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

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ANIRBAN C

A dissertation submitted in part fulfillment for the degree of

Master of Science (Audiology)

University of Mysore, Mysore

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APRIL 2007.

Dedicated to
My dear
Maa and Baba
&
Vanaja Mam

CERTIFICATE

This is to certify that this dissertation entitled **"Brainstem and Cortical Responses to Speech Stimuli in Individuals with Cochlear Hearing Loss"** is a bonafide work in part fulfillment for the degree of Master of Science (Audiology) of the student Registration no: 05AUD003. This has been carried 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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DECLARATION

This is to certify that this master's dissertation entitled **"Brainstem and Cortical Responses to Speech Stimuli in Individuals with Cochlear Hearing Loss"** is the result of my own study and has not been submitted earlier to any other university for that award of any degree or diploma.

Register Number. 05 AUD003

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April 2007

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"Gratitude is the hardest of all emotions to express.
There is no word capable of conveying all that one feels.
Until we reach a world where thoughts can be adequately expressed in words.
'Thankyou (will have to do). "

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Introduction

Listeners with cochlear hearing loss have difficulty in understanding speech (Plomp, 1978, 1986; Dreschler & Plomp, 1980; Glasberg & Moore, 1989). They have greater difficulty in understanding speech in the presence of background noise, especially when the background noise is competing speech (Festen & Plomp, 1990; Dijkhuizen, Festen & Plomp, 1990). This difficulty has been linked to reductions in audibility and to spectrotemporal processing deficits associated with cochlear hearing loss (Humes , Dirks, Bell & Kincaid, 1987; Zurek & Delhorne, 1987).

An individual with normal hearing uses a number of acoustic cues to classify speech signals based on the place and manner of articulation. Burst and formant transition serves as separate cue for perception of place and manner of articulation (Benki, 2001; Blumstein & Stevens, 1980). Voiced and voiceless burst serves as cue for perception of manner (Stevens, 1998). Many studies have shown that transition of second and third formants are sufficient cues for the place distinction (Delattre, Liberman & Cooper, 1955; Liberman, Delattre, Cooper & Gerstrian, 1954). The transitions are also used as voicing cue for the stop consonants (Stevens & Klatt, 1974; Lisker, Liberman, Erickson, Dechovitz & Mandler, 1977; Summerfield & Haggard, 1977).

Cochlear hearing loss effects the perception of the cues used for speech perception. The acoustic cues used for the perception of place of articulation are particularly susceptible to the effects of sensorineural hearing loss (Boothroyd, 1984; Dubno, Dirks, & Langhofer, 1982). Initially, place cues may be simply inaudible to person with hearing loss in the frequency regions containing these cues (Dubno, Dirks & Langhofer 1982.). But the sensorineural hearing loss may also distort supra threshold

speech cues in various ways. It has been reported that the perception of place of articulation is effected more than the perception of manner of articulation and voicing in individuals with sensorineural hearing loss (Plomp, 1978). Plomp (1986) reported that the ability to categorize consonants by place of articulation is significantly effected in hearing impaired regardless of audiometric configuration. Turner and Robb (1987) reported that while the audibility of individual stop consonants is an important factor influencing recognition perception in hearing impaired subjects, it is not always sufficient to explain the effects of sensorineural hearing loss. Turner, Chi and Flock (1999), Turner, Souza and Forget (1995) reported that listeners with sensorineural hearing loss utilize the relative amplitude cue in making place distinction. Zeng and Turner(1990) and Hedrick (1997) reported that sensorineural hearing loss may disrupt formant transient coding or any type of dynamic process in periphery (i.e. rapidly changing aspects of speech signal is not being coded).

Recent advances in the field of electrophysiology have shown that auditory evoked potentials, both brainstem and cortical responses can be recorded reliably for speech stimuli (Song, Banai, Russo & Kraus, 2006; Khaladkar, Karthik & Vanaja, 2005; Krishnan, 2002; Russo, Nicol, Mussacchia & Kraus, 2004; King, Warrier, Hayes & Kraus, 2002). As brainstem responses can be best recorded using short duration signals, burst or transition portion have been used to elicit brainstem responses in some of these studies.

The studies carried out on children with learning disability have shown that responses to speech stimuli were deviant in these children even when responses to nonspeech stimuli were normal (Khaladkar, 2005; King, Warrier, Hayes & Kraus, 2002;

Russo, Nicol, Mussacchia & Kraus, 2004). Effect of hearing loss on brainstem responses to speech stimuli is yet to be explored.

Need for the study

A review of literature shows that there is a dearth of studies on speech evoked auditory potentials in subjects with sensorineural hearing loss. Khaladkar, Karthik and Vanaja (2005) reported that there was a significant correlation between speech identification scores and speech burst ABR in subjects with sensorieneural hearing loss. Transition portion of speech also contains important cue for speech identification (Blumstein & Stevens, 1980). So, response to transition portion of speech also probably will correlate well with speech identification score of a subject. But there is dearth for investigation comparing the relationship of SIS with brainstem response evoked by burst portion and transition portion of speech stimuli.

There is also a need to study the cortical representation of burst and transition of speech stimuli in subjects with normal hearing and those with hearing loss. Studies also need to be carried out to investigate the relationship of cortical responses to speech burst and transition with speech identification scores.

Aim of the study

This study was designed to investigate the following aims:

- To study if there is a difference between subjects with normal hearing and those with cochlear pathology in the following responses:
    - Brainstem responses to speech burst.
    - Brainstem responses to transition of speech.

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

To investigate the relationship between the following, in subjects with cochlear pathology:

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

Review of Literature

Various auditory evoked potentials have been explored as a measure of objectively predicting the behavioral audiogram or speech perception in groups of subjects who cannot provide reliable or accurate behavioral results. Auditory Evoked potentials are electrical potentials generated by structures from the auditory nerve to the auditory cortex and other cortical areas, including the ABR at 0 to 10 ms after stimulus onset, the middle latency response (MLR) at 10 to 60 ms, and the long latency response at 60 to 250 ms (Ponton, 1996).

Brainstem response to speech

Jewett and Williston (1971) were the first to definitively describe far-field scalp-recorded auditory brainstem responses (ABR). The ABR is a phasic response to a transient acoustic event, occurring within the initial 10-15 ms after the event (e.g., stimulus onset). Till decades ABR has been proven itself as a sensitive tool to assess integrity of auditory pathway. This sensitivity results from the high replicability and temporal precision of ABR components commonly labeled as waves I to VII. These waves represent neural activity from auditory nerve to inferior colliculus.

Transient acoustic events induce a pattern of voltage fluctuations in the brain stem resulting in a familiar waveform, yielding information about brainstem 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). Disruptions in this systematic progression in the order of fractions of milliseconds are clinically significant in the diagnosis of hearing loss and brain stem pathology. Studies also have been done to find out how brainstem timing

contributes to neural encoding of 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, 1984, Young & Sachs, 1979). 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 (Young & Sachs, 1979, Sachs, 1984.). 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.

Speech stimuli also have been used to study brainstem potentials in humans (Krishnan, 2002; Russo, Nicol, Musacchia & Kraus, 2004). The speech-evoked ABR recorded in human brainstem can be divided into transient and sustained portions, specifically the onset response and the frequency-following response (FFR) (Johnson, Nicol, & Kraus, 2005; Kraus & Nicol, 2005). Onset responses are transient, similar to click evoked ABR, with peak durations lasting tenths of milliseconds. The FFR arises from the harmonic portion of the stimulus and is characterized as a series of transient neural events phase locked to periodic information within the stimulus (Batra, Kuwada, & Maher, 1986; Marsh & Worden, 1968; Sohmer & Pratt, 1977). Frequency following responses (FFR) in humans was first reported by Moushegian, Rupert and Stillman (1973). Galbraith, Arbagey, Branski, Comerci,& Rector (1995) demonstrated that the FFR elicited by word stimuli reflects the stimulus accurately enough to allow it to be recognized as intelligible speech when "played back" as an auditory stimulus. More

recently, Galbraith, Amaya,,Rivera, Donan, Duong & Hsu, (2004) have suggested that based on the FFR pattern of activation for forward and backward speech, synaptic processing at the level of the brain stem is more effective for forward speech stimuli characterized by highly familiar prosodic and phonemic structure, than to backward speech. Krishnan (2002) has studied the FFR elicited by synthetic vowels to relate phase-locking characteristics of brainstem neurons to individual harmonics of a complex sound. Results suggest that human FFR spectra show clear and distinct peaks corresponding to formant frequencies of stead-state synthetic vowels. Subsequent studies by Krishnan and colleagues used Chinese syllables to show that pitch representation in the auditory brain stem is based on temporal patterns of phase locked neural activity of the fundamental frequency, as represented by the FFR (Krishnan et al., 2004).

Because rapid temporal changes and complex spectral distributions are inherent in speech, both micromeasures (transient) and macromeasures (sustained) of timing and magnitude are used to describe the response (Johnson, Nicol & Kraus,2005) Timing measures provide insight into the accuracy with which brain stem nuclei synchronously respond to acoustic stimulation (e.g., peak latency, interpeak interval, and slope) and the fidelity with which the response mimics the stimulus i.e. stimulus to response correlation. Magnitude measures provide information about the robustness with which the brain stem nuclei respond to acoustic stimulation (e.g., peak amplitude and root-mean-square amplitude of activation) and the size of spectral components within the response (e.g., frequency-domain analysis.

Many of the studies to record brainstem potentials have been carried out using the stimuli /da/ (Cunningham, Nicol, Zecker & Kraus, 2001; Russo, Nicol, Musacchia &

Kraus, 2004). Russo, Nicol, Musacchia and Kraus (2004) reported that the period between response peaks D, E, and F corresponds to the wavelength of the F0 of the utterance for stimulus /da/. Moreover, Fourier analysis of this portion of the response confirms a spectral peak at the frequency of F0. Waves V, A, C, and O are events that occur in response to transient stimulus events separate from the periodic acoustic events in the stimulus. The VA complex reflects a highly synchronized neural response to the onset of the stimulus. Peak C probably is a response to the onset of the voicing that occurs at 10 ms after stimulus onset. Wave O probably is a response to the cessation of sound, as it corresponds temporally to the offset of the stimulus. Wible, Nicol and Kraus (2004) on the other hand marked the negative trough after the V peak as Vn and did not marked the other peaks of FFR individually.

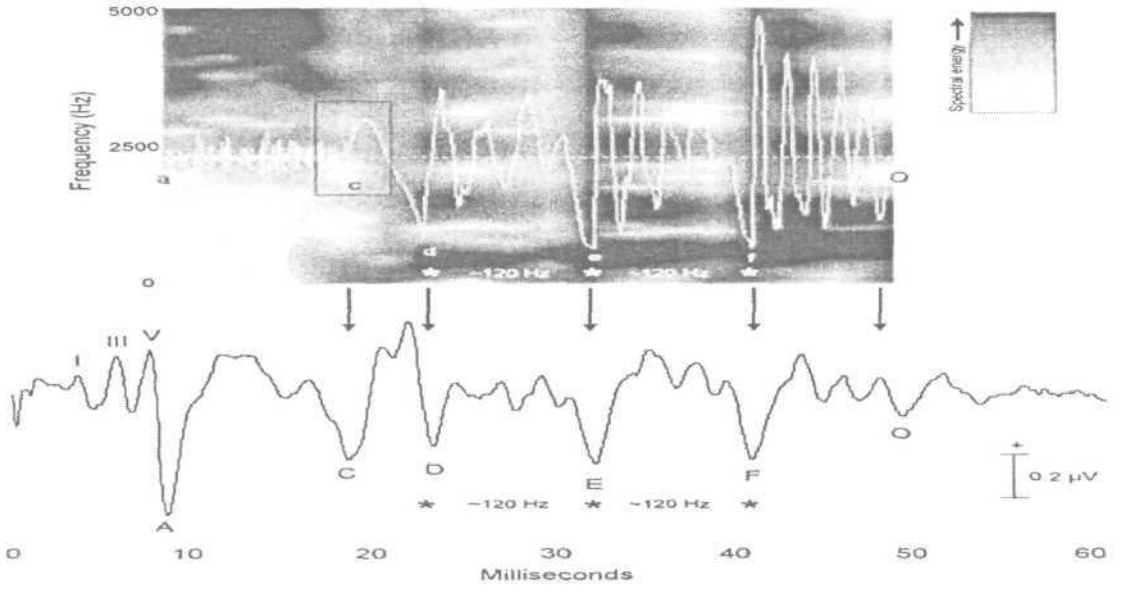

Fig 1: A sample of brainstem response to speech (Adopted from Johnson, Nicol & Kraus, 2005). It shows the correspondence between brainstem. Neural events (uppercase letters) in the response reflect a direct mapping of stimulus characteristics (lowercase letters). The stimulus waveform has been shifted 7 msec to compensate for neural lag in the

response. Note that the wavelengths between peaks d, e, and f (the FO of the stimulus) correspond to peaks D, E, and F of the response (marked by asterisks). Also note that waves C and O correspond to major stimulus feature changes (wave C: transition between onset burst and more periodic portion; wave O: stimulus offset). It has been observed that brain stem response demonstrates remarkable fidelity to the stimulus in the frequency domain for both FO and F1 (Johnson, Nicol & Kraus, 2005).

Russo, Nicol, Mussacchia and Kraus (2004) recorded ABR for synthesized /da/ stimuli to speech syllables presented in quiet and in background noise from 38 normal children. Both the transient and sustained portion of brainstem response was recorded. Transient peak responses were analyzed with measures of latency, amplitude, area, and slope. Magnitude of sustained, periodic frequency-following responses was assessed with root mean square, fundamental frequency, and first formant amplitudes; timing was assessed by stimulus-to response and quiet-to-noise inter-response correlations. They found that 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 the 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.

Subsequently in another study, Reddy, Kumar and Vanaja (2004) attempted to study speech evoked ABR using burst portion of naturally produced speech syllables. They used 4 different speech stimuli to extract burst, i.e. /t/, /th/, /p/, /k/and compared responses across stimuli. The results of their study indicated that overall wave morphology of ABR evoked by speech burst was similar to that of clicks. Almost all,

ABR evoked by *III* and /th/ had better morphology when compared to response evoked by /p/ and *Ikl.* All the 5 peaks in the response could be easily identified. It was also deserved that there were robust VI and VII peaked with better morphology than of a click. With respect to latency measure, It was observed that the latency of ABR evoked with click stimuli was shortest followed by that of /k/, */t/*, /th/, and /p /.

Thus a review of literature shows that brainstem response can be recorded reliably for burst and transition portions of speech stimuli in normal hearing individuals.

Cortical response to speech*:*

The long latency auditory evoked potentials (LLR) are characterized by components comprising the time domain of 50-500 ms (Me Pherson & Starr, 1993) and are labeled according to their polarity and latency at the vertex (Picton et al., 1978). The LLR has enjoyed widespread clinical application as an objective measure of hearing sensitivity in adult subjects (Coles & Mason, 1984; Tickards et al., 1996; Hyde, 1997). The LLR is a wave form consisting of three main peaks: a positive (P1), followed by a negative (Nl), and a second positive (P2). The amplitude of the N1-P2 response is typically 25uV for high intensity stimuli. The response is typically evoked by a range of transient stimuli including clicks, tone bursts, noise bursts, syllables, and transient changes in the auditory environment (Naatanen & Picton, 1987). Speech evoked LLR are frequently used to study the neural representation of speech-sound in populations with impaired speech understanding. The underlying assumption is that speech perception is dependent on the neural detection of time-varying spectral and temporal cues contained in the speech signal (Tremblay, Billings & Rohila, 2004). The P1-N1-P2 complex reflects

the neural detection of time-varying acoustic cues. Because abnormal P1-N1-P2 response patterns have been reported in children and adults with varying types of speech perception impairments, there is a current surge of interest in learning more about this brain-behavior relationship (Gravel, Kurtzberg, Stapells, Vaughan & Wallace, 1989; Kurtzberg, 1989; Gravel & Stapells, 1993; Klein et al., 1995; Kraus, 2001; Rance, Wesson, Wunderlich & Dowell, 2002).

Speech evoked ABR in clinical population

A number of studies have been carried out on children with learning disability using speech evoked ABR. Results of the studies shows that a subset of children with learning disabilities show abnormal neural encoding of speech syllable at the level of brainstem (speech evoked ABR) (Cunningham et al., 2001; Johnson, Nicol, & Kraus,., 2005; Song, Banai, Russo & Kraus, 2006). Abnormal speech elicited ABR has been observed in children with learning disability though they had typically normal click-evoked ABRs (Wible, Nicol & Kraus, 2005; Song, Banai, Russo & Kraus, 2006; Khaladkar, 2005).

Auditory processing deficit is seen in case of sensorineural hearing loss also, and they may have difficulty in understanding speech (Plomp, 1978, 1986; Dreschler & Plomp, 1980, 1985; Glasberg & Moore, 1989). There is a dearth of studies on speech evoked auditory potential on sensorineural hearing loss. However studies on click evoked ABR reveal that there is an effect of hearing loss on latency and amplitude of ABR. Increasing high frequency loss is reported to have clear prolongation effects on the wave V latency (Watson, 1996; Oates & Stapells, 1992; Elberling & Parbo, 1987; Gorga,

Worthington, Reilnad, Beauchaine & Goldgan, 1985; 1981; Bausch & Olsen, 1986; Hall, 1992; Coats, 1978).

Selters and Brackman (1977) proposed that if the threshold is 55-65 dBHL at 4 kHz there will be a 0.1ms latency delay and if threshold is over 65 dBHL there will be 0.2ms delay. Hyde and Blair, 1981 reported 0.1ms delay in latency per 5dB, as loss exceeds 55dBHL at 4 kHz. But these studies lack validation. Watson (1999) studied effects of degree slope audiogram shape on ABR measures in 306 patients with cochlear hearing loss. It was reported that wave V latency was increased as the hearing loss and slope increased. Hence the specificity was decreased with increasing loss and slope, maximum effect was seen in losses greater than 70dBHL. I-V interval identification was reduced as high frequency loss and slope increases. For cochlear loss less than 60dB HL click evoked ABR showed near normal latencies for the stimulus that was 20dB higher than the threshold at 2-4 kHz, but for stimuli below 20dB of threshold then latencies may be prolonged (Durrant & Fowler, 1996).

Latency Intensity function (L-I) was steeper than normal in subjects with sloping hearing loss (Coats & Martin, 1977; Hall, 1992). The steepness of the L-I function increases as the magnitude of the hearing loss increases (Coats, 1978; Oates & Shapelles, 1992). They also studied L-I function for patients with high frequency hearing loss and reported that wave V tended to be delayed at low intensities but was normal or near normal at high levels.

Khaladkar, Karthik and Vanaja (2005) obtained speech burst ABRs for 20 ears with mild to moderate sensory neural hearing loss. Two stimuli were used to evoke the ABR; a standard acoustic click and the burst portion of the syllable *Ixl*. The result of their

study indicate that while click evoked ABRs exhibited latency values within normal limits, speech burst evoked ABRs showed more deviant results. There was a significant correlation between speech identification score and speech burst ABR, perhaps suggesting that using speech sounds to elicit the ABR offers an opportunity to better isolate normal speech processing from abnormal speech processing.

Speech evoked cortical response in clinical population

Cortical responses also have been studied to correlate with auditory processing in individuals with learning disability and sensory neural hearing loss (e.g., Kraus, McGee , Carrell, Zecker, Nicol & Koch, 1996; Kraus, McGee., Carrell, Sharma, Micco, Nicol, 1993; Martin, Sigal, Kurtzberg, & Stapells, 1997; Novak, Kurtzberg, Kreuzer, & Vaughan, 1989; Whiting, Martin, & Stapells, 1998).

Oates, Kurtzberg and Stapells (2002) investigated the effects of sensory neural hearing loss (ranged from mild to moderate defined as 25 to 49 dBHL to severe/profound loss defined as 75 to 120 dBHL) on cortical event related potentials (ERPs) N1, MMN, N2 and P3 and their associated behavioral measures (d' sensitivity and reaction time) to the speech sounds /ba/ and /da/ presented at 65 and 80 dB ppe SPL. They found that both ERP amplitude and behavioral discrimination (d') scores were lower for listeners with sensory neural hearing loss than for listeners with normal hearing. Their results indicate that latency measures are more sensitive indicators of the early effect of decreased audibility than are response strength (amplitude, d' or percent correct) measures. The amplitude and latency response changes, that occurred with sensory neural hearing loss were significantly greater for the later ERP peaks (N2/P3) and behavioral discrimination

measures (d' and RT) in comparison with earlier (Nl/MMN). That means sensory neural hearing loss has a greater impact on higher level or 'nonsensory' cortical processing in comparison with lower level or 'sensory' cortical processing.

Tremblay, Kalsstein, Billings and Souza (2006) studied cortical potentials in seven hearing aid user adults (50-76 years) with mild to severe sensorineural hearing participated in their study. Two identifiable consonant-vowel (CV) syllables ("shee" and "see"), were presented. P1-N1-P2 response was different for each speech sound. The latency was prolonged for "shee" (transition duration is more) than "see". That means that central auditory system of persons with sensorineural hearing loss also is capable of representing and integrating spectral and temporal information present in stimuli like normal hearing listeners.

From all these current studies, it is evident that speech evoked auditory evoked potential can provide valuable information about auditory processing that may be missed out by the conventional click evoked auditory evoked potential.

Method

Participants

Two groups of participants were included for the study. The clinical group included 10 subjects with cochlear pathology in the age range of 18 to 50 years. The control group included 12 age matched participants with normal hearing.

Participants' selection criteria: Participants included in clinical group had to meet the following criteria:

- Should have pure tone threshold between 25 to 50 dB HL
- Should have air- bone gap less than 10 dB
- Should not have any history of any neurological involvement
- Should not have any abnormality on click evoked ABR
- The hearing impairment should be post lingual
- Should not have any history of speech and language problem
- Should be a native speaker of Kannada

Participants included in clinical group had to meet the following criteria

- Should have hearing thresholds less than or equal to 15 dB HL
- Should not have any history of neurological or otological problem
- Should not have any abnormality on click evoked ABR
- Should not have any history of speech and language problem
- Should be a native speaker of Kannada

Instrumentation

The present study was carried out using the following instruments:

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

Materials

The following stimuli were used to evoke brainstem and cortical responses:

Stimulus 1: Extracted transition portion of naturally produced syllable /pa/, /ta/, /ka/ by an adult female speaker.

Stimulus 2: Extracted burst portion of naturally produced syllable /pa/, /ta/, /ka/ by an adult female speaker.

Stimulus preparation:

A female adult Kannada speaker was asked to produce the three stop consonants. A Madsen Electronics condenser mic (MD 21) was used to pick the stimuli. Speech sounds were recorded on to a computer using Creative sound blaster card (Creative technology, Japan). The sampling frequency was 14000 Hz at a quantization rate of 16-bits. To view and edit the speech sounds, PRAAT (version 4.4.27) (GNU scientific library, USA) was used. Both spectral and waveform view of the speech sounds were used to identify the burst and formant transition portion. The stimuli were then converted

into .stm format using IHS stimulus converter utility. All the stimulus was calibrated in dB nHL.

The following stimuli were used for speech audiometry

- Paired words in Kannada to determine the Speech Reception Thresholds (SRT).
- Recorded version of speech identification test word list in Kannada developed by Vandana(1998).

Table 1: Details of stimuli used for recording ABR and LLR:

	Duration of burst (in msec)	Duration of transition (in msec)	FO (in Hz)	Fl(in Hz)
/pa/	8.483	25.313	230	450-800
/ta/	9.687	49.375	230	450-800
Ikal	23.625	41.688	230	500-781

Test environment

All the tests were carried out in an acoustically treated room with adequate lighting.

Test procedure

Behavioral testing

Pure tone thresholds were assessed using modified Hughson Westlake method (Carhart & Jerger, 1959) for air conduction stimuli from 250 Hz to 8000 Hz and for bone conduction stimuli from 250 Hz to 4000 Hz. Speech Reception Threshold (SRT) was obtained using paired words in Kannada.

Speech identification scores (SIS) was obtained at 40 dB SL (ref: SRT) for stimuli presented through earphones in the following conditions:

- SIS in quiet for PB word list developed by Vandana (1998).

- SIS in noise (speech babble) with signal-to-noise ration of 0 for PB word list developed by Vandana(1998).

The subjects were instructed to repeat the words verbally and the percentage of correct identification was calculated.

AEP recording

Subjects were seated in a reclining chair in an electrically shielded and acoustically treated room. Two channel AEP recording was carried out with vertex as noninverting site, right and left mastoid as inverting sites and low forehead as site for common electrode. Auditory Evoked potentials were evoked using the protocol given in Table 1 and Table 2. It was ensured that the electrode impedance at each site was <5 *KQ* and inter electrode impedance was *<2KQ.*

Table 2: Protocol for recording brainstem responses:

Parameter	Stimuli		
	Click	Burst	Transition
Polarity	Rarefaction	Rarefaction	Rarefaction
Transducer	Insert earphone	Insert earphone	Insert earphone
Intensity	40 dBSL*	40 dBSL*	40 dBSL*
Repetition rate	11.1/sec	11.1/sec	11.1/sec
Filter setting	100 Hz-3 kHz	100 Hz-3 kHz	100 Hz-3 kHz
No of sweeps	1500	1500	1500
Amplification	100K times	100K times	100K times
Analysis time	15ms	20 ms	50 ms

* Re: Speech recognition threshold

Table 3: Protocol for recording cortical responses:

Parameter	Stimuli	
	Burst	Transition
Polarity	Rarefaction	Rarefaction
Transducer	Insert earphone	Insert earphone
Intensity	40 dBSL*	40 dBSL*
Repetition rate	3.1/sec	3.1 /sec
Filter setting	1-30 Hz	1-30 Hz
No of sweeps	300	300
Amplification	50 K times	50 K times
Analysis time	300	300

* Re: Speech recognition threshold

Waveform analysis

Waveform analysis was done offline and manually. The wave V was identified for each participant for all stimuli. Presence of ABR in each stimulus was determined by replicating the wave V vertex. Wave V and its negative trough (wave A) of ABR evoked by burst were marked. The peak to trough amplitude of the wave V was measured.

Similarly the Vth peak for transition evoked ABR was also identified and amplitude is measured. In addition, since transition ABR has a steady state portion FFR was also analyzed as described by Kraus (2000). For the transition evoked ABR, response measures that were considered were latency of wave V, wave A, wave C, wave D, wave E and wave F and amplitude of wave V (refer Figure 2). Replicable discrete

peaks subsequently after peak V were marked as peak A, C, D, E and F. The offset response O as described by Kraus (2002) was not always replicable in the present study and so was not considered in the analysis.

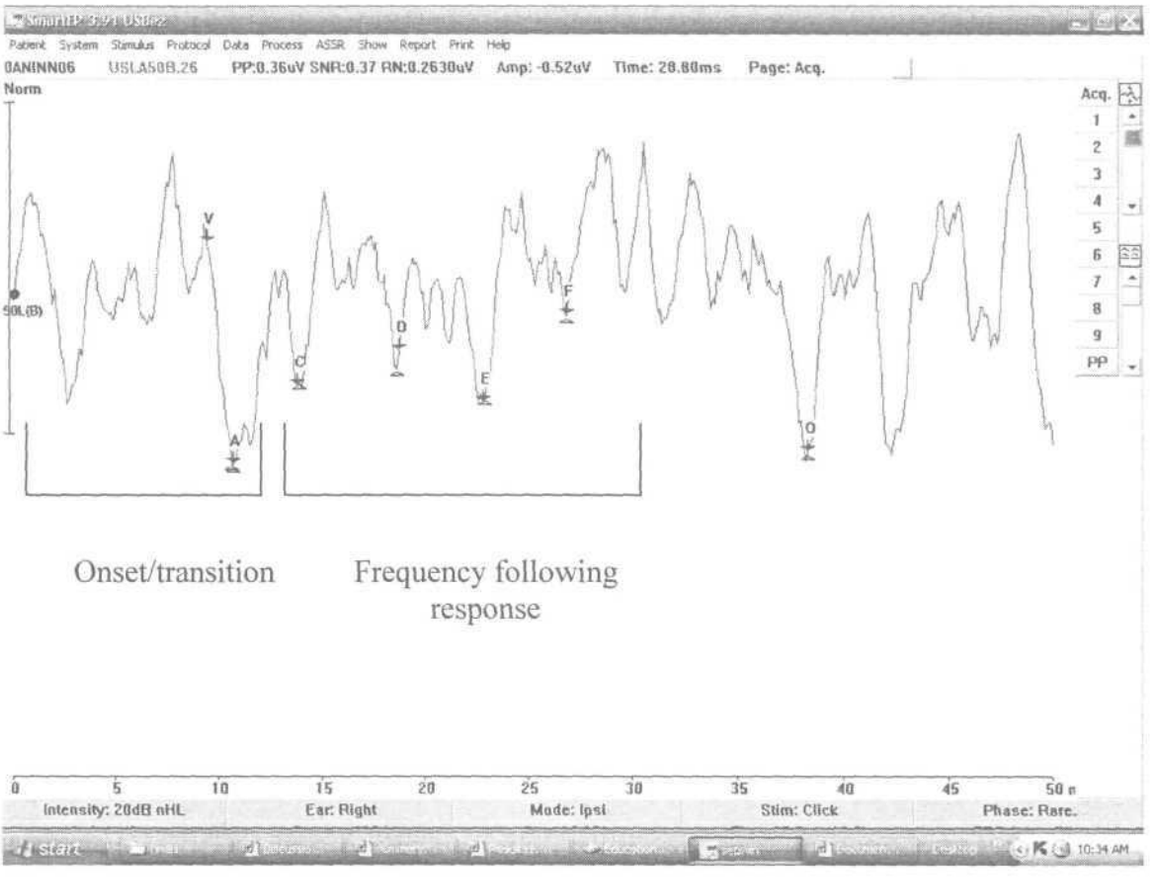

Figure 2: Transient and sustained responses for the transition of/ka/ consonant.

For LLR, peak PI, N1, P2, and N2 were measured. The peak with the highest amplitude after the stimulus onset was considered as peak P1, the large negative peak immediately after the PI was considered as N1, the positive peak after the N1 was marked as P2 and the negative peak immediately after the P2 was marked as N2. Replicability of peaks was considered to mark the peaks. 50 ms Pre stimulus averaging was taken in to consideration as baseline during recording and analysis and was subtracted from amplitude of peak N1 and P2 to get corrected amplitude.

Results

The aim of the present study was to evaluate the effect of cochlear hearing loss on speech burst and transition evoked brainstem and cortical responses. Also the relationship of speech identification scores with brainstem and cortical responses were investigated. The data obtained were tabulated and statistical analysis was carried out using SPSS software (VI5, SPSS Inc). Multivariate analysis of variance was used to assess the significant effect of groups and stimuli on brainstem potentials as well as cortical potentials. Pearson product moment correlation was administered to see correlation between SIS scores with brainstem and cortical potentials.

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

Table 4 Show the mean latency and amplitude of wave V evoked by bursts of /pa/, /ta/ and /ka/ in individuals with normal hearing and those with cochlear hearing loss. It can be noted from the table that latency of wave V for /p/ and /k/ was similar but *l\l* latency was shorter in both the groups. The amplitude of the /p/ and /k/ was similar but *iXl* amplitude was lesser than /p/ & /k/ in both the groups. It can be observed from the table that the latency is longer in individuals with hearing impairment than that of normal hearing, for all the stimuli. The amplitude of the normal hearing subjects was higher than that of subjects with hearing impairment. Figure 3 shows a representative sample of ABR for burst portion of /pa/, /ta/ and /ka/.

Table-4: Mean and SD of latency and amplitude of wave V

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

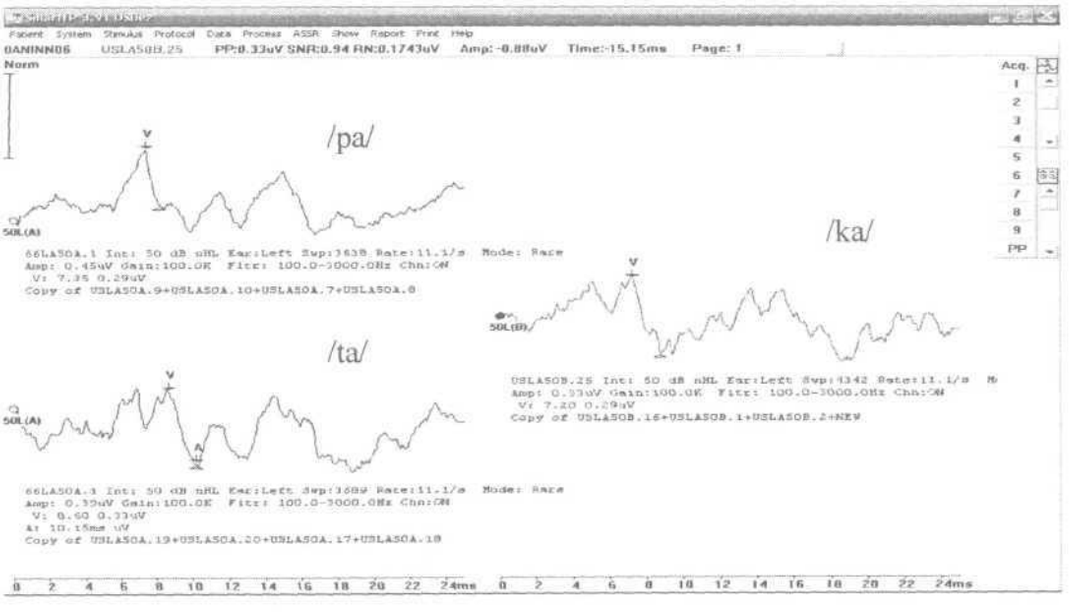

Fig 3: ABR evoked by burst of /pa/, /ta/ and /ka/ in normal hearing participants

Multivariate Analysis of Variance was administered to assess the effect of groups and three stimuli on latency and amplitude for wave V. Results revealed that there was a significant effect of cochlear hearing loss on latency (F= ; $p<0.05$) but there was no significant difference in amplitude of wave V between the groups ($p>0.05$). Also there was no interaction of stimulus with group for latency and amplitude. Scheffe's post hoc

showed no significant effect of stimulus on latency and amplitude of wave V in both groups.

Latency and amplitude of wave V in individuals with normal hearing and individuals with cochlear hearing loss for transition evoked ABR and FFR

For all the three transitions, onset response peak V and A as well as sustained response, peak C, D, E and F could be identified. Figure 4 shows the transient and sustained portion of response for the transition portion of consonants /pa/, /ta/ and /ka/. In the sustained portion of the FFR, the difference between D and E, E and F was on an average around 4 msec.It approximated the FO (230 Hz) of the stimulus.

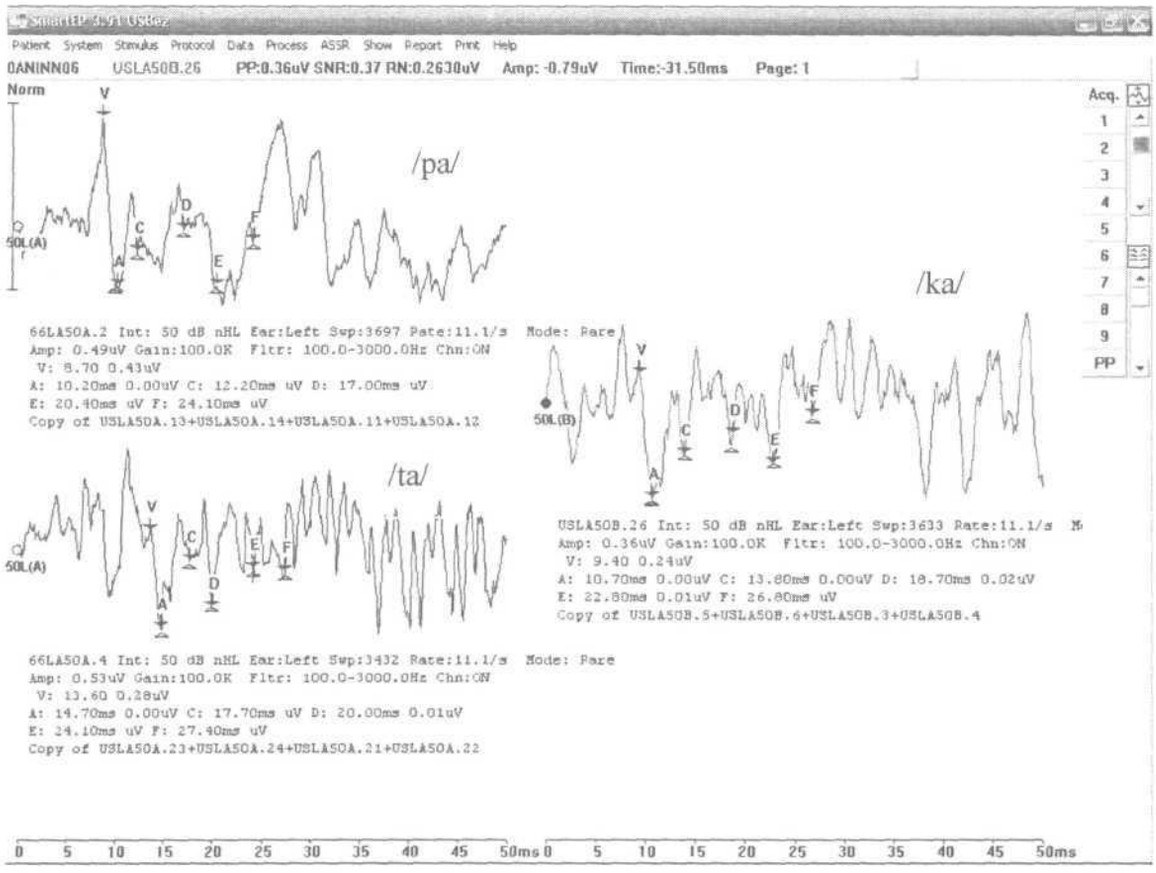

Fig 4: ABR evoked by transition of /pa/, /ta/ and /ka/ in normal hearing participants

Table 5 shows the mean latency of peaks V, A, C, D, E, F and amplitude of peak V, elicited by transition portion of/pa/, /ta/ and /ka/ in normal hearing subjects. The latency of wave V for /pa/ and /ka/ was similar but /ta/ latency was longer than the other two stimuli. The amplitude of the *Ik/* was higher than *III* and /p/. However, the standard deviation for amplitude of/ k/ was larger indicating greater variability.

Table-5: Latency of wave V, A, C, D, E, F and amplitude of wave V in individuals with normal hearing.

	V		A	C	D	E	F
	Latency in msec	Amplitude in μv	Latency in msec	Latency in msec	Latency in msec	Latency in msec	Latency in msec
pa	9.9 (2.3)	0.3 (0.04)	11.1(2.3)	13.8(2.7)	17.2(3.09)	21.4(3.1)	25.6 (3.2)
ta	12.5(1.6)	0.3 (0.07)	13.66(1.8)	18.3(2.7)	21.9(2.9)	25.5 (3)	29.8 (3.3)
ka	9.8 (2.4)	0.39 (0.2)	11.5(2.4)	14.8(1.7)	18.3(1.5)	22.4(1.6)	26.8(1.6)

Table 6 shows the latency and amplitude of waves (V, A, C, D, E, F), elicited by transition portion of /pa/, /ta/ and /ka/ in participants with hearing impairment. The trend obtained for different stimuli is similar to that observed for participants with normal hearing. The latency of wave V for /pa/ and /ka/ was similar but /ta/ was longer than the other two stimuli. The amplitude of the /pa/ and /ka/ was similar but /ta/ amplitude is lesser than /pa/ & /ka/.

Table 6: Latency of peaks V, A, C, D, E, F and amplitude of wave V in subjects with hearing impairment.

	V		A	C	D	E	F
	Latency in msec	Amplitude in μv	Latency in msec	Latency in msec	Latency in msec	Latency in msec	Latency in msec
Pa	15.4(2.5)	0.2(0.07)	17.2(2.9)	22.1(2.66)	25.7(2.6)	30.9(3.4)	35.1(3.6)
Ta	19.6(1.3)	0.22(0.2)	21.7(1.6)	27.3(2.0)	31.4(2.0)	36(1.3)	40.4(1.6)
Ka	16.2(1.7)	0.28(0.2)	18.1(1.9)	22(1.4)	26.6(2.0)	30.9(1.9)	35.2(1.8)

Multivariate analysis of variance was administered to assess the significant difference between groups for three stimuli in latency and amplitude. There was a main effect of group (cochlear hearing loss) on latency of all the peaks and amplitude of wave V ($p<0.01$) and there was no interaction between stimulus and group. Scheffe's Post Hoc analysis of variance revealed that the amplitude and latency of /ta/ differed significantly from that of/pa/ and /ka/ ($p<0.01$) for wave V of ABR and other waves of FFR but there was no significant difference between /pa/ and /ka/ ($p>0.05$).

Table 7: F, df and p values between group for latency and amplitude of transition evoked brainstem responses

	F	df	**P**
V latency	119.2	1	0.01
V amplitude	2.9	1	0.09
A latency	117.5	1	0.01
C latency	151.1	1	0.01
D latency	155.9	1	0.01
E latency	161.2	1	0.01
F latency	153.5	1	0.01

Long latency responses evoked by speech bursts

Table 8 and 9 shows the mean for latencies for the components (PI, NI, P2, N2) of LLR and amplitude of NI-P2 complex in individuals with normal hearing and individuals with cochlear hearing loss across three speech burst stimuli. Multivariate analysis of variance was carried out to check if there is a main effect of cochlear hearing loss on latencies of components of LLR and N1-P2 amplitude. Results (refer table 10) revealed that there was no significant main effect of group (cochlear hearing loss) for its measure on latency of all the peaks ($p>0.05$) but N1-P2 amplitude differed significantly ($p<0.01$) and no interaction was observed between stimulus and group. Figure 5 shows a representative sample of LLR evoked by bursts of /pa/, /ta/ and /ka/.

Table 8: Mean (SD) of Latency for components of LLR and N1-P2 amplitude recorded with burst in normal hearing subjects.

	PI	Nl	P2	N2	N1 P2 amp
pa	86.7(13.8)	127.2(12.3)	188.2(14.7)	197.6(24.1)	1.3(0.3)
ta	82.3(8.3)	128.4(16.7)	181.8(14.09)	229.2(11.8)	1.5(0.4)
ka	86.9(18.6)	130.7(12.4)	187.1(25.4)	225.4(20.5)	1.3(0.5)

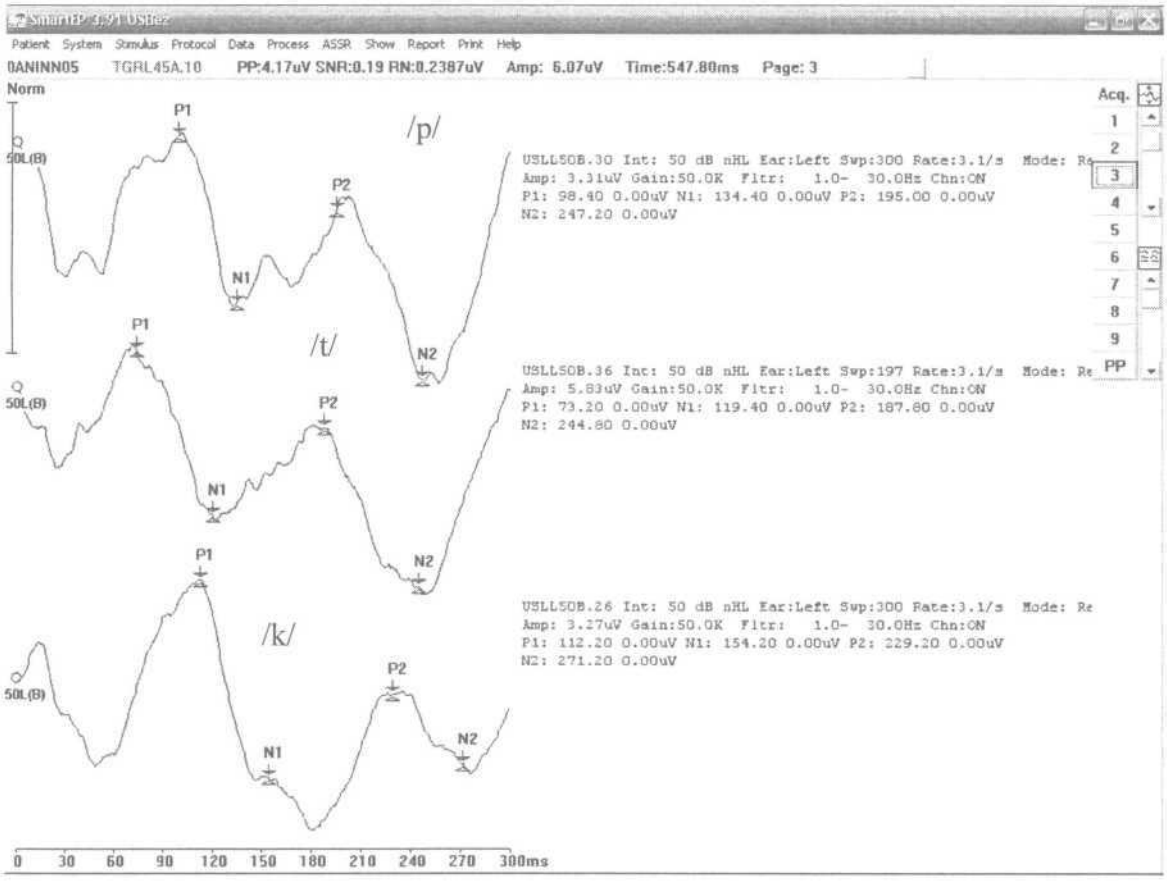

Fig 5: LLR evoked by burst of /p/, /t/ and / *k* / in normal hearing participants

Table 9: Mean (SD) of latency for PI, Nl, P2, N2 and amplitude of N1-P2 for bursts in hearing impaired subjects.

	P1	N1	P2	N2	N1 P2 amp
pa	87.3(34.2)	127.12(37.93)	184.65(42.06)	235.42(43.52)	0.98(0.17)
ta	83.3(22.7)	121.32(17.99)	173.25(16.19)	230.02(11.13)	0.83(0.16)
ka	86.07(21.90)	123.2(25.80)	177.47(17.91)	233.42(13.06)	1.04(0.30)

Table 10: F, df and p values between group for latency and amplitude of LLR peaks evoked by bursts

	F	df	P
P1 latency	0.143	1.0	0.9
N1 latency	0.25	1.0	0.6
P2 latency	1.1	1.0	0.2
N2 latency	3.6	1.0	0.06
N1-P2 amplitude	5.52	1.0	0.01

Long latency responses evoked byformant transition

Table 11 shows latencies for the components (P1, N1, P2, N2) of LLR and amplitude of NI-P2 complex in individuals with normal hearing and individuals with cochlear hearing loss across three speech formant transitions. Multivariate analysis of variance was carried out to check if there was a main effect of cochlear hearing loss on latencies of P1, N1, P2, N2 and amplitude of N1-P2. Results revealed that there no

significant main effect of cochlear hearing loss (p> 0.05) and no interaction was observed between group and stimulus. Sheffec's Post Hoc analysis revealed no significant effect of stimulus on latency or amplitude of LLR. Figure 7 shows a representative sample of LLR evoked by transition of/pa/, /ta/ and /ka/.

Table 11: Mean and SD of latency (in ms) and amplitude (in u V) of LLR peaks elicited by transition

		P1 latency	N1latency	P2 latency	N2 latency	N1 1P2 amp
Normal hearing subjects	pa	92.75(15.24)	139.31(2)	194.46(24.83)	251.31(21.29)	1.07(0.34)
	ta	95.6(24.82	142.91(29.02	212.12(48.43	240.36(36.17)	1.33(1.33)
	ka	97.1(9.88)	143.19(13.07)	211.94(31.01)	242.94(32.91)	1.47(0.4)
Hearing impaired subjects	pa	90.82(25.92)	123.52(24.82)	176.10(31.82)	230.85(44.91)	0.94(0.17)
	ta	86.62(25.53)	119.87(23.95)	178.57(35.64)	227.92(41.21)	1.01(0.16)
	ka	96.3(19.11)	137.07(19.66)	202.12(10.06)	257.4(15.99)	1.04(0.22)

Table 12: F, df and p values between group for latency and amplitude of LLR peaks evoked by transition

	F	df	P
P1 latency	0.025	1	0.87
N1 latency	1.4	1	0.29
P2 latency	0.12	1	0.47
N2 latency	1.4	1	0.68
N1-P2 amplitude	13.1	1	0.01

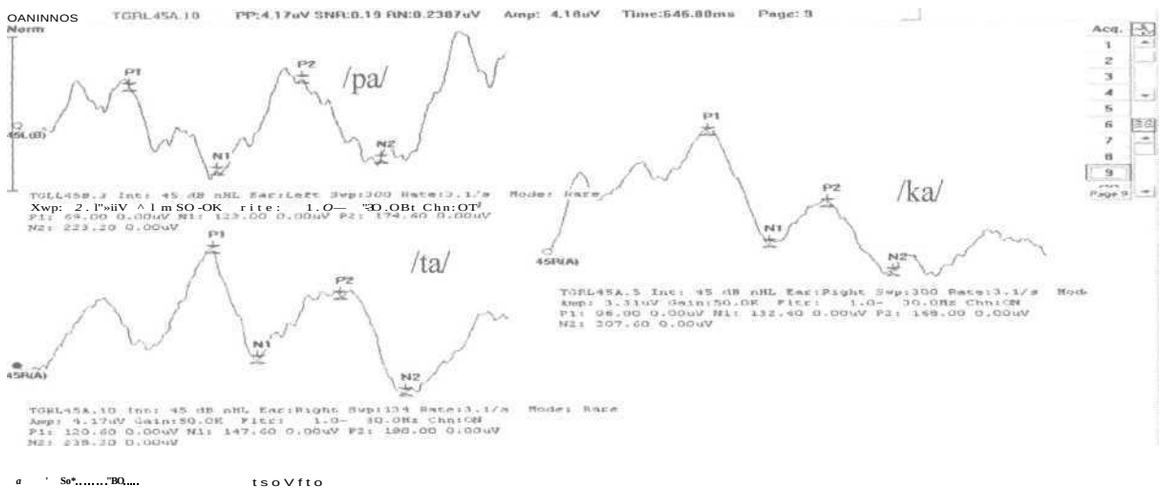

Fig 6: LLR evoked by transition of/p/, *It/* and /ka/ in normal hearing participants

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

Table 13 shows the speech identification scores in quiet and in presence of noisy condition for the participants with normal hearing and those with hearing impairment. Independent sample t test revealed that there was a significant difference between the scores of participants with normal hearing and participants with hearing impairment in both quiet ($t = 13.0$, $p<0.01$) and noisy condition ($t=19.9$, $p<0.01$).

Table 13: Mean (SD) of speech identification scores in quiet and in the presence of noise

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

Relationship between speech identification scores and brainstem and cortical responses

Pearson product moment correlation analysis was carried out to investigate the relationship of latency and amplitude of brainstem potentials for the three stimuli with speech identification scores (SIS) in quiet and in the presence of noise. Results revealed that there was SIS in noise correlated significantly with formant transition evoked FFR, and wave V for all the three stimuli (refer Table 14 for r values), but SIS score in quiet did not show a significant correlation. Speech burst evoked ABR and LLR, as well as transition evoked LLR did not show a significant correlation with SIS scores in quiet or in the presence of noise.

Table 14: Correlation of SIS scores with brainstem responses evoked by transition of /pa/, /ta/ and /ka/

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

*p<0.05 and **p<0.01

To summarize, the results of the present study revealed that brainstem and cortical responses to bursts and transition of speech stimuli can be recorded from participants with normal hearing as well as those with hearing loss. There was a significant effect of hearing loss on brainstem responses to speech but cortical responses to speech were not effected by hearing loss. Speech identification scores obtained in the presence of noise showed a significant correlation with wave V and FFR evoked by transition of speech.

Discussion

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

The prolongation of latencies in subjects with hearing impairment may be due to the high frequency hearing loss or overall reduction in audibility. Previous studies on click evoked ABR have also reported that the latency of all the peaks increase with increase in hearing threshold (Oates & Stapells, 1992; Gorga, Worthington, Reilnad, Beauchaine and Goldgan, 1985). Though statistically not significant, the mean amplitude was lesser in subjects with hearing impairment when compared to those with normal hearing. This is probably due to reduction in number of nerve fibers responding for the stimuli. It has been reported in literature that the amplitude of ABR depends on the number of nerve fibers firing (Hecox, Squires & Galambox, 1976). Thus the results of the present study suggest that, coding of the processing of burst is effected in subjects

with hearing impairment. However, speech identification scores in quiet or in the presence of noise did not show a significant correlation with latency or amplitude of ABR elicited by burst. These results contradict the report of Khaladkar, Karthik and Vanaja (2005) who observed that there was a significant correlation between SIS and speech burst ABR in subjects with sensorineural hearing loss.

The latency of the onset response (Wave V and A) for the transition portion of the signal in the present study was longer than that reported by King, Warrier, Hayes and Kraus (2002) but the latency for the other peaks (C, D, E and F) was shorter. It has been reported that the wave V and A signal the onset of sound at the brainstem whereas wave C is the response to the onset of the vowel (Kraus & Nicol, 2005). The other peaks, D, E and F are responses to sustained portion of the signal. So probably the difference in latency reflects the difference in the stimulus used in the two studies. King, Warrier, Hayes and Kraus (2002) used synthesized transition of/da/ with 40msec duration. On the other hand in the present study a natural stimulus was taken and the transition part was extracted. The duration of transition in the present study was around 25 msec for /pa/, 49 msec for /ta/ and 41 msec for /ka/. The fundamental frequency ranged from 103 to 121 Hz in their study and it was around 230 Hz in the present study.

The latency of the FFR portion in hearing impaired subjects was prolonged compared to normal hearing subjects and the amplitude was significantly reduced in these subjects. This suggests that the encoding of the sustained portion was effected in the participants with hearing impairment. The interpeak latency difference between D and E as well as E and F were around 4 msec in subjects with normal hearing whereas it was around 5 msec in subjects with hearing loss. This indicates that processing of the

fundamental frequencies was effected in subjects with hearing impairment. It has been reported in literature that the FO and Fl coding are effected in persons with hearing impairment at the brainstem level and this is reflected in the abnormalities in the waveform of ABR (Kraus & Nicol, 2005).

Auditory system encodes the FO from fine structure, but it can also encode the FO from the envelope but encoding of FO from the envelope is weaker when compared to that extracted from the fine structure (Fulkunar et al., 2005; Zeng et al., 2004). In addition psycho acoustical studies have shown that cochlear hearing impaired subjects are impaired in coding the temporal fine structure of the speech signal, which contains the F0 and harmonics (Lorenzi, Gilbert, Cam. Gamier & Moore 2006; & Moore, Glasberg, Hopkins 2006). This indicates a greatly reduced ability to use temporal fine structure speech in individuals with moderate hearing loss. This loss of ability to use temporal fine structure information perhaps was related to a loss of neural synchrony (Woolf, Ryan, Bone, 1981). This would have contributed for reduced amplitude and prolonged latencies in subjects with cochlear hearing loss.

The recent studies have shown that speech in quiet could be completely understood with only envelope cues (amplitude variation of the speech signal) (Shannon et al., 1995; Name et al., 2006; Smith, et al, 2002). But understanding of speech in noise depends on the encoding of the fine structure of the speech signal as well as envelope. It has been reported that coding of envelope of the speech signal is normal in cochlear hearing loss subjects, but processing of temporal fine structure is impaired. The results of correlation also revealed that SIS scores in noise were correlated well with components

of FFR. This support the hypothesis that processing of temporal fine structure is effected in subjects with cochlear hearing loss.

There is dearth of study investigating LLR with burst and transition in subjects with hearing loss. However the results obtained in this study are comparable with those reported in literature for other stimuli. There was no significant difference in latency of LLR for the participants with normal hearing and those with hearing impairment. This may be because the degree of hearing loss was less than moderate degree. Mild to moderate degree of hearing impairment do not significantly influence the latency of LLR (Albera et al., 1991). It has been reported that at suprathreshold levels the latency of LLR is not significantly effected by intensity of the stimulus (Picton et al., 1977). Variability of the LLR latency in normal subjects is also high. This may have been one of the reasons for obtaining no significant difference in the latency of LLR in the two groups. The N1-P2 amplitude was significantly better in subjects with hearing loss when compared to that of normal heating subjects. This suggests that probably less number of cortical cells were responding in subjects with hearing loss. It has been reported that the amplitudes of LLR depends on the number of cells responding for the stimulus. It has been reported that long deprivation of auditory stimuli may lead to loss of cells at the cortical level (Irvine, 2000). However the duration of hearing impairment in a majority of subjects in the present study was not more than 9 months. Probably there would have been a significant effect on LLR if the duration of hearing impairment was more. No significant correlation betweens SIS and LLR measures suggests that probably that poor speech perception in the subjects was mainly due to abnormal encoding of speech at the cortical and brainstem level.

Summary and conclusion

Cochlear hearing loss effects the perception of the cues used for speech perception. So individuals with cochlear impairment find it difficult to understand speech in quiet as well is in the background noise. In the recent years it has been reported that burst and transition portion of the speech stimuli can be used to record brainstem as well as cortical potentials. However, there is a dearth of studies on speech evoked auditory potentials in subjects with sensorineural hearing loss. Hedrick and Jesteadt (1996) reported that sensorineural hearing loss may disrupt formant transient coding or any type of dynamic process in periphery (i.e. rapidly changing aspects of speech signal is not being coded). So it can be hypothesized that formant transition evoked ABR may provide useful information about processing of speech at brainstem level.

Khaladkar, Karthik and Vanaja (2005) reported that ABR for speech bursts were abnormal in subjects with cochlear pathology. They also found that there was a significant correlation between speech identification score and speech burst ABR in subjects with sensorineural hearing loss. However there is a dearth of studies correlating SIS with ABR evoked by transition portion of the stimuli. Comparison needs to be made between correlation of speech identification score with speech burst and transition evoked brainstem responses. Research is needed to study the cortical representation of burst and transition of speech stimuli in subjects with normal hearing and those with hearing loss. Studies also need to be carried out to investigate the relationship of cortical responses to speech burst and transition with speech identification scores.

Hence the current study aimed to,

- To study if there is a difference between subjects with normal hearing and those with cochlear pathology in the following responses:
    - Brainstem responses to speech burst.
    - Brainstem responses to transition of speech.
    - Cortical responses to speech burst.
    - Cortical responses to transition of speech.
- To investigate the relationship between the following, in subjects with cochlear pathology:
    - Brainstem responses to speech burst and speech identification scores,
    - Brainstem responses to transition of speech and speech identification scores.
    - Cortical responses to speech burst and speech identification scores
    - Cortical responses to transition of speech and speech identification scores.

10 adult subjects with cochlear pathology served as the clinical group and 12 age matched normal hearing subjects served as a control group. Stimuli used to evoke brainstem and cortical potentials were synthesized using software PRAAT (version 4.4.27) software. Burst and transition portions were extracted separately from the stimuli /pa/, /ta/, /ka/ spoken by an adult female speaker. Auditory evoked potentials were recorded using IHS Smart Evoked Potential System (Version 2.39). Burst evoked brainstem responses were analyzed for wave V, transient evoked brainstem responses were analyzed for peak V, A, C, D, E and F and cortical evoked potentials were analyzed

for P1, N1, P2, and N2. Speech identification scores in quiet and in presence of noise (speech babble noise) were obtained for bisyllabic word list in Kannada presented through an audiometer at 40 dB SL (ref: SRT).

The data obtained were statistically analyzed using SPSS software to investigate the aims of the study. The analyses of the data revealed the following results:

- There was a significant difference in burst evoked wave V latency between cochlear hearing loss group and normal hearing group but no significant difference was found in terms of wave V amplitude.
- For the transition stimuli, latencies of wave V, A, C, D, E, & F and amplitude of wave V were significantly different between the two groups.
- All the components (V, A to F) evoked by transition stimuli significantly correlated with SIS scores in noise. But no correlation was observed for burst evoked brainstem responses.
- There was no significant difference between groups for all the components of LLR (P1, Nl, P2, & N2) but N1-P2 amplitude was significantly different between groups.
- Pearson product moment correlation revealed no correlation with SIS in quiet as well as in noise.

It can be concluded from the results of the present study that, cochlear hearing loss impairs the processing of the burst and transition portion of speech signal. The brainstem measures are more reliable measures for assessing the processing of these cues in cochlear hearing loss population. Furthermore, LLR may not be a reliable measure for assessing the processing of this specific portion of speech signal.

Future Direction

1. The present study can be replicated on large population.
2. Studies can be carried out on subjects with different degrees, configurations and type of hearing impairment.

References

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

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

Bauch, C. D. & Olsen, W. O. (1986). The effect of 2000-4000 Hz Hearing Sensitivity on ABR Results. *Ear & Hearing, 7(5),* 314-317.

Benki, J. (2001). Place of articulation and first formant transition pattern both affect perception of voicing in English. *Journal of Phonetics, 29,* 1-22.

Blumstein, S. E. & Stevens, K. N. (1980). Perceptual invariance and onset spectra for stop consonants in different vowel environments. *Journal of Acoustical Society of America, 67(2),* 648-62

Boothroyd, A. (1984). Auditory perception of speech contrasts by subjects with sensorineural hearing loss. *Journal of Speech and Hearing Research,* 27, 134—144.

Coats, A. C. (1978). Human auditory nerve action potentials and brain stem evoked responses. *Archives of Otolaryngology, 104(12),* 709-17.

Coats, A. C. & Martin, J. L. (1977). Human auditory nerve action potentials and brain stem evoked responses: effects of audiogram shape and lesion location. *Archives of Otolaryngology Head and Neck Surgery, 103(10),605-622.*

Coles, R. R. A. & Mason, S. M. (1984). The results of cortical electric response auditory in medico-legal investigations. *British Journal of Audiology, 18,* 71-78.

Cunningham, J., Nicol, T. G., Zecker, S. G. & Kraus, N. (2001). Neurobiologic responses to speech in noise in children with learning problems: Deficits and strategies for improvement. *Clinical Neurophysiology, 112,* 758-767.

Delgutte, B. (1984). Speech coding in the auditory nerve II: Processing schemes for vowel-like sounds. *Journal of the Acoustical Society of America, 75,* 879-886.

Delgutte, B. & Kiang, N. Y. (1984). Speech coding in the auditory nerve. Vowel-like sounds. *Journal of the Acoustical Society of America, 75,* 866—878.

Delattre, P. C, Liberman, A. M. & Cooper, F. S. (1955). Acoustic loci and transitional cues for consonants. *Journal of Acoustical Society of America, (27),169-113*

Dreschler, W. A., Plomp, R. (1980). Relation between psychophysical data and speech perception for hearing-impaired subjects. I. *Journal of Aoustical Society of America,* 68(6),1608-1615.

Durrant, C. G. & Fowler, J. D. (1996). ABR Protocols for Dealing with Asymmetric Hearing Loss. *American Journal of Audiology, 5,* 5-6.

Elberling, C, & Parbo, J. (1987). Reference data for ABRs in retrocochlear diagnosis. *Scandinavian Audiology, 16(1),* 49-55.

Festen, J. M, Plomp, R. (1990). Effects of fluctuating noise and interfering speech on the speech-reception threshold for impaired and normal hearing. *Journal of Acoustical Society of America, 88(4),* 1725-36.

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

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

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

Gorga, M. P., Worthington, D. W., Reiland, J. K., Beauchaine, K. A. & Goldgar, D. E. (1985). Some comparisons between auditory brainstem response thresholds, latencies, and the pure-tone audiogram. *Ear and Hearing, 6(2),* 105-12.

Gorga, M. P., Beauchaine, K. A., Reiland, J. K., Worthington, D. W., & Javel, E.. (1984).Effects of stimulus duration on ABR threshold and behavioral threshold. *Journal of the Acoustical society of America. 76,6*16-619

Gravel, J., Kurtzberg, D., Stapells, D. R., Vaughan, H. G., & Wallace, I. F. (1989) Case studies. *Seminar in Hearing, 10*, 272-287.

Gravel, J. & Stapells, D. R. (1993). Behavioral, electrophysiologic and otoacoustic measures from a child with auditory processing dysfunction: case report. *Journal of American Academy of Audiology, 4,* 412-419.

Hall, J. (1992): Handbook of Auditory Evoked Responses. Masssachutus: Allyn and Bacon.

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

Hood, L. J. (1998). Clinical applications of the auditory brainstem response. San Diego, Singular publishing group, Inc.

Humes L. E., Dirks D. D., Bell T. S. & Kincaid G. E. (1987). Recognition of nonsense syllables by hearing-impaired listeners and by noise-masked normal hearers. *Journal of American Academy of Andiology,* 81(3):765-73

Hyde, M. (1997). The Nl response and its applications. *Audiology & Neuro-otology, 2(5),* 281-307.

Jacobson, J. T. (1985). The auditory brainstem response. San Diego, CA: College-Hill Press.

Johnson, K. L., Nicol, T. & Kraus, N. (2005). The brainstem response to speech: a biological marker of auditory processing. *Ear and Hearing, 26,* 424-434.

Jewett, D. L. & Williston, J. S. (1971). Auditory-evoked far fields averaged from the scalp of humans. *Brain, 94,* 681-696.

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

King, C, Warrier CM., Hayes, E., Kraus, N (2002) Deficits in auditory rainstempathway encoding of speech sounds in children with learning problems, *Neuroscience Letter* 319:111-115.

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

Klein, S. K., Kurtzberg, D., Brattson, A., Kreuzer, J. A., Stapells, D. R., Dunn, M. A., Rapin, I. & Vaughan, H. G. (1995). Electrophysiologic manifestations of impaired temporal lobe auditory processing in verbal auditory agnosia. *Brain & Language, 51(3),* 383-405.

Kraus, N. (2001). Auditory pathway encoding and neural plasticity in children with learning problems. *Audiology and Neurotology. 6(4),* 221-7.

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

Kraus, N., McGee, T., Carrell, T.D., Sharma, A., Micco, A., & Nicol, T. (1993). Speech evoked cortical potentials in children. *Journal of American Academy of Audiology, 4,* 238-248.

Kraus, N., McGee, T. J., Carrell, T. D., Zecker, S. G., Nicol, T. G., & Koch, D. B. (1996). Auditory neurophysiologic responses and discrimination deficits in children with learning problems. *Science, 273,* 971-973.

Krishnan, A. (2002). Human frequency-following responses: representation of steady-state synthetic vowels. *Hearing Research,* 7(5(5,192-201.

Krishnan, A., Xu, Y., Gandour, J. T. & Cariani, P. A. (2004). Human frequency-following response: representation of pitch contours in Chinese tones. *Hearing Research, 189,* 1-12.

Liberman, A. M., Delattre, D. C, Cooper, F. S. & Gerstman, L. J. (1954). The role of consonant vowel transitions in the perception of the stop and nasal consonants. *Psychological Monogram, 68,* 1-13.

Lisker, L., Liberman, A. M, Erickson, D. M., Dechovitz, D. & Mandler, R. (1977). On pushing the voice onset-time (VOT) boundary about. *Language and Speech, 20,* 209-216.

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

Marsh, J. T. & Worden, F. G. (1968). Sound evoked frequency following responses in the central auditory pathway. *Laryngoscope, 78,* 1149-1163.

Martin, B. A., Sigal, A., Kurtzberg, D. & Stapells, D. R. (1997). The effects of decreased audibility produced by high-pass noise masking on cortical event-related potentials to speech sounds /ba/ and /da/. *Journal of the Acoustical Society of America, 101,* 1585-1599.

McPherson, D. L. & Starr, A. (1993). Auditory evoked potentials in the clinic. In A. M. Halliday (Ed.), *Evoked Potentials in Clinical Testing.* Edinburgh: Churchill Livingstone.

Moushegian, G., Rupert, A. & Stillman, R. (1973). Scalp-recorded early response in man to frequencies in the speech range. *Electroencephalography and Clinical Neurophysiology 35,* 665-667.

Naatanen, R., & Picton, T., (1987). The N1 wave of the human electric and magnetic response to sound: A review and analysis of the component structure. *Psychophysiology, 24*, 375-425.

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

Novak, G. P., Kurtzberg, D., Kreuzer, J. A. & Vaughan, H. G. (1989). Cortical responses to speech sounds and their formants in normal infants: maturational sequence and spatiotemporal analysis. *Electroencephalography Clinical Neurophysiology, 73(4)*, 295-305.

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

Oates, P. A., Kurtzberg, D. & Stapells, D. R. (2002). Effects of sensorineural hearing loss on cortical event-related potential and behavioral measures of speech-sound processing. *Ear and Hearing, 23(5)*, 399-415.

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

Plomp, R. A. (1978). Auditory handicap of hearing impairment and the limited benefit of hearing aids. *Journal of Acoustical Society of America, 63(2)*, 533-49.

Plomp, R. A. (1986). Signal-to-noise ratio model for the speech-reception threshold of the hearing impaired. *Journal of Speech and Hearing Research, 29(2)*, 146-54.

Sohmer, H. & Pratt, H. (1977). Identification and separation of acoustic frequency following responses (FFRS) in man. *Electroencephalography and Clinical Neurophysiology, 42,* 493-500.

Song, J. H., Banai, K., Russo, N. M. & Kraus, N. (2006). On the Relationship between Speech- and Nonspeech-Evoked Auditory Brainstem Responses. *Audiology & Neurotology*, 77,233-241.

Stevens, K. N. (1998). Acoustic Phonetics, MIT Press, Cambridge, MA

Stevens, K. N. & Klatt, D. H. (1974). Role of formant transitions in the voiced-voiceless distinction for stops. *Journal of Acoustical Society of America, 55,* 653-659

Summerfield, Q. & Haggard, M. (1977). On the dissociation of spectral and temporal cues to the voicing distinction in initial stop consonants. *Journal of Acoustical Society of America, 62,*435-448.

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

Tremblay, K. L., Kalstein, L., Billings, C. J. & Souza, P. E. (2006). The neural representation of consonant-vowel transitions in adults who wear hearing aids. *Trends in Amplification, 10(3),* 155-62.

Turner, C. W., Chi, S-L., & Flock, S. (1999). Limiting spectral resolution in speech for listeners with sensorineural hearing loss. *Journal of Speech, Language, and Hearing Research,* 42, 773-784.

Turner C. W & Cummings (1999). Speech Audibility for Listeners With High-frequency Hearing Loss. *American journal of Audiology, 8,* 47-56.

Ponton, C. W. (1996). Possible application of functional imaging of human auditory system uh the study of acclimatization and late onset deprivation. *Ear and Hearing, 17,* 78S-86S.

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

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

Russo, N., Nicol, T. G., Musacchia, G. & Kraus, N. (2004). Brainstem responses to speech syllables. *Clinical Neurophysiology, 115,* 2021-2030.

Sachs, M. B., (1984). Neural coding of complex sounds: speech. *Annual Review Physiology, 46,* 261-73.

Syka. J.,(2002) Plastic Changes in the Central Auditory System After Hearing Loss, Restoration of Function, and During Learning. *Physiological Review, 82(3).* 601 - 636

Selters, W. A. & Brackmann, D. E. (1977). Acoustic tumor detection with brain stem electric response audiometry. *Archives of Otolaryngology, 103(4),* 181-7.

Shannon, R. V., Zeng, F.G., Kamath, V., Wygonski, J., & Ekelid, M. (1995). Speech recognition with primarily temporal cues, *Science* 270, 303-304.

Sininger, Y., & Starr, A. (2001). Auditory neuropathy: A new perspective on hearing disorders. San Diego, CA: Singular publishing group.

Sohmer, H. & Pratt, H. (1977). Identification and separation of acoustic frequency following responses (FFRS) in man. *Electroencephalography and Clinical Neurophysiology, 42,* 493-500.

Song, J. H., Banai, K., Russo, N. M. & Kraus, N. (2006). On the Relationship between Speech- and Nonspeech-Evoked Auditory Brainstem Responses. *Audiology & Neurotology,* 77,233-241.

Stevens, K. N. (1998). Acoustic Phonetics, MIT Press, Cambridge, MA

Stevens, K. N. & Klatt, D. H. (1974). Role of formant transitions in the voiced-voiceless distinction for stops. *Journal of Acoustical Society of America, 55,* 653-659

Summerfield, Q. & Haggard, M. (1977). On the dissociation of spectral and temporal cues to the voicing distinction in initial stop consonants. *Journal of Acoustical Society of America, 62,* 435-448.

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

Tremblay, K. L., Kalstein, L., Billings, C. J. & Souza, P. E. (2006). The neural representation of consonant-vowel transitions in adults who wear hearing aids. *Trends in Amplification, 10(3),* 155-62.

Turner, C. W., Chi, S-L., & Flock, S. (1999). Limiting spectral resolution in speech for listeners with sensorineural hearing loss. *Journal of Speech, Language, and Hearing Research,* 42, 773-784.

Turner C. W & Cummings (1999). Speech Audibility for Listeners With High-Frequency Hearing Loss. *American journal of Audiology, 8, 47-56.*

Turner, C. W., Souza, P. E., & Forget, L. N. (1995). Use of temporal envelope cues in speech recognition by normal and hearing-impaired listeners. *Journal of the Acoustical Society of America,* 97, 2568-2576

Watson, D. R. (1996). The effects of cochlear hearing loss, age and sex on the auditory brainstem response. *Audiology, 35(5),* 246-58.

Watson, D. R. (1999). A study of the effects of cochlear loss on the auditory brainstem response (ABR) specificity and false positive rate in retrocochlear assessment. *Audiology, 38(3),* 155-64.

Whiting, K. A., Martin, B. A. & Stapells, D. R. (1998). The effects of broadband noise masking on cortical event-related potentials to speech sounds /ba/ and /da/. *Ear & Hearing, 19(3),* 218-31.

Wible, B., Nicol, T. & Kraus, N. (2004). Atypical brainstem representation of onset and formant structure of speech sounds in children with language-based learning problems. *Biological Psychology, 67,* 299-317.

Young, E. D. & Sachs, M. B. (1979). Representation of steady-state vowels in the temporal aspects of the discharge patterns of populations of auditory-nerve fibers. *Journal of Acoustical Society of America, 66(5),* 1381-1403.

Zurek P. M. & Delhorne L. A. (1987). Consonant reception in noise by listeners with mild and moderate sensorineural hearing impairment. *Journal of American Academy of Audiology,* 82(5): 1548-59

Zeng F.G, Christopher W. Turner (1990). Recognition of Voiceless Fricatives by Normal and Hearing-Impaired Subjects. *Journal of Speech and Hearing Research, 3 3,* 440-449

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