Effect of Envelope Enhancement on Speech Reception and Late Latency Response Measures in Subjects with Auditory Dys-Synchrony

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Abstract

Auditory neuropathy is a disorder characterized y the impairment of the peripheral auditory function with the preservation of OHCs (Starr, et al. 1996; Berlin et al., 1998;) It is known fact that these individuals have problem with speech discrimination.

Aim: To compare the effect of envelope enhancement on speech perception and LLR in subjects with auditory neuropathy, with those associated with normal hearing.

Method: 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 10 dB 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 fr non enhanced and enhanced stimulus. The testing was done for AN/AD subjects as well as age and gender matched subjects with normal hearing.

Results: analysis was done using SPSS version 15, which revealed that there is decease in latency and better amplitude in both groups with enhancement, but it was not significant for all the peaks. Mean and standard deviation are given for each analysis.

Conclusions : The envelope enhancement did tend to decrease the latency and increase the amplitude of LLR, but the effect was not much significant for all the peaks, hence more advanced study with better control over the variables is advocated.

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, Sininger, Pratt, 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, Picton, Sininger, Hood, Berlin, 1996; 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).

Physiological tests generally used in diagnosing auditory dys-synchrony are auditory brainstem response and otoacoustic emissions. Another physiologic test which has been studied widely in individuals with AN/AD is the auditory late latency responses (LLR). 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).

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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-synchrony (Sininger & Oba, 2001). 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 and 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 and 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 and 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, 2004; Liu, Del Rio, Bradlow and Zeng, 2004; Payton, Uchanski and 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, Berthommier, Apoux, and Bacri, 1999; Apoux, Tribut, Dehruille, & Lorenize, 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, (2006) 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, et al., 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.

Method

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.

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 dyssynchrony 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 years
- 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.

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.

In perceptual testing client had to repeat whatever was heard to them. Following were the objective protocol.

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

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

SPSS 15 was used, mixed ANOVA, independent sample t test and paired t test were done, and Latencies were measured for all the four peaks of LLR in non enhanced and enhanced condition.

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.

	Peak Latencies (msec)								
Groups	F	P ₁		N ₁		P ₂		N ₂	
	NEL	EL	NEL	EL	NEL	EL	NEL	EL	
Experimental	71.11	67.27	121.66	123.50	185.00	183.50	235.33	232.17	
	(27.01)	(23.86)	(36.25)	(41.54)	(42.43)	(45.70)	(47.34)	(53.89)	
Control	72.11	70.11	129.61	123.00	180.22	170.72	228.44	208.83	
	(10.30)	(13.41)	(16.24)	(17.20)	(28.21)	(31.07)	(27.86)	(34.19)	

Absolute amplitude for all the peaks (P_1 , N_1 , P_2 & N_2) 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) inboth groups. Values in parenthesis are standard deviation.

	Peak Amplitudes (uV)								
Groups	F) 1	N_1		P_2		N ₂		
	NEA	EA	NEA	EA	NEA	EA	NEA	EA	
Experimental	1.23	1.73	-3.57	-3.92	1.74	1.99	-3.41	-2.67	
	(0.73)	(0.76)	(2.26)	(2.03)	(1.24)	(1.30)	(1.66)	(1.97)	
Control	1.37	1.40	-4.47	-3.84	2.67	2.02	-3.86	-2.76	
	(1.01)	(0.67)	(1.59)	(1.10)	(1.64)	(1.17)	(2.04)	(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.





Figure 1: (a) & (b): LLR waveforms recordings for (a) non enhanced and (b) enhanced //da/ stimulus in control group (normal hearing).



Figure 2 (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							
P ₁ N ₁ P ₂ N ₂							
Enhancement effect	1.717	0.585	1.776	6.174*			
Group effect	0.094	0.154	0.551	1.677			
Interaction	0.170	1.826	0.940	1.505			

F value for amplitude measures in LLR peaks							
Enhancement effect 2.898 0.191 0.562 4.837*							
Effect of group.	0.183	0.656	1.779	0.203			
Interaction 2.264 2.196 2.881 0.171							

From Table 3 it can be seen that the latency as well as amplitude of N_2 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 N_2 peak. However there was no significant difference in terms of latency and amplitude of other peaks i.e., P_1 , N_1 and P_2 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 P_1 , N_1 , P_2 and N_2 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).

"t" value for LLR peaks in latency measures between both groups.								
	P ₁	N ₁	P ₂	N ₂				
Non enhanced	0.147	0.848	0.398	0.532				
Enhanced	0.439	0.047	0.981	1.539				
"t" value for L	<i>"t" value for LLR peaks in amplitude measures between both groups.</i>							
Non enhanced	0.468	1.375	1.930	0.713				
Enhanced	1.363	0.155	0.872	0.117				

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)

Paired sample *t* test 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 P_1 amplitude in non enhanced condition when compared with enhanced condition, but was not seen for any other peaks i.e. N_1 , P_2 and N_2 . Though there was increase in amplitude for all the peaks, it was statistically significant only for peak P_1 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						
	P ₁	N_1	P ₂	N_2		
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 N_1 , P_2 and N_2 (p < 0.05). However, it was not so for P_1 latency. For amplitude there was significant difference in N1 peak (p < 0.05) and there was no significant difference for peaks P_1 , P_2 and N_2 .

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, 1998; 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. (2000) and Wunderlich and 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., 1999). 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 differentiation".

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:

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		

Table 7 : Peak Latencies (msec) in different age groups for synthetic CV syllable /GA/. (Cunningham et al 2000)

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:

Group age	Peaks (uV)				
Years	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		

Table 8: Peak amplitude (uV) in different age groups for synthetic CV syllable/GA/.(Cunnigham et al 2000)

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: 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. In control group 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.

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 and 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 10 dB 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 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. (1999). Auditory neuropathy: using OAEs and ABRs from screening to management. *Seminars in Hearing*, 20,307-3 15.
- Butinar D, Zidar J, Leonardis L, Popovic M, Kalaydjieva L, Angelicheva D, et al.(1999) Hereditary auditory, vestibular, motor, and sensory neuropathy in a Slovenian Roma (Gypsy) kindred. *Annals of Neurology; 46:3644*.
- Carhart, R. & Jerger, J.F. (1959). Preferred method for clinical determination of puretone thresholds. *Journal of Speech and Hearing Disorder*, 24,330-345.
- Cody D.T. & Klass DWz(1968) Cortical audiometry: Potential pitfalls in testing. Archives of tolaryngology. Oct; 88(4):396-406.
- Cunningham, J., Nicol, T. & Kraus, N. (2000). Neurophysiologic representation of clear speech in noise. *Association. Research of Otolaryngology.* 1. 23, 86.
- Cunnigham, Nicol, King, Zecker & Kraus (2002). Effect of noise and cue enhancement on neural responses to speech in auditory midbrain, thalamus and cortex. *Hearing Research*, 169, 97-111.
- 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*, 11 2, 259-2 71.
- Hood L.J. (1998). Auditory Neuropathy: What is it and what can we do about it? *The Hearing Journal* 5(8): 10-18.

- 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.
- Kruase, J.C. & Braida, L.D. (2004). Acoustical properties of naturally produced clear speech at normal speaking rates. *f i e Journal of the Acoustical Society of America*, *11 5*, 362-378.
- Kurtzberg, D., Hilpert , P.L, Kreuzer, J.A. & Vaughan, H.G Jr.(1984)Differential maturation of cortical auditory evoked potentials to speech sounds in normal full term and very low-birth weight infants. *Developmental Medicine of Child Neurology*, 26(4):466-75.
- Li, F., Wang, H., Chen, J. & Liang, R. (2005). Auditory neuropathy in children (Analysis of 4 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*, 11 6(4, Pt. 1), 23 74-2383.
- Lorrenzi, C., Berthommier, F., Apoux, F. & Bacri, N. (1999). Effects of envelope expansion on speech recognition. *Hearing Research*, *136*, 13 1-138.
- Payton, K.L., Uchanski, R.M. & Braida, L.D. (1994).Intelligibility of conversational and clear speech in noise and reverberation for listeners with normal and impaired hearing. *Journal of the Acoustical Society of America*, 95, 1581-1 592.
- Pearce W., Golding, M. & Dillon H. (2007). Cortical auditory evoked potentials in the assessment of auditory neuropathy: two case studies. *Journal of American Academy of Audiology*, 18(5):380-90.
- Picheny, M.A., Durlach, N.I. & Braida, L.D. (1989).Speaking clearly for the hard of hearing. III: An attempt to determine the contribution of speaking rate to differences in intelligibility between clear and conversational speech. *Journal of Speech and Hearing Research*, 32, 60&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. 11: Acoustical characteristics of clear and conversational speech, *Journal* of Speech and Hearing Research, 29, 434-446.
- Ponton, C.W, Eggermont, J.J, Kwong B, & Don, M. (2000). Maturation of human central auditory system activity: evidence from multi-channel evoked potentials. *Clinical Neurophysiology*. *III* (2):220-36.
- 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.
- Rapin, I. & Gravel, J. (2003). Auditory neuropathy: Physiologic & pathological evidence calls for more diagnostic specificity. *International journal of pediatric otorhinolaryngology*, 67,707-728.
- Salvi, R.J., Wang, J., Ding, D., Stecker, N. & Arnold, S. (1999) Auditory deprivation of central auditory system resulting from selective inner hair cell loss : animal model of auditory neuropathy. *Scandivian Audiology*; 28 (supplement 51):1-12.
- Simmons, J. L. & Beauchaine, K. L. (2000). Auditory neuropathy: Case study with hyperbilirubinemia. *Journal of the American Academy of Audiology*, *1*, *337*-347.
- 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. (199 1). Absence of both auditory evoked potential and auditory percepts dependent on timing cues. *Brain*, 114, 1157-1 180.
- Starr, A., Michdewski, 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 Phamocology*, 11, 2 15-229.

- 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, 8 1-84.
- Wunderlich J.L. & Cone-Wesson B.K. (2006) Maturation of CAEP in infants and children: a review . *Hearing Research. Fe b; 212 (I -2): 2 12-23.*
- 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*, 3429-343 5.