

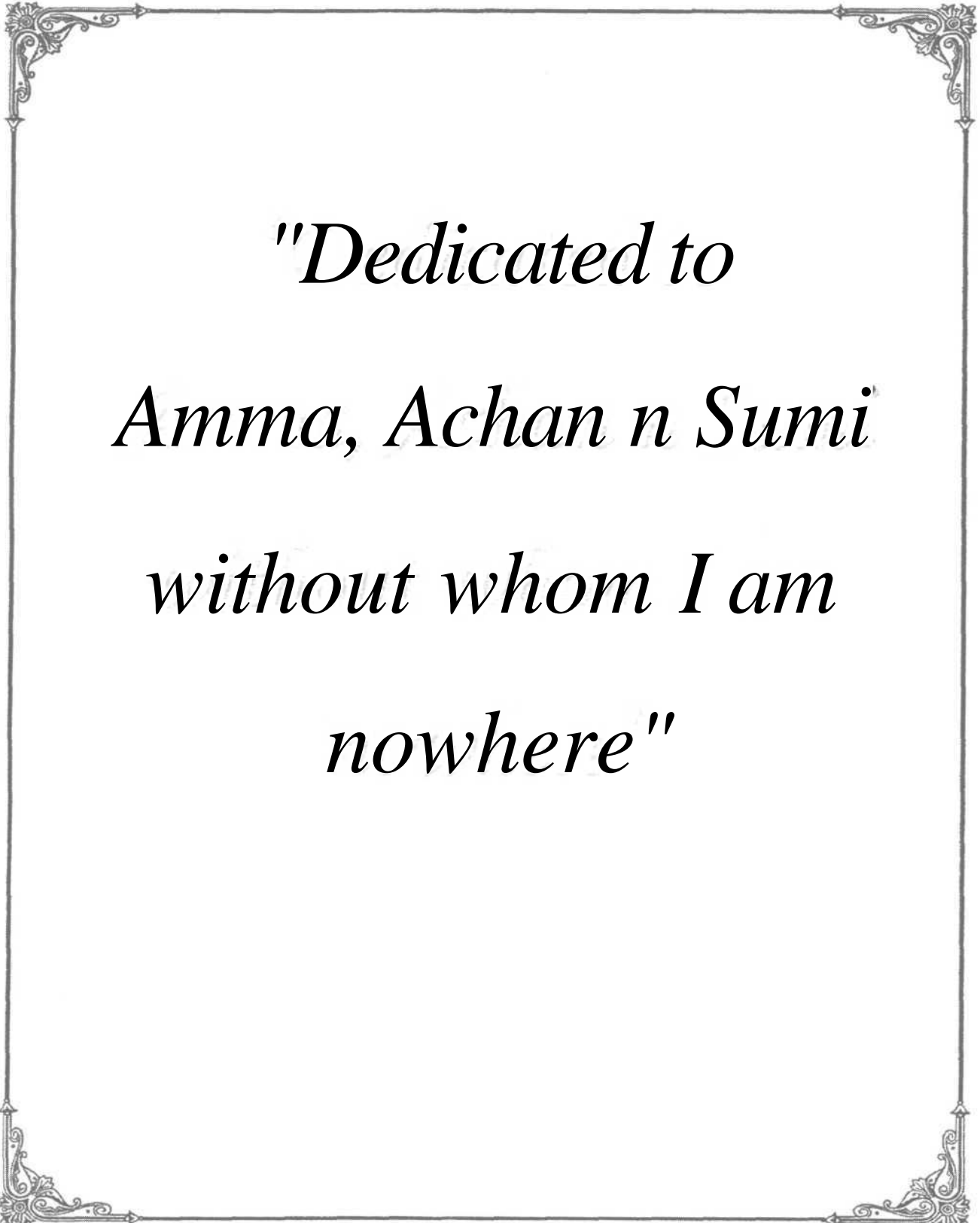
**BRAINSTEM RESPONSES TO SPEECH IN NORMAL
HEARING AND COCHLEAR HEARING LOSS INDIVIDUALS**

**Register Number: 05AUD017
Sumesh . K**

**A dissertation submitted in part fulfillment for the degree of
Master of Science (Audiology)
University of Mysore, Mysore.**

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MANSAGANGOTHRI, MYSORE-570 006**

APRIL 2007.

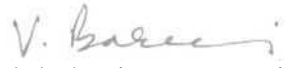


*"Dedicated to
Amma, Achan n Sumi
without whom I am
nowhere"*

CERTIFICATE

This is to certify that this dissertation entitled "*Brainstem responses to speech in normal hearing and cochlear hearing loss individuals*" is a bonafide work submitted in part fulfillment for the degree of Master of Science (Audiology) of the student with Registration No: 05AUD017. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any diploma or degree.

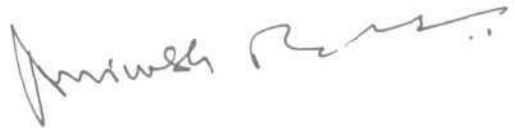
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CERTIFICATE

This is to certify that this dissertation entitled "*Brainstem responses to speech in normal hearing and cochlear hearing loss individuals*" has been prepared under my supervision and guidance. It is also certified that this dissertation has not been submitted earlier to any other university for the award of any diploma or degree.



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DECLARATION

This is to certify that this master's dissertation entitled "*Brainstem responses to speech in normal hearing and cochlear hearing loss individuals*" is the result of my own study and has not been submitted earlier to any other university for the award of any degree or diploma.

Mysore
April 2007

Register Number: 05 AUD017

ACKNOWLEDGEMENTS

I extend my heartfelt gratitude to my teacher, my guide Mr. Animesh Barman for all the support, guidance, help, motivation through out this project and through out my AIISH days. Your patience and dedication to work and research never failed to amaze me! You the person whom I am most comfortable to work with, I will surely miss your scoldings sir. . . .

I am thankful to Dr. Vijayalakshmi Basavaraj, Director, AIISH, for permitting me to conduct this study.

My sincere thanks to Dr. K. Rajalakshmi, HOD, Dept. of Audiology, AIISH for permitting me to use the department facilities for my data collection. You are a person who I always love to adore. Thanks a lot ma'm.

My sincere thanks to Prof. C.S.Vanaja, Ms P. Manjula, Prof. Asha Yathiraj for their guidance, support, through out my academic years and for enlightening my knowledge in the field of Audiology.

I am also thankful to, Dr. Sundaraju, Dr. Venkateshan, Dr. G. Jayaram, Prof. Savithri, Prof. K.C. Shyamala & Ms. Yeshoda for their help and support in innumerable ways through out my AIISH years. 'Thanks' is too small a word for your unconditioned encouragement, faith and support, which has made me a different individual,

I thank all the participants of this study for having accepted to under go the long process of the study with lot of enthusiasm and support.

I thank Nicol Russo, Northern Western University for her kindly help and support through out my dissertation.

Dearest amma & Achan. . . U r the best, n no one can have a better mom and dad as mine. I thank god for choosing me to be ur son. What I am now is jus a mere reflection of your love, encouragement, belief, guidance, blessings and support with which I have grown up. I can never thank you . . .

Dearest Sumi, I might have achieved a lot in my life, but my life would have stayed incomplete but for a sweet sister like you. Since childhood u stayed as my role model, my inspiration, my friend, what not. God was very partial towards me in making you as my sister. . . .u r so sweet!

Dear Prakash etta,.. Thanks for your immeasurable love, affection, care and support you have been showing towards me.

Dearest Reddy sir, the greatest friend of mine, who always wants me to achieve the best in my life. . . . Nothing equals the support, care, and encouragement you show towards me sir.

Friends are like Crayons. Vinaya, Kanthi, , Nambi (vengal), Kishna, KD, Yatin (punjabi). . . you guys are vibrant colors of my box of crayons. I am blessed to have such smart and caring friends.

Alfi, Thanks for being with me all the time. Rest is self explanatory rite....

Pavan, Keerthi, & dear Kid... my dear little juniors who have always supported me, believed me, helped me., shared my best n worst times with me. It was a great time being with you. U all mean a lot to me.

Rahana, Sheetal, Neethi, Pri, I need not thank you all. You were really great and hope that we stay as friends forever.

My heartfelt thanks to Vijay sir and sandeep sir for being with me when ever I needed help. Found a good friend in you and hope this relation stay forever.

My sincere thanks and gratitude to Ajith sir, & vinay sir for their kind and priceless support. You will stay as an inspiring model for us forever

My dear friends Niraj, Radz,, Puru Sushmit, Bijan, Manas, Vijay, Ravi, Abhay, Kunal, Ani, palash... it was a real fun filled stay with you guys. Learnt a lot from you all, gained lot of love & support. So glad to be your friend

How can I forget my posting mates Dg (paapi), shibasis (kannada teacher), My bees (shruthi, gungi, Pri). Had a great time with you all during postings and would wish that our relation should not be constrained to just postings....

My class beauties (Anusha, Swapu, Deeps) you all were so lively in the class. It was really great time with you all,

Thanks to my Msc and Bsc Clasmates we had a memorable and funfilled time together...

Thanks is also due to all the tiny tots for making my stay at AIISH memorable Ismail, Sudipto, Dhiraj, Sangamesh, Arun, Poorna, Vivek, Sriram, Ramesh, Naresh, Balaji, karthi, jukku, Kishoru, Manuj, Chandrakant, Ankit, Priyanka, Achaya, Mohan, Akshay, Vipin the list is endless...

Thanks Ms. Vasantha lakshmi for helping me out with the statistics

Thanks the Librarians, Mr. Lokesh, Mr. Nanjundaswamy, Mr. Mahadevappa and Raju fpr their timely help and support.

Thanks Mr. Shivappa & Co., for the kind cooperation throughout.....

Above All I thank ALL MIGHTY, for showering his abundant blessing on me at each step of my life....

TABLE OF CONTENTS

CHAPTER	TITLE	PAGE NO
I	INTRODUCTION	1
II	REVIEW OF LITERATURE	6
III	METHOD	22
IV	RESULTS AND DISCUSSION	35
V	SUMMARY AND CONCLUSION	54
	REFERENCES	59

LIST OF TABLES

TABLE No.	DESCRIPTION	PAGE No.
1	Parameters used for immittance evaluation	24
2	Stimulus and acquisition parameters used to record click ABR	25
3	Stimulus and recording parameters used to record speech evoked ABR/ FFR	27
4	The Mean, Standard Deviation and t-values of the various latency and amplitude parameters of brainstem responses to speech at 80 dB nHL and 40 dB SL obtained in the Control group.	37
5	The Mean, Standard Deviation and t-values of the various latency and amplitude parameters of brainstem response of speech at 80 dB nHL and 40 dB SL in Group I.	41
6	The Mean, Standard Deviation and t-values of the various latency and amplitude parameters of brainstem response of speech at 80 dB nHL and 40 dB SL in Group II.	43
7	The mean and Chi-square values across the three groups for various parameters recorded at 40 dB SL.	46

8	The Z values (Mann Whitney U test) between the control group, Group I and Group II	47
9	The mean and Chi-square values across the three groups for various parameters recorded at 80 dB nHL.	50
10	The Z values (Mann Whitney U test) between the control group, Group I and Group II at 80 dBnHL	51

LIST OF FIGURES

FIGURE No.	DESCRIPTION	PAGE No.
1	Response waveform showing the wave V followed by the negative peaks A, C, D, E and F.	3
2	The stimulus waveform and the brainstem responses to speech stimulus /da/	13
3	The spectral analysis of FFR showing maximal amplitude at FO, followed by F1 in quiet and in presence of background noise	14
4	The wave form representation of the stimulus /da/.	26
5	The recording of brainstem response to speech in a normal hearing individual recorded at 40 dBSL.	30
6	The upper panel shows the brainstem response to speech and the lower panel shows the portion of the sustained responses in the brainstem responses considered for the objective measure	32
7	The FFT of the sustained portion of the brainstem response to speech	32

8	The 500 Hz tone burst artifact recorded at 90 dBnHL, and FFT of the 500 Hz tone burst artifact	33
9	The 1000 Hz tone burst artifact recorded at 90 dBnHL, and FFT of the 1000 Hz tone burst artifact	34

CHAPTER I

INTRODUCTION

Sensory experiences of all types contribute to how an organism will react to the surrounding environment. For example, a distinct odor will warn a predator not to attack a skunk; a moving shadow will allow a hawk to spot prey; touching a hot pan will warn a child of danger. Appropriate reaction is an important outcome to almost all events, and sensory systems are especially equipped to respond well to rapidly occurring stimuli exhibiting distinct temporal features. Exposure to sensory stimuli from all modalities is important from an evolutionary standpoint. However, there is one exceptional stimulus which, although not mandatory for survival, is an essential part of everyday life: speech.

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.

Frequency encoding is manifested in speech-evoked auditory responses both in the latency (Steinschneider et al., 1993; Martin et al., 1997; McGee et al., 1996) and the amplitude (Steinschneider et al., 1995) 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 interpeak intervals between these peaks correspond precisely to the wavelength of the F0 of the utterance. Wave O is a response to the cessation of sound. The small higher-frequency fluctuations between waves D, E and F,

corresponds in frequency to that of the first formant (F1) of the stimulus, which, along with F2, primarily shapes the vowels.

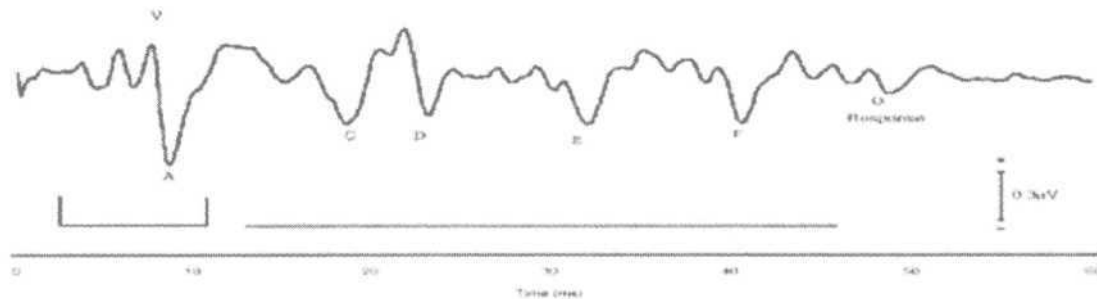


Figure. 1: depicts the wave V followed by the negative peaks A, C, D, E and F. The onset response is bracketed, while the region containing the FFR is indicated with a horizontal line

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, 2001; Cunningham, Nicol, Zecker, Bradlow, & Kraus, 2001). Some also experience abnormal susceptibility to the demands placed on the auditory system by rapidly presented temporal information (Wible, Nicol, & Kraus, 2002; Nagarajan, Mahncke, Salz, Tallal, Roberts, & Merzenich, 1999).

Plyler and Ananthanarayan (2001) studied whether FFR can encode the time-varying second formant transitions in synthetic stop consonant stimuli in normal-hearing and hearing-impaired listeners (age range 20 to 67 yrs). The results demonstrated that the FFR did encode the second formant transition in normal-hearing listeners. However, FFR encoding was severely degraded in most of the listeners with a hearing loss.

In addition, a major difference between the onset and FFRs measured was noted under a stressed circumstance like speech in noise. 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.

Need for the study

Individuals with cochlear hearing loss have consistently shown difficulties in perceiving place (Revoile, Pickett, Holden-Pitt, Talkin & Brandt, 1987) and manner cues (Danahauer, 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.

Most of the objective studies to understand the processing deficits are done using late evoked potentials like MMN. Studies have shown that cochlear hearing loss have its manifestations as prolonged latencies and reduced amplitude of auditory late latency potentials such as the mismatch negativity recorded for speech (Oates, Kurtzberg &



Stapells, 2002; Sivaprasad, 2006). These studies imply the presence of neural coding deficits at the level of auditory cortex.

However, it is very important to understand whether these individuals 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 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.
- 3) To establish norms for the brainstem responses to speech.

CHAPTER II

REVIEW OF LITERATURE

Human auditory system is designed not only to pass on the external sounds to the brain but also to analyze them during the transmission. Analysis of the incoming sound starts with simple frequency analysis to very complex analysis of binaural inputs and extracting signals from the surrounding background noise. The auditory system is equipped with structures ranging from mere transmitters to neurons responding to selective sound signals. The auditory system is mainly divided into peripheral and central system. The peripheral auditory system consists of structures outside the brainstem or brain - that is the external ear, the middle ear, the inner ear, and the cochlear nerve. The external ear provides directional cues and transfers these cues to the middle ear with in turn covert these sound waves into mechanical vibrations and transmits to the inner ear. The cochlea transforms the mechanical vibrations into electrical and sends it to the cochlear nerve toward the central auditory pathway.

The central portion of the auditory system encompasses all the auditory structures located beyond the cochlear nerve. It consists of nuclei, fibers, tracts and commissures in ipsilateral and contralateral pathways. The levels in the auditory pathway are interlinked and if any problem arises at any point, then the auditory perception can be affected. These problems can arise not only in children but also be seen in elderly individuals. Some of the speech understanding difficulties expressed by elderly adults may be related to impaired temporal precision in the aging auditory system. This might explain why

older individuals with normal peripheral hearing encounter difficulty in understanding speech in unfavorable listening conditions.

The recent development of neuroimaging techniques such as PET, fMRI and auditory evoked potentials such as FFR and MMN enables the study of sound processing in auditory pathway.

Auditory Evoked Potentials

An auditory evoked potential (AER) is activity within the auditory system (the ear, the auditory nerve, or auditory regions of the brain) that is produced or stimulated by sounds. In the simplest term AER are brain waves generated when a person is stimulated with sounds. These sounds may range from click to tones, and even speech sounds. Evoked potential recording is a non-invasive method that has been widely used by researchers as a tool for threshold estimation in the difficult to test population, to find out the site of lesion and subtle auditory processing deficits. These tests have been also used to study the processing from many discrete and neural generating sites along the auditory pathway from the cochlea to the cerebral cortex. The cortical responses (N1 P2, MMN, and P300) and the brainstem responses (ABR, FFR) are some tools which can be used to study the processing at the cortical and brainstem levels respectively.

Brainstem responses to sounds

Recording brainstem responses to sound has long been established as a valid and reliable means to assess the integrity of the neural transmission of acoustic stimuli. Transient acoustic events induce a pattern of voltage fluctuations in the brain stem resulting in a familiar waveform, yielding information about brain stem nuclei along the

ascending central auditory pathway (Hood, 1998; Jacobson, 1985). An accurate manifestation of stimulus timing in the auditory brain stem is a hallmark of normal perception (Sininger & Starr, 2001).

Brainstem responses for non-speech stimuli

Auditory brainstem responses

The work of Jewett and Williston (1971); Jewett et al, (1970); Lev and Sohmer, (1972) was the first to definitively describe far-field scalp-recorded auditory brainstem responses (ABR). The ABR is a phasic response to a transient acoustic event (Click, Tone burst), occurring within the initial 10-15 ms after the event i.e., the stimulus onset. In the years since Jewett's work, advancements in recording and analysis techniques, in combination with corroborating evidence of generator loci from animal, imaging, and intra-operative studies, have fostered development of ABR into a highly sensitive index of the integrity of the auditory periphery and brainstem. This sensitivity results from the high replicability and temporal precision of ABR components commonly identified as waves I—VII, demonstrated to represent activity at distal auditory nerve (I), proximal auditory nerve (II), cochlear nucleus (III), superior olivary complex (IV), lateral lemniscus (V) and inferior colliculus (Vn, VI, VII) (Hall, 1992).

The replicability of these features within and across subjects, their relatively early maturation and their independence from higher cognitive function (e.g., they can be recorded in sleeping or anesthetized subjects), have allowed establishment of normative data to which subjects suspected of auditory dysfunction can be compared. ABR as a measure has been used successfully in threshold estimation (Galambos & Hecox, 1978;

Smith & Simmons, 1982; Sorensen, Christensen & Parving, 1988; Swoboda-Brunner, Swoboda, Neuwirth-Riedl & Turk, 1989) as well as site of lesion testing (Selters & Brackmann, 1977; Chandrasekhar, Brackmann & Devgan, 1995; Selesnick & Jackler, 1992; Welling, Glasscock, Woods & Jackson, 1990; Barrs, Blackmann & Olsen, 1985; Jerger, Oliver, Chmiel & Rivera, 1986; Starr et al, 1996).

Frequency Following Brainstem Responses (FFR)

Continuous presentation of low-frequency tone stimuli can produce a scalp-recorded evoked response of the same frequency. Moushegian, Rupert and Stillman (1973) adopted the term 'frequency following response' abbreviated FFR, to refer to this type of evoked response. They recorded the scalp response to low frequency sinusoidal signal using signal averaging techniques. These scalp potentials were periodic, following the frequency of the signal upto 2 kHz and were abolished in the presence of masking noise. Their onset latency was approximately 6 msec, which is consistent with an upper auditory brainstem source. Smith (1975) tested this hypothesis by comparing FFR recorded from the scalps of cats with FFR recorded from brainstem nuclei of the same animals. The mean latency (5.3 msec) of FFR recorded from the Inferior Colliculus (IC) was found to be similar to the latency of the scalp potential (5.6 msec). The similarity in latencies is consistent with the IC being the primary source of the scalp recorded FFR.

Greenberg et al (1986) recorded FFR for two types of complex tones lacking the fundamental frequency and pure tones equal in frequency to the missing fundamental. The stimuli were (a) four component complex tones, consisting of the second through fifth harmonics of a common fundamental (244, 366, or 488 Hz) and (b) sinusoidal

signals equal in the frequency to the fundamental of the complex tones. The results clearly showed that the complex tones generated FFR whose predominate energy is centered at the frequency of the fundamental despite the fact that this component was absent in the stimuli. The FFR to the sinusoidal component, although similar to the potential generated by the complex tone of equivalent fundamental frequency, differs with respect to response latency and amplitude.

Krishnan and Parkinson (2000) investigated the FFRs to a rising and a falling tone in 8 normal-hearing adults at 95, 85, 75 and 65 dBnHL. Their results clearly demonstrated that the human FFR does indeed follow the trajectory of the rising and falling tones. Also, amplitude changes in the FFR supported the view that neural phase locking decreases with increasing frequency. The relatively smaller FFR amplitude for the falling tone compared to its rising counterpart lends further support to the notion that rising tones produce greater neural synchrony than falling tones. These results indicated that the human FFR may be used to evaluate encoding of time-varying speech sounds like diphthongs and certain consonant-vowel syllables.

Brainstem responses for speech stimuli

Speech evoked ABR/FFR

Sivaprasad, Kumar and Vanaja (2004) recorded ABR waveforms for click and speech burst in normal hearing individuals. The speech stimuli they used were the burst portion of the stop consonant /tha/, /ta/, /pa/, /ka/. Further, they analyzed the latency and peak to peak amplitude of wave V and found the mean latency for the click as 5.61 ms also for speech burst present at 6.18 ms in normal hearing adults. Also they reported that

the place cues were reflected in the wave V latencies. Hence they argued that brainstem plays a role in speech sound processing.

Speech stimuli have been extensively used in humans to study the response characteristics of the frequency following response (FFR) (Galbraith et al., 2004; Galbraith, Arbagey, Branski, Comerci, & Rector, 1995; Krishnan, 2002; Krishnan, Xu, Gandour, & Cariani, 2004). Galbraith et al. (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 and colleagues (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 affective for forward speech stimuli characterized by highly familiar prosodic and phonemic structure, than to backward speech.

Animal models have been used to describe auditory nerve and cochlear nucleus single-unit response properties for synthetic speech-like sounds (Delgutte, 1984; Delgutte & Kiang, 1984a, 1984b, 1984c, 1984d; Young & Sachs, 1979). The studies indicated that not only do auditory nerve and cochlear nucleus fibers show increased phase-locked activity to the formant harmonics in the stimulus, but separate populations of neurons appear to encode the first and second formant. Neural encoding of speech in more rostral structures such as the lateral lemniscus and inferior colliculus has not been studied extensively. Moreover, based on the phase-locking limitations of these structures, it is assumed that neural encoding of the periodic acoustic properties of speech at such rostral areas would be limited to temporal events well below the second formant. The FFR arises from the harmonic portion of the stimulus, is characterized as a series of transient

neural events phase locked to periodic information within the stimulus (Batra, Kuwada, & Maher, 1986; Marsh & Worden, 1968; Sohmer & Pratt, 1977).

Krishnan (1999) recorded FFRs to three different two-tone approximations of vowels were obtained from 10 normal-hearing human adults at 85, 75, 65 and 55 dBnHL. Spectrum analyses of the FFRs revealed distinct peaks at frequencies corresponding to the first and the second formants across all levels suggesting that phase-locked activity among two distinct populations of neurons are indeed preserved in the FFR. Also, the FFR spectrum for vowels revealed a robust component at $2F_1-F_2$ frequency suggesting that the human FFR contains a neural representation of cochlear nonlinearity. Comparison of FFRs to the vowel approximations and the individual components at F_1 and F_2 revealed effects that may be suggestive of two-tone synchrony suppression and/or lateral inhibition. They also suggested that scalp-recorded FFR may be used to evaluate not only neural encoding of speech sounds but also processes associated with cochlear nonlinearity.

Krishnan (2002) evaluated FFRs to the more complex steady-state synthetic English back vowels (/u/, /ɔ/, and /a/). FFRs were obtained from 10 normal-hearing human adults at 85, 75, 65, and 55 dB nHL. Spectrum analyses of the FFRs revealed distinct peaks at harmonics adjacent to the first and the second formants across all levels suggesting that phase-locked activity among two distinct populations of neurons is indeed preserved in the FFR. For each vowel the spectral peaks at first formant harmonics dominated the spectrum at high stimulus levels suggesting formant capture. The observation of less robust peaks for harmonics between the formants may very well suggest selective suppression to enhance spectral peaks at the formant frequencies.

Kraus and Nicol (2005); Russo, Nicol, Musacchia, and Kraus (2004); King, Warder, Hayes, and Kraus (2002); Johnson, Nicol, Zecker, and Kraus (2007) studied the brainstem response to a speech stimulus /da/ of 40 msec in duration. The consonant contained an initial 10 ms burst; the frequencies of which were centered around the beginning frequencies of formants 3-5, thus in the range of 2580-4500 Hz and the F0 of the utterance, ramping from 100 Hz to 120 Hz.. The response waveform (figure 2) includes transient peaks as well as sustained elements that comprise the FFR. The response to the onset of the speech stimulus /da/ includes a positive peak (wave V), likely analogous to the wave V elicited by click stimuli, followed immediately by a negative trough (wave A).

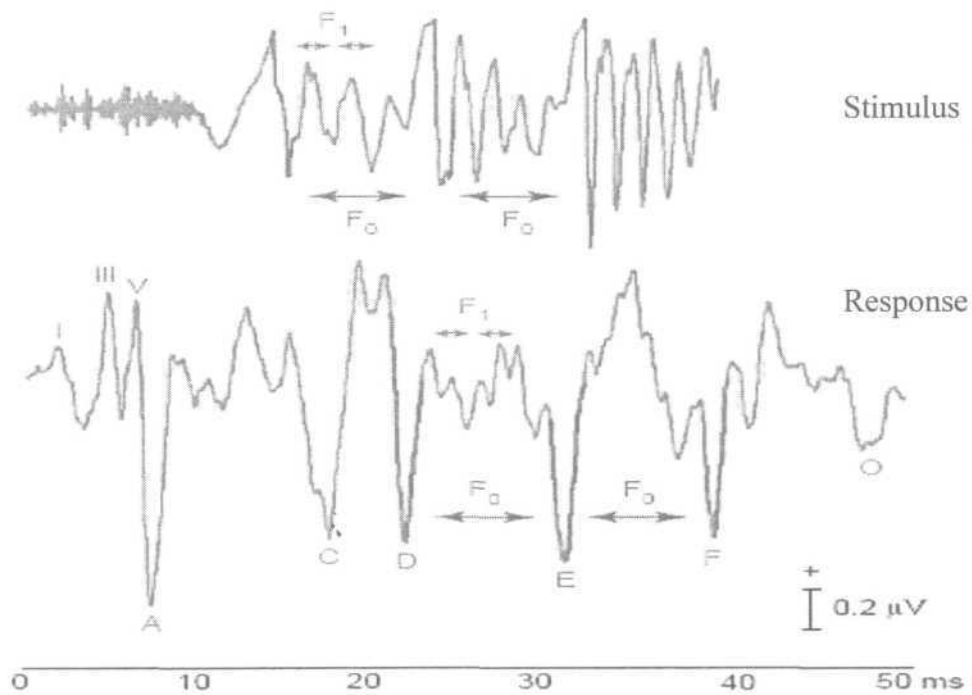


Figure 2: The stimulus waveform and the brainstem responses to speech stimulus /da/

Wave C is probably a response to the onset of the vowel - the release of the tongue from the roof of the mouth. Wave O is a response to the cessation of sound.

Together, these transient peaks, and small higher-frequency fluctuations between waves D, E and F are sensitive to stimulus spectrum. The spacing between the high frequency fluctuations corresponds in frequency to that of the first formant (F1) of the stimulus, which, along with F2, primarily shapes the vowel sound /a/.

Peaks - D, E and F represent vibrations of the vocal folds and are considered as the sustained responses. The defining feature of the sustained portion of the response is its periodicity, which follows the frequency information contained in the stimulus. Neural conduction accounts for a delay of approximately 7ms between stimulus and response. The fundamental frequency occurs at approximately 15msec, 24msec and 33msec in stimulus corresponding to wave D (22msec), E (31msec) and F (40msec) in response. These FFR peaks involve the encoding of periodicity, and are prominent enough to provide reliable latency measurements.

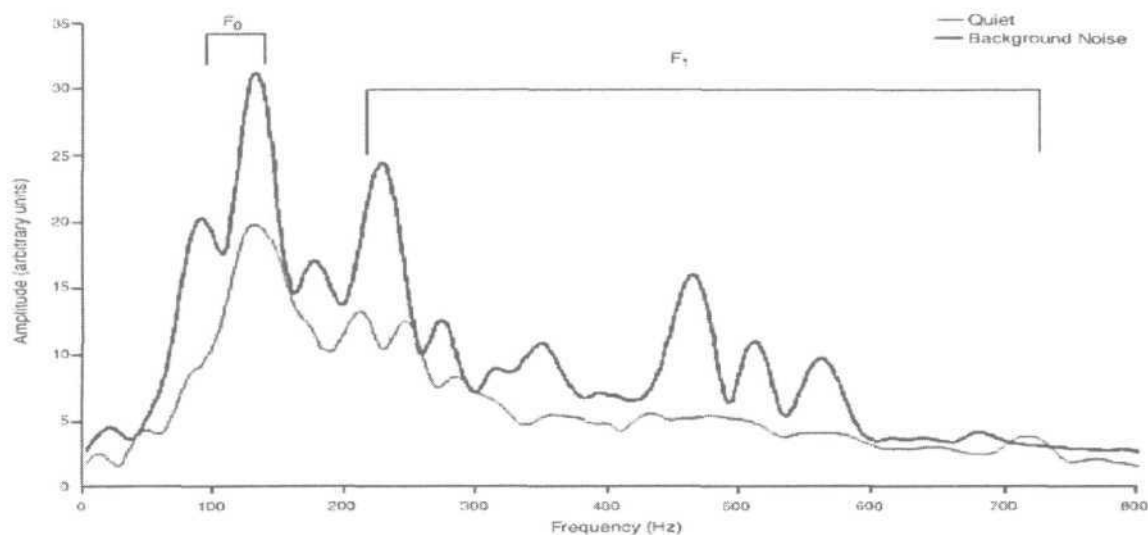


Figure 3: The spectral analysis of FFR showing maximal amplitude at FO, followed by F1 in quiet and in presence of background noise

Further, the FFR encompassing Fundamental frequency (FO) and first formant (F1) peaks, viewed in the frequency domain (figure 3), thereby quantifying the amount of

neural activation at particular frequencies in the stimulus revealed greatest amount of energy in the F0 region followed by F1 region in quiet. Noisy condition disrupted the F0, F1 amplitude as shown in the figure 3. Response frequencies corresponding to higher stimulus formants were not significantly above the noise floor.

Measures of transient and sustained components of the brainstem response to speech syllables were reliably obtained with high test-retest stability and low variability across subjects. All components of the brainstem response were robust in quiet. Background noise disrupted the transient responses whereas the sustained response was more resistant to the deleterious effects of noise. The authors concluded that the speech syllable evoked brainstem response faithfully reflects many acoustic properties of the speech signal with remarkable precision in both frequency and time domains.

Brainstem responses to speech in clinical population

Khaladkar, Kartik and Vanaja (2005) investigated the perceptual deficits in 20 ears with mild to moderate SNHL using ABR. The stimuli they used were standard acoustic click and the burst portion of the syllable /t/. They found that click ABRs exhibited latency value within normal limits, where as speech burst evoked ABRs showed more deviant results, suggesting that using speech sounds to elicit the ABR offers an opportunity to better isolate normal speech processing from abnormal speech processing. The researchers further suggested that it would be useful for evaluating patients with possible auditory processing disorders.

Plyler and Krishnan (2001) investigated FFR to determine (1) if FFR can encode the time-varying second formant transitions in synthetic stop consonant stimuli in normal-hearing and hearing-impaired listeners, (2) if hearing-impairment causes

degradation of this neural representation, and (3) if the degraded representation is correlated with reduced consonant identification in hearing-impaired listeners. FFRs were obtained from normal-hearing and hearing-impaired listeners in response to a 15-step /ba/-/da/-/ga/ continuum generated by varying the onset frequency of the second formant from 900 to 2300 Hz. Their results demonstrated that the FFR did encode the second formant transition in normal-hearing listeners. However, FFR encoding was severely degraded in most of the hearing-impaired listeners. Further, comparison of identification and FFR data for individual hearing-impaired listeners appears to suggest that degradation in the neural representation of the second formant transition may be accompanied by reduction in identification performance.

King et al (2002) recorded Auditory brainstem responses in normal children and children clinically diagnosed with a learning problem. These responses were recorded to both a click stimulus and the formant transition portion of a speech syllable /da/. While no latency differences between the Normal and learning disabled populations were seen in responses to the click stimuli, the syllable /da/ did elicit latency differences between these two groups. Deficits in cortical processing of signals in noise were seen for learning disabled subjects with delayed brainstem responses to the /da/, but not for learning disabled children with normal brainstem measures. Over all they concluded that the onset synchrony of auditory brainstem neurons differs between normal children and some children with learning impairments. In addition, children with delayed onset responses to a speech stimulus also have delays in the brainstem FFR. The effect of these brainstem neural timing deficits on speech perception in quiet is not evident. However,

in the presence of noise, the deficits seen at the level of the brainstem appear to have a deleterious effect on cortical responses to the same stimulus.

Song, Banai, Russo and Kraus (2006) explored the relationship between brainstem encoding of click and speech signals in normal- learning children and in those with language- based learning problem. They found that the normal pattern of correlation between click and speech evoked ABR was disrupted with speech- evoked ABRs having delayed latency. These findings suggest that while there may be some shared processing reflected in the click and speech onset latency measures, there is also a separate component unique to the processing of more complex auditory signals, such as speech. Wible, Nicol and Kraus (2005) showed similar result in learning disabled children.

Khaladkar (2005) evaluated the efficacy of two speech stimulus, extracted burst portion and extracted transition portion of naturally produced syllable ΛJ . ABR evoked by click and the extracted burst and transition portion was recorded in normal and learning disabled children. There was a significant difference in latencies between the responses evoked by the click stimulus and the two speech stimuli (burst and transition in both ears. With increase in duration of stimulus, the latencies were increased. Transition, which had long duration, evoked longer latencies of responses, followed by bursts and then by clicks. It was observed that only speech evoked ABR showed significant difference across the three type of stimulus. In children with learning disability, more number of children showed prolonged latency for transition. However the number of children who showed prolonged latency for bursts was greater than the children who showed deviant responses for click evoked ABR

Effect of cochlear hearing loss on the auditory system

In humans, degeneration of the neurons secondary to cochlear lesion is shown in the spiral ganglion cells (Hinjosa, Blough & Mhoon, 1987; Nadol, Young & Glynn, 1989), cochlear nucleus, medial superior olivary complex and the inferior colliculus (Moore, Niparko, Perazzo, Miller & Linthicum, 1997). The number of spiral ganglion cells surviving after the damage to the cochlea, are reported to have correlations with the degenerative changes seen in the nuclei of the central auditory pathway. Raj an and Irvine (1996) showed that the neurons in the primary auditory cortex showed broadened frequency characteristics when the ear contralateral to the lesion was stimulated. From the review on the existing literature on this issue, it may be said that the cochlear lesions induce changes in the physiology and the structure of the central auditory nuclei and the auditory cortex, which may reflect in auditory perception skills.

Effects of SN hearing loss on speech perception

The vowel and consonant perception in individuals with hearing impairment is far from a simple attenuation of audibility. They also show that the inter-individual differences in perceptual abilities can be explained by the degree and configuration of hearing loss.

Lorenzi, Gilbert, Carn, Garnier and Moore (2006) studied the role of temporal fine structure of sounds in speech perception in hearing impaired. Speech sounds were processed by filtering them into 16 adjacent frequency bands. The signal in each band was processed using the Hilbert transform so as to preserve either the envelope (the relatively slow variations in amplitude over time) or the temporal fine structure (the rapid

oscillations with rate close to the center frequency of the band). The band signals were then recombined and the stimuli were presented to subjects for identification. After training, normally hearing subjects scored perfectly with unprocessed speech, and about 90% correct with envelope and temporal fine structure speech. Both young and elderly subjects with moderate flat hearing loss performed almost as well as normal with unprocessed and envelope speech, but performed very poorly with temporal fine structure speech, indicating a greatly reduced ability to use temporal fine structure. For the younger hearing-impaired group, temporal fine structure scores were highly correlated with the ability to take advantage of temporal dips in a background noise when identifying unprocessed speech. This is in support to the studies done by (Shannon, Zeng, Kamath, Wygonski & Ekelid, 1995; Drullman, 1995; Dorman, Loizou & Tu, 1998; Nelson, Jin, Carney & Nelson, 2003) where they suggested that temporal fine structure cues are important for the intelligibility of speech in noise.

Vowel Perception in Cochlear Hearing Loss

Listeners with hearing impairment show considerably better perception for vowels than for consonants (Revoile & Pickett, 1982). Pickett et al (1972) studied four groups of hearing impaired children with mean hearing losses of 67, 73, 82, and 88 dB HL. They were presented with 50 monosyllabic words in a closed-set format to the better ear at 6 dB above each listener's most comfortable level. The vowel recognition scores for these groups were 91%, 76%, 62%, and 48% respectively. It was concluded that with increasing hearing loss more vowel confusions were observed for those vowels with low frequency F1.

Fourcin (1976) investigated vowel recognition with two-formant synthesized vowels /i/, /u/, and /a/ in listeners with hearing loss. The vowels had the same F1 values but were distinguishable by the amplitude of F2. He showed that those listeners with a severe hearing loss had difficulty in recognizing /i/ and /u/, but could correctly identify /a/. Turner and Henn (1989) compared vowel recognition with measures of frequency resolution in hearing and listeners with hearing impairment. They found that differences in frequency resolution together with the vowel spectra information correlated with vowel recognition scores and thus accounting for individual differences.

Overall, vowel perception in listeners with hearing loss is generally affected by the degree of hearing loss. Significant inter-individual differences have been noticed in vowel perception scores even if the degree of hearing loss is controlled. The perceptual scores correlate well with the spectral resolution rather than the degree of hearing loss.

Consonant Perception in Cochlear Hearing Loss

Godfrey and Millay (1978) asked listeners with mild and moderate SN hearing loss to identify synthesized /be/ and /we/ syllables across a range of transition durations from 10 to 120 ms in 10 ms steps. Two kinds of responses were seen one group attained maximum score with transitions of 40 ms or less and 80 ms or more for /be/ and /we/ respectively. The other group did not perform above chance level for all transition durations.

Hedrick, Schulte and Jesteadt (1995) found that burst/vowel relative amplitude seemed important for discriminating synthetic /pa/-/ta/ contrast pair by mild-to-moderate adults with hearing loss. In contrast, the control group of normally hearing adults

showed dependence on the vowel transitions rather than the burst/ vowel amplitude. Bennett and Ling (1973) studied voicing perception for initial stops using CV monosyllabic words in children with normal-hearing and with a severe hearing loss. Stimuli were prepared with systematic variations in voice-onset-time (VOT) were presented at comfortable listening levels. Normal-hearing children distinguished voiced from unvoiced by VOTs between 20 ms to 40 ms. Children with a hearing loss showed inconsistency in responses and tended to identify more unvoiced than voiced stops at VOTs of 60 ms or more.

The use of acoustic cues by listeners with moderate-to-severe hearing loss was studied by Revoile, Pickett, Holden-Pitt, Talkin and Brandt (1987). This study used stops with varying VOT and other cues such as flattening of F0. Results confirmed that VOT was the strongest cue used by this population to identify voiced stops. This study did not support the study by Bennett and Ling (1973), and the differences may be because of the subjects used in the latter study had more severe hearing loss.

From the above review it can be summarized that the acoustic cues for place perception used among listeners with hearing loss are different from those used by listeners with normal-hearing. The listeners with hearing loss required additional cues for perception of voicing in consonants. Brainstem evoked responses can be used to probe brainstem roots for the speech perception deficits in cochlear hearing loss. Both transient and sustained components of the ABR can be a viable clinical tool to study the cochlear hearing loss group.

CHAPTER III

METHOD

The following method was adopted to investigate the effect of cochlear hearing loss, and presentation level on brainstem responses to speech.

a) Subjects

Forty four ears of adults with normal hearing and cochlear hearing loss between the ages of 16 to 50 years participated in the study. They were classified into two groups - Control group and clinical group.

Clinical Group

Twenty two ears with cochlear hearing loss were taken. The following criteria were considered while recruiting the participants into this group:

- Diagnosed as having sensori-neural hearing loss
- Pure tone average (PTA, 500 Hz, 1000 Hz, 2000 Hz) between 26 to 55 dB HL at least in one ear
- The Air borne gap less than 10 dBHL
- Speech identification scores proportional to pure tone average of 500Hz, 1000Hz & 2000Hz.
- Normal middle ear functioning as assessed by tympanometry and acoustic reflex threshold ('A' type tympanogram with present, elevated or absent acoustic reflexes)
- No abnormality in click-evoked ABR
- Absence of TEOAEs in the ear with hearing loss

- No history of other otological and neurological problems
- No history of congenital or pre-lingual hearing loss (to rule out the effects of deviant/delayed language)

The clinical group was further sub divided into two subgroups based on their PTA,

Group I: Individuals with minimal to mild cochlear hearing loss (N=11 ears) with pure tone average less than 41 dBHL.

Group II: Individuals with moderate cochlear hearing loss (N=11 ears) with pure tone average between 41 dBHL and 55dBHL.

Control Group

Group consisted of twenty two ears of normal hearing individuals. These participants were recruited in to this group if they passed the following criteria,

- Hearing sensitivity less than or equal to 15 dB HL at octave frequencies between 250 Hz and 8000 Hz.
- 'A' type tympanogram and acoustic reflexes present at normal levels.
- No history of neurological or otological problems.

b) Instrumentation

- A calibrated diagnostic audiometer, (GSI - 61) with TDH-39 earphones was used for estimating the air conduction thresholds. Radio ear B-71 bone vibrator was used for bone conduction testing.
- A calibrated middle ear analyzer, (GSI tymptar) was used to record tympanogram and acoustic reflexes

- Brainstem responses to speech and click stimuli was 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)

c) Procedure

Subject selection

Pure tone Audiometry: Pure tone thresholds were obtained at octave frequencies between 250Hz and 8000Hz for air conduction stimuli and between 250Hz to 4000 Hz for bone conduction stimuli using modified Hughson-Westlake method (Carhart & Jerger, 1959).

Immittance evaluation: Tympanometry and reflexometry were carried out to rule out any middle ear pathology. The parameters used for the immittance measurement are shown in table 1. The Intensity of the reflex eliciting tone was varied to elicit the acoustic reflex threshold.

Probe tone frequency	226 Hz
Probe tone intensity	85 dBSPL
Reflex eliciting tone	500 Hz, 1000 Hz, 2000 Hz, 4000 Hz
Mode of eliciting reflexes	Ipsilateral, Contralateral

Table. 1. Parameters used for immittance evaluation

Auditory Brainstem Responses: Auditory brainstem response for click was recorded using the following parameters.

Stimulus parameters	Intensity	90 dBnHL
	Repetition rate	11.1/sec; 90.1/sec
	Polarity	Rarefaction
	Click duration	100 μ sec
Acquisition parameter	Analysis time	10 msec
	Filter setting	100 to 3000 Hz
	Electrode placement	Cz - Non-inverting (+ve); Both mastoids - Inverting (-ve); Forehead - Ground
	Notch filter	On
	Artifact rejection	40 μ V

Table.2. Stimulus and acquisition parameters used to record click ABR

The wave I, III, and V, absolute latencies, Interpeak latencies, latency variation across the repetition rate were considered to rule out retrocochlear lesion.

Oto Acoustic Emission: The Transient Oto acoustic emissions were recorded for clicks using non linear mode presented at 85 dB peSPL. The responses of 256 sweeps were averaged to obtain the emissions and amplitude of TEOAEs and noise were measured. The responses were considered to be present when the emission amplitude was more than 3 dB above the noise floor and had reproducibility more than 70%. The absences of TEOAEs in the presence of hearing loss were considered as indicators of cochlear damage.

Experiment conducted

The study was carried out in two phases: I. Stimulus preparation and II. Recording of evoked potentials.

1. Stimulus preparation

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 stimulus was constructed to include an onset burst frication at F3, FA, and F5 during the first 10 msec, followed by 30-msec F1 and F2 transitions ceasing immediately before the steady-state portion of the vowel. The fundamental frequency (F0) and the first three formants (F1, F2, F3) changed linearly over the duration of the stimulus: 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 4.

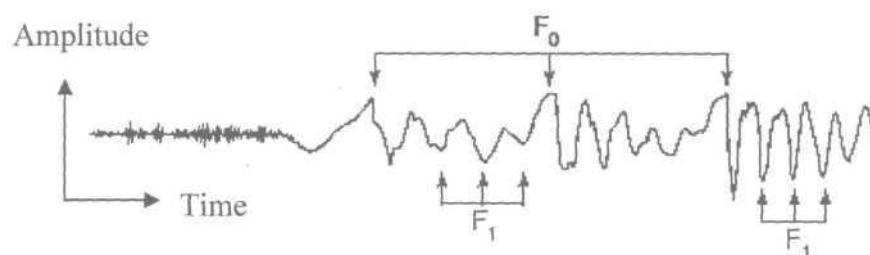


Figure.4. The wave form representation of the stimulus /da/. Fundamental frequency (F0) is seen in periodicity of major peaks. The first formant (F1) is seen as periodically occurring smaller peaks.

//. Recording of evoked potentials

Evoked potentials were recorded in a special facility where subjects were seated comfortably in an acoustically and electrically shielded room. They were instructed to relax and refrain from extraneous body movements to minimize movement artifacts. Subjects were also asked to be awake. The stimulus and recording parameters used are described in Table.3.

Stimulus parameters	Speech stimulus	/da/ (synthesized)
	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
Acquisition parameters	Transducer	Insert ear phones ER-3 A
	Analysis time	70 msec (includes 10 ms pre-stimulus period)
	Band pass filter	30 to 3000 Hz
	Electrode placement	Cz - Non-inverting (+ve); Both mastoids - Inverting (-ve); Forehead - Ground
	Sweeps	1500
	Electrode impedance	< 10 k Ω
	Inter-electrode impedance	< 3k Ω

Table.3. Stimulus and recording parameters used to record speech evoked ABR/ FFR

The skin surface at the mastoids, vertex and forehead was cleaned with a skin abrasive, until a skin impedance of less than 10 kilo Ohms was obtained with disc electrodes. The electrodes dipped in skin conduction paste were fixed in place at the scalp sites using a surgical tape. Each of the recordings was repeated at least twice to have a check on the repeatability.

d) Analysis of the ABR/ FFR recordings

The electrophysiological brainstem response to a speech sound is a complex waveform. This includes transient peaks as well as sustained elements that comprise the FFR.

Onset responses

While analyzing the ABR waveforms off-line the recording window was maintained from -10 ms to 15 ms for clear visibility of the ABR wave V. The following wave V parameters were measured from the each of the ABR recordings:

- i) Peak latency of wave V: It is defined as the duration in ms between the onset of the stimulus and the highest amplitude of the wave V
- ii) Peak amplitude of wave V: It is defined as the amplitude difference in uV between the highest point of the wave V and the point where the lowest amplitude seen in the following trough. This point of minimum amplitude is considered as wave A.

Sustained responses

The recording window was maintained from -10 msec to 60 msec for the clear visibility of the later peaks of the brainstem response to speech and the following Wave C, D, E and F, O parameters were measured.

- i) Peak latency is defined as the duration in ms between the onset of the stimulus and the highest amplitude of the wave C, D, E and/or F, O
- ii) Peak amplitude is defined as the amplitude difference in uV between the highest point of the wave A, C, D, E, and F and the point where the lowest amplitude seen in the following trough.

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 ms- 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.

Waveform Analysis

Three experienced judges were involved in peak picking. The following criteria were used in the waveform analysis.

Repeatability of atleast two waveforms is a typical prerequisite for wave form analysis. Those waveforms that meet this criterion could only be considered as response. The figure 3 is a recording of brainstem response to speech in a normal hearing individual recorded at 40 dBSL. The wave V, A, C, D, E, F, O are marked on the recording.

Wave V the peak latency is calculated as the time difference between the onset of the stimuli and the maximum amplitude of the peak and the Wave V amplitude is calculated as the amplitude difference between the highest amplitude of Wave V to the

minimum amplitude in the following trough. This point of minimum amplitude is marked as the peak A the latency of which is calculated.

The peaks C to F are considered in the FFR. The region following the onset responses were considered as FFR. The defining feature of the sustained portion of the response is its periodicity, which follows the frequency information contained in the stimulus. So the peaks D, E and F are picked based on there periodicity. To consider the presence of FFR, the fluctuations in the brainstem activity should repeat itself with a time period of approximately 10 milliseconds. This time period would correspond to the F0 (100 Hz) of the stimulus used (frequency = 1/time period). So 3 major peaks which repeated it self at the time period of 10 msec were considered as D, E and F. The peak C which is considered as the response to the onset of vowel occurred previous to the onset of the periodic responses. The amplitude of these peaks are calculated as the difference between the highest amplitude of the peak to the minimum amplitude in the following trough. The figure 5 displayed below shows the latency and amplitude

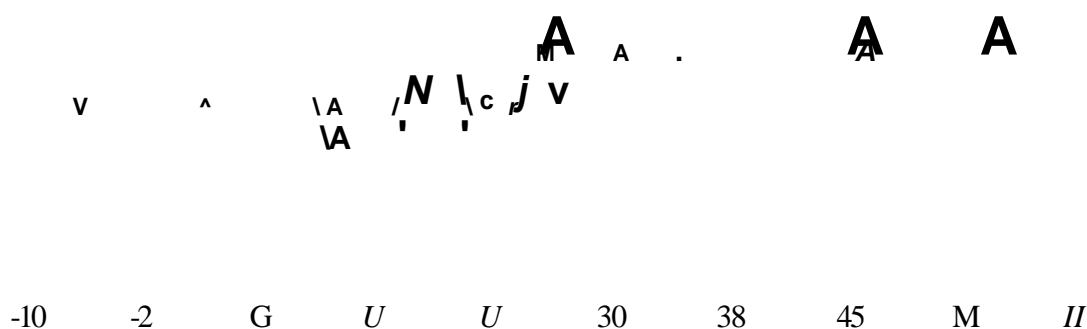


Figure 5: The recording of brainstem response to speech in a normal hearing individual recorded at 40 dB SL.

Objective Measures for Frequency Following Responses

The region following the onset responses was defined as the FFR. The spectral measures performed to analyze the sustained FFR (an epoch of 15 ms- 54 ms) were, the amplitude of the spectral component corresponding to the stimulus fundamental frequency (F0 amplitude) and first formant (F1 amplitude).

Amplitude of the Fundamental Frequency and First Formant

FFR consists of energy at fundamental frequency and its harmonics (Worden & Marsh, 1968). Recently Russo et al. (2004) reported that, speech evoked brainstem responses contained energy at fundamental frequency and first harmonic. The F0 amplitude provides a gauge of the specific portion of the sustained response devoted to encoding the fundamental frequency of the speech sound, while the F1 amplitude is devoted to encoding the first formant. 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.

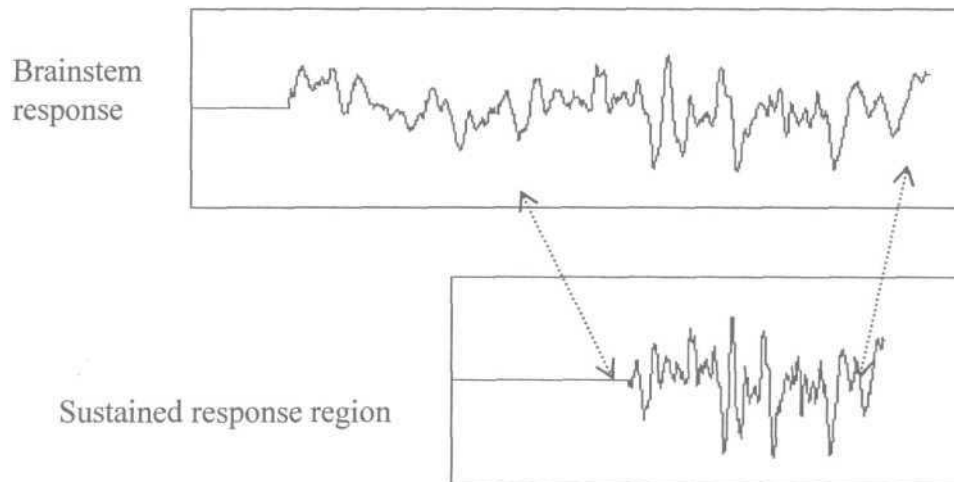


Figure 6: The upper panel shows the brainstem response to speech and the lower panel shows the portion of the sustained responses in the brainstem responses considered for the objective measure

In the above figure the upper panel represents the brainstem responses to speech recorded at 40 dB SL for /da/ stimulus. The lower panel represents the portion of sustained responses (approximately, 15 msec to 54 msec) which occurs immediately after the onset responses. This portion of the responses is subjected to FFT and the Figure 7 represents the FFT for the sustained portion.

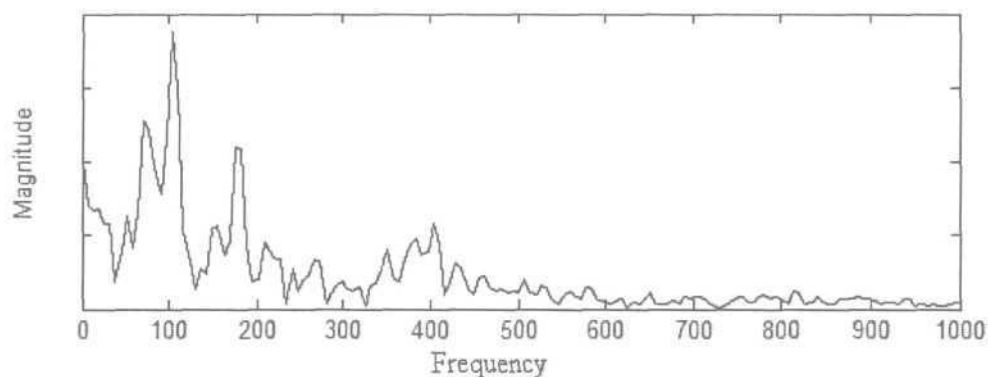


Figure 7: The FFT of the sustained portion of the brainstem response to speech

The above figure shows a maximum amplitude in the FO region i.e., around 100 to 120 Hz. We can also notice some amount of energy in the FI region i.e., from 200 to 720 Hz.

This program was validated with recordings with known spectral characteristics and the following procedure was adopted, An EEG recording of 500 Hz and 1000 Hz tone burst artifact were analysed using this program and are depicted in the figure 8 & 9.

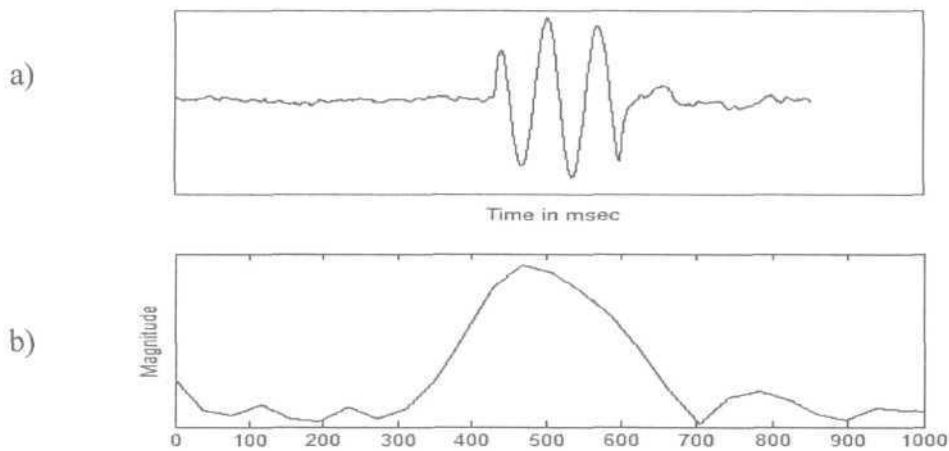


Figure 8: a) The 500 Hz tone burst artifact recorded at 90 dBnHL, b) FFT of the 500 Hz tone burst artifact showing maximum energy at 500 Hz region

The above figure shows that FFT done using the custom made program run in MATLAB, for an EEG recording done using a 500 Hz tone burst at 90 dBnHL. The EEG recording consisted of the artifact of 500 Hz tone burst. The FFT in the lower panel showed maximum amplitude at the 500 Hz region corresponding to the tone burst frequency.

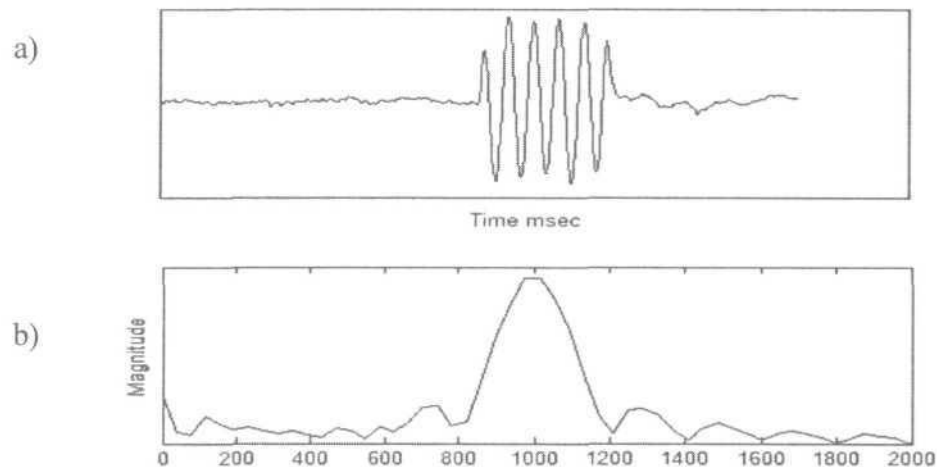


Figure 9: a) The 1000 Hz tone burst artifact recorded at 90 dBnHL, b) FFT of the 1000 Hz tone burst artifact showing maximum energy at 1000 Hz region

The above figure shows that FFT done using the custom made program run in MATLAB, for an EEG recording done using a 1000Hz tone burst at 90 dBnHL. The EEG recording consisted of the artifact of 1000 Hz tone burst. The FFT in the lower panel showed maximum amplitude at the 1000 Hz region corresponding to the tone burst frequency. Thus it validates the custom made program.

Data Analysis

The amplitude and latencies of waves V, A, C, D, E, F and O and also the F0 and F1 amplitude were considered for analysis. Comparison across the two presentation level was done using paired sample *t* test. Recordings for both the groups were compared against the normal group at 40 dBSL and 80 dBnHL. Kruskal-Wallis test was used to check if there any significant difference between the three groups. Mann Whitney U test was carried out to check whether the Group I and Group II differed significantly from that of control group.

CHAPTER IV

RESULTS AND DISCUSSION

The results of the brainstem responses to speech stimuli were discussed separately in normal hearing and cochlear hearing loss. The aim of the present study was to study the effects cochlear hearing loss on brainstem responses to speech and to study the effects of stimulus presentation level (equal SL and equal SPL) on the brainstem responses to speech in normal and cochlear hearing loss and also to establish norms for brainstem response to speech.

The participants of the present study were divided into two groups - the Clinical group consisting of individuals with cochlear hearing loss (N=22 ears) and the Control Group consisting of individuals with normal hearing (N=22 ears). The sub groups of the clinical group were: Group I (N=11 ears) and Group II (N=11 ears).

The brainstem responses to speech were recorded at two presentation levels. The discrete peaks - V, A, D, E, F, and O were identified and their latency and the amplitude were measured. Fast Fourier Transforms were done to find the raw amplitude of F0 and F1 frequency components using custom made program run on a MATLAB platform. Mean and Standard Deviation (SD) values for these parameters were calculated for all the groups.

Comparison of the latency and the amplitude parameters of the brainstem responses to speech, between the groups and within the groups were carried out. The following statistical tests were taken up to attain the difference between the groups. The Kruskal-Wallis test was performed to check whether there is any significant difference between the three groups for the above mentioned parameters. The Mann-Whitney U test

was administered to check whether the Group I and Group II differed significantly from the control group for these parameters. Also, an independent sample t-test was performed to check if there is a significant difference within the groups across the two presentation level.

I. Brainstem responses to speech in individuals with normal hearing

The table.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 22 ears with normal hearing. The table also includes the results of paired sample t test across the two presentational levels.

Parameters		80 dB nHL		40 dB SL		t-values
		Mean	SD	Mean	SD	
Latency Parameters	Wave V	8.15	0.29	9.35	0.27	11.94*
	Wave A	9.09	0.31	10.82	0.42	15.07*
	Wave C	19.87	0.33	21.67	0.91	8.86*
	Wave D	26.66	0.58	29.15	0.84	11.60*
	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*
Amplitude Parameters	Wave V	0.27	0.09	0.24	0.07	1.02
	Wave C	0.41	0.13	0.30	0.08	3.94**
	Wave D	0.48	0.12	0.33	0.12	4.43*
	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 Results	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

Table 4: The Mean, Standard Deviation and \pm -values of the various latency and amplitude parameters of brainstem responses to speech at 80 dB nHL and 40 dB SL obtained in the Control group.

From the table it's clear that 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 would approximately correlate to 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.

Comparison of the latencies and the amplitude across the presentation level using paired sample t-test showed (i) a significant increase in the latency and (ii) a significant decrease in the amplitude of all the parameters when the intensity was varied from 80dB nHL to 40 dB SL except for the wave V amplitude. The variation in latency and amplitude could be due to a mere difference in the audibility of the stimulus.

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). Similar results were also shown by Picton et al (1974) where he reported that the ABR latencies decrease with an increase in the intensity level. 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.

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 is supported by Picton et al (1974); 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 would result in the high amplitude evoked responses. This reasons out why the amplitude measure showed an increase with the increase in intensity.

Wave V showed a reduction in amplitude when the intensity was varied from 80 dBnHL to 40 dBSL, however it was not significant. This may be due to the high variability in the Wave V amplitude (Chiappa, Gladstone & Young, 1979; Edwards et al, 1982; Rowe, 1978). Also, Hecox and Galambos (1974) reported that the amplitude of ABR showed no consistent values in the amplitude growth as a function of intensity. Thus, the wave V amplitude is considered to have less diagnostic significance.

The peak latencies of the transient and the sustained responses show an over all delay in comparison with the latencies reported in literature by Kraus & Nicol (2005); Russo, Nicol, Musacchia & Kraus (2004); King, Warner, Hayes & Kraus (2002); Johnson, Nicol, Zecker & Kraus (2007). This delay in latencies could be attributed to few factors such as the usage of a synthetic stimuli synthesized developed based on the western norms of speech perception and the length of the insert tube used for stimulus delivery. However these factors are uniform through the study and it may not have an effect on the comparison of results between the groups.

FFT analysis of FFR - F0 and F1 amplitudes

The FFR consists of energy at the fundamental frequency of the stimulus and its harmonics. It is a phase-locked response that 'follows' the waveform of the stimulating sound up to a frequency of approximately 1000 Hz (Hoormann, Falkenstein, Hohnsbein & Blanke, 1992)

FFR, encompassing F0 and F1 peaks, can be viewed in the frequency domain, thereby quantifying the amount of neural activation at particular frequencies in the stimulus. The F0 and F1 amplitude given in the table clearly shows that the F0 region has the greatest amount of response energy at both the presentation level this is consistent with the study done by Russo, Nicol, Musacchia & Kraus (2004) where they reported that the brainstem response to synthetic /da/ stimuli had greatest amount of energy present in the F0 region. This could be due to stimulus characteristics and the phase locking properties of the neuron. The stimulus has a higher energy at the F0 region compared to its harmonics (Ladefoged, 1996) and higher energy components are represented better at the neuronal level. Also, the F0 has a lower frequency compared to its harmonics and we know that lower the frequency better will be the phase locked response (Gelfand, 1998). Thus the F0 having greater energy and better phase locking is coded more robustly than its harmonics.

Comparison between the presentation level using the paired sample t test showed a significant reduction in the response amplitude in the F0 region when the presentation level was varied from 80 dBnHL to 40 dBSL, but the reduction was not significant in the F1 region. The reduction in F0 and F1 amplitude may be due to the reduction in the amount of acoustic energy reaching the neurons at 40 dBSL compared to 80 dBnHL.

Only a minor decline in the F1 amplitude may be because of the less significant drop of F1 energy in the stimulus, from 80 dBnHL to 40 dB SL. The data obtained in the normals can be used as norms keeping in mind the delay in latencies found for all parameters.

II. Brainstem responses to speech in cochlear hearing loss

Group I: Table.5 shows the mean amplitude and latencies of the discrete peaks - the waves V, A, C, D, E, F, O and the mean F0, F1 amplitude of the sustained responses for 11 ears with hearing loss less than 41 dBHL. It also includes the results of the paired sample t-test done between two presentation levels.

Parameters		80 dB nHL		40 dB SL		t-values
		Mean	SD	Mean	SD	
Latency Parameters	Wave V	9.23	0.76	9.81	0.73	4.69**
	Wave A	10.35	0.77	10.80	0.66	3.23***
	WaveC	20.61	0.95	22.03	0.95	4.29**
	Wave D	28.32	1.09	29.70	1.04	3.25**
	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***
Amplitude Parameters	Wave V	0.32	0.12	0.24	0.13	2.11
	Wave C	0.28	0.13	0.24	0.10	2.34***
	Wave D	0.36	0.15	0.32	0.30	1.25
	Wave E	0.40	0.19	0.30	0.12	1.90
	Wave F	0.30	0.16	0.27	0.15	1.29
FFT	FO 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 5 : The Mean, Standard Deviation and *t*-values of the various latency and amplitude parameters of brainstem response of speech at 80 dBnHL and 40 dB SL in Group I.

Sustained responses D, E, F maintain a periodicity approximately equivalent to the F0 of the stimulus (100 Hz), which is in correspondence to the study done by Russo, Nicol, Musacchia and Kraus (2004) and Kraus and Nicol (2005), where they said that the Peaks D, E and F represents F0 of the speaker. The current results shows that the periodicity coding of F0 is maintained even in the minimal to mild hearing loss individuals.

The comparison across the presentation level using paired sample *t* - test revealed a significant difference between the two presentation level within the group, in most of the parameter except for the Wave V, D, E and F amplitude i.e., there was an 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.

This increase in latency and reduction in amplitude could be associated to the audibility of the stimulus reaching the neural fibers. The individuals included in this group had a hearing loss less than 41 dBHL, most of them having thresholds between 20 to 35 dBHL. Even when the input given to these individuals is at 40 dBSL, still the energy reaching the neurons would be less than that of an 80 dBnHL input, thus making it less audible compared to the 80 dBnHL. Hence audibility is a factor affecting the latencies and amplitude of the responses. This is supported by Moller (1981) where he reported that the Compound action potential latency is directly dependent on how quickly the generator potential and the excitatory post synaptic potential reach the threshold for firing and so higher the intensity, shorter would be the latency of the responses.

The Wave V, D, E and F showed a reduction in amplitude when the presentation level was varied from 80 dBnHL to 40 dBSL, however insignificant. This may be due to the high variability in the AER amplitude (Chiappa, Gladstone & Young, 1979; Edwards et al, 1982; Rowe, 1978). Hall (1992) reported that the rate of decrease in amplitude with decreasing intensity is more rapid for earlier waves of the brain stem responses compared to the later peaks. Also, the energy difference between the two levels may not be sufficient enough to bring about any significant changes in the amplitude measures.

FFT analysis of FFR - F0 and F1 amplitudes

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

Comparison across presentation level using paired sample t - test revealed no significant decrease in the F0 and F1 amplitude with the change in the presentation level from 80 dBnHL to 40 dBSL. This could be due to the same reason explained for the Wave V, D, E and F amplitude measure mentioned for the control group. Also, the indifference between the low and high intensity values may attributed to the disturbed intensity processing in the mild hearing loss group (Florentine, 1993).

Group II: Table 6 shows the mean amplitude and latencies of the discrete peaks - waves V, A, C, D, E, F, O and the mean F0, F1 amplitude of the sustained responses for 8 out of 11 ears with hearing loss between 41 to 55 dBHL. Table also includes the results of paired sample t - test done across two presentation level.

Parameters		80 dB nHL		40 dB SL		t-values
		Mean	SD	Mean	SD	
Latency Parameters	Wave V	10.30	0.73	9.91	0.48	3.94***
	Wave A	11.57	0.98	10.83	0.76	3.80**
	Wave C	22.33	0.79	23.54	1.13	2.25
	Wave D	29.79	1.03	29.95	1.00	0.53
	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***
Amplitude Parameters	Wave V	0.21	0.05	0.21	0.10	1.74
	Wave C	0.26	0.09	0.24	0.05	0.40
	Wave D	0.28	0.05	0.22	.076	2.3
	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
	F1 amplitude	8.90	2.28	10.03	2.53	14

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

Table 6: The Mean, Standard Deviation and t-values of the various latency and amplitude parameters of brainstem response of speech at 80 dB nHL and 40 dB SL in Group II.

As seen from the data given in the above table its clear that the sustained responses D, E, F have a periodicity of approximately 100 Hz, however the standard deviation of these peak latencies are very high. Also, in 3 out of the 11 subjects taken, the transient and the sustained peaks could not be identified and in 2 out of the 8 subjects consider, wave D, E, F, O were not identifiable. Thus, the high variability in the std deviation of these peaks latency and absences of identifiable responses in certain individuals may indicate inaccurate coding of F0 and its harmonics.

Comparison across presentation level using paired sample t - test showed no significant difference for most of the parameters except for the Wave V, A, and O latency and also there was no particular trend in the change in the latency and amplitude of most of the parameters with the change in the presentation level. The Wave V and wave A latency showed a decrease in latency and wave O latency showed a increase in latency, when the level was varied from 80 dBnHL to 40 dBSL. . This may be simply because little differences between the two presentation levels. All the subjects in this group had thresholds between 41 dBHL to 55 dBHL. If a participant has a PTA of 41 dBHL, the 40 dBSL would range to 81 dBnHL which is approximately equivalent to the 80 dBnHL presentation level. Thus, due to greater degrees of hearing loss, the SL and HL values may be hardly different. Also a high variability in most of the parameters across the participants could be a reason for the little difference seen across presentation level. Over all, the parameters did not show any particular trend in the latency and amplitude measures.

FFT analysis of FFR - F0 and F1 amplitudes

The F0 and F1 amplitude given in the table 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 may be due to the high energy in the F0 region of the stimulus (Ladefoged, 1996) and also due to the better phase locking of the lower frequencies (Gelfand, 1998). The paired sample t-test revealed no significant difference between the F0 and F1 amplitudes, across the two presentation levels as both the levels would cause an equal sensation in these hearing loss individuals.

III. Comparison of brainstem responses to speech between Control and

Clinical groups

Non parametric tests were used to compare the recordings of the control group with that of the clinical groups (Groups I & II) due to the unequal sample size. Kruskal-Wallis test was carried out to check whether there is any significant difference between the three groups. The Mann-Whitney U test used to check whether the Groups I and II differed significantly from that of the control group.

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

For the comparison across equal sensation level, the brainstem response to speech was recorded at 40 dB above the pure tone average. The Table 7 gives the mean latency and amplitude of the discrete peaks and the F0, F1 amplitude. Results of Kruskal-Wallis test (Table 7) 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 F0 amplitude.

Parameters		Control	Group I	Group II	Chi-Square
Latency Parameters	Wave V	9.35	9.81	9.91	7.32***
	Wave A	10.65	10.80	10.83	0.26
	Wave C	21.67	22.03	23.54	1.48
	Wave D	29.15	29.70	29.95	3.97
	Wave E	39.49	40.09	40.97	2.32
	Wave F	49.64	49.84	50.92	5.08
	Wave O	59.09	58.79	58.96	2.34
Amplitude Parameters	Wave V	0.24	0.24	0.21	0.89
	Wave C	0.30	0.24	0.24	4.34
	Wave D	0.33	0.32	0.22	7.89***
	Wave E	0.25	0.30	0.22	2.06
	Wave F	0.34	0.27	0.21	9.05***
FFT	F0 amplitude	24.1299	23.9992	14.9401	11.95**
	F1 amplitude	12.7849	13.0542	10.0335	5.69

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

Table 7: The mean and Chi-square values across the three groups for various parameters recorded at 40 dB SL.

The parameters (latencies of waves V and F; amplitudes of wave D and F; and F0 amplitude) which revealed a significant difference in the Kruskal-Wallis test were only considered for the Mann-Whitney U test to find out if there is any significant difference

between any two groups. The Man-Whitney U test was carried out to study the pair wise comparison for the 3 groups. The F1 amplitude was also considered for the Man-Whitney U test for the pair wise comparison for the groups as it just missed the 0.05 level of significance (Sig - 0.058) in the Kruskal - Wallis test.

Between group comparison at equal SL

Table. 8 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.

Parameters		z-values		
		Control Vs Group I	Control Vs Group II	Group I & II
latency	Wave V	-1.12	-2.98**	-0.28
	Wave F	-0.86	.2.09***	-1.63
Amplitude	Wave D	-0.13	-2.61**	-2.39***
	Wave F	-1.62	-3.05**	-0.41
FFT	Fo amplitude	-0.30	-3.25**	-2.81**
	F1 amplitude	-0.30	-2.03***	-2.25***

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

Table 8: Z values between the control group, Group I and Group II

Control group Vs Group I

Results of Mann Whitney U test revealed no significant difference between the groups for all the parameters, i.e., there was no significant increase in the latency or reduction in the amplitude when the results obtained from normal hearing was compared with the group I. However there a minimal increase in the latency and reduction in the

amplitude for the group I compared to normal hearing. 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 showed in their data 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.

Control group Vs Group II

Results of Mann Whitney U test revealed significant difference between the groups for all the tested parameters i.e., in spite of the equal sensation of the stimulus, the latency of the peaks were prolonged and the amplitude was reduced in group II when compared with individuals with normal hearing. Also, the F0 and F1 amplitude showed a significant reduction when the data of hearing loss of moderate degree was compared with the normal hearing group. This could be due to reduced temporal processing in higher degree of hearing loss.

This could be supported by a study done by Lorenzi, Gilbert, Carn, Garnier and Moore (2006), they 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. 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. 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).

Group I Vs Group II

Results of Mann Whitney U test revealed significant difference between the groups for Wave D amplitude, F0 and F1 amplitude i.e., there was a reduction in D amplitude and the F0, F1 amplitude when there was an increase in the degree of hearing loss. This shows 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, 2003a). 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.

Though there was a minimal reduction in the F amplitude and increase in the wave V and F latency in the group II compared to group I, it failed to show a significant difference.

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

Comparison across the groups (at equal Hearing Levels, HL - 80 dBnHL)

For the comparison across equal Hearing level, the brainstem response to speech was recorded at 80 dBnHL. Results of Kruskal-Wallis test to find out any significant difference between the control group, group I and group II are given in table 9.

Parameters		Control	Group I	Group II	Chi-Square
Latency Parameters	Wave V	8.15	9.23	10.30	22.85*
	Wave A	9.09	10.35	11.57	24.16*
	Wave C	19.87	20.61	22.33	20.41*
	Wave D	26.66	28.32	29.79	26.19*
	Wave E	37.25	38.93	39.86	20.22*
	Wave F	47.35	48.77	50.03	23.66*
	Wave O	56.95	58.12	58.20	21.08*
Amplitude Parameters	Wave V	0.27	0.32	0.21	3.76
	Wave C	0.41	0.28	0.26	11.82**
	Wave D	0.48	0.36	0.28	14.48**
	Wave E	0.41	0.40	0.23	10.35**
	Wave F	0.44	0.30	0.21	14.64**
FFT	F0 amplitude	30.40	23.73	15.25	21.50*
	F1 amplitude	15.29	15.10	8.90	12.99**

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

Table 9: The mean and Chi-square values across the three groups for various parameters recorded at 80 dBnHL.

Results of the Kruskal - Wallis test revealed significant difference between the 3 groups for all parameters except Wave V amplitude. The parameters which revealed a significant difference in the Kruskal - Wallis test were considered for the Mann Whitney U test to find out if there is any significant difference between any two groups.

Between group comparison at equal nHL

Table. 10 shows the results of the Mann-Whitney U test, for the pair wise comparison of all parameters of control group, group I and group II

Parameters		z-values		
		Control Vs Group I	Control Vs Group II	Group I Vs Group II
Latency Parameters	Wave V	-3.46**	-3.93*	-1.96
	Wave A	-3.58*	-3.95*	-2.49***
	Wave C	-2.21***	-4.07*	-3.11**
	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
Amplitude Parameters	Wave C	-2.58***	-2.95**	0.20
	Wave D	-1.98***	-3.82*	-1.11
	Wave E	-0.591	-3.36**	-1.87
	Wave F	-2.16***	-3.70*	-1.24
FFT	F0 amplitude	-2.36***	-4.27*	-2.74**
	F1 amplitude	-0.53	-3.57*	-2.60**

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

Table 10: Z values between the control group, Group I and Group II at 80 dBnHL

Control group Vs Group I

Results of Mann Whitney U test showed a significant difference between the groups 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 significance. This could be due to the inadequate audibility of the sound due to certain degree of hearing loss, which in turn has impaired the temporal coding.

Control group Vs Group II

Results of Mann Whitney U test revealed a significant difference between the normal hearing group and the group II for all the parameter tested. The latencies significantly increased and the amplitude reduced in the moderate hearing loss group. Also, there is drastic reduction in the F0, F1 amplitude in the moderate hearing loss group. This suggests that the inadequate audibility would affect the temporal processing to a great extent in moderate hearing loss group.

Group I Vs Group II

Comparison between group I and group II revealed increase in the latency and decrease in the amplitude of all parameters though significant only for wave A, C, D, F latency. The F0, F1 amplitude also showed a significant reduction in the group II as revealed by Mann Whitney U test. This again shows that as the hearing loss increases, the audibility reduces and this would affect the temporal processing and F0, F1 coding.

However the most of the amplitude parameters which are highly variable failed to show any significant reduction.

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's clear that as the degree of hearing loss increases, the temporal processing degrades due to reduced audibility. Thus, in day today situation hearing impaired individuals miss out temporal cues, which essential for the speech intelligibility.

Over all, we know this temporal fine structure of speech is very important for coding the F0 and its formant. 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 adverse environments.

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CHAPTER V

SUMMARY AND CONCLUSIONS

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. Less well-defined is how the brainstem responds to complex stimuli such as a speech syllable. Studying the neural encoding of speech sounds provides insight into some of the auditory processes involved in normal communication. Brainstem responses to speech can be divided into onset response and the frequency following response which acts as a measure of both spectral and periodicity encoding. Various researchers (Revoile et al, 1987; Danhauer, Hiller & Edgerton, 1984) had reported that the cochlear hearing loss individuals have difficulty in perceiving place and manner cues. Moore, Glasberg and Hopkins (2006) reported temporal fine structure processing deficit in cochlear hearing loss. There is a need to assess whether this temporal processing deficit is due to the reduced audibility or due to cochlear distortion. Objective assessment of temporal processing in brainstem for hearing loss individuals has seldom been studied. So this study was primarily designed to study the brainstem responses to speech syllable in subjects with normal hearing and cochlear hearing loss. The aims of the study were to:

- Study the effects cochlear hearing loss on brainstem responses to speech, and
- Study the effects of stimulus presentation level (equal SL and equal SPL) on the brainstem responses to speech in normal and cochlear hearing loss.
- Establish norms for brainstem responses to speech.

Participants in the present study were divided into two groups - the Clinical group consisting of individuals with cochlear hearing loss (N=22 ears) and the Control Group consisting of individuals with normal hearing (N=22 ears). The sub groups of the Clinical group were: Group I (N=11 ears) with less than 41 dB HL thresholds and Group II (N=11 ears) with thresholds between 41 dB HL to 55 dBHL.

The subjects were prepared using conventional procedure to record auditory brainstem responses. A speech syllable /da/ extensively used by Kraus and colleagues was chosen as the stimulus for recording the brainstem responses. The syllable was presented at two levels - at 40 dB SL (ref. to pure tone average) and at 80 dBnHL. The ABR waveforms contained both the onset (amplitude and latency of peaks V and A), the sustained (amplitude and latencies of peaks - C, D, E, F) and the offset responses (peak O). In addition, Fast Fourier Transformation of the sustained potentials were done to evaluate the amplitudes of the fundamental frequency (F0) and the first formant (F1) coded in the auditory system.

The data obtained from the participants of the study was subjected to statistical analysis using SPSS version 15.0 for windows. The mean, standard deviation values were calculated for all the groups. Independent samples *t* test was carried out to find if there is a significant difference in amplitude and latency of the various onset and the sustained responses of individuals with normal hearing and those with cochlear hearing loss. Kruskal-Wallis test was carried out to check whether there is any significant difference between the three groups. The Mann-Whitney U test used to check whether the Groups I and II differed significantly from that of the control group.

In line with other evoked potentials the latencies and amplitudes of all ABR components obtained in this study showed well-known trading patterns in normal hearing individuals. With an increase in intensity from 40 dB SL to 80 dBnHL, the latencies of all peaks significantly reduced and the amplitudes increased. This can be attributed to the properties of the compound action potentials of the nerve fibres. These patterns were somewhat different in the mild hearing loss group (Group I), where they showed significant decrease in latencies in all the latency parameters with a change in presentation level from 40 dB SL to 80 dBnHL. However, the amplitude parameters did not show any significant changes except for wave C amplitude. The results obtained from the Group II showed very different patterns. Most of the latency and amplitude parameters did not change significantly with a variation the presentation level and there was no trading pattern seen in them.

Further analysis indicated that at equal sensation level (at 40 dBSL), the amplitude and latency parameters obtained from the control group and Group II were significantly different for some of the parameters and the group II exhibited poor temporal fine structure coding in coherence with other psychophysical studies done in moderate cochlear hearing loss. The differences in amplitude and latency measures between the control and clinical group widened when comparisons were made at equal hearing level (at 80 dBnHL) across the groups. Both the group I and Group II were significantly different in latency and amplitude parameters, from the normal hearing group. The group II showed a larger difference than the group I.

From this study following conclusions can be made:

1. All the latency, amplitude and spectral parameters used in the study showed some significant differences between the subject groups
2. The latency parameters generally showed decreasing trends while the amplitude parameters increased with increase in presentation level from 40 dB SL to 80 dBnHL in the control group and the group I.
3. The cochlear hearing loss affected the brainstem responses to speech sounds. Amplitude parameters reduced and latency parameters prolonged with an increase in degree of hearing loss
4. The temporal fine structure coding is minimally affected in group I and significantly affected in the group II indicating that, temporal fine structure coding degrades with the increase in the degree of loss.
5. Hearing loss individuals having degraded temporal processing also due to reduced audibility and this becomes more adverse with the increase in the degree of loss. So in a daily listening situation the hearing loss individuals tend to miss out the temporal cues to a great extent.
6. Sustained responses were worse affected than the transient responses to speech sounds as a result of hearing loss

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