Brainstem Responses to Speech in Normal Hearing and Cochlear Hearing Loss Individuals

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Abstract

Studying the neural encoding of speech sounds provides insight into some of the auditory processes involved in normal communication. Auditory brainstem evoked responses to speech provide direct information about how the sound structure of a speech syllable is encoded in the auditory system. Individuals with cochlear hearing loss have consistently shown difficulties in perceiving place and manner cues of consonants. The current study aimed at determining the effect of cochlear hearing loss, stimulus presentation level (equal SL and equal SPL) on brainstem responses to speech. The ABR and FFR were recorded for the synthetic speech stimuli /da/ in 22 normals and 22 cochlear hearing loss (PTA < 55 dBHL) individuals at 80 dBnHL and 40 dB SL. Results revealed that the cochlear hearing loss showed reduced amplitude and prolonged wave latency even at equal sensational level. This effect was adverse with the increase in severity of hearing loss which is reflected by the poor coding of F0 and its formant (F1).

Introduction

The neural encoding of sound stimulus begins at the auditory nerve and continues till the cortex via the auditory brainstem. Brainstem responses to simple stimuli (e.g., clicks, tones) are well defined and widely used in clinical practice in the evaluation of auditory pathway integrity (Moller, 1999; Starr & Don, 1988). However, the role of brainstem in processing a complex signal, varying in many acoustic dimensions continuously over time, such as a speech syllable have recently become a subject of great interest with the help of conventional techniques of recording evoked potentials.

Studying the neural encoding of speech sounds provides insight into some of the auditory processes involved in normal communication. Auditory brainstem evoked responses (ABR) provide more direct information about how the sound structure of a speech syllable is encoded by the auditory system. A handful of studies have been done in similar lines to understand the brainstem processing of speech signal (Russo, Nicol, Musacchia & Kraus, 2004; Kraus & Nicol, 2005). Based on these studies brainstem responses to a speech syllable can be divided into - transient and sustained portions, namely the onset response and the frequency-following response (FFR). The response functions as a gauge both of spectrum encoding and periodicity encoding.

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Frequency encoding is manifested in speech-evoked auditory responses both in the latency (Martin et al., 1997; McGee et al., 1996) and the amplitude of transient responses. The onset responses are transient, akin to the well-documented clinical measure that uses click or tonal stimuli as a tool for assessing both peripheral hearing and retrocochlear lesions such as tumors of the auditory nerve or brainstem (Hall, 1992). The sustained frequency-following response (FFR) is a phase-locked response that 'follows' the waveform of the stimulating sound up to a frequency of approx 1000 Hz (Hoormann et al., 1992). It must be noted that although the FFR is a sustained response it might be considered a series of repeated transients. Thus, the FFR can be treated as a measure of both periodicity and spectral processing.

Russo, Nicol, Musacchia and Kraus (2004) have designed a method to evaluate both the periodicity and spectral encoding in far-field FFR recordings. The markings used in the system are shown in the Figure.1. It contains a series of peaks ranging from peak V, A, C, D, E, F and O. Waves V and A signal the response to the onset of sound. Wave C is thought as a response to the onset of the vowel. Peaks - D, E and F represent vibrations of the vocal folds. The fundamental frequency occurs at approximately 15 msec, 24 msec and 33 msec in stimulus corresponding to wave D (22 msec), E (31 msec) and F (40 msec) in response. Neural conduction accounts for a delay of approximately 7 ms between stimulus and response. Wave O is a response to the cessation of sound. The small higher-frequency fluctuations between waves D, E and F correspond in frequency to that of the first formant (F1) of the stimulus which, along with F2, primarily shapes the vowels.



Figure.1: Depicts the wave V followed by the negative peaks A, C, D, E and F. The onset response is bracketed while the region containing the FFR is indicated with a horizontal line

The significance of these peaks is now well established by its application in clinical population. FFR has been used to study the brainstem coding deficits in several communication disorders such as children with learning problems and adults with cochlear hearing loss. Some children with language-based learning problems exhibit abnormal neural encoding of the spectral and temporal information crucial for accurate perception of sounds (King, Warrier, Hayes & Kraus, 2002; Cunningham, Nicol, Zecker, Bradlow & Kraus, 2001). Some also experienced abnormal susceptibility to the demands placed on the auditory system by rapidly presented temporal information (Wible, Nicol & Kraus, 2005).

Khaladkar, Kartik and Vanaja (2005) suggested that using speech sounds to elicit the ABR offers an opportunity to isolate normal speech processing from abnormal speech processing better. The researchers further suggested that it would be useful for evaluating patients with possible auditory processing disorders. Plyler and Ananthanarayan (2001) reported that the FFR can encode the second formant transition in normal-hearing listeners. However, FFR encoding seems to be severely degraded in most of the listeners with hearing loss.

Russo, Nicol, Musacchia and Kraus (2004) found that the addition of background noise interfered with normal brainstem encoding of the speech stimulus /da/. Most affected were the onset responses V and A which were severely degraded and completely obscured in more than 40% of the subjects. Peaks C and F however remained present in noise in most subjects. Their peak amplitudes were also affected.

Individuals with cochlear hearing loss have consistently shown difficulties in perceiving place (Revoile, Pickett, Holden-Pitt, Talkin & Brandt, 1987) and manner cues (Danhauer, Hiller & Edgerton, 1984) of consonants. These difficulties increased with the degree of hearing loss. Moore, Glasberg and Hopkins (2006) reported that subjects with moderate hearing loss performed much worse in the difference limen for F0 compared to normally hearing subjects at the same center frequency, suggesting that most of the hearing-impaired subjects had a poor ability to use temporal fine structure. The temporal fine structures are important for the coding of F0 and its harmonics.

It is very important to understand whether the individuals with cochlear hearing loss exhibit any encoding deficits at the level of the brainstem as a result of distortion at the cochlea. Speech-evoked brainstem responses provide a unique opportunity to explore this possibility in a non-invasive manner. Since there is a dearth of literature on the brainstem processing for speech stimulus in individuals with hearing loss there is a need for exploring the brainstem bases for speech perception deficits in individuals with hearing loss. Also, there is a need to understand whether the temporal processing difficulties in the cochlear hearing is due to the reduction in the audibility only or does the temporal processing deficit exist even when the audibility of stimulation is controlled. Thus to test these needs the present study was designed with the following objectives.

Aim of the study

- 1. To study the effects of cochlear hearing loss on brainstem response to speech.
- 2. To study the effects of stimulus presentation level (equal SL and equal SPL) on brainstem responses to speech.

Method

Participants of the present study were divided into two groups, experimental and control group. The control group included 22 ears of normal hearing individuals aged 16-50 years and hearing sensitivity with in 15dB HL. The experimental group included 22 ears with cochlear hearing loss of subjects aged from 16-50 years with hearing sensitivity within 55dB HL. Speech

identification scores of all 22 subjects were proportional to their pure tone average of 500, 1000 and 2000 Hz. There was no abnormality indicated on click evoked ABR and the absent TEOAE indicated the presence of cochlear pathology. The experimental group is further divided group I (PTA >15 dB HL & \leq 41 dB HL) and group II (PTA > 41 dB HL & \leq 55 dB HL).

Instrumentation

A calibrated diagnostic audiometer (GSI-61) was used for estimating the pure tone thresholds and a calibrated middle ear analyzer (GSI Tympstar) to rule out middle ear pathology. The brainstem responses to speech and click stimuli were recorded using Intelligent Hearing Systems (IHS Smart EP windows USB version 3.91) evoked potential systems. The Oto acoustic emissions were recorded using Intelligent Hearing Systems (IHS Smart TrOAE windows USB version 2.62) to check for the outer hair cell functioning.

Procedure

Stimulus used

The stimulus /da/, extensively used by Kraus and her colleagues, was used for recording the speech-evoked ABR. A Klatt formant synthesizer (Klatt, 1980) was used to synthesize a 40-msec speech-like /da/ syllable at a sampling rate of 10 KHz. The F0 changed from 103 to 120 Hz, F1 from 200 to 720 Hz, F2 from 1700 to 1240 Hz and F3 from 2580 to 2500 Hz. F4 and F5 remained constant at 3600 and 4500 Hz respectively. The time-amplitude waveform of the stimulus is shown in Figure 2.



Figure 2: The wave form representation of the stimulus /da/. Fundamental frequency (F0) is seen in periodicity of major peaks. The first formant (F1) is seen as periodically occurring smaller peaks.

Analysis of the ABR/ FFR recordings

The peak latency and the peak to trough amplitude of Wave V, A, C, D, E, F, O were measured. Fast Fourier Transform (FFT) was performed to obtain the information regarding spectral characteristics of the FFR - Frequency and Amplitude of spectral peaks. FFT was performed on all evoked potential recordings for an epoch of 15-54 ms using a custom-made program run in MATLAB platform. The Peak amplitude corresponding to F0 and F1 region was also calculated using a custom made program file in the MATLAB platform.

II. Recording of evoked potentials: The brainstem response to speech was recorded for speech using the test protocol given in table 1.

	Speech stimulus	/da/ (synthesized)		
Stimulus	Duration of the stimulus	40 msec		
	Speech stimulus levels	40dB SL and 80 dBnHL		
	Polarity	Alternate		
	Mode of presentation	Ipsilateral (monaural)		
	Repetition rate	9.1		
ters	Transducer	Insert ear phones ER-3A		
	Analysis time	70 msec (includes 10 ms pre-stimulus period)		
m	Band pass filter	30 to 3000 Hz		
ara	Electrode placement	Cz – Non-inverting (+ve);		
d u		Both mastoids – Inverting (-ve);		
itio		Forehead – Ground		
cquisi	Sweeps	1500		
	Electrode impedance	$< 10 \text{ k}\Omega$		
Ā	Inter-electrode impedance	$< 3 \text{ k}\Omega$		

Table 1: Stimulus and acquisition parameters used to record ABR and FFR

The figure 3 represents the brainstem response to speech recorded in normal hearing individual at 40 dBSL.



Figure 3: The recording of brainstem response to speech in a normal hearing individual at 40 dBSL

Objective Measures for Frequency Following Responses (using MATLAB platform)

The region following the onset responses was defined as the FFR. The spectral measures performed to analyze the sustained FFR (an epoch of 15-54 ms) were the amplitude of the spectral component corresponding to the stimulus fundamental frequency (F0 amplitude) and first formant (F1amplitude).

The sustained portion of the responses (FFR) was passed through 100 -120 Hz and 200 to 720 Hz band pass 4th order Butterworth filters in order to obtain the energy at fundamental frequency and first formant respectively. The Fourier analysis was performed on the filtered

signal. A subject's responses were required to be above the noise floor in order to include in the analysis. This was performed by comparing the spectral magnitude of pre-stimulus period to that of the response. If the quotient of the magnitude of the F0 and F1 frequency component of FFR divided by the pre-stimulus period was greater than one the responses was deemed to be above the noise floor. The raw amplitude value of the F0 and F1 frequency component of the response was then measured. This program was validated with recordings with known spectral characteristics.

Results and Discussion

To understand the effect of cochlear hearing loss and the severity of hearing loss on the brainstem responses to speech, the clinical group was divided into 2 groups, Group I (N = 11 ears) having PTA >15 dB HL & \leq 41 dB HL and Group II (N = 11 ears) having PTA > 41 dB HL & \leq 55 dB HL. The data obtained from the groups were then compared with the control group (N = 22 ears).

The tables 2, 3, 4 shows the mean amplitudes and latencies of the discrete peaks - waves V, A, C, D, E, F, O and the F0, F1 amplitude for normal hearing Group I and Group II respectively. The tables also include the results of paired sample t-test across the two presentational levels.

Table 2: Mean, SD and t-values of the various waves latency and amplitude of brainstem responses to /da/ at 80 dB nHL and 40 dB SL obtained in the Control group

Parameters		80 dB r	nHL	40 dB SL t-value		t values
		Mean	SD	Mean	SD	t-values
	Wave V	8.15	0.29	9.35	0.27	11.94*
	Wave A	9.09	0.31	10.82	0.42	15.07*
cy	Wave C	19.87	0.33	21.67	0.91	8.86*
iten	Wave D	26.66	0.58	29.15	0.84	11.60*
Γ_{c}	Wave E	37.25	0.54	39.49	0.86	10.52*
	Wave F	47.35	0.48	49.64	0.69	16.07*
	Wave O	56.95	0.69	59.09	0.47	12.41*
	Wave V	0.27	0.09	0.24	0.07	1.02
nde	Wave C	0.41	0.13	0.30	0.08	3.94**
plit	Wave D	0.48	0.12	0.33	0.12	4.43*
Am	Wave E	0.41	0.11	0.25	0.08	4.81*
	Wave F	0.44	0.13	0.34	0.11	2.58***
FFT	F0 amplitude	30.40	7.86	24.12	7.34	2.69***
	F1 amplitude	15.29	3.33	12.78	4.17	1.40

*p<0.001, **p<0.01, ***P<0.05

Parameters		80 dB nHL		40 dB SL		t voluce
		Mean	SD	Mean	SD	t-values
	Wave V	9.23	0.76	9.81	0.73	4.69**
	Wave A	10.35	0.77	10.80	0.66	3.23***
cy	Wave C	20.61	0.95	22.03	0.95	4.29**
aten	Wave D	28.32	1.09	29.70	1.04	3.25**
Ľ	Wave E	38.93	1.12	40.09	1.19	2.59***
	Wave F	48.77	0.99	49.84	0.87	2.94***
	Wave O	58.12	0.78	58.79	0.53	2.67***
	Wave V	0.32	0.12	0.24	0.13	2.11
ude	Wave C	0.28	0.13	0.24	0.10	2.34***
ıplit	Wave D	0.36	0.15	0.32	0.30	1.25
Am	Wave E	0.40	0.19	0.30	0.12	1.90
	Wave F	0.30	0.16	0.27	0.15	1.29
FF T	F0 amplitude	23.73	7.89	23.99	8.49	0.51
	F1 amplitude	15.10	5.19	13.05	3.42	1.64
*p<0.001, **p<0.01, ***P<0.05						

Table 3: Mean, SD and t-values of the various wave latency and amplitude of brainstem responses to /da/ at 80 dBnHL and 40 dB SL in Group I

Table 4: Mean, SD and t-values of the various waves latency and amplitude of brainstem response to /da/ at 80 dB nHL and 40 dB SL in Group II

Parameters		80 dB nHL		40 dB SL		t voluos
		Mean	SD	Mean	SD	t-values
	Wave V	10.30	0.73	9.91	0.48	3.94***
	Wave A	11.57	0.98	10.83	0.76	3.80**
cy	Wave C	22.33	0.79	23.54	1.13	2.25
ten	Wave D	29.79	1.03	29.95	1.00	0.53
La	Wave E	39.86	1.07	40.97	1.26	0.76
	Wave F	50.03	1.22	50.92	1.66	2.09
	Wave O	58.20	0.56	58.96	0.81	2.81***
	Wave V	0.21	0.05	0.21	0.10	1.74
nde	Wave C	0.26	0.09	0.24	0.05	0.40
plit	Wave D	0.28	0.05	0.22	0.076	2.3
Am	Wave E	0.23	0.08	0.22	0.05	0.69
	Wave F	0.21	0.08	0.21	0.05	0.12
FFT	F0 amplitude	15.25	4.00	14.94	4.59	0.15
	F1amplitude	8.90	2.28	10.03	2.53	1.4

*p<0.001, **p<0.01, ***P<0.05

The peaks D, E, F which are considered as the sustained brainstem responses occurred periodically at a periodic interval of approximately 10 msec. This time period when converted into frequency values (Frequency = 1/time period) it correlated with the F0 of the speech stimuli (100 Hz). Russo, Nicol, Musacchia & Kraus, (2004); Kraus, Nicol, (2005) reported that the peaks D, E, F in the sustained FFR represents the vibration of the vocal folds i.e., the F0 of the speaker.

The results showed that this periodicity was coded effectively in normal hearing individuals and group I (minimal to mild hearing loss). However, in group II high variability in the standard deviation of these peaks latency and absences of identifiable responses in certain individuals could be the indication of inaccurate coding of F0 and its harmonics.

The comparison across the presentation level revealed a significant increase in the latency and decrease in the amplitude of the responses when the presentational level was varied from 80 dBnHL to 40 dBSL in normals for all wave parameters except for wave V amplitude. However, Group I showed significant difference in most of the wave parameters except for the wave V, D, E and F amplitude and Group II showed no significant difference for most of the parameters except for the wave V, A, and O latency.

Decrease in the latency with an increase in the stimulus intensity is due to a progressively faster rising generator potential within the cochlea and similarly faster development of excitatory post synaptic potential (Moller, 1981). Latency of the compound action potential directly depends on how quickly the generator potential and the excitatory post synaptic potential reach the threshold for firing leading to reduced wave latency.

Increase in the amplitude parameters with the increase in the stimulus intensity may be because of the increase in the audibility of the stimulus. This supports the finding by Hall (1992) where he says that the Auditory evoke potential amplitude increases with the increase in the intensity. The amplitude of an AER is decided by the number of neurons firing for particular stimulus intensity. At higher intensities the number of neuron beginning to fire will be more and amplitude of the compound action potential thus generated will be high. This had resulted in the high amplitude evoked responses.

In the clinical group some parameters did not show a significant difference across two presentation level because of little difference across the presentation level and this was negligible in the group II who had higher thresholds. It could also be due to a high variability in most of the parameters in the participants.

The F0 and F1 amplitude given in the tables clearly shows that the F0 region has the greatest amount of response energy compared to its harmonics at both the presentation level which is consistent with the study done by Russo, Nicol, Musacchia and Kraus (2004). They reported that F0 region in the responses showed a greater energy compared to its harmonics. It is due to the high energy level in the F0 region of the stimulus (Ladefoged, 1996) and also due to the better phase locking of the lower frequencies (Gelfand, 1998).

Comparison between the presentation level using the paired sample t-test in normal hearing individuals showed a significant reduction in the response amplitude in the F0 region when the presentation level was varied from 80 dB nHL to 40 dB SL but the reduction was not significant in the F1 region.

The reduction in amplitude may be due to the reduction in the amount of acoustic energy reaching the neurons at 40 dB SL compared to 80 dB nHL. The clinical group revealed no significant decrease in the F0 and F1 amplitude with the change in the presentation level. This could be due to the little differences across the presentation level. Also, the indifference between the low and high intensity values may attributed to the disturbed intensity processing in the hearing loss group (Florentine, et al., 1993).

Comparison across groups at equal Sensation Levels (40 dB SL)

Kruskal-Wallis test was carried out to check whether there is any significant difference between the three groups. The Mann-Whitney U test was carried out for those parameters which revealed significant difference with the Kruskal- Wallis test to check whether the Groups I and II differed significantly from that of the control group.

romotoro	z-values				
Tameters	Control Vs Group I	Control Vs Group II	Group I & II		
Wave V	-1.12	-2.98**	-0.28		
Wave F	-0.86	-2.09***	-1.63		
Wave D	-0.13	-2.61**	-2.39***		
Wave F	-1.62	-3.05**	-0.41		
F0 amplitude	-0.30	-3.25**	-2.81**		
F1 amplitude	-0.30	-2.03***	-2.25***		
	rameters Wave V Wave F Wave D Wave F F0 amplitude F1 amplitude	rameters Control Vs Group I Wave V -1.12 Wave F -0.86 Wave D -0.13 Wave F -1.62 F0 amplitude -0.30 F1 amplitude -0.30	z-values z-values Control Vs Group I Wave V -1.12 -2.98** Wave F -0.86 -2.09*** Wave D -0.13 -2.61** Wave F -1.62 -3.05** F0 amplitude -0.30 -3.25** F1 amplitude -0.30 -2.03***		

Table 5: Z-values between the groups at 40 dB SI
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*p<0.001, **p<0.01, ***P<0.05

Results of Kruskal-Wallis test for the latency and amplitude parameters of discrete peaks and the F0, F1 amplitude revealed a significant difference between the three groups (Control group, Group I and Group II) for the latencies of wave V and F; wave D and F amplitude; and the amplitude of the F0. Table 5 shows the results of the Mann-Whitney U test for the pair wise comparison of the control group, group I and group II for the latencies of waves V and F; amplitudes of waves D and F; and amplitudes of F0 and F1.

Results of Mann Whitney U test revealed no significant difference between the group I and the control group for all the parameters. However, there was a minimal increase in the latency and reduction in the amplitude for the group I. This may be due to the lesser degree of hearing loss which has minimal or no effect in the temporal processing. This is consistent with the study done by Bus, Hall and Grose (2004). They reported that individuals with mild cochlear impairment are minimally affected in coding temporal fine structure compared to individuals

with moderate cochlear impairment. Also few of the mild hearing loss individuals in their study had near normal performance in temporal fine structure coding.

The group II revealed a significant amplitude reduction and latency prolongation when compared with individuals with normal hearing. Also the F0 and F1 amplitude showed a significant reduction. This could indicate reduced temporal processing in higher degree of hearing loss. This supports the study done by Lorenzi, Gilbert, Carn, Garnier and Moore (2006) who reported that both young and elderly subjects with moderate cochlear hearing loss performed very poorly with temporal fine structure speech which is very important for the coding of F0 and its formants. This loss of ability to use temporal fine structure information perhaps was related to a loss of neural synchrony (Woolf, Ryan & Bone, 1981).

Comparison of Group I and II showed a reduction in wave D amplitude and the F0 and F1 amplitude with the increase in severity of hearing loss. This indicated that the degree of hearing loss has an effect on temporal processing and coding temporal fine structure of speech (Lorenzi, Gilbert, Carn, Garnier, & Moore 2006; Moore & Moore, 2003). Effect of degree of hearing loss on temporal fine structure coding can be understood from the study done by Bus, Hall and Grose (2004). Their data revealed that individuals with mild cochlear impairment are minimally affected in coding temporal fine structure compared to individuals with moderate cochlear impairment. Also a few of the mild hearing loss individuals in their study had near normal performance in temporal fine structure coding.

Overall we can conclude that though the audibility of the stimulus was same across the three groups, still the clinical group had some deficit in coding information at the auditory nerve which was reflected in the latency and amplitude measures. The minimal to mild hearing loss group had minimal loss of information and were almost similar to the normal group. This deficit was more pronounced in the moderate hearing loss group.

Comparison across the groups at equal Hhearing levels (80 dB nHL)

Results of the Kruskal-Wallis test revealed significant difference between the 3 groups for all parameters except for the wave V amplitude. Table 6 shows the results of the Mann-Whitney U test for the pair-wise comparison of all parameters for between the groups. Results of Man Whitney U test showed a significant difference between the group I and control group for most of the parameter except the Wave E amplitude, F1 amplitude. As expected the normals had shorter latencies and higher amplitude of the peaks compared to the Group I. This is due to higher audibility in normal hearing individuals compared to group I. Also, there was a significant reduction in the F0 amplitude in the group I. Though F1 amplitude showed a slight reduction in amplitude in Group I it failed to show any significant difference.

In group II the wave latencies increased and the amplitude reduced significantly in the compared to control group. Also, there was a drastic reduction in the F0, F1 amplitude. This suggests that the inadequate audibility would affect the temporal processing to a great extent in moderate hearing loss group.

Parameters		<i>z</i> -values				
		Control Vs Group I	Control Vs Group II	Group I Vs Group II		
	Wave V	-3.46**	-3.93*	-1.96		
	Wave A	-3.58*	-3.95*	-2.49***		
	Wave C	-2.21***	-4.07*	-3.11**		
Latency	Wave D	-3.88*	-3.99*	-2.73**		
	Wave E	-2.83**	-4.14*	-1.53		
	Wave F	-3.54*	-4.04*	-1.98***		
	Wave O	-3.63*	-3.76*	0.00		
	Wave C	-2.58***	-2.95**	-0.20		
Amplitudo	Wave D	-1.98***	-3.82*	-1.11		
Ampiltude	Wave E	-0.591	-3.36**	-1.87		
	Wave F	-2.16***	-3.70*	-1.24		
EET	F0 amplitude	-2.36***	-4.27*	-2.74**		
1.1.1	F1 amplitude	-0.53	-3.57*	-2.60**		
		*** <0.001 **** <0.0)1 ***D $< 0 \frac{05}{05}$			

Table 6: Z-values between the groups at 80 dB nHL

*p<0.001, **p<0.01, ***P<0.05

Comparison between group I and group II revealed increase in the latency and decrease in the amplitude of all parameters though significant difference was seen only for wave A, C, D, F latency. A significant reduction in F0, F1 amplitude was also seen in group II. This again shows that as the hearing loss increases the audibility reduces and this would have affected the temporal processing and F0, F1 coding.

Conclusion

To conclude the comparison across the groups at equal hearing level were done in order to see the kind of difficulties that the hearing impaired individuals will face in day to day situation. As we know that in day to day situation both normal and hearing impaired individuals will be exposed to sounds at equal hearing levels and not equal sensation level. From the results above it is clear that as the degree of hearing loss increases the temporal processing degrades due to reduced audibility or could be due to the altered physiology of the inner ear. Thus, in day to today situation hearing impaired individuals might miss out lot of the temporal cues which essential for the speech perception. The cochlear hearing loss individuals will most often have degraded coding of F0 and its harmonics and this is more pronounced for a higher degree of hearing loss.

From this we can conclude that as the degree of hearing loss increases the ability to process temporal fine structure of speech degrades, thus, compromising the speech intelligibility in quiet as well as in adverse environments.

Clinical implications

1. Brainstem responses to speech syllables can throw more light to understand the role of brainstem processing of speech sounds.

- 2. FFT analysis of the brainstem responses is a useful tool in detecting deficits in speech sound processing. Amplitudes of F0 and F1 peaks proven to be useful for this type of evaluation.
- 3. It can be used as an objective tool to assess temporal processing in difficult to test population.
- 4. It can also be used as a tool for hearing aid selection or to check for benefit from hearing aid or rehabilitation.

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