Brainstem and Cortical Responses to Speech Stimuli in Individuals with Cochlear Hearing Loss

Anirban Chaudhury & Vanaja C S*

Abstract

This study investigated the effect cochlear pathology on brainstem and cortical responses to speech burst and transition. The relationship between these potentials and speech identification scores was also investigated. Ten adult subjects with cochlear pathology and 12 age matched normal hearing subjects were included in the study. Burst and transition portions were extracted separately from the stimuli /pa/, /ta/, /ka/. Burst evoked brainstem responses were analyzed for wave V, transient evoked brainstem responses were analyzed for peak V, A, C, D, E and F and cortical evoked potentials were analyzed for P1, N1, P2 and N2. Speech identification scores in quiet and in the presence of noise were obtained for bisyllabic word list in Kannada. Burst evoked responses showed a significant difference between the latency of wave V obtained in subjects with cochlear hearing loss and those with normal hearing group but no significant difference was found in terms of wave V amplitude. For the transition stimuli, latencies of wave V, A, C, D, E, and F as well as the amplitude of wave V were significantly different between the two groups. All the components (V, A to F) evoked by transition stimuli significantly correlated with SIS scores in noise. But no correlation was observed for burst evoked brainstem responses. There was no significant difference between groups for all the components of LLR (P1, N1, P2 & N2) but N1-P2 amplitude was significantly different between the groups. These findings suggest that cochlear hearing loss impairs the processing of the burst and transition portion of speech signal mainly at the brainstem level.

Introduction

Individuals with sensorineural hearing loss have difficulty in understanding speech (Glasberg & Moore, 1989). Behavioral tests have been devised and used to assess these speech processing difficulties. But these types of behavioral tests cannot be used in some of the difficult-to-test population. In such individuals objective electrophysiological tests may be helpful in predicting speech perception.

Conventionally brief acoustic signals such as clicks, tone bursts and tone pips have been used to elicit the ABR. Recent investigations have shown that brainstem responses to speech stimuli can also be reliably recorded and analyzed (Khaladkar, Karthik & Vanaja, 2005). As brainstem responses can be best recorded using short duration signals, burst or transition portion

* Professor of Audiology, School of Audiology and Speech Language Pathology, Bharathiya Vidya Peet University, Katra-Dhanakawadi, Pune, India. e-mail: csvanaja@rediffmail.com

have been used to elicit brainstem responses in these studies. The speech-evoked ABR recorded in human brainstem can be divided into transient and sustained portions, specifically the onset response and the frequency-following response (FFR) (Kraus & Nicol, 2005). Onset responses are transient, similar to click evoked ABR with peak durations lasting tenths of milliseconds. The FFR arises from the harmonic portion of the stimulus and is characterized as a series of transient neural events phase locked to periodic information within the stimulus (Batra, Kuwada & Maher, 1986). Galbraith, Arbagey, Branski, Comerci and Rector (1995) demonstrated that the FFR elicited by word stimuli reflects the stimulus accurately enough to allow it to be recognized as intelligible speech when "played back" as an auditory stimulus. More recently, Galbraith, Amaya, Rivera, Donan, Duong and Hsu, (2004) have suggested that based on the FFR pattern of activation for forward and backward speech, synaptic processing at the level of the brain stem is more effective for forward speech stimuli characterized by highly familiar prosodic and phonemic structure than to backward speech. The studies carried out on children with learning disability have shown that responses to speech stimuli were deviant in these children even when responses to nonspeech stimuli were normal (Khaladkar, 2005).

Khaladkar, Karthik and Vanaja (2005) obtained speech burst ABRs for 20 ears with mild to moderate sensorineural hearing loss. Two stimuli were used to evoke the ABR; a standard acoustic click and the burst portion of the syllable /t/. The result of their study indicate that while click evoked ABRs exhibited latency values within normal limits, speech burst evoked ABRs showed more deviant results. There was a significant correlation between speech identification score and speech burst ABR, perhaps suggesting that using speech sounds to elicit the ABR offers an opportunity to better isolate normal speech processing from abnormal speech processing. Hedrick and Jesteadt (1996) reported that sensorineural hearing loss may disrupt formant transient coding or any type of dynamic process in periphery (i.e. rapidly changing aspects of speech signal is not being coded). So it can be hypothesized that the transition responses evoked by ABR may provide useful information about processing of speech at brainstem level. There was also a need to study the cortical representation of burst and transition of speech stimuli in subjects with normal hearing and those with hearing loss.

Speech evoked LLR were frequently used to study the neural representation of speech sound in populations with impaired speech understanding. The underlying assumption is that speech perception is dependent on the neural detection of time-varying spectral and temporal cues contained in the speech signal (Tremblay, Billings & Rohila, 2004). The P1-N1-P2 complex reflects the neural detection of time-varying acoustic cues. Because abnormal P1-N1-P2 response patterns have been reported in children and adults with varying types of speech perception impairments, there is a current surge of interest in learning more about this brain-behavior relationship (Rance, Wesson, Wunderlich & Dowell, 2002). There is a dearth of studies correlating both brainstem and cortical responses with SIS in subjects with SN hearing loss. Also research has not been carried out to study the cortical responses for only burst or transition portion of a syllable. Hence the present study aimed to investigate if there is a

difference between subjects with normal hearing and those with cochlear pathology in the following responses:

- Brainstem responses to speech burst
- Brainstem responses to transition of speech
- Cortical responses to speech burst
- Cortical responses to transition of speech

The study also investigated the relationship between the following in subjects with cochlear pathology:

- Brainstem responses to speech burst and speech identification scores
- Brainstem responses to transition of speech and speech identification scores
- Cortical responses to speech burst and speech identification scores
- Cortical responses to transition of speech and speech identification scores

Method

Participants:

Participants of the present study were divided into two groups. Control group included twelve ears of normal hearing individuals aged 15-50 years and hearing sensitivity within 15 dBHL. The clinical group included twenty two ears with cochlear hearing loss of subjects aged 15-50 years with hearing sensitivity within 55 dBHL. The hearing impairment was post-lingual. Participants had no history of speech and language problem and all of them were native speakers of Kannada.

Instrumentation:

A calibrated dual channel OB922 clinical audiometer (Version 2) with TDH 39 earphones housed in MX/41 AR ear cushions and Radio ear B 71 bone vibrator was used for estimating pure tone threshold and speech audiometry. A calibrated GSI Tympstar middle ear analyzer was used for tympanometry and acoustic reflex measurement to rule out middle ear pathology. The IHS smart EP, version 2.39 (Intelligent Hearing systems, Florida, USA) with Eartone 3A insert earphones was used to record and analyze auditory evoked potentials.

Materials:

Extracted transition and burst portion of naturally produced syllable /pa/, /ta/, /ka/ by an adult female Kannada speaker was used to elicit brainstem and cortical response. The syllables were spoken into a unidirectional microphone connected to the computer. To view and edit the speech sounds, PRAAT (version 4.4.27) was used. The wave file was then converted to stimulus file for ALLR recording using ‘Stim conv’ provided by the Intelligent Hearing System (version 2.39). All the stimuli were calibrated in dB nHL. Paired words in Kannada were used to

determine the Speech Reception Thresholds (SRT) and recorded version of the word list from speech identification test in Kannada developed by Vandana (1998) was used to determine SIS.

Test procedure:

Pure tone thresholds were assessed using modified Hughson Westlake method (Carhart & Jerger, 1959) for air conduction stimuli from 250 Hz to 8000 Hz and for bone conduction stimuli from 250 Hz to 4000 Hz. Speech Reception Threshold (SRT) was obtained using paired words in Kannada. Speech identification scores (SIS) were obtained at 40 dB SL (ref: SRT) in both quiet and noise (speech babble, 0 dB SNR) with PB word list developed by Vandana (1998). All the auditory evoked potentials were recorded using conventional electrode montage with the noninverting electrode on vertex, inverting electrode on mastoid and common electrode on the forehead. Stimuli were presented at 40 dBSL (ref. SRT). Repetition rate of the stimuli was 11.1/sec for brainstem responses and 3.1/sec for cortical responses. The analysis window was 20 ms for brainstem responses to burst, 50 ms for brainstem responses to transition and 300 ms to cortical responses. The analysis included 50 ms pre stimulus window while recording cortical responses. Responses for 1500 stimuli were filtered using a band pass filter of 100 Hz to 3 KHz and amplified 100 K times for brainstem responses. Cortical responses for 300 stimuli were filtered using a band pass filter of 1-30 Hz and amplified 50 K times.

Results

Wave V and its negative trough (wave A) of ABR evoked by burst were marked. The peak to trough amplitude of the wave V was measured. Similarly the Vth peak for transition evoked ABR was also identified and amplitude was measured. In addition since transition ABR has a steady state portion FFR was also analyzed as described by Kraus (2000). For the transition evoked ABR latency of wave V, wave A, C, D, E and F and amplitude of wave V were considered. For LLR, peak P1, N1, P2 and N2 were measured. The data obtained were tabulated and statistical analyses were carried out using SPSS software (V15, SPSS Inc).

1. Latency and amplitude of wave V in individuals with normal hearing and individuals with cochlear hearing loss for burst evoked ABR

Table 1 Shows the mean latency and amplitude of wave V evoked by bursts of /pa/, /ta/ and /ka/ in individuals with normal hearing and those with cochlear hearing loss. It can be noted from the table that latency of wave V for /p/ and /k/ was similar but /t/ latency was shorter in both the groups. The amplitude of the /p/ and /k/ was similar but /t/ amplitude was lesser than /p/ and /k/ in both the groups. It can be observed from the table that the latency was longer and amplitude was lesser in individuals with hearing impairment than that of normal hearing, for all the stimuli.

Multivariate Analysis of Variance was administered to assess the effect of groups and three stimuli on latency and amplitude for wave V. Results revealed that there was a significant effect of cochlear hearing loss on latency ($p<0.05$) but there was no significant difference in

amplitude of wave V between the groups (p>0.05). Also there was no interaction of stimulus with group for latency and amplitude. Scheffe's post hoc showed no significant effect of stimulus on latency and amplitude of wave V in both groups.

Table-1: Mean and SD of latency and amplitude of wave V

Group	pa		ta		ka	
	Latency in msec	Amplitude in µv	Latency in msec	Amplitude in µv	Latency in msec	Amplitude in µv
Normal hearing	7.1 (0.5)	0.44 (0.04)	6.2 (0.4)	0.4 (0.2)	7.4 (0.7)	0.5 (0.3)
Hearing impairment	7.2 (0.02)	0.38 (0.8)	6.99 (0.7)	0.35 (0.1)	7.7 (1.6)	0.4 (0.2)

2. Latency and amplitude of peaks in individuals with normal hearing and individuals with cochlear hearing loss for transition evoked ABR and FFR

Table 2 shows the mean latency of peaks V, A, C, D, E, F and amplitude of peak V, elicited by transition portion of /pa/, /ta/ and /ka/ in individuals with normal hearing and hearing impairment. For individuals with normal hearing the latency of wave V for /pa/ and /ka/ was similar but /ta/ latency was longer than the other two stimuli. The amplitude of the /k/ was higher than /t/ and /p/. However, the standard deviation for amplitude of /k/ was larger indicating greater variability. For individuals with hearing impairment the trend obtained for different stimuli was similar to that observed for participants with normal hearing. The latency of wave V for /pa/ and /ka/ was similar but /ta/ was longer than the other two stimuli. However, the amplitude of the /pa/ and /ka/ was similar but /ta/ amplitude was lesser than /pa/ and /ka/ in this group.

Table-2: Latency of wave V, A, C, D, E, F and amplitude of wave V in individuals with normal hearing and hearing impairment

Subjects and Stimuli		V		A	C	D	E	F
		Latency in msec	Amplitude in µv	Latency in msec	Latency in msec	Latency in msec	Latency in msec	Latency in msec
normal hearing	pa	9.9 (2.3)	0.3 (0.04)	11.1 (2.3)	13.8(2.7)	17.2(3.09)	21.4 (3.1)	25.6 (3.2)
	ta	12.5 (1.6)	0.3 (0.07)	13.66 (1.8)	18.3 (2.7)	21.9 (2.9)	25.5 (3)	29.8 (3.3)
	ka	9.8 (2.4)	0.39 (0.2)	11.5 (2.4)	14.8 (1.7)	18.3 (1.5)	22.4 (1.6)	26.8 (1.6)
hearing impaired	pa	15.4(2.5)	0.2(0.07)	17.2(2.9)	22.1(2.66)	25.7(2.6)	30.9(3.4)	35.1(3.6)
	ta	19.6(1.3)	0.22(0.2)	21.7(1.6)	27.3(2.0)	31.4(2.0)	36(1.3)	40.4(1.6)
	ka	16.2(1.7)	0.28(0.2)	18.1(1.9)	22(1.4)	26.6(2.0)	30.9(1.9)	35.2(1.8)

Multivariate analysis of variance was administered to assess the significant difference between groups for three stimuli in latency and amplitude. There was a main effect of group (cochlear hearing loss) on latency of all the peaks and amplitude of wave V (p<0.01) and there was no interaction between stimulus and group. Scheffe's Post Hoc analysis of variance revealed

that the amplitude and latency of /ta/ differed significantly from that of /pa/ and /ka/ ($p<0.01$) for wave V of ABR and other waves of FFR but there was no significant difference between /pa/ and /ka/ ($p>0.05$).

3. Long latency responses evoked by speech bursts

Table 3 shows the mean for latencies for the components (P1, NI, P2, N2) of LLR and amplitude of NI-P2 complex in individuals with normal hearing and individuals with cochlear hearing loss across three speech burst stimuli. Multivariate analysis of variance was carried out to check if there is a main effect of cochlear hearing loss on latencies of components of LLR and N1-P2 amplitude. Results revealed that there was no significant main effect of group (cochlear hearing loss) for its measure on latency of all the peaks ($p>0.05$) but N1-P2 amplitude differed significantly ($p<0.01$) and no interaction was observed between stimulus and group.

Table 3: Mean (SD) of Latency for components of LLR and N1-P2 amplitude recorded with burst in individuals with normal hearing and hearing impairment

		P1	N1	P2	N2	N1P2 amp
Individuals with normal hearing	pa	86.7(13.8)	127.2(12.3)	188.2(14.7)	197.6(24.1)	1.3(0.3)
	ta	82.3(8.3)	128.4(16.7)	181.8(14.09)	229.2(11.8)	1.5(0.4)
	ka	86.9(18.6)	130.7(12.4)	187.1(25.4)	225.4(20.5)	1.3(0.5)
Individuals with hearing impairment	pa	87.3(34.2)	127.12(37.93)	184.65(42.06)	235.42(43.52)	0.98(0.17)
	ta	83.3(22.7)	121.32(17.99)	173.25(16.19)	230.02(11.13)	0.83(0.16)
	ka	86.07(21.90)	123.2(25.80)	177.47(17.91)	233.42(13.06)	1.04(0.30)

4. Long latency responses evoked by formant transition

Table 4 shows latencies for the components (P1, NI, P2, N2) of LLR and amplitude of NI-P2 complex in individuals with normal hearing and individuals with cochlear hearing loss across three speech formant transitions. Multivariate analysis of variance was carried out to check if there was a main effect of cochlear hearing loss on latencies of P1, N1, P2, N2 and amplitude of N1-P2.

Table 4: Mean and SD of latency (in ms) and amplitude (in µ V) of LLR peaks elicited by transition

		P1 latency	N1 latency	P2 latency	N2 latency	N1P2 amp
Normal hearing subjects	pa	92.75(15.24)	139.31(2)	194.46(24.83)	251.31(21.29)	1.07(0.34)
	ta	95.6(24.82	142.91(29.02	212.12(48.43	240.36(36.17)	1.33(1.33)
	ka	97.1(9.88)	143.19(13.07)	211.94(31.01)	242.94(32.91)	1.47(0.4)
Hearing impaired subjects	pa	90.82(25.92)	123.52(24.82)	176.10(31.82)	230.85(44.91)	0.94(0.17)
	ta	86.62(25.53)	119.87(23.95)	178.57(35.64)	227.92(41.21)	1.01(0.16)
	ka	96.3(19.11)	137.07(19.66)	202.12(10.06)	257.4(15.99)	1.04(0.22)

Results revealed that there was no significant main effect of cochlear hearing loss ($p > 0.05$) and no interaction was observed between group and stimulus. Sheffec's Post Hoc analysis revealed no significant effect of stimulus on latency or amplitude of LLR.

5. Speech identification scores in individuals with normal hearing and individuals with cochlear hearing loss

Table 5 shows the speech identification scores in quiet and in presence of noisy condition for the participants with normal hearing and those with hearing impairment. Independent sample t test revealed that there was a significant difference between the scores of participants with normal hearing and with hearing impairment in both quiet ($t = 13.0$, $p < 0.01$) and noisy condition ($t = 19.9$, $p < 0.01$).

Table 5: Mean (SD) of speech identification scores in quiet and in the presence of noise

Group	In Quiet	In Noise
Normal Hearing	100	97 (3.9)
Hearing Impaired	76.2 (6.4)	15.6 (13.4)

6. Relationship between speech identification scores and brainstem and cortical responses

Pearson product moment correlation analysis was carried out to check the relationship of latency and amplitude of brainstem potentials for the three stimuli with speech identification scores (SIS) in quiet and in the presence of noise. Results revealed that SIS in noise correlated significantly with formant transition evoked FFR and wave V for all the three stimuli (refer Table 6 for r values) but SIS score in quiet did not show a significant correlation. Speech burst evoked ABR and LLR as well as transition evoked LLR did not show a significant correlation with SIS scores in quiet or in the presence of noise.

To summarize the results of the present study revealed that brainstem and cortical responses to bursts and transition of speech stimuli can be recorded from participants with normal hearing as well as those with hearing loss.

Table 6: Correlation of SIS scores with brainstem responses evoked by transition of /pa/, /ta/ and /ka/

Latency & Amplitude	pa		ta		ka	
	In quiet	In noise	In quiet	In noise	In quiet	In noise
V latency	-0.217	-0.740**	-0.281	-0.896**	-0.235	-0.813**
V amplitude	0.286	0.640**	0.129	0.491	0.190	-0.251
A latency	-0.293	-0.728**	-0.394	-0.909**	-0.287	-0.778**
C latency	-0.325	-0.726**	-0.341	-0.862**	-0.397	-0.829**
D latency	-0.288	-0.630**	-0.354	-0.867**	-0.413	-0.829**
E latency	-0.303	-0.644**	-0.365	-0.862**	-0.416	-0.837**
F latency	-0.368	-0641**	-0.345	-0.845**	-0.387	-0.820**

*p<0.05 and **p<0.01

There was a significant effect of hearing loss on brainstem responses to speech but cortical responses to speech were not affected by hearing loss. Speech identification scores obtained in the presence of noise showed a significant correlation with wave V and FFR evoked by transition of speech.

Discussion

In the present study brainstem responses and cortical responses could be recorded for all the stimuli from all the participants with normal hearing as well as those with mild to moderate hearing loss. The latencies of peak V for different stimuli obtained in the present study are comparable with those reported by Reddy, Kumar and Vanaja (2004) except for /ka/ which had longer latency in the present study. This could be due to the difference in the stimuli used in the two studies. The duration of the signals used in the present study was longer than those used by the earlier study and the difference was largest for /ka/. ABR is an onset response and the latency and amplitude of the response depends on stimulus onset/rise time, spectrum of the response and the duration of the signal (Gorga et al., 1984). Differences in latencies can be attributed to the differences in spectrum, rise time of the stimulus and durational differences of the stimuli used in the two studies.

The prolongation of latencies in subjects with hearing impairment may be due to the overall reduction in audibility. Previous studies on click evoked ABR have also reported that the latency of all the peaks increase with increase in hearing threshold (Oates & Stapells, 1992). Though statistically not significant the mean amplitude was lesser in subjects with hearing impairment when compared to those with normal hearing. This is probably due to reduction in number of nerve fibers responding for the stimuli. It has been reported in literature that the amplitude of ABR depends on the number of nerve fibers firing (Hecox, Squires & Galambox, 1976). Thus the results of the present study suggest that coding of the processing of burst is effected in subjects with hearing impairment. However, speech identification scores in quiet or in the presence of noise did not show a significant correlation with latency or amplitude of ABR elicited by burst. These results contradict the report of Khaladkar, Karthik and Vanaja (2005) who observed that there was a significant correlation between SIS and speech burst ABR in subjects with sensorineural hearing loss.

The latency of the onset response (Wave V and A) for the transition portion of the signal in the present study was longer than that reported by King, Warrier, Hayes and Kraus (2002) but the latency for the other peaks (C, D, E and F) was shorter. It has been reported that the wave V and A signal the onset of sound at the brainstem whereas wave C is the response to the onset of the vowel (Kraus & Nicol, 2005). The other peaks, D, E and F are responses to sustained portion of the signal. So probably the difference in latency reflects the difference in the stimulus used in the two studies. King, Warrier, Hayes and Kraus (2002) used synthesized transition of /da/ with 40 msec duration. On the other hand in the present study a natural stimulus was taken and the transition part was extracted. The duration of transition in the present study was around 25 msec

for /pa/, 49 msec for /ta/ and 41 msec for /ka/. The fundamental frequency ranged from 103 to 121 Hz in their study and it was around 230 Hz in the present study.

The latency of the FFR portion in hearing impaired subjects was prolonged compared to normal hearing subjects and the amplitude was significantly reduced in these subjects. This suggests that the encoding of the sustained portion was affected in the participants with hearing impairment. The inter-peak latency difference between D and E as well as E and F were around 4 msec in subjects with normal hearing whereas it was around 5 msec in subjects with hearing loss. This indicates that processing of the fundamental frequencies was affected in subjects with hearing impairment. It has been reported in literature that the F0 and F1 coding are affected in persons with hearing impairment at the brainstem level and this is reflected in the abnormalities in the waveform of ABR (Kraus & Nicol, 2005).

Auditory system encodes the F0 from fine structure but it can also encode the F0 from the envelope but encoding of F0 from the envelope is weaker when compared to that extracted from the fine structure (Zeng et al., 2004). In addition psycho-acoustical studies have shown that cochlear hearing impaired subjects are impaired in coding the temporal fine structure of the speech signal which contains the F0 and harmonics (Lorenzi, Gilbert, Carn, Garnier & Moore 2006). This indicates a greatly reduced ability to use temporal fine structure speech in individuals with moderate hearing loss. This loss of ability to use temporal fine structure information perhaps was related to a loss of neural synchrony (Woolf, Ryan & Bone, 1981). This would have contributed for reduced amplitude and prolonged latencies in subjects with cochlear hearing loss.

The recent studies have shown that speech in quiet could be completely understood with only envelope cues (amplitude variation of the speech signal) (Nambi, Mahajan, Narne & Vanaja, 2007). But understanding of speech in noise depends on the encoding of the fine structure of the speech signal as well as envelope. It has been reported that coding of envelope of the speech signal is normal in cochlear hearing loss subjects but processing of temporal fine structure is impaired. The results of correlation also revealed that SIS scores in noise were correlated well with components of FFR. This supports the hypothesis that processing of temporal fine structure is affected in subjects with cochlear hearing loss.

There is dearth of study investigating LLR with burst and transition in subjects with hearing loss. However the results obtained in this study are comparable with those reported in literature for other stimuli. There was no significant difference in latency of LLR for the participants with normal hearing and those with hearing impairment. This may be because the degree of hearing loss was less than moderate degree. Mild to moderate degree of hearing impairment do not significantly influence the latency of LLR (Albera et al., 1991). It has been reported that at suprathreshold levels the latency of LLR is not significantly affected by intensity of the stimulus (Picton et al., 1978). Variability of the LLR latency in normal subjects is also high. This may have been one of the reasons for obtaining no significant difference in the latency of LLR in the two groups. The N1-P2 amplitude was significantly better in subjects with hearing

loss when compared to that of normal hearing subjects. This suggests that probably less number of cortical cells were responding in subjects with hearing loss. It has been reported that the amplitudes of LLR depends on the number of cells responding for the stimulus and that long deprivation of auditory stimuli may lead to loss of cells at the cortical level (Polley, Chen-Bee & Frostig, 1999). However, the duration of hearing impairment in a majority of subjects in the present study was not more than 9 months. Probably there would have been a significant effect on LLR if the duration of hearing impairment was more. No significant correlation between SIS and LLR measures suggests that probably the poor speech perception in the subjects was mainly due to abnormal encoding of speech at the cortical and brainstem level.

Conclusion

In this study there was a significant difference in burst evoked wave V latency between cochlear hearing loss group and normal hearing group but no significant difference was found in terms of wave V amplitude. For the transition stimuli, latencies of wave V, A, C, D, E & F and amplitude of wave V were significantly different between the two groups. All the components (V, A to F) evoked by transition stimuli significantly correlated with SIS scores in noise. But no correlation was observed for burst evoked brainstem responses. There was no significant difference between groups for all the components of LLR (P1, N1, P2 & N2) but N1-P2 amplitude was significantly different between groups. No correlation found with SIS in quiet as well as in noise. It can be concluded from the results of the present study that cochlear hearing loss impairs the processing of the burst and transition portion of speech signal.

References

Albera, R., Roberto, C., Magnano, M., Lacilla, M., Morra, B. & Cortesina, G. (1991). Identification of the waveform of cortical auditory evoked potentials. *Acta Otorhinolaryngologica Italica, 11(6),* 543-9.

Batra, R., Kuwada, S. & Maher, V. L. (1986). The frequency following response to continuous tones in humans. *Hearing Research, 21*, 167–177.

Galbraith, G. C., Amaya, E. M., de Rivera J. M., Donan, N. M., Duong, M. T. & Hsu, J N. (2004). Brainstem evoked response to forward and reversed speech in humans. *Neuroreport, 15*, 2057–2060.

Galbraith, G. C., Arbagey, P. W., Branski, R., Comerci, N. & Rector, P. M. (1995). Intelligible speech encoded in the human brain stem frequency-following response. *Neuroreport, 6,* 2363–2367.

Glasberg, B. R. & Moore, B. C. (1989). Psychoacoustic abilities of subjects with unilateral and bilateral cochlear hearing impairments and their relationship to the ability to understand speech: *Scandanavian Audiology (Suppliment), 32*, 1-25.

Gorga, M. P., Beauchaine, K. A., Reiland, J. K., Worthington, D. W. & Javel, E.. (1984). Effects of stimulus duration on ABR and behavioral threshold. *Journal of the Acoustical society of America. 76,*616-619.

Hecox, K., Squires, N. & Galambos, R. (1976). Brainstem evoked responses in man: I Effect of stimulus rise-fall time and duration. *Journal of the Acoustical society of America, 60,* 1187-1192.

Khaladkar, A. A. (2005). Speech elicited ABR: An exploratory study in normals and in children with learning disability. *Unpublished Master's dissertation submitted to University of Mysore*, Mysore.

Khaladkar, A. A., Kartik, N. & Vanaja, C.S. (2005). Speech Burst and Click Evoked ABR. Scientific paper presented at the 37th National conference of the Indian Speech & Hearing Association (Indore).

King, C., Warrier C.M., Hayes, E. & Kraus, N (2002) Deficits in auditory brainstem pathway encoding of speech sounds in children with learning problems, *Neuroscience Letter* 319:111–115.

Kraus, N. & Nicol, T. G. (2005). Brainstem origins for cortical 'what' and 'where' pathways in the auditory system. *Trends in Neurosciences, 28,* 176–181.

Lorenzi, C.G. Gilbert, H. Carn, S. Garnier. & Moore, B. C. J. (2006). Speech perception problems of the hearing impaired reflect inability to use temporal fine structure. *Proceedings of the National Academy of Sciences*. 103(49): 18866 - 18869**.**

Nambi, P. A., Mahajan, Y., Narne V. & Vanaja, C. S. (2007). Importance of amplitude and frequency modulation cues for speech recognition. *Paper presented at 39th annual conference of Indian Speech and Hearing Association*, Calicut.

Oates, P. & Stapells, D. R. (1992). Interaction of click intensity and cochlear hearing loss on auditory brain stem response wave V latency. *Ear and Hearing. 13(1),* 28-34.

Picton, T. W. & Smith, A. D. (1978). The practice of evoked potential audiometry. *Otolaryngologic Clinics of North America, 11(2),* 263-82.

Polley, D.B., Chen-Bee, C.H. & Frostig, R.D. (1999). Two directions of plasticity in the sensory-deprived adult cortex. *Neuron. 24(3)* ,623-37.

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

Reddy, S. M., Kumar, U. A. & Vanaja, C. S. (2004). Characteristics of ABR evoked by speech bursts. *Scientific paper presented at the 36th National conference of the Indian Speech & Hearing Association*, Mysore.

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

Woolf, N. K., Ryan, A.F. & Bone, R.C. (1981). Neural phase-locking properties in the absence of cochlear outer hair cells. *Hearing Research, 4(3-4),* 335-46.

Zeng F.G., Nie K., Liu S., Stickney G.S., Del Rio E., Kong Y.Y. & Chen H.B. (2004). On the dichotomy in auditory perception between temporal envelope and fine structure cues. *Journal of the Acoustical Society of America, 116(3),* 1351-1354.