EABR) in AN/AD patients post-implant; furthermore, they obtained neural response telemetry (NRT) that measures responses from the auditory nerve using the intracochlear electrode. They report that post-implant AN/AD patients show synchronous NRT responses similar to those observed in non-AN/AD cochlear implant patients (Shallop et al, 2001). These results are consistent with Trautwein et al, (2000) who discussed a single child who demonstrated synchronous responses on (NRT) and performance on behavioral tests comparable to cochlear implant patients without AN/AD (Trautwein et al, 2000). In addition, Berlin (2001), reported that out of 19 children with cochlear implants, 17 of whom were demonstrating good auditory and speech results (Berlin, 2001).

1.5. c. Communication strategies

There are some communication strategies which could be a viable option for AN/AD subjects, such as cued speech, sign language, modification of environment etc.

Cued Speech has been recommended as a habilitation method for children with AN/AD (Berlin, 1999). Cued speech is a method in which the speaker makes distinctive hand shapes near the face and lips to cue phonetic information such as the presence of voicing or to disambiguate speech sounds that cannot be discriminated for lip-reading. Cued speech is a tool to help the child absorb language visually, it is not mutually exclusive, nor is there compelling evidence that children delay the acquisition of spoken language (Hood et al, 2002). It should to be used with either hearing aid or cochlear implant.

The use of sign language has been popular. For the English speaking population it is strongly recommended that children using sign language use an English-based system. Manually Coded English (MCE), Pidgin Signed English (PSE), and Conceptually Accurate Signed English (CASE) are examples of English-based sign systems (Stredler-Brown, 2002). Spoken English can be paired with each one of these systems. It is important to pair signs with spoken English if the family wants to support speech development in the future. It is important that the child should have a foundation in the English language, in order to be prepared to learn spoken English (Stredler-Brown, 2002). Berlin, Hood, Morlet, Rose and Brashears, (2001), studied AN/AD cases for its diagnosis and management options. They recommended use of sign-language, cued speech, baby sign etc for AN/AD subjects.

1.5. d. Spectral/ envelope enhancement

Modern hearing aids have used amplification and compression as the primary methods for accommodating sensorineural hearing loss (SNHL), providing significantly improved speech audibility without causing discomfort. Despite of restoration of speech audibility, it doesn't give 100% speech understanding, especially when there is adverse situation.

Plomp (1978) described two components of SNHL: (a) attenuation and (b) distortion. Hearing aids compensate for the attenuation factor but do not overcome the distortion component of hearing loss. Recently signal processing techniques have been suggested to improve the effective signal-to-noise ratio (SNR), which partially compensates for the distortion factor. One processing technique is the spectral enhancement. When a signal consisting of speech and noise is input to a digital spectral enhancement system, the formants (or spectral peaks) in the speech are selectively amplified, while the spectral valleys between the peaks are either unaffected or attenuated. The peak-to-trough decibel difference is increased through spectral enhancement, which effectively improves the local speech to noise ratio.

Several groups have implemented algorithms for increasing spectral contrast, but the results have been mixed (Baer, Moore, & Thomas , 1993; Franck, Sidonne van Kreveld-Bos, Dreschler & Verschurre, 1999). Baer et al (1993) studied 11 subjects with hearing loss of cochlear origin and found that moderate degree of spectral enhancement benefits in quality and speech intelligibility, whereas greater degree of spectral enhancement has deleterious effect on speech quality and intelligibility.

In the study by Franck et al (1999), the separate and combined effects on speech perception of compensation of the reduced dynamic range by compression and compensation of the reduced frequency resolution by spectral enhancement was investigated. The study was designed to compare the effects of signal processing on monosyllabic consonant–vowel–consonant words for hearing-impaired listeners in conditions of quiet, fluctuating noise, and continuous noise. Speech perception of spectrally enhanced speech was compared with unprocessed speech. They found better scores for vowels in spectrally enhanced signals while less for consonants.The reason for the lack of its success is not clear, but it may be that spectral enhancement is a theoretically flawed strategy or that the implementation of spectral enhancement may be that spectral enhancement has been done ineffectively.

Numerous studies of psychophysical tasks have shown difference between normal and impaired listeners in spectral-resolution abilities. These investigations provide theoretical motivation for the idea that enhancing peaks in a complex spectrum may be beneficial in restoring speech intelligibility for listeners with SNHL. Leek, Dorman, and Summerfield (1987) showed that hearing-impaired listeners required greater relative intensity of spectral peaks when identifying simulated two formant vowels. They observed that normal hearing listeners identified vowels with 90 % accuracy when the peak-to-trough ratio reached 6 dB or greater. 6 hearing impaired listeners, however, identified vowels about 70% correctly with a peak-to-trough ratio of 8 dB. Hearing impaired listeners therefore required a grater peak-to-trough ratio to accurately identify vowels accurately.

Physiological studies also are relevant and offer evidence in support of the potential benefits of spectral enhancement. Investigators measuring the neural representation of speech have shown that decreased spectral causes inaccurate representation of complex signal in the auditory nerve and that increasing spectral contrast in complex signals can compensate for that loss (Geisler, 1989;Miller, Schilling, Frank & Young, 1997). Models of cochlear function also suggested that SNHL might significantly affect the frequency-specific neural representation of speech sounds (e.g., Geisler, 1989). Measurements made on acoustically traumatized cats have confirmed these models. Millet et al. 1997 measured the representation of a synthesized vowel in normal and acoustically traumatized cat's auditory nerve fibers and found strong synchronization places of stimulation corresponding to formant peaks for cats with normal neurons. Neural synchrony was not seen in traumatized cats even when the speech levels were high enough to overcome the loss of sensitivity. Miller et al. 1999 subsequently observed that acoustic trauma eliminated the ability to discriminate F2 frequency via discharge-rate-patterns. Thus, Miller et al 1997 have demonstrated that reduced frequency selectivity associated with SNHL degrades the neural representation of complex signals and that amplification alone is insufficient to overcome processing limitations in damaged cochlea. Some authors argued that such a strategy won't be helpful, whereas some have supported the strategy. Speech perception tests of spectral

enhancement algorithms however have shown outcomes that ranged from somewhat successful to unsuccessful. Simpson, Moore and Glasberg (1990) tested nine listeners with SNHL on speech-in-noise tests. They found that spectral enhancement improved identification of words and sentences.

Till date majority of the study have focuses on SNHL individuals, very few study has been done with subjects having AN/AD. Individuals with auditory dys-synchrony have been reported to have temporal deficits, and hence have difficulty recognizing short signals effectively. Therefore, there is a need to study whether spectral enhancement of signal improves speech recognition in such individual or not. It will be interesting and relevant to study the effect, if any, both in terms of subjective perceptual measures (SIS) as well as objective measures, cortical potential (LLR).Hence there is the need for the present study.

METHOD

The present study was conducted to see whether the spectral enhancement of speech stimulus in individuals with auditory neuropathy/dys-synchrony improves their speech perception ability or not. This was done both using behavioral measures as well as electrophysiolgical tests. The study was done in two parts.

Part I: preparation of the speech stimuli

11 VCV syllables were recorded in an adult male voice using PRATT software. All the recording was carried in a sound treated room. The stimuli chosen were /aba/, acha/, ada/, /adha/, /aga/, /aka/, /ala/, /ama/, /ana/, /apa/ and /ara/. The consonant chosen represents different place and manner of articulation. Stimuli were restricted to less number due to time constraint issue in the testing.

These stimuli were further enhanced using PRATT software, with 16 bits sampling rate (as it gives better waveform) and 22050 sampling frequency and 2to 32 Hz modulation frequency. The stimuli were later mixed up with speech noise in preset proportion to make it 10 dB SNR condition, using MATLAB software version 6. The software calculates the root mean square for signal and noise and then does the mixing.

Once the stimuli preparation was over, the stimuli were copied over a CD, for testing, and of 11 stimuli one stimulus /ada/ was used for the LLR recording (objective testing). In this only /da/ portion was retained, to load on to the instrument (IHS) as the instrument takes stimuli up to 250 ms. This editing work was done using PRATT software and later the wave file was converted into stimulus file using waveform converter in the instrument itself. Unenhanced /da/ and enhanced /da/ were both loaded in the instrument as stimulus file for objective recording.

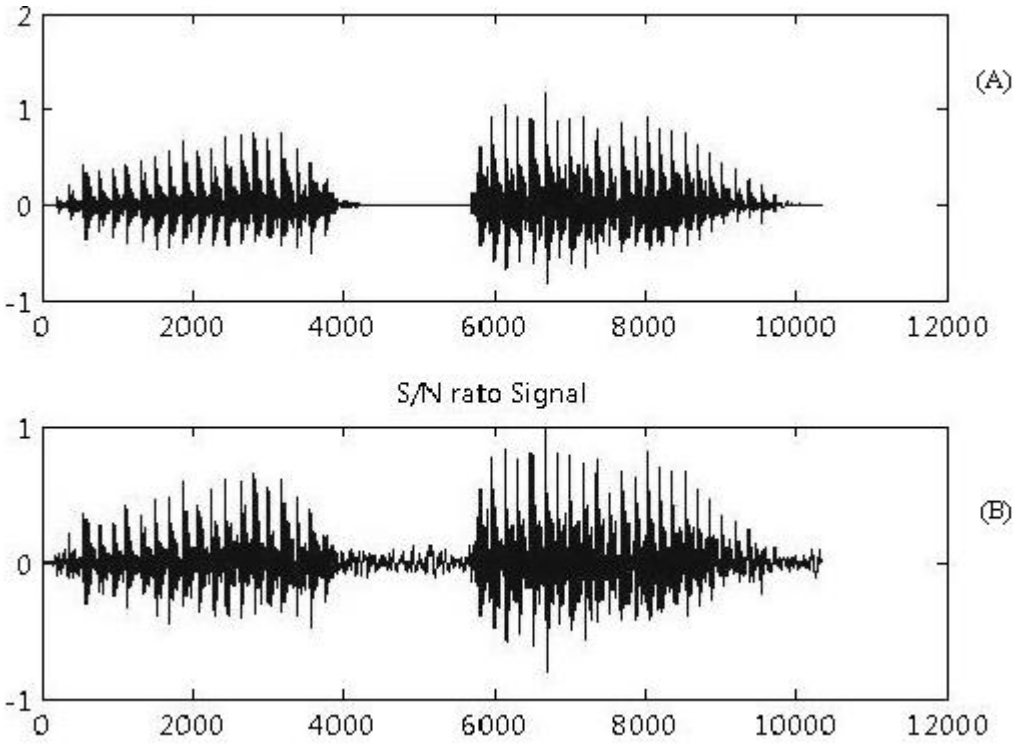


Figure 1 : Showing (A) Non enhanced /ada/ and (B) Enhanced /ada/, which was used for testing.

Justification for the selection of /da/ stimulus.

The acoustic characteristics of speech give cues enabling identification of both the phonetic content of message as well as information about who is speaking. Linguistic information give meaning of message while paralingutic information gives emotion etc. acoustically these cues are conveyed by "source" and "filter" characteristics.

A visual analysis of the /da/ stimulus waveform and its corresponding brain stem response reveals several similarities. Also /da/ stimulus has good onset and sustained neural response pattern (Johnson, Nicol & Kraus ., 2005). Till date most of the studies have incorporated the same stimulus because of its transient and periodicity nature to represent it neutrally.

Part II: Testing the subjects with AN/AD and age/gender matched subjects with normal hearing.

Subjects:

Subjects were divided in two groups; experimental group and control group.

Experimental group: Nine subjects (18 ears) diagnosed as having auditory dys-synchrony were taken for the study. Inclusion criteria were as follows:

- Age ranging from 10 to 26 years old with a mean age of .19.77 yrs
- Normal to moderately severe hearing loss (based on the pure tone average of 500 Hz, 1 kHz & 2 kHz).
- Speech identification Score disproportionate to pure tone average of 500 Hz, 1 kHz & 2 kHz.
- "A" type tympanogram indicating normal middle ear functioning.
- Absent of both ipsilateral and contralateral acoustic reflexes.
- No history of any middle ear problems, and no misarticulations.
- Presence of Otoacoustic emissions.
- Absence of auditory brainstem responses.

Below table shows the details of the subjects with AN/AD.

subjects	Age(yrs) / gender	Degree of loss (dB)	Configuration of loss	SIS (%)	Acoustic reflexes	Otoacoustic emissions	ABR
S1-R	10/ male	35	Sloping	Poor	Absent	Present*	absent
S1-L	10/male	36.66	Sloping	Poor	Absent	Present*	absent
S2-R	14/female	58.3	Saucer shaped	20	Absent	Absent	Absent
S2-L	14/female	41.6	Saucer shaped	30	Absent	Absent	Absent
S3-R	25/male	28.3	Rising	45	Absent	Present	Absent
S3-L	25/male	18.3	Rising	55	Absent	Present	Absent
S4-R	19/female	28.3	Flat	70	Absent	Present	Absent
S4-L	19/female	18.3	Flat	90	Absent	Present	Absent
S5-R	21/male	38.3	Rising	75	Absent	Present*	Absent
S5-L	21/male	36.6	Rising	75	Absent	Present*	Absent
S6-R	19/male	28.33	Rising	60	Absent	Present	Absent
S6-L	19/male	25	Flat	65	Absent	Present	absent
S7-R	22/male	31.6	Flat	50	Absent	Present	Absent
S7-L	22/male	26.5	Flat	55	Absent	Present	absent
S8-R	26/female	55	Flat	25	Absent	absent	Absent
S8-L	26/female	60	Flat	35	Absent	absent	Absent
S9-R	22/female	11.6	Flat	50	Absent	Present	absent
S9-L	22/female	3.3	Flat	60	Absent	Present	absent

*Table 1: showing the details of subjects with AN/AD. * denotes the subjects where DPOAE was done instead of TEOAE.*

Control Group: Control group consisted of 9 age and gender matched subjects with normal hearing sensitivity. The inclusion criteria for the control group were as follows:

- Hearing threshold <15 dB HL from 250 Hz to 8 kHz , at octaves and interoctaves.
- Good speech identification score of more than 90%.
- "A" type tympanogram with present Ipsilateral and Contralateral reflexes, and no history of middle ear problem.
- Presence of OAEs.
- Presence of ABR response.
- No history/presence of any neurological deficits.

Instrumentation:

- A calibrated (ANSI S3.6-1996), two channel clinical audiometer OB922 with TDH-39 headphones housed in Mx-41/AR ear cushions with audio cups were used for puretone audiometry. Radioear B-71 bone vibrator was used for measuring bone conduction threshold.
- A calibrated middle ear analyzer, (GSI tympstar) using 226 Hz probe tone was used for tympanometry and reflexometry.
- Oto acoustic emissions were recorded using either Intelligent Hearing System Smart OAE windows USB version 2.62 or otodynamics ILO V6 OAE instrument.
- Intelligent Hearing System (Smart EP windows USB version 3.91) evoked potential system with insert ear ER-3A receiver was used for recording auditory brainstem responses and late latency responses.
- Perceptual testing for the speech reception was carried out with the help of CD which was played through Pentium IV computer, routed through OB922 audiometer with head phone output.
- Late Latency Response for speech stimulus was recorded using Intelligent Hearing System (Smart EP windows USB version 3.91) evoked potential system.

Test environment:

All the audiological tests were carried out in an acoustically treated room (as per ANSI, 1996) with adequate illumination.

Procedure:

- Pure tone audiometry was done from 250 Hz to 8 kHz at octaves and interoctaves for air conduction stimuli and from 250 Hz to 4 kHz for bone conduction stimuli. All the testing was done using Modified Hughson-Westlake Method (Carhart & Jerger, 1959). Speech audiometry was also done using modified Olsen –Tillman method (1973). Inbuilt talk back system was used for speech audiometry.
- Tympanometry and reflexometry was done to check to rule out middle ear pathology. 226 Hz was the probe frequency and 85 dB SPL was the level used. Reflex eliciting signal was at 500 Hz, 1000 Hz and 2000 Hz. It was checked for ipsilateral and contralateral mode of stimulation.
- Otoacoustic emissions evoked by clicks presented at 85 dBpeSPL for the linear clicks were recorded. The probe with a tip was positioned in the external ear canal and was adjusted to give flat stimulus spectrum across the frequency range. The response was acquired using the linear averaging method. The two averaged TEOAE waveforms of each memory buffer composed of 256 accepted click trains, were automatically cross-correlated and used to determine the reproducibility of the measured TEOAEs by the software. Responses were accepted when the reproducibility was 70% or greater. A total of two responses were recorded to ensure the stability of the response. A minimum of one minute gap was given between any two recordings to reduce the influence of the one recording over another recording. Care was taken to ensure that the position of probe was not altered.
- Auditory brainstem responses were recorded from one channel using ER-3A insert receiver. The site of electrode placement was prepared with skin preparation gel. Silver chloride disc electrode was used with a conducting gel.

The following protocol was used to record auditory brainstem responses.

Table2. Parameters for recording ABR

Stimulus parameter	Intensity	90 dB nHL
	Repetition rate	11.1/sec
	Polarity	Rarefaction
	Duration	100 u sec
Acquisition Pararmeters	Analysis time	10 m sec
	Filter setting	100 - 3000 Hz
	Electrode montage	Fpz : Non Inverting (+ve) Test ear : Inverting (-ve) Non test ear: Ground
	Notch Filter	On
	Artifact rejection	40 u V

.

Perceptual testing:

- The stimuli were presented to the Clients from computer routed through audiometer via headphone. The subject's task was to repeat the stimuli presented to them.
- Stimuli were presented at 40 dB SL referenced to pure tone average. The responses given by client were marked correct or wrong for all the conditions.
- All together 11 stimuli were presented in 4 different context as: Non enhanced, Enhanced, 10 dB SNR Non Enhanced and 10 dB SNR Enhanced condition. Total presentations made were 44 times. List was randomized to overcome the order effect. Also the ears tested were randomly chosen to overcome the ear effect.
- For the entire testing, client's verbal response was taken, and it was made clear that none of them had misarticulations.
- Stimulus presentation was done in random order to avoid order and sequence effect.

Objective testing:

Clients were instructed to seat themselves comfortably in the reclining chair for the LLR recording. The site for the electrode placement (test ear mastoid, forehead & Nontest ear mastoid) was cleaned using Skin prep gel and cotton. The silver disc electrodes were placed using conducting paste, and plaster was used to make it secure at the required site. Clients were instructed to be quiet throughout the testing. Each recording was done 3 times using the same protocol to ensure the reproducibility of the waveform. Whole testing took almost 60 minutes including client preparation to marking of the peaks. Consent was taken from all the clients in written form before the testing. The following protocol was used to record late latency responses.

Table 3: showing parameter for LLR recording

Stimulus Parameters	Speech stimulus	/da/ Enhanced & Non enhanced
	Duration	230 ms
	Level	90 dB nHL.
	Polarity	alternating
	Mode of presentation	Ipsilateral
	Repetition rate	1.1/s
Acquisition Parameters	Transducer	ER-3A insert receiver
	Analysis time	0-500 msec with -50 pre stimulus period
	Filter setting & Gain	1-30 Hz, 50,000
	Electrode placement	Inverting(-ve): Test ear Noninverting(+ve): FPz Ground Non Test ear.
	Sweeps, Artifact rejection	150 sweeps & 40 uV
	Electrode Impedance	< 10 kHz
	Inter Electrode Impedance	< 3 kHz.

Latency and amplitude of P1, N1, P2 & N2 peaks were measured for both enhanced as well as for non enhanced speech stimulus. The absolute amplitude was measured for all the waveforms for both normal hearing subjects as well as for individuals with auditory neuropathy/dys-synchrony. Also the morphology of the waveforms was rated on a 3 point rating scale; as Good, Average or poor. Two audiologists independently analyzed the waveform.

The following data were generated for analysis:

1) Speech identification score results in non enhanced and enhanced condition in quiet as well as in 10 dB SNR condition for subjects with AN/AD and normal hearing.
2) LLR latencies in non enhanced and enhanced condition in quiet condition for subjects with AN/AD and normal hearing.
3) LLR amplitude LLR latencies in non enhanced and enhanced condition in quiet condition for subjects with AN/AD and normal hearing
4) Morphology status in non enhanced and enhanced condition in quiet condition for subjects with AN/AD and normal hearing.

RESULTS AND DISCUSSION

The present study investigated the effect of envelope enhancement of speech stimuli in the perception of speech in individuals with AN/AD (experimental group) and individual with normal hearing (control group). This was evaluated in two ways, namely perceptual measures (asking the subject to repeat the speech sound which is heard) and objective measures using late latency responses (LLR). The effect of enhancement of speech was studied electrophysiologically. The amplitude and latency of late latency potential were recorded in non enhanced and enhanced condition for /da/ stimulus, for normal hearing group as well as for the individuals with AN/AD. All the statistical analysis was done using statistical package for social sciences (SPSS 15) version. The independent variables were the speech stimuli in non enhanced and enhanced condition, age and sex of the subjects in both groups. The dependent variables were the latencies and amplitudes obtained for each peak in the LLR measure and, the responses given by the subjects for perceptual measure.

The following analyses were carried out between and within the group:

- Descriptive analysis for all the parameters
- Mixed ANOVA, to see for the main effect of enhancement (irrespective of group), group effect (experimental Vs control group) and interaction effect of variables.
- Independent sample" t" test, to compare the data across the two groups for the non enhanced and enhanced conditions.
- Paired sample "t" test, to see the significance difference within the groups for the non enhanced and enhanced conditions.

Latency and Amplitude Measures of Late latency responses

Latencies were measured for all the four peaks of LLR i.e P1, N1, P2, N2 in enhanced and non enhanced condition. Late latency responses were present in all the normal hearing individuals as well in all individuals with AN/AD. Overall mean value for the latencies for the enhanced stimulus was less compared to the non enhanced signal for all the four peaks and across two groups as shown in Table 1. Also the standard deviation (given in parenthesis) was more in AN/AD group than in the normal hearing group

Table 1: Mean and standard deviation of latencies of LLR peaks in non enhanced (NEL) and enhanced (EL) conditions for experimental (AN/AD) and control (Normal) group.

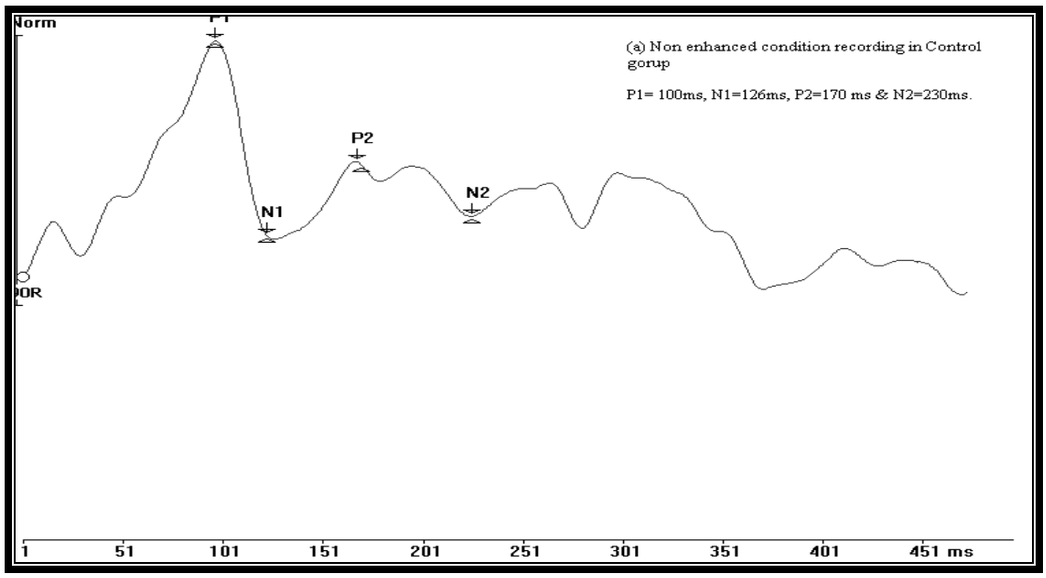
Groups	**Peak Latencies (msec)**							
	P1		N1		P2		N2	
	NEL	EL	NEL	EL	NEL	EL	NEL	EL
Experimental	71.11 (27.01)	67.27 (23.86)	121.66 (36.25)	123.50 (41.54)	185.00 (42.43)	183.50 (45.70)	235.33 (47.34)	232.17 (53.89)
Control	72.11 (10.30)	70.11 (13.41)	129.61 (16.24)	123.00 (17.20)	180.22 (28.21)	170.72 (31.07)	228.44 (27.86)	208.83 (34.19)

Absolute amplitude for all the peaks (P1, N1, P2 & N2) were measured in non enhanced and enhanced condition for both the groups. Mean and standard deviation for amplitude of different peaks in non enhanced and enhanced stimulus within each group is mentioned in the Table 2. It was found that within the enhanced condition there was increase in the amplitude in both the groups. Again, there was more standard deviations for the AN/AD group was larger than that of the normal hearing group.

Table 2: Absolute amplitude for the peaks in LLR for both conditions (NEA & EA) in both groups. Values in parenthesis are standard deviation.

Groups	**Peak Amplitudes (uV)**							
	P1		N1		P2		N2	
	NEA	EA	NEA	EA	NEA	EA	NEA	EA
Experimental	1.23 (0.73)	1.73 (0.76)	-3.57 (2.26)	-3.92 (2.03)	1.74 (1.24)	1.99 (1.30)	-3.41 (1.66)	-2.67 (1.97)
Control	1.37 (1.01)	1.40 (0.67)	-4.47 (1.59)	-3.84 (1.10)	2.67 (1.64)	2.02 (1.17)	-3.86 (2.04)	-2.76 (2.88)

LLR sample recording of the experimental and control group showing the change in latency and amplitude for the non enhanced and enhanced conditions are shown in Figure 1 (a) & (b) and Figure 2 (a) & (b) respectively.



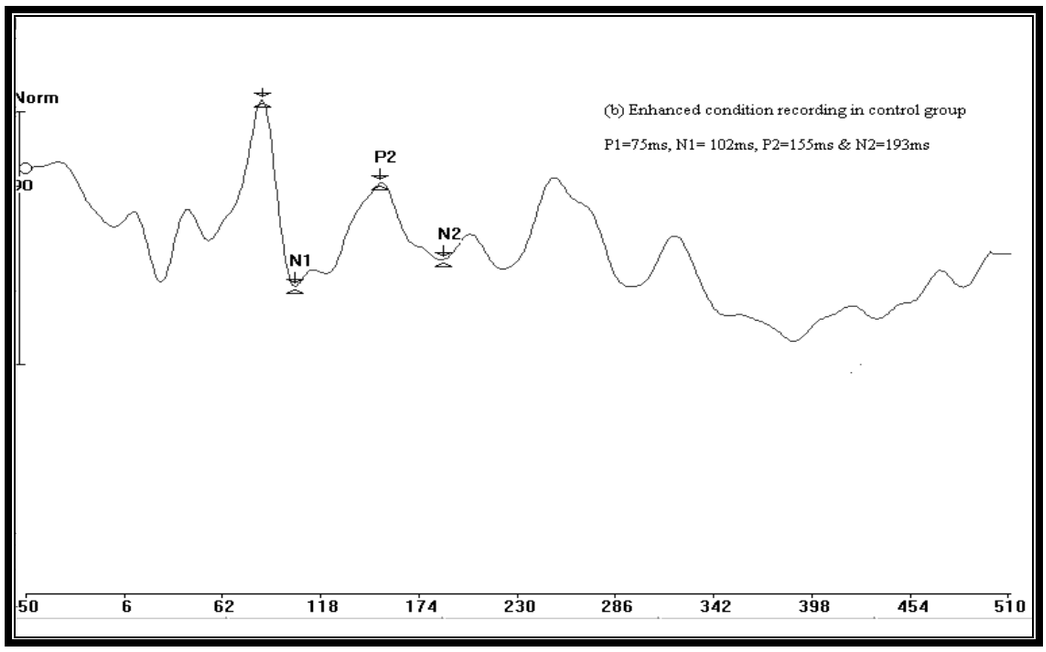


Figure1 (a) & (b): LLR waveforms recordings for (a) non enhanced and (b) enhanced /da/ stimulus in control group (normal hearing).

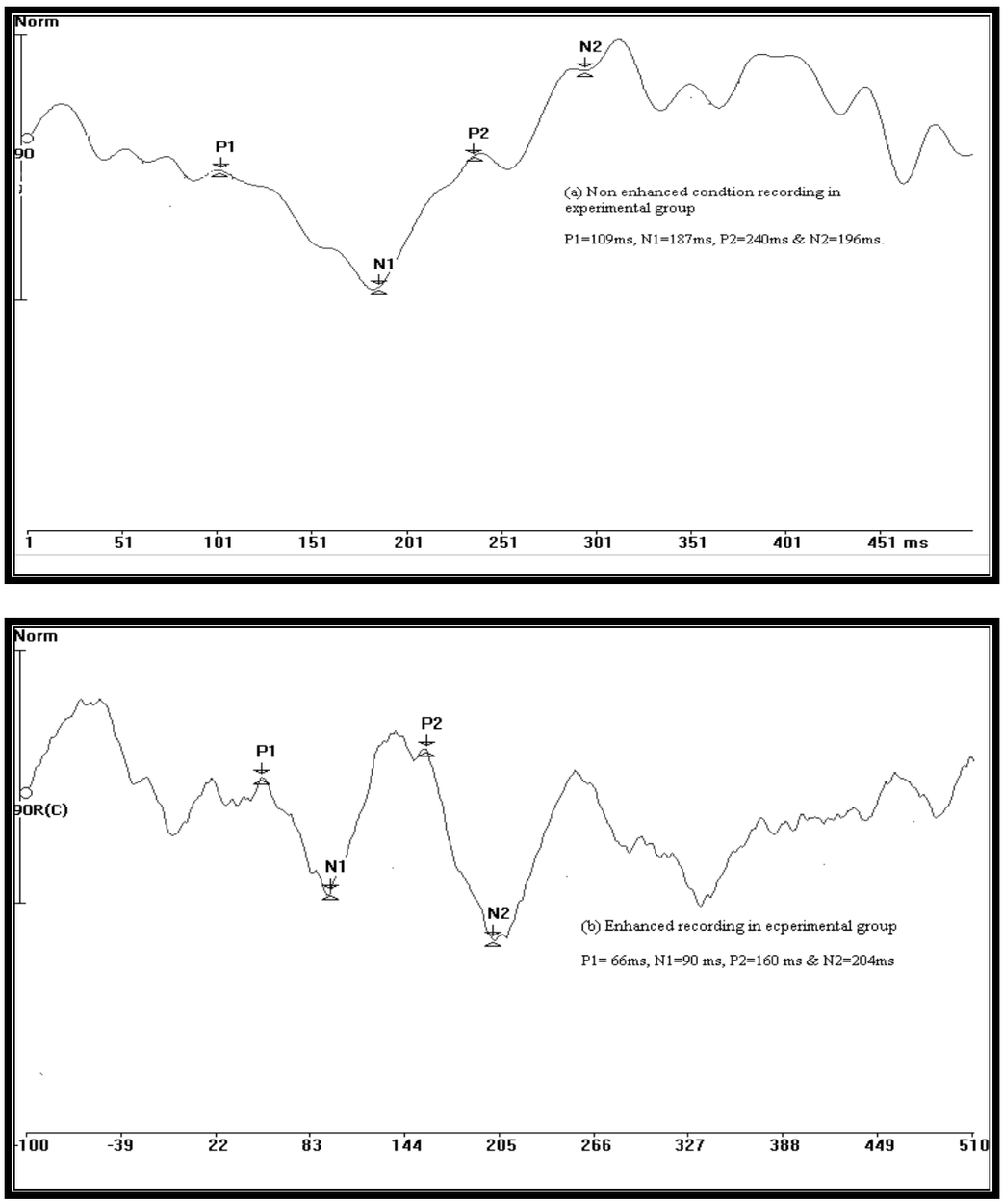


Figure2 (a) & (b): LLR waveform recorded for (a) non enhanced and (b) enhanced /da/ stimulus in experimental group (AN/AD).

Mixed ANOVA was done to find out (i) main effect of enhancement i.e., the difference between non enhanced and enhanced conditions when both the groups were combined, (ii) main effect of group i.e., the effect of group AN/AD and normal group when non enhanced and enhanced are compared and (iii) Interaction effect of enhancement and group for both latency and amplitude of LLR.

Table 3: Mixed ANOVA results for each parameter of peaks in terms of F (1, 34) value. . Shaded box shows significant results ($p < 0.05$)

F value for latency measures in LLR peaks				
	P1	N1	P2	N2
Main effect of enhancement	1.717	0.585	1.776	6.174*
Main effect of group.	0.094	0.154	0.551	1.677
Interaction of enhancement & group	0.170	1.826	0.940	1.505
F value for amplitude measures in LLR peaks				
Main effect of enhancement	2.898	0.191	0.562	4.837*
Main effect of group.	0.183	0.656	1.779	0.203
Interaction of enhancement & group	2.264	2.196	2.881	0.171

From Table 3 it can be seen that the latency as well as amplitude of N2 was significantly different for the effect of enhancement when both the group were combined. There were positive results of enhancement meaning to say enhancement did decrease the latency and increased the amplitude for all the peaks but it was statistically significant for N2 peak. However there was no significant difference in terms of latency and amplitude of other peaks i.e., P1, N1 and P2 between non enhanced and enhanced conditions. There was no group effect or interaction effect (between enhancement & group) found for any of the peaks parameter (latency & amplitude).

Once the overall results is calculated (main effect), further analysis was done to see the significant difference if any for the effect of enhancement in both the groups and across the groups.

Independent sample *t* test was done to find out whether there is any significant difference between the groups in terms of latency and amplitude of P1, N1, P2 and N2 considering the non enhanced and enhanced conditions separately. The results are shown in Table 4.It was found that there is no significant difference for any peak parameters in either of the conditions between groups ($p>0.05$).

Table 4 shows the "t" value for latency and amplitude parameters in both conditions when comparison was made between the groups.(Independent "t" test result)

"t" value for LLR peaks in latency measures between both groups.				
	P1	N1	P2	N2
Non enhanced	0.147	0.848	0.398	0.532
Enhanced	0.439	0.047	0.981	1.539
"t" value for LLR peaks in amplitude measures between both groups.				
Non enhanced	0.468	1.375	1.930	0.713
Enhanced	1.363	0.155	0.872	0.117

Paired sample *t t*est was done to find out whether there is any significant difference within group when compared between non enhanced and enhanced conditions.

Table 5: Paired t test results in AN/AD (experimental group) between non enhanced and enhanced conditions for the peaks of LLR parameters in terms of t (17) value. Shaded box represents significant results (p < 0.05 level).

"t" values for latency of LLR peaks				
	P1	N1	P2	N2
NEL Vs EL	1.207	0.327	0.214	0.706
"t" values for amplitude of LLR peaks				
NEA Vs EA	2.596	0.578	0.755	1.428

It can be seen from table 5 that there is significant difference for P1 amplitude in non enhanced condition when compared with enhanced condition, but was not seen for any other peaks i.e. N1, P2 and N2. Though there was increase in amplitude for all the peaks, it was statistically significant only for peak P1 in AN/AD group. Also there was no statistically significant difference in latency parameter for any of the peaks when comparison was made though there was increase in latency.

Table 6: Paired t test results in normal's (control group) between non enhanced and enhanced conditions for the peaks of LLR parameters in terms of t (17) value. Shaded box represents significant results (p < 0.05 level).

Latency				
	P1	N1	P2	N2
NEL Vs EL	0.641	2.383	2.177	3.808
Amplitude				
NEA Vs EA	0.126	2.234	1.571	1.674

From table 6 it can be seen that the latencies for the two conditions were significantly different for peaks N1, P2 and N2 ($p < 0.05$) .However, it was not so for P1 latency. For amplitude there was significant difference in N1 peak ($p < 0.05$) and there was no significant difference for peaks P1, P2 and N2.

Late latency responses was recordable in all the subjects with AN/AD and also in all the normal hearing individuals. Earlier studies have also reported the presence of late latency responses in individuals with AN/AD (Starr et al.1996; Hood, 1999; Kraus et al., 2000; Rance et al., 2002; Pearce, Golding & Dillon, 2007) and also in normal hearing subjects(Kurtzberg, Hilbert, Kreuzer, and Vaughan, 1984). The late latency responses may be present in the individuals with AN/AD due to the fact that the disruption of peripheral function which often leads to absence of ABRs, does not necessarily affect the later responses as these are not reliant on timing as the earlier evoked responses (Hood, 1998, Rapin & Gravel, 2003).

However the hit rate of LLR in the present study is higher than that reported in literature. In the present study LLR was present in all the AN/AD subjects. Rance et al. (2002) reported the presence of LLR in 50% of the AN/AD individuals. The difference may be due to the difference between the subject selection criteria in the two studies. The subjects in the study by Rance et al (2002) were aged between 3.4 years to 9 years, who were born prematurely, whereas in the present study all the subjects were aged above 10 years, with no history of prematurity. Ponton et al., (1996); Ponton et al., (2000); Ponton et al., (2002) and Wunderlich & Cone-Wesson, (2006) report about the absence of LLR due to maturational factors. It is possible that the auditory development was still underway in the subjects of Rance et al (2002) study and hence LLR was absent.

The results of the present study reveal that there is no difference in terms of latency of LLR in normals and the AN/AD group. This is comparable to the previous study by Starr et al. (2003). Starr et al also reported no significant latency differences in LLR between normals and the AN/AD group at higher intensities whereas there was a significant difference in latency at the lower intensities. In present study, a high intensity (90 dB nHL) presentation was used to record LLR. Starr et al.(2003) reports "the no significant difference in terms of latency between the two groups at higher intensities may be due to the fact that in AN there may be a form of `central recruitment' which may accompany hearing impairment at higher intensities. Cody et al. (1968) described an abnormal growth of N100 amplitude as a function of signal intensity in individuals with `sensorineural' hearing loss, and speculated as to its relationship to abnormal growth of loudness often encountered in such patients. For AN subjects, however psychoacoustic measures of intensity processes are normal in contrast to their marked abnormality of temporal processes (Zeng et al., 2000). The mechanisms underlying altered cortical excitability in AN may reside within the cortex. An animal model of AN showing increased excitability of auditory cortex did not have a corresponding excitability change of inferior colliculus (Salvi et al., 1999). The abnormal excitability of auditory cortex in AN may be likened to the central excitability changes encountered in disorders of other sensory systems following deafferentation".

The mean latencies for the LLR for non enhanced signal in the present study for the AN/AD group was 71.11 msec for P1, 121.66 msec for N1, 185 msec for P2 and 235.33 msec for N2. The latencies for LLR are lesser than reported by Rance et al (2002). Rance et al (2002) reported 140.2 msec for P1, 227.7 msec for N1 and 320.9 msec for P2. The difference in latencies may be attributable to the difference in the subject's selection criteria and the stimulus used between the two studies. As mentioned earlier in the study of Rance et al (2002) the subjects had the history of prematurity but there was no such history of premature birth in the subjects for the present study. The stimulus used by Rance et al (2002) was 440 Hz tone burst and /daed/ whereas in the present study the speech stimulus /da/ was used. The latencies for the normal hearing group in the present study was 72.11msec for P1, 129.61msec for N1, 180.22msec for P2 and 228.44msec for N2 respectively. However the mean latencies in the study of Rance et al (2002) for normal hearing group was 100msec for P1, 200msec for N1 and 301.5 msec for P2, while Cunnigham, Nicol, Zecker and Kraus (2000) reported latencies for the different age groups for a synthetic syllable (CV) as follows :

Table 6 : Peak Latencies (msec) in different age groups for synthetic CV syllable /GA/.

(Cunningham et al 2000)

Age groups in years	Peak Latencies in msec		
	P1	N1	N2
11-12	88	137	228
13-15	80	120	226
19-27	64	122	203
55-78	68	121	198

Again these differences between the present study and the study reported could be due to the wide range of subjects (10 yrs to 26 yrs) which were age and gender matched when selecting for the control group. It could also be due to the stimuli used for the testing.

Cunnigham, Nicol, King, Zecker and Kraus 2002 reported that “stimulus modifications that improve the temporal precision of individual neural firing patterns can enhance neural synchrony, across a population of cortical neuron, leading to large amplitude aggregating neural response”. So if there is large amplitude due to aggregation of neural response there has to be reduced latencies, as it was seen in the present study for both the groups. However, when latency was compared for non enhanced and enhanced stimulus there was no significant difference in AN/AD group for any of the peaks recorded, while there was significant difference in normal group for N1, P2 and N2 peaks. This difference in the presence of the significant enhancement effect of the peaks in normal’s could be due to the preserved synchrony which was absent for the AN/AD group. Though the LLR was present in the AN/AD group, the enhanced condition did result in betterment of the latency but it was not significant ($p > 0.05$). it is possible that the reduced synchrony in subjects with AN/AD did not facilitate improvement in latency or amplitude. It is also possible that the amount of enhancement was not adequate to bring about such a change.

In the present study mean absolute amplitude for the AN/AD group was 1.23 uV for P1, -3.57uV for N1, 1.74uV for P2 and -3.41uV for N2. Rance et al (2002) reported 4.1 uV for P1N1 and 3.4 uV for N1P2. Cunnigham et al (2000) gives the baseline amplitude as follows:

Table 7: Peak amplitude (uV) in different age groups for synthetic CV syllable/GA/.

(Cunnigham et al 2000)

Group age years	Peaks (uV)		
	P1	N1	N2
11-12	1.5	0.8	2.0
13-15	1.0	0.4	1.8
19-27	0.8	0.8	1.0
55-78	1.3	1.1	0.8

The difference in amplitude between the present study and the reported studies could be due to the subject selection criteria, the stimulus used the method of marking amplitude. Relative amplitude was considered by Rance et al (2002) whereas absolute amplitude was considered by Cunnigham et al (2000) study as done in the present study.

When amplitude was compared between non enhanced and enhanced conditions there was increment in the enhanced condition for both AN/AD group as well as the Normal group. The reason for this was explained earlier as per the study by Cunnigham et al 2000 which is due to better synchrony. In the present study there was significant difference between non enhanced and enhanced stimuli for P1 ($p < 0.05$) in AN/AD group, whereas in normal group it was N1 ($p < 0.05$). This result may be due to the difference in the feature of synchrony preserved i.e normal hearing group had better synchrony than the AN/AD group, which could have lead to better amplitude. The significant difference is seen only for N1 peak, is not explainable, more research is needed to discuss for the same.

LLR waveform morphology analysis was done by two judges (audiologist) on 3 point rating scale namely, good, average and poor. It was found that enhancement gave poorer waveform compared to non enhanced stimulus recording in 50 % of the subjects and for the rest 50 % it was similar morphology

irrespective of using enhanced or non enhanced stimuli. The results are inconclusive to say regarding the changes in the waveform morphology due to enhancement.

Results on perceptual testing: The experimental group and control group were tested with non enhanced and enhanced stimuli in quiet and in 10dB SNR condition. As mentioned in the method, 11 stimuli were presented through headphones in /aCa/ context. Scores depicted in table 8 are correct responses for 11x 2 (right ear & left ear) stimuli.

Table 8: Scores and total scores for AN/AD subjects for non enhanced (NE) and enhanced stimuli (E) in quiet and in 10 dB SNR conditions

Subjects	Test conditions			
	Quite		10 dB SNR	
	Non enhanced	Enhanced	Non enhanced	Enhanced
S1	3	5	0	5
S2	7	13	2	8
S3	13	13	10	12
S4	16	19	13	16
S5	13	13	5	6
S6	18	10	16	16
S7	7	12	4	9
S8	16	19	15	17
S9	13	15	15	16
Mean & its (%)	11.77 (53.55%)	13.22 (60 %)	8.8 (40.36 %)	11.66 (53%)
Range	3-18	5-19	0-16	5-16

Total scores shows that there is improvement in scores in quiet from 53.55 % to 60 % between non enhanced and enhanced condition whereas in 10 dB SNR condition it was from 40.36 % to 53% between non enhanced and enhanced condition. The range calculated for non enhanced and enhanced conditions clearly shows that in enhanced condition the range has reduced in both quiet as well as 10 dB SNR condition. Perceptual testing results revealed that there is less improvement in the quite condition i.e., 6.45 % whereas in 10 dB SNR condition it was 12.64 % which is almost double than in quiet condition.

Table 9: Scores and total score for non enhanced and enhanced stimuli in quiet and in 10 dB SNR conditions in control group

SUBJECTS	Quite		10 dB SNR	
	Non enhanced	Enhanced	Non enhanced	Enhanced
S1	20	21	20	20
S2	20	19	22	19
S3	22	22	22	22
S4	22	22	22	22
S5	22	22	22	22
S6	22	22	19	22
S7	22	22	21	22
S8	21	22	22	22
S9	22	22	20	22
Mean % its (%)	21.44 (97.47%)	21.55 (97.97%)	21.11 (95.95%)	21.44 (97.47 %)
Range	20-22	19-22	19-22	19-22

Total scores shows that there is improvement in scores in quiet from 97.47 % to 97.97 % between non enhanced and enhanced condition. In 10 dB SNR it is from 95.95 % to 97.47 % between non enhanced and enhanced condition.. In the normal group there was more improvement in the 10 dB SNR condition i.e., 1.52% than 0.50% in quiet, but it shows that there is marginal improvement.

Study by Digiovanni, Nelson & Schlauch (2005) reported that the improvement in detection and frequency discrimination of narrow band signal, in presence of broad band noise, after spectral enhancement was more in normal group than in the sensory neural hearing loss group, which was explained saying that normal have broader auditory filters, which would have helped them to attain better scores.

Thus, this study gives the idea that enhancement of signal does improve the latency and amplitude of LLR waveform and also in terms of speech reception gives better score in both the groups, but the effect found was not significant. Therefore, more in-depth study is required to study the effect of same taking various disorder with more number of cases to give a better picture of the enhancement.

Summary and conclusions

Auditory neuropathy/dys-synchrony is a disorder characterized by the impairment of the peripheral auditory function with the preservation of outer hair cell integrity (Starr, Sininger, Picton, Hood & Berlin, 1996; Berlin et al., 1998; Berlin, 1999). It is a known fact that these individuals have problem with speech discrimination. Speech identification scores of subjects with AN/AD are widely documented to be disproportionate to their degree of hearing loss. To overcome this difficulty many management options have been advocated from sign language to cochlear implant, but none of them have given 100% success.

Research is underway on the management issues of subjects having AN/AD. Enhancement of the cues in the speech is reported to make speech identification better. This has been tested with subjects having normal hearing and cochlear hearing loss, wherein improvement in speech identification has been reported.

AN/AD group have been reported to have temporal deficits, and hence have difficulty in recognizing short signals. Therefore, present study was carried out to see whether enhancing the speech temporal envelope will improve the speech perception or not. This was done both objectively (LLR) and subjectively (SIS).

11 VCV syllables were recorded using an adult male voice by using PRAAT software. These syllables were further mixed up in preset proportion of speech noise to make it in 10dB SNR condition. In perceptual testing the subjects task was to repeat the stimuli is heard, in both for quiet as well as 10 dB SNR condition for non enhanced and enhanced stimuli, whereas objective recording was done using only one stimulus /da/, where latency, amplitude and morphology of LLR were recorded in quiet condition for non enhanced and enhanced

stimulus. The testing was done for AN/AD subjects as well as age and gender matched subjects with normal hearing.

Perceptual testing was done using OB 922 clinical audiometer and a Pentium IV computer to route the recorded speech stimuli. This testing was done using TDH-39 headphones at 40 dB SL to the pure tone average. In objective recording was done using Intelligent Hearing System (Smart EP windows USB version 3.91). The stimulus /da/ was loaded in the software and then the LLR testing was carried out at 90 dB nHL with repetition rate of 1.1/s and alternating polarity. 3 site electrode placements were used, and the mode of presentation of kept ipsilateral, filter setting 1-30 Hz, with a gain of 50,000. All together 150 sweeps were considered with artifact rejection at 40 uV.

The latency and absolute amplitude were noted, with comment over morphology in both non enhanced and enhanced condition. All the recording was done twice to check for the replicibality. SPSS version 15 was used for the analysis of the data obtained. Mixed ANOVA, Independent sample “t” test and Paired “t” test was done. Results revealed that LLR was present in all the subjects taken for the study with the following effect:

1) Mean values of Latency in enhanced condition was lesser in experimental as well as control group, with a larger standard deviation in experimental group implying heterogeneity of the experimental group.
2) Also the mean value for amplitude was higher in enhanced condition for both experimental and control group with larger standard deviation in experimental group.

3) There was significant difference for latency and amplitude of peak N2 between non enhanced and enhanced condition, when both experimental and control group were combined.
4) But there was no significant difference in latency or amplitude for any other peaks of the LLR , when tested between the groups for the non enhanced and enhanced condition.
5) On comparison between non enhancement and enhancement within experimental group it was found that there is significant difference in amplitude of P1 peak only.
6) Whereas comparison between non enhancement and enhancement within control group revealed that there is significant difference in latency of N1, P2 and N2 peaks, also in amplitude of N1 peak.
7) Perceptual testing showed the improvement with enhancement, more in 10 dB SNR condition, in both experimental as well as control group, though it was very less.
8) Morphology of the waveform was degraded in 50 % of the subjects and remained same in another 50 % of the subjects over a 3 point rating scale in enhanced condition.

Based on the results following conclusions were made:

1) Enhancement does help in improvement of speech identification scores majorly, for AN/AD in 10 dB SNR condition.
2) Enhancement lead to the decrease in latency and increase in the amplitude of LLR peaks in AN/AD group and normal hearing group.
3) Few more studies on the similar topic are advocated taking more number of subjects and more stimuli to record the LLR further, to illustrate the effect of enhancement , so that if there is significant improvement this strategy may help subjects with AN/AD.

Limitations of the study:

1) The configuration of hearing loss was not controlled in the experimental group.
2) The speech identification scores also varied in the experimental group.
3) If the subjective recording were also done in 10 dB SNR condition the results would have made provision to make observation on the enhancement effect.
4) The stimulus used for objective recording (LLR) was only /da/, more number of stimuli would have given better information on the effect of envelope enhancement in subjects with AN/AD.

References.

Apoux, F., Tribut, N., Dehruille, X., & Lorenizie, C. (2004). Identification of envelope expanded sentences in normal hearing and hearing impaired listeners. *Hearing Research, 189,* 13-24.

Berlin, C. I., Bordelon, J., St John, P., Wilensky, D., Hurleym, A., Kluka, E., et al. (1998). Reversing click polarity may uncover auditory neuropathy in infants. *Ear and Hearing, 19,* 37-47.

Berlin, C.I., Hood, L., & Rose, K. (2001). On renaming auditory neuropathy as auditory dys-synchrony. *Audiology Today, 13 (6),* 15-17.

Collet, L., Kemp, D.T., Veuillet, E, et al. (1990). Effect of contralateral auditory stimuli on active cochlear micromechanical properties in human subjects. *Hearing Research, 43, 251-262.*

Ferguson, S. H., & Kewley-Port, D. (2002). Vowelintelligibility in clear and conversational speech fornormal-hearing and hearing-impaired listeners. *Journal of the Acoustical Society of America, 112, 259–271.*

Govil S. (2001) Auditory middle latency and long latency responses in patients with auditory neuropathy. *An unpublished master's independent project,* University of Mysore: Mysore, India.

Harrison RV. (1998) Animal model of auditory neuropathy. *Ear and Hearing* 19: 355-361.

Hood LJ. (1998). Auditory Neuropathy: What is it and what can we do about it*? The Hearing Journal* 5(8): 10-18.

Hood L., Berlin C., Morlet T., Brashears S., Rose K., Tedesco S. (2002). Considerations in the Clinical Evaluation of Auditory Neuropathy/Auditory Dys-Synchrony. *Seminars in Hearing* 23(3):201-208.

Kraus, N., Bradlow, A.R., Cheatham, M.A., Cunningham, J., King, C.D., & Koch, C.D. (2000). Consequences of neural asynchrony: A case of auditory neuropathy. *Journal of Association for Research in Otolaryngology, 1,* 33-45.

Kraus, N., Ozdamar O., Stein L., & Reed N. (1984). Absent auditory brainstem response: Peripheral hearing loss or brainstem dysfunction. *Laryngoscope, 94,* 400-406.

Kruase, J.C., & Braida, L.D. (2004). Acoustical properties of naturally produced clear speech at normal speaking rates. *The Journal of the Acoustical Society of America, 115,* 362-378.

Li, F., Wang, H., Chen, J., & Liang, R. (2005). Auditory neuropathy in children (Analysis of 14 cases). *Lin chuang er bi yan hou ke za zhi,19,* 19-21.

Liu, S., Del Rio, E., Bradlow, A. R., & Zeng, F. G. (2004).Clear speech perception in acoustic and electric hearing.*Journal of the Acoustical Society of America, 116(4, Pt. 1),2374–2383.*

Lorrenzi, C., Berthommier, F., Apoux , F., & Bacri, N. (1999). Effects of envelope expansion on speech recognition. *Hearing Research, 136,* 131-138.

Mott, J.B., Norton, S.J., Neely, S.T., Warr, W.B. (1989). Changes in spontaneous Otoacoustic emissions produced by acoustic stimulation of the contralateral ear. *Hearing Research, 38, 229-242.*

Payton, K. L., Uchanski, R. M., & Braida, L. D. (1994).Intelligibility of conversational and clear speech in noiseand reverberation for listeners with normal and impaired hearing. *Journal of the Acoustical Society of America, 95, 1581–1592.*

Picheny, M. A., Durlach, N. I., & Braida, L. D. (1989).Speaking clearly for the hard of hearing. III: An attempt todetermine the contribution of speaking rate to differences in intelligibility between clear and conversational speech. *Journal of Speech and Hearing Research, 32, 600–603.*

Picheny, M.A., Durlach, N., & Braida, L.D. (1985). Speaking clearly for the hard of hearing I: Intelligibility difference between clear and conversational speech. *Journal of Speech and Hearing Research, 28,* 96-103.

Picheny, M.A., Durlach, N., & Braida, L.D. (1986). Speaking clearly for the hard of hearing. II: Acoustical characteristics of clear and conversational speech, *Journal of Speech and Hearing Research, 29,* 434-446.

Puel , J.L., Rebillard, G. (1990). Effect of contralateral sound stimulation on the distortion product 2 F1-F2 : evidence that the medial efferent system is involved. *Journal of Acoustical Society of America 87, 1630-1635.*

Rance, G., Beer, D.E., Cone-Wesson, B., Shepherd, R.K., Dowell, R.C., King, A.M., Rickards, F.W., & Clark, G.M. (1999). Clinical findings for a group of infants and young children with auditory neuropathy. *Ear and Hearing, 20,* 238-252.

Rance, G., Cone-Wesson, B., Wunderlich, J., & Dowell, R. (2002). Speech perception and cortical event related potentials in children with auditory neuropathy. *Ear and Hearing, 23,* 239-253.

Rance, G., McKay, C., & Grayden, D. (2004). Perceptual characterization of children with auditory neuropathy. *Ear and Hearing, 25,* 34-46.

Shallop, J.K., Peterson, A., Facer, G. W., Fabry, L.B., & Driscoll, C. L. (2001). Cochlear implants in five cases of auditory neuropathy: post operative findings and progress. *Laryngoscope, 111,* 555-562.

Shivprakash, K. (2003). *Gap detection test- Development of normative data.* Unpublished independent project, University of Mysore, Mysore, India.

Sininger Y. (2002). Identification of Auditory Neuropathy in Infants and Children. *Seminars in Hearing* 23(3):193-200.

Sininger, Y., & Oba, S. (2001). Patients with auditory neuropathy: Who are they and what can they hear? In Y. Sininger, & A. Starr (Eds.), *Auditory neuropathy: A new perspective on hearing disorder* (pp. 15-36). Canada: Singular publishing group.

Starr, A., McPherson, D., Patterson, J., Don, M., Luxford, W., Shannon, R, Sininger, Y., Tonakawa, L., et al. (1991). Absence of both auditory evoked potential and auditory percepts dependent on timing cues. *Brain, 114,* 1157-1180.

Starr, A., Michalewski, H.J., Zeng, F.G., Brooks, S.F., Linthicum, F. Kim, C.S., Winnier, D., & Keats, B. (2003). Pathology and physiology of auditory neuropathy with a novel mutation in the MPZ gene**.** *Brain, 126***,** 1604-1619..

Starr, A., Picton, T.W., Sininger, Y., Hood, L., & Berlin, C.I. (1996). Auditory neuropathy. *Brain, 119,* 741-753.

Starr, A., Sininger, Y.S., & Praat (2000). Varieties of Auditory neuropathy. *Journal of Basic Clinical Physiology and Pharmocology, 11,* 215-229.

Tallal, P., Miller, S.T., Bedi, G., Byma, G., Wang, X., Nagarajan, S., Schreiner, C., Jenkins, W., et al. (1996). Language comprehension in language learning impaired children improved with acoustically modified speech. *Science, 271,* 81-84.

Zeng, F. G., Kong, Y. Y., Michalewski, H. J., & Starr, A.(2005). Perceptual consequences of disrupted auditory nerve activity. *Journal of Neurophysiology, 93, 3050–3063.*

Zeng, F.G., & Liu, S. (2006). Speech perception in individuals with Auditory Neuropathy. *Journal of Speech Language and Hearing Research, 49,367-380.*

Zeng, F.G., Oba, S., Garde, S., Sininger, Y., & Starr, A. (1999). Temporal and speech processing deficits in auditory neuropathy. *NeuroReport, 10,* 3429-3435.

Vanaja CS, & Manjula P: **LLR as a measure of benefit derived from hearing devices with auditory dys-synchrony**. In first conference on *Auditory Neuropathy.* Edited by N. Shivashanker & H.R.Shashikala. Bangalore: department of Speech pathology and Audiology, National Institute of Mental Health and Neuro sciences;
2004, 136-146.

Simmons, J. L., & Beauchaine, K. L. (2000). Auditory neuropathy: Case study with hyperbilirubinemia. *Journal of the American Academy of Audiology*, *11,* 337–347.

Abdala, C., Sininger, Y., & Starr, A. (2000). Distortion product Otoacoustic suppression in subjects with auditory neuropathy. *Ear and Hearing*, 21 (6), 542-553.

Berlin, C.I. (1999). Auditory neuropathy: using OAEs and ABRs from screening to management. *Seminars in Hearing*, 20,307-315.

Rapin, I., & Gravel, J. (2003). Auditory neuropathy: Physiologic & pathological evidence calls for more diagnostic specificity. *International journal of pediatric otorhinolaryngology*, 67,707-728.

Trautwein,P.G., Sininger, Y.S., & Nelson, R. (2000). Cochlear implantation of auditory nueropathy. *Journal of American Academy of Audiology, 11,* 309-315.

Plomp, R. (1988). The negative effect of amplitude compression in multichannel hearing aids in the light of the modulation-transfer function. *Journal of the Acoustical Society of America*, *83*, 2322-2327.

Kruase, J.C., & Braida, L.D. (2004). Acoustical properties of naturally produced clear speech at normal speaking rates. *The Journal of the Acoustical Society of America, 115,* 362-378.

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

Butinar D, Zidar J, Leonardis L, Popovic M, Kalaydjieva L, Angelicheva D, et al. Hereditary auditory, vestibular, motor, and sensory neuropathy in a Slovenian Roma (Gypsy) kindred. Ann Neurol 1999;46:3644.

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Berndt, T. J. (2002). Friendship quality and social development. *Current Directions in Psychological Science, 11*, 7-10.

Two Authors

List by their last names and initials. Use the ampersand instead of "and."

Wegener, D. T., & Petty, R. E. (1994). Mood management across affective states: The hedonic contingency hypothesis. *Journal of Personality & Social Psychology, 66*, 1034-1048.

Three to Six Authors

List by last names and initials; commas separate author names, while the last author name is preceded again by ampersand.

Kernis, M. H., Cornell, D. P., Sun, C. R., Berry, A., & Harlow, T. (1993). There's more to self-esteem than whether it is high or low: The importance of stability of self-esteem. *Journal of Personality and Social Psychology, 65*, 1190-1204.

More Than Six Authors

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Harris, M., Karper, E., Stacks, G., Hoffman, D., DeNiro, R., Cruz, P., et al. (2001). Writing labs and the Hollywood connection. *Journal of Film and Writing, 44*(3), 213-245.

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Berndt, T.J. (1981).

Berndt, T.J. (1999).

When an author appears both as a sole author and, in another citation, as the first author of a group, list the one-author entries first.

Berndt, T. J. (1999). Friends' influence on students' adjustment to school. *Educational Psychologist, 34*, 15-28.

Berndt, T. J., & Keefe, K. (1995). Friends' influence on adolescents' adjustment to school. *Child Development, 66*, 1312-1329.

References that have the same first author and different second and/or third authors are arranged alphabetically by the last name of the second author, or the last name of the third if the first and second authors are the same.

Wegener, D. T., Kerr, N. L., Fleming, M. A., & Petty, R. E. (2000). Flexible corrections of juror judgments: Implications for jury instructions. *Psychology, Public Policy, & Law, 6*, 629-654.

Wegener, D. T., Petty, R. E., & Klein, D. J. (1994). Effects of mood on high elaboration attitude change: The mediating role of likelihood judgments. *European Journal of Social Psychology, 24*, 25-43.

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Berndt, T. J. (1981a). Age changes and changes over time in prosocial intentions and behavior between friends. *Developmental Psychology, 17*, 408-416.

Berndt, T. J. (1981b). Effects of friendship on prosocial intentions and behavior. *Child Development, 52*, 636-643.

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Edited Book, No Author

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Edited Book with an Author or Authors

Plath, S. (2000). *The unabridged journals* (K.V. Kukil, Ed.). New York: Anchor.

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O'Neil, J. M., & Egan, J. (1992). Men's and women's gender role journeys: Metaphor for healing, transition, and transformation. In B. R. Wainrib (Ed.), *Gender issues across the life cycle* (pp. 107-123). New York: Springer.

Multivolume Work

Wiener, P. (Ed.). (1973). *Dictionary of the history of ideas* (Vols. 1-4). New York: Scribner's.