RELATIONSHIP BETWEEN SPEECH IDENTIFICATION SCORES AND AUDITORY EVOKED POTENTIALS IN CHILDREN WITH LEARNING DISABILITY

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Certificate

This is to certify that this dissertation entitled “Relationship between Speech Identification Scores and Auditory Evoked Potentials in children with learning disability” is a bonafide work in part of fulfillment for the degree of Master of Science (Audiology) of the student Registration no: 07AUD016. 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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This is to certify that this master's dissertation entitled "Relationship between Speech Identification Scores and Auditory Evoked Potentials in children with learning disability" 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.

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1. Introduction

The role of an audiologist is to identify and rehabilitate individuals with a hearing problem. Some of these problems may be very obvious and easy to identify such as a severe-to-profound hearing loss, or the presence of a conductive hearing loss. The major challenge faced is when s/he has to identify hearing problem which is subtle in nature such as the presence of a central auditory processing disorder (C) APD, or auditory neuropathy.

There are various behavioral tests which have been developed to identify different auditory processes like gap detection test which assesses the temporal integrity and dichotic CV test which assesses the binaural integration deficits are few to name. There is a surfeit of literature available to prove the sensitivity and specificity of these tests. Most of these tests are time consuming, during which there is high possibility for the child to get distracted or lose his attention. It is also possible that some children may not understand the complex instructions in which case the testing would be difficult. In Indian context, where many languages are spoken it is difficult to develop a test in each language. Also, for children less than 7 years of age there is lack of normative data available. In case the normative data is available, a wide range of scores makes it difficult to identify a child with an auditory processing disorder.

The prevalence of a central auditory processing deficit is more in children with learning disability. The National Joint Committee on Learning Disabilities (NJCLD, 1990) defines the term learning disability as: “a heterogeneous group of disorders manifested by significant difficulties in the acquisition and use of listening, speaking, reading, writing, reasoning or mathematical abilities. These disorders are intrinsic to the

individual and presumed to be due to the Central Nervous System Dysfunction" (NJCLD, 1990).

The concept of LD covers an extremely wide range of characteristics. One of the earliest profiles, developed by Clement (1966), includes the following 10 frequently cited attributes:

- Hyperactivity
- Perceptual-motor impairments
- Emotional liability
- Co-ordination problems
- Disorders of attention
- Impulsivity
- Disorders of memory and thinking
- Academic difficulties
- Language deficits

There have been many studies done to evaluate the usefulness of auditory evoked potentials (AEPs) in discriminating children having a learning problem with those having no learning problem. Majority of these studies have recorded AEPs using speech as a stimulus as it represents the signals encountered in daily living situation.

The studies have shown that in the auditory brainstem responses the latency of wave V, and the wave V slope latency and amplitude are sensitive measures to differentiate between children having learning problem from those having no learning problem (Wible, Nicol, & Kraus, 2005; Cunningham, Nicol, Zecker, & Kraus, 2001). In

the auditory late latency responses it is the amplitude of the N1-P2 complex which is sensitive to children with a learning problem (Putter-Katz et al, 2005; Purdy, Kelly, & Davies, 2002; & Cunningham et al, 2001).

Behavioral measures of speech intelligibility show that children with a learning problem have poorer speech perception ability than the children without a learning problem. This difference in the perception is enhanced in stressful environmental conditions like listening in the presence of background noise, degrading the perception abilities of children with a learning problem even more (Wible, Nicol, & Kraus, 2005; Cunningham et al, 2001).

Various studies have been done using AEPs to tap the exact nature of deficit responsible for degrading speech perception in children with learning problem. No one study has been able to find the exact deficit (Song, Banai, & Kraus, 2008; Russo, Nicol, Musacchia, & Kraus, 2004; & Cunningham et al, 2001).

Need For the Study

Learning disability (LD) is a very heterogeneous group; it has a lot of subgroups. Some children having LD may exhibit auditory processing deficits, while some may not exhibit auditory processing. There are various tests which enable us to differentiate the kind of deficit the child has. Most of these tests are subjective in nature and require complete attention and concentration of the child. The results of the tests would be invariably affected by the variables such as the attention span of the child, his/her willingness to co-operate in the testing. The child might be wrongly diagnosed as having

LD based purely on these subjective tests if the above mentioned variables are not controlled. Hence, there is a need for an objective test which will help us in accurately diagnosing these children.

In literature there are many studies done to find the neurophysiological responses i.e., auditory evoked potentials (AEPs), in children without a learning problem and in children with learning disability. Majority of these studies have been done under quite background conditions and not in adverse listening conditions (Wible, Nicol, & Kraus, 2005). The learning disabled population performs well in quite situations; whereas the major problem faced by them is in adverse listening situations (Russo et al, 2004; Cunningham et al, 2001). Hence these study over estimate their performance. AEPs for speech stimuli in adverse conditions could be sensitive in identifying auditory processing deficits, as most often this population does not exhibit abnormality in quite conditions.

As both the speech perception abilities and the AEPs are affected in the learning disabled group to a larger degree than compared to normals there is a need to relate both.

Aim of the Study

The aim of the present study was to:

1) Find a relationship between the latency of the ABR wave V, ALLR waves N1 and P2 with the SIS scores obtained in different conditions independently for both the groups separately.
2) Find a relationship between the amplitude of N1-P2 complex with the SIS scores obtained in different conditions independently for both the groups separately.

3) Know whether the latency of ABR wave V vary in the three different conditions and also between the two groups.
4) To know whether the latency of N1 and P2 waves vary in the three different stimulus conditions and also between the two groups.
5) Similarly to know whether the amplitude of the N1-P2 complex differ in different conditions and also between the two groups.

2. Review of Literature

An auditory evoked response (AER) is activity within the auditory system (the ear, the auditory nerve or auditory regions of the brain) that is produced or stimulated (evoked) by acoustic signal. In simpler words AER's are the brain waves (electrical potentials) generated when a person is stimulated with sounds. The stimuli that can be used to elicit AER range from clicks to tones, and even to speech sounds. AER can be recorded in a non invasive way via evoked potential recording. It can be used for differential diagnosing different neurological disorders and also to estimate the threshold (Hall, 2007).

The auditory evoked potentials that are clinically useful can be classified along various dimensions:

1) Classification based on latency.
2) Classification based on source.
3) Endogenous and exogenous potentials.

I. Classification of AEPs based on peak response latency distinguishes-

- *Very short* : <2-3 msec; e.g., Electro Cochleo Graphy (ECochG)
- *Short latency* : < 10msec; e.g., Auditory Brainstem Response (ABR), Frequency Following Response (FFR)
- *Middle latency* : 10 to 50msec e.g., Middle Latency Response (MLR), 40 Hz response
- *Long latency* : 50 to 250 msec e.g., Long Latency Response (LLR)

- *Very long latency:* > 300 msec e.g., Mismatch Negativity (MMN), P300, Contingent Negative Variance (CNV).

II. Classification of AEPs based on source:

- *Compound action potentials* generated in the nerve tracts [Compound Action Potential (CAP) and ABR].
- *Compound postsynaptic potentials* generated in cell dendrites (MLR and LLR).

III. Endogenous vs. Exogenous Potentials:

- *Endogenous potentials:* Auditory evoked responses that occur between 200 and 600ms are considered endogenous or more related to intrinsic factors. Endogenous refers to the event related potentials whose response is primarily mediated through cognition. MMN, Processing negativity, N200, P300, N400, P500, CNV are the endogenous potentials (Hall, 2007).
- *Exogenous potentials:* Exogenous refers to the evoked potentials whose response is primarily mediated by stimulus parameters. Also referred to as sensory (auditory) evoked potentials. They are unaffected by attention and state of the subject. This includes ECochG, ABR, FFR, MLR, 40 Hz, ASSR, P60, N100, P160, and T-Complex (Hall, 2007).

2.1 Short Latency Potentials (ABR, FFR)

The Auditory brainstem response (ABR) is a series of vertex positive waves that occur within 10-15 msec of the onset of a click stimulus in human adults. These peaks are typically labeled by the Jewett and Williston (1971) convention, using sequential capital roman numerals. Although seven peaks are often seen, in most cases only waves I to V

are evaluated. As waves II and IV are quite variable in amplitude and identifiability, the most commonly evaluated ABR peaks include I, III, and V (Hall, 2007).

2.1.1 Factors Affecting ABR Recording

ABR can be affected by several factors such as stimulus factors, acquisition factors etc. The information gathered about each factor has been given below.

Stimulus factors

Acoustic stimulus used to elicit can affect ABR depending on its characteristics. Type of stimulus, frequency, duration etc has significant impact on ABR recording.

Type of stimuli: The ABR is typically measured clinically with 100 μsec click signals, generated by higher frequencies in the click spectrum, roughly from 1000 – 8000 Hz (Hall, 2007). The major drawback of click as a stimulus for clinical assessment auditory function of infants and young children is the lack of its frequency specificity. For that reason, the use of tone burst ABR is preferred technique for frequency-specific estimation of auditory function. Recently ABR has also been recorded for speech stimuli (Hall, 2007).

Intensity: All waves of the ABR show a systematic increase in latency and decrease in amplitude as stimulus intensity decreases from 80 dB nHL to the threshold of detectibility (Picton, Hillyard, Kravsz, & Galambos, 1974). Wave V is the most visible at lower intensity levels, whereas the earlier components tend to become indistinguishable at intensities of 25 to 35 dB nHL. Near the threshold of the response, Wave I occurs at approximately 4.0 ms and wave V at about 8.0 ms in adults (Hall, 2007).

Duration*:* Click duration does not have a marked influence on ABR latency and amplitude. There is no latency change for stimulus durations ranging from 25 to 100 µsec, although with an increase in duration from 100-200 µsec the latency increases by about 0.1 msec (100 µsec) and, with increase in duration from 100-400 µsec the latency increases by about 0.2 msec (200 µsec) (Beattie & Boyd, 1984).

Rise time of the stimulus*:* Salt & Thornton (1984) reported that there is a slight, but unspecified, increase in the latency as the rise time is varied from 170 to 580 µsec. Similar results were also reported by Suzuki & Horiuchi (1981).

Stimulus rate and inter-stimulus interval: The rate at which test stimuli are presented affects both the latency and the amplitude of the components of the ABR. At stimulus rates above approximately 30/sec, the latency of all components of the ABR increases and the amplitude of the earlier components decreases (Don, Allen, & Starr, 1977).

Polarity: Some investigators reported significantly shorter ABR wave V latency values for rarefraction than for condensation clicks in most individuals with normal hearing sensitivity (Emerson, Brooks, Parker, & Chiappa, 1982; Stockard, Stockard, Westmoreland, & Corfits, 1979). Other authors have noted the opposite effect- i.e., shorter latency values for condensation than for rarefraction clicks (Coats & Martin, 1977). The most consistent polarity related ABR findings is shorter latency for wave I (0.07 msec) for rarefraction clicks, but a condensation click advantage still occurs in some subjects (Hall, 2007).

Acquisition factors

Several acquisition parameters also affect ABR. There have been extensive studies by several researchers: factors which affect the ABR have been reviewed and given below.

***Electrode*:** The ABR is recorded minimally with non-inverting electrode located at the vertex or high forehead, the inverting electrode locate near the ear (earlobe, mastoid) on the stimulation side and, a ground (common) electrode can be located at the non stimulating ears mastoid or earlobe or low forehead (Hall, 1992).

***Analysis time*:** For routine clinical ABR assessment, a minimal analysis time of 10 msec is necessary and recommended. An analysis time of 15 msec, however, is recommended because it encompasses ABR latencies for virtually all patients, including infants and patients with hearing loss (Hall, 2007).

***Filter*:** Band pass filter settings of 30/100 – 1500/3000 Hz are appropriate to encompass spectrum of response while reducing undesirable electrical activity. Changes in the high-pass filter settings exert pronounced effects on the ABR. As the cutoff is increased from a very low value (e.g., 0.05 Hz) up to a restricted value of 500 Hz, wave V ABR amplitude is reduced by 50% and latency progressively decreases (Cacace, Shy & Satya-Murthi, 1980). For the low-pass filter, decreasing the cutoff frequency from a very high value of 10,000 Hz to 3000 Hz eliminates noise and thereby enhances waveform analysis without distorting ABR latency or amplitude. Further reduction in the low pass filter cutoff from 3000 Hz to 1500 Hz produces an increase in absolute latencies of major ABR wave components, and produces a smoothening effect (Hall, 2007).

Subject Related factors

Other than stimulus and acquisition factors additional factors such as age, gender, and arousal state, etc., can also influence ABR. It is important to keep these factors in mind before interpreting the ABR findings of a person.

Age: The ABR changes as a function of age, particularly during the first 12-18 months of life, as the central auditory system continues to mature (Hall, 2007). Reliable ABR components for 65 dB nHL clicks have been reported in newborns of approximately 28 weeks gestational age (Starr, Amlie, Martin, & Sanders, 1977). Waves I, III and, V are most visible in infant recordings and the normal wave V absolute latency for a newborn is approximately 7.0 msec at 60 dB nHL. Wave I may be slightly prolonged in infants, but generally is not as prolonged as wave V, This leads to a longer inter-wave latency of about 5.0 msec compared to 4.0 msec in adults (Hecox & Galambos, 1974). Various studies have also shown that with the advancement in age there is a prolongation in the absolute and inter wave latency (Oku & Hasegewa, 1997). Hall (2007) stated that within the age range of 25 to at least 55 years, latency increases by approximately 0.2 msec.

Gender**:** Females tend to have shorter latency and higher ABR amplitude than males (Allison, Wood, & Goff, 1983; Jerger & Hall, 1980). Wave V latency is on an average 0.2 msec shorter in females, and amplitude is higher, particularly for Waves IV, V, VI, and VII. It has been suggested that the source of the differences in latency and amplitude in the ABR between males and females may be related to shorter cochlear response times in females than males (Don, Ponton, Eggermont, & Masuda, 1994).

***Attention and state of arousal*:** ABR waveforms are not affected by sleep (Jewett & Williston, 1971). For the clinical purpose, the ABR does not differ significantly as a function of attention of the patient (Picton & Hillyard, 1974).

***Drugs*:** studies have reported that the ABR is not influenced by sedatives, relaxants, barbiturates, or anesthesia (Sanders, Duncan, & McCullough, 1979). However, abnormal ABRs have been reported in conjunction with medications such as phenytoin, lidocaine, and diazepam. Also, carbamazepine (CBZ) monotherapy results in prolongation of peak latencies of wave I, III, and V and prolongation of waves I-III and I-V intervals.

Brainstem response to speech stimuli in quiet

Animal models have been used to describe auditory nerve and cochlear nucleus single-unit response properties for synthetic speech like sounds (Delgutte, 1984). Not only do auditory nerve and cochlear nucleus fibers show increase phase locked activity to the formant harmonics of the stimulus, but separate populations of neurons appear to code the 1^{st} and the 2^{nd} formant (Johnson, Nicol, & Kraus, 2005)

Speech stimuli have also been used in humans to study the response characteristics of the Frequency Following Response (FFR) (Galbraith et al.., 2004; Krishnan, 2002). 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. Krishnan has studied the FFR elicited by synthetic vowels to relate phase locking characteristics of brain stem neurons to individual harmonics of a complex sound. Results suggest that human FFR spectra show clear and distinct peaks corresponding to formant frequencies of steady state synthetic vowel.

Johnson, Nicol, & Kraus (2005) studied the brain stem response to a speech stimulus /da/ of 40 msec in duration. The consonantal segment contained an initial 10 msec burst. The frequencies of the consonantal segment were centered around the 3rd to the 5th formants, thus in the range of 2580-4500 Hz. The F_o of the utterance ranged between 100 Hz to 120 Hz. The neural response can be described morphologically in terms of an onset transient response (ABR) and a sustained element that comprises the FFR, as seen in Figure 2.1. The robust onset response is similar to that observed in response to a tone or click stimulus, consisting of waves I, III, and the VA complex. The voiced portion of the stimulus evokes the periodic portion of the response, the FFR, which reflects phase-locking to the waveform of the stimulus.

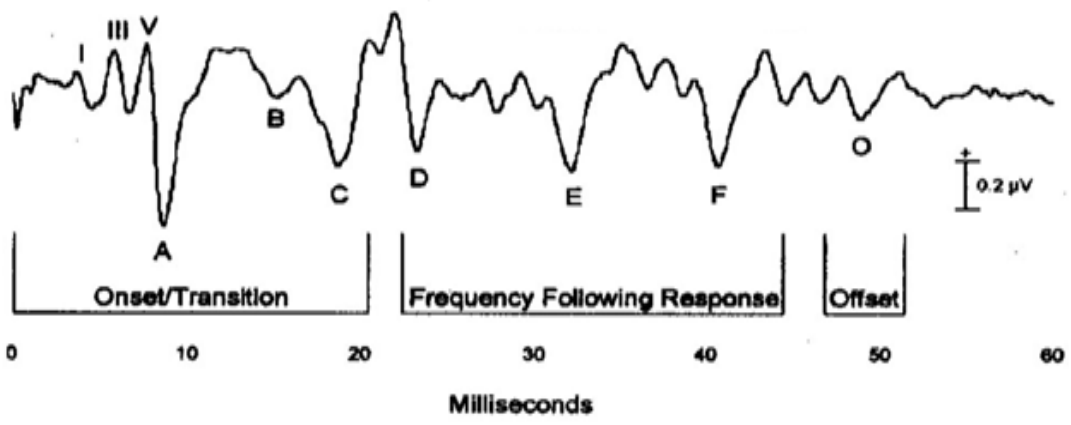

Figure 2.1: Auditory brain stem response to the speech syllable /da/. Waves I, III, and V are standard nomenclature for the onset response complex.

A visual analysis of the /da/ stimulus waveform and its corresponding brain stem response reveals several similarities. Shifting the stimulus waveform by approximately 7

msec to account for neural conduction time reveals an even more striking match (Johnson, Nicol, & Kraus, 2005).

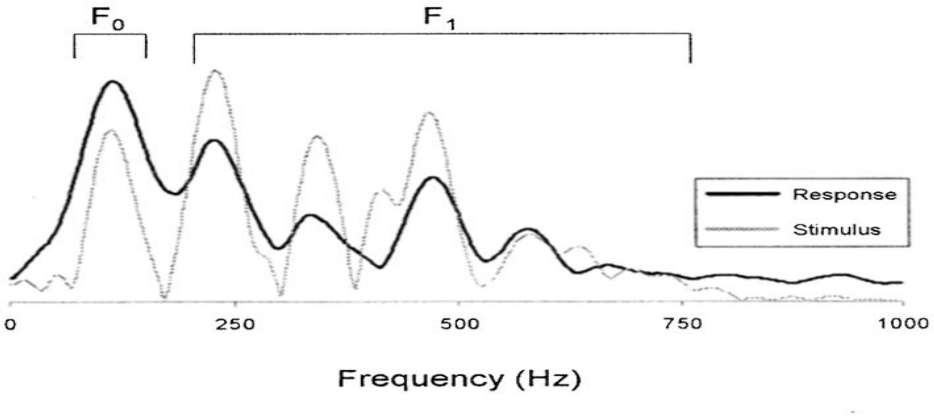

Figure 2.2: Fourier analysis of the stimulus (light line) and the brain stem response (dark line).

The period between response peaks D, E, and F corresponds to the wavelength of the *F*0 of the utterance. These peaks represent the glottal pulsing of the vocals folds and are thus representing source information. Fourier analysis of this portion of the response confirms a spectral peak at the frequency of *F*0. 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 msec after stimulus onset.

Wave O probably is a response to the cessation of sound, as it corresponds temporally to the offset of the stimulus. Together, these transient peaks, the timing of which is sensitive to stimulus spectrum, comprise responses to the acoustic filter characteristics of the syllable. Additionally, the spacing of the small, higher-frequency

fluctuations between waves D, E, and F correspond in frequency to the *F*1 of the stimulus. *F*2 is also an important acoustic cue for identifying linguistic content. However, *F*2 and the higher formants in the /da/ are beyond the phase- locking limit of the rostral brain and, consequently, are not evident in the response.

Brainstem response to speech stimuli in background noise

In the presence of background noise, brainstem encoding of speech is disrupted. In particular, noise interferes with the onset response. In the majority of normal subjects the onset response is severely degraded, while in 40% of subjects it is completely abolished. On the other hand, the FFR portion of the response is less susceptible to noise and the FFR peaks are identifiable in cases where the onset has disappeared (Russo et al, 2004).

Russo et al, (2004) reported degraded ABR response to speech stimuli in the presence of noise. They recorded brainstem response to speech syllables in 38 children with normal hearing and no learning problem. They evaluated the effect of adding background noise on the normal brainstem encoding of the speech stimulus /da/. They found that the onset responses V and A were most affected, being severely degraded and completely obscured in more than 40% of the subjects. The peaks C and F, however, remained present in noise in most subjects (100 and 86%, respectively). There was a reduction in the peak amplitude of all the peaks ($p< 0:001$). The latencies of onset peaks V and A, and FFR peak C were delayed in comparison to quiet. In contrast, peak F showed resilience to background noise in that its latency did not change with the addition of the noise and remained easily identifiable in most subjects. F0 and F1 amp were also significantly affected by the presence of background noise.

Abnormal transient responses to stimulus onset (Purdy, Kelly, & Davies, 2002; Jerger, Martin, & Jerger, 1987) and abnormal phase-locked representation of stimulus harmonic structure (McAnally & Stein, 1997) have been reported in subjects diagnosed with learning or auditory-processing problems.

Cunningham et.al (2001) reported poorer ABR responses for children with learning disability when compared with children having no learning problem. They compared the neurophysiological responses to speech in noise for children with learning disability (LP) to those with no learning problems (NL). They recorded ABR, FFR, and LLR and also found behavioral JND's. For the FFR they performed a Fast Fourier Transform (FFT), wherein they divided the FFR in 3 frequency bins (0-200, 250-400 and 450-750 Hz). In quiet, they found no difference in the magnitude of the spectral components (FFT) for the response to /da/ between normal and LP children. In noise, for the NL group there was a significant reduction in the magnitude of the spectral content of bin 1, whereas bins 2 and 3 remained stable. In contrast, all 3 bins, reflecting the fundamental frequency and F1 transition (0±750 Hz), were significantly diminished in LP children in noise. In quiet, there were no significant differences in wave V latency or amplitude elicited by /da/ between normal and LP children. With the addition of background noise, both normal and LP children displayed a prolongation in wave V latency and a reduction in wave V amplitude. However, comparison of wave V latency to /da/ in noise between normal and LP children revealed that LP children exhibited significantly longer wave V latencies on the order of 0.41 msec. There were no significant group differences in wave V amplitude in noise.

Purdy, Kelly, & Davies, (2002) compared the ABR, MLR and the LLR recorded from children being diagnosed as having learning disability with those recorded from children with no learning problem. They found that there were minor differences in the ABR latency between the two groups. The children diagnosed as having learning disability were also suspected as having (C) APD based on the SCAN and SSW test.

Wible, Nicol, & Kraus, (2002) found mixed results on comparing neurophysiologic responses to repeated speech stimuli, presented in quiet and noise, in normal children and children with learning disability. They found that the learning disability group consisted of two subgroups, one whose responses appeared relatively normal, and another whose responses were severely degraded by repetition in noise.

Wible, Nicol, & Kraus, (2004) on comparing and correlating brainstem (ABR) and cortical (ALLR) auditory processes in normal (NL) and language impaired (LP) children found that the duration of the wave V slope was more prolonged in LP children compared with NL children. No group differences were observed for wave V and wave V slope latencies or amplitudes. Averaged waveforms for NL and LP groups revealed the prolonged relative latency of wave V slope with respect to wave V in the group of LP children. In the presence of noise the correlation between cortical responses to repeated stimuli reduced, and this reduction was more pronounced in LP children compared with NL children. A strong correlation between brainstem and cortical auditory processing was demonstrated by the NL children; decreased duration of the ABR wave slope related to decreased differences in inter-response correlations between quiet and noise conditions. Furthermore, the LP group as a whole failed to demonstrate a relationship between

brainstem and cortical measures that was demonstrated to be quite strong across all normal children. However, a subset of roughly three-quarters of the LP children appeared to demonstrate the normal relationship between brainstem and cortical processing, suggesting that they share a common functional connection with NL children.

Johnson, Nicol, & Kraus, (2005) reviewed the various studies evaluating brainstem responses to speech sounds in quiet and in presence of background noise between two groups of children, one having a learning problem and the other without any learning problem. The speech stimulus used in majority of the studies was the speech sound /da/ which was of 40 msec. They report that one third of the group having learning problem exhibit delayed peak latency or shallower slope measures of the VA onset complex and of waves C and O, indicating poor synchrony to transient events. They also report that children with learning problem displayed diminished F_1 spectral content which is one of the filter cues. In the presence of background noise for the group with learning problem they say that spectral cues present in the F_1 region were diminished but not in F_0 region.

Song, Banai, Russo, & Kraus, (2006) reported that although click ABR in children with learning disability are typically within clinical norms, they tend to be delayed. It is therefore possible that scalp-measured ABR are not sensitive enough to document this minute effect and that deficits are therefore observed only in response to more complex stimuli. The speech-evoked ABR may be conceptualized as the neural code of speech syllable (Johnson, Nicol & Kraus, 2005). Reading-impaired children

demonstrate delays in the latencies of waves V and reduction in the wave V slope (Banai et al., 2005).

Song et al. (2008) evaluated brainstem responses to speech sounds to find the origin of the deficits in children with learning problem. They compared the latency and amplitude of the ABR waves in both children having a learning problem (LP) and those having no learning problem (NL). They found that many of the LP children's scores overlapped with those of NL subjects. However, a large subgroup had abnormally delayed latencies of waves V and A, or extremely imprecise responses as reflected by reduced VA slopes and prolonged duration of the VA transition. For the wave I they found no evidence for meaningful group differences between NL and LP children at the most peripheral level of the auditory brainstem. Wave III was reliably evoked in the large majority of subjects of both groups and its timing did not differ between them. They concluded that the auditory deficits in the majority of LP children with abnormal speech-evoked ABR originate from corticofugal modulation of sub cortical activity.

2.2 Auditory Long Latency Potentials (ALLR)

The cortical auditory evoked potentials (CAEPs) are scalp recorded evoked potentials that occur in response to variety of stimuli (Naatanen & Picton, 1987). CAEPs can be classified into 'obligatory' and 'discriminative' potentials. Discriminative potentials are evoked by the change from frequent 'standard' stimulus to infrequent 'deviant' stimulus. The discriminative potentials consist of MMN, P300. The 'obligatory' CAEP are classified in terms of their latencies or the time of occurrence after

presentation of a stimulus (Hall, 2007). The obligatory CAEP is also called auditory long latency responses (ALLR) or long latency responses (LLR).

The long latency auditory evoked potentials are characterized by components comprising the time domain of 50-500 ms (Mc Pherson & Starr, 1993) and are labeled according to the polarity and latency at the vertex (Picton et al., 1978). Major components in the long latency auditory evoked potentials include a positive component at about 60 ms, another positive component at 160 ms and two negative components at about 100 and 200 ms (Mc Pherson & Starr, 1993).

2.2.1. Factors Affecting ALLR Recording

There are several factors which can affect ALLR recording such as stimulus factors, acquisition factors and, subject related factors.

Stimulus factors

There are various types of acoustic stimuli that can be used to elicit ALLR, based on the property of the stimulus being used the ALLR parameters such as its latency, amplitude and morphology will be different.

Type of stimuli: ALLR can be evoked by a wide variety of transient sounds such as Click, Tone burst, noise burst, and syllable and also by sudden changes in continuous sounds such as in amplitude or frequency spectrum (Jerger & Jerger, 1970). Amplitude of ALLR components vary as a function of nature of stimulus (Ceponiene, Alku, Westerfield, Torki, & Townsend, 2005; Ceponiene et al., 2001). Ceponiene et al. (2001) reported that the amplitude of N1 to P2 complex is larger for speech sounds than for single frequency tonal stimuli, but latency values for N1 and P2 are usually earlier for

tonal versus speech stimuli. It has been reported by several authors that the ALLR response vary according to the kind of stimulus used for recording.

***Frequency of the stimulus*:** Stimulus frequency can alter the amplitude of N1-P2 even when the loudness of the stimulus is controlled. In contrast to the amplitude, latencies increase as the frequencies increases, particularly when high intensity stimulus is used. Sugg and Polich (1995) reported that the amplitude for the N1 and P2 components is larger, and the latency is longer for low frequency tonal signal in comparison to high frequency signals. However, Rothman (1970) reported that inter subject mean of N1-P2 amplitude is negatively correlated with frequency in the range of 500 Hz to 2 KHz. But he also reported that this function vary considerably between subjects. Grimes and Fieldman (1971) reported that no effect of frequency change from 500 Hz to 4000Hz on the difference between the behavioral threshold and ALLR threshold.

***Intensity*:** It is one of the most important parameter encountered mostly with the clinical protocol. One of the first observation made about ALLR was that the amplitude of ALLR is increased in an essentially linear fashion as stimulus intensity increased, where as latency decreased over the same intensity range (Rothman, 1970). Changes in the amplitude as a function of stimulus intensity tended to level off, or saturates for moderate to high intensities approximately above 70 dBnHL (Beagley & Knight, 1967). Rapin, Schinnel, Tourk, Krasneger and Pollak (1966) reported that ALLR latency changes with intensity vary for clicks versus tonal stimuli. For an ALLR evoked by a click stimulus, latency for the N1 or P2 components changes relatively little as stimulus intensity increases, except at the intensity closer to auditory threshold. They also found that the largest amplitude changes with intensity for 1000 Hz, less for 250 Hz, and least for 6000

Hz. It has been concluded that there is considerable inter and intra subject variability in the amplitude – intensity relationship of ALLR.

***Duration*:** The effect of stimulus duration on ALLR was studied extensively since 1960. Onishi and Davis (1968) reported that amplitude of ALLR increases as the stimulus duration increases to approximately 30-50 ms. Most of the studies concentrated on explaining the effect of stimulus duration on ALLR based on the temporal integration time. Alain, Woods and Covarrubias (1997) discovered that changes in signal duration produced different scalp distribution for the ALLR N1 wave and P2 wave i.e. the fronto - central region for ALLR N1 wave and the posterior electrode site for the P2 wave. These findings were based on the temporal integration properties of ALLR. Onishi and Davis (1968) used 1000Hz tone burst with linear on and off ramps. They observed that varying the rise/fall and plateau times had complex effects on the ALLR latency and amplitude. On varying the duration from 0 – 300 msec while keeping the rise/fall time constant (30 msec), there was no effect on the latency and amplitude of the ALLR components. However, for a brief rise/fall time of 3 msec, reduction of the plateau time from 30 – 0 msec produced a reduction in the amplitude of the ALLR component. They also reported that with longer plateau amplitudes were nearly constant.

Rate and Inter-stimulus interval (ISI): Rothman, Davis & Hay (1970) reported that longer inter stimulus interval and concomitantly, slower stimulus rates produced substantially larger amplitudes for N1 and P2 components of ALLR. However the latency of these ALLR components had little effect. Conversely, Davis and Zerlin (1966) reported that with increase in ISI there is predictable increase in ALLR amplitude. The increased ISI is required for increased amplitude of ALLR is due to the refractory period

of the auditory nerve. Roth, Ford, Lewis & Kopell (1976) reported that differential effect of interstimulus interval for the amplitude of N1 versus P2. The amplitude of P2 increases rather systematically with stimulus rate, where as the N1 amplitude remains relatively stable for ISI within the range of 0.75 to 1.5 ms. Bruneau, Roux, Guerin, Barthelemy and Lelord (1997) reported that ISI of longer than 1 second is required to consistently record an N1 component from children. Picton, Woods, Baribear, and Healey (1977) also reported that reduction in the ISI from 4 sec to 1 sec may lead to reduction in the amplitude in the order of 50 percentages or more. On the other hand, the N2 appears to be relatively unaffected by increasing the stimulus rate.

Contralateral signals: The ALLR may be altered by sounds presented to the non stimulus ear. The contralateral sound may be tones, some type of noise or speech. Competing sounds presented to one ear appear to interfere with subject attention (Hall, 2007). Many studies have shown that the effect of contralateral sound was different for N1 and P2 waves. In addition, the effect of contralateral signal on ALLR varies as a function of the interaction of the various factors, including characteristics of the target stimulus, difficulty of the listening task, and subject factors such as age (Fisher, Morlet, & Giard, 2000; Hymel, Canford & Stuart, 1998). Cranford, Rothernel, Walker, Stuart, & Elargovan, (2004) further investigated the effect of competing noise on N1 and P2 components of ALLR on 10 normal hearing female adults. The task involved was to discriminate between two frequencies. The amplitude of N1 and P2 were compared between two conditions i.e. with the competing noise and without the competing noise. They found that there is no change in the amplitude of N1, but there is a reduction in the amplitude of P2 when compared between two conditions. Based on this study they

pointed out towards the independence of the N1 and P2 waves and argued against the simple analysis of N1-P2 complex within the ALLR waveform.

Acquisition Factors

Acquisition factors also have an impact on the ALLR being recorded. There have been extensive studies by several researchers regarding how acquisition factors affect the ALLR.

***Electrode*:** Electrode placement may affect the ALLR because of its site of generation. Cody, Jacobson, Waller, and Bickford (1964) reported that response amplitude was largest when recorded at vertex. Many investigators presented evidence confirming that the vertex, or a location within two or three centimeters lateral or anterior to vertex, is an optimal electrode site (Picton & Hillyard 1974; Ruhm, 1971; Varghan & Ritter, 1970; Teas, 1965).

Analysis time: The ALLR should be analyzed with a pre-stimulus average of 100ms and post stimulus analysis of 1000 to 1500 ms (Hall, 2007). ALLR analysis time should be extended at least for 500 ms after stimulus (Hall, 2007). Pre stimulus analysis gives an idea about the variation of EEG such as the alpha rhythmic activity of the patient.

***Filter*:** The filter setting for evoked potential is selected according to the frequency content of the response. The frequency composition or spectrum for ALLR response is in the frequency region below 30 Hz (Yamamoto, Sakabe & Kaliho, 1979; Sayers, Beagley, & Henshall, 1974). Hence band pass filter setting of less than 1 Hz to 30 or 100 Hz is typically employed in ALLR recording (Hall, 2007).

Subject characteristics

Not only the stimulus factors or acquisition factors can affect ALLR, several other subject realted factors have an effect on ALLR. Factors related to subjects that can also affect ALLR recording are age, gender, subject status etc.

Age: The Prominent changes in ALLR waves occur within the five years of life, and to a lesser extent within 2-5 of years age range (Suzuki & Taguchi, 1968). The N1 wave is not present in infants and young children, and for children age 3 to 10 years it is recorded only with extended inter stimulus interval of 1 second or longer (Ponton et al., 2000; Sharma, Kraus, Gee, & Nicol., 1997). Latency decreases and amplitude increases as a function of age during childhood (Tanguchi, Picton, Orpin & Goodman, 1969). ALLR can be recorded from premature, full term, newborn and older children also (Hall, 2007). Barnet, Ohlrich, Weiss, and Shanks, (1975) reported that the latency of P2 shortens from 230 to 150 ms; N2 from 535 to 320 ms during the age range of 15 days to 3 years. McPherson, Tures, & Starr, (1989) reported that ALLR or at least one component of ALLR could be recorded from normal hearing infants at birth. Some investigators have shown a general increase in the latency and decrease in the amplitude with advancing age (Callaway, 1975). However, Spink, Johannsen and Pirsig (1979) reported that shorter P2 latency for older subjects of 65 years of age compared to younger age group. On the other hand Amenedo and Diaz (1998) reported that P2 latency does not change with aging.

***Gender*:** Although gender effects have been suspected by many of the investigators, it has rarely been investigated. The gender effect in the brain structure and function are well documented (Witelson, 1991). The result of many studies emphasized on the complexity

of interactions among ALLR, stimulus conditions, age and gender. There are other investigators who report that no consistent gender effect for ALLR N1, P2, and N2 components exists (Hall, 2007). Onishi and Davis (1968) reported that ALLR amplitude in general tended to be larger for females and also reported that the amplitude versus intensity function steeper for females than males. Shucard, Shucard, & Cummins, (1981) found in both verbal and nonverbal conditions that females had higher amplitude responses from left hemisphere than male subjects, where as males showed higher amplitude responses than females from the right hemisphere.

***Attention and state of arousal*:** Most of the reports showed that apparent increase in the amplitude of ALLR response with increased stimulus oriented attention. Picton, et al. (1974) reported that the amplitude changes are most marked at the stimulus level near threshold. They also found an increase in the amplitude with increased stimulus oriented attention and, progressively diminished amplitude of N1 from the awake state to sleep stage 4. During transition to deep sleep, P2 amplitude may actually increase although agreement on this trend is lacking (Campbell, Bell & Bastein, 1992). Amplitude of ALLR generally becomes more variable in sleep (Rapin, Schimmels & Cohen, 1972). Colaria, Diparsia and Gora (2000) reported that probably some of the sleep related changes in ALLR waveforms are related to the underlying fluctuations in the EEG activity.

***Drugs*:** Drugs such as anesthetic agents, tranquilizers and psychotherapeutic agents may influence the ALLR recording (Hall, 2007). Apart from this other like alcohol also influences ALLR recording. Mendel, Hasick, Davis, Hirsh, & Dinges, (1975) reported that sub cortical induced sleep was associated with increased variability in ALLR and it is less accurate. The N1, P2, N2 components amplitude was reduced by benzodiazepine but

the latency of these components is been less affected (Lader, 1977). There are some other sedative such as meperidine like morphine has no apparent effect on ALLR (Hall, 2007). Properidol produced a latency prolongation of about 10 ms for P1 and N1 of the ALLR components and amplitude reduction (Hall, 2007). Anesthetic agents also produce diverse effect on different components of ALLR. Skinner and Shinota (1975) reported that during sedation with chloral hydrate, ALLR variability is increased. Halliday and Manson (1964), found no significant decrease in ALLR amplitude. Different tranquilizers produce mixed effects on ALLR components such as chlorpromazine increases latency of waves P2 and N2 without affecting the latency of N1, or the amplitude of any components (Lader, 1977). Another tranquilizer such as lithium increases ALLR latency without affecting the amplitude (Hall, 2007).

***Alcohol*:** The amplitude of ALLR is decreased by acute alcohol intoxication (Porjesz & Begleiter, 1981; Wolpaw & Penry, 1978). Murata, Araki, Tanigawa, & Uchida, (1992) investigated the effect of acute alcohol ingestion on the ALLR N1 and P2 latency. It was found that latency of the N1 was significantly prolonged when the recording was done after 2 hours of alcohol ingestion, where as the P2 latency was found to be unchanged after the alcohol intake.

***Handedness*:** Handedness along with the different electrode placement influences the ALLR recording. Alexander and Polich (1997) investigated the possible influence of handedness on N1, P2 and N2 components of auditory late responses. They found that there was no handedness effect for N1 amplitude, but the latency of N1 component was shorter for left handed versus right hander. P2 amplitude was smaller for left handed

subjects, where as P2 latency was not related to handedness. They also reported that handedness was not a factor in N2 amplitude.

Learning Disability: Putter-Katz et al. (2005) found latencies of auditory evoked responses (AERPs) recorded during syllable identification of the learning disabled group were prolonged relative to those of children with no learning problem. Also, N1 amplitudes were larger and P3 amplitudes were smaller for children with learning disability. They concluded that the latency and amplitude of AERPs are sensitive measures of the complexity of phonological processing in children with and without a learning disability.

Brief Audiological Profile of children with Central Auditory Processing Disorder

While testing for (C) APD there are various factors which can affect the outcome of the test. Some of these factors may be known the age of the child, the presence or absence of a peripheral hearing loss, the language ability of the child and, the cognition are some of them. Other than this also there can be variables which affect the results of the test, but they remain unknown to the clinician. These can be the motivation of the child, the attention span, memory. It is possible that either of these variables may contribute to the poor scores of the child in a (C) APD test, and thus wrongly labeling the child as having a central auditory processing disorder.

Among the AEPs, the ABR is the most likely to be normal in children with APD leading to the view that sub cortical structures in the ascending auditory pathway are not involved in APD. Hall & Mueller (1997) report findings for the ABR, AMLR and P300

in a series of over 200 children evaluated for APD. Fewer than 10% had abnormal ABR findings, whereas majority of the children had abnormal findings for cortical AEPs. Abnormal ABR consisted of modest delays in inter-peak latencies.

LLR in presence of background noise

One of the well recognized challenges encountered by the children with (C) APD is to comprehend speech in the presence of background noise and competing speech signals (Jerger & Musiek, 2000). Kraus and colleagues in 2001 found smaller amplitude of the P2 and N2 wave complex recorded for children with auditory learning problems when speech signal /ga/ was presented at 0 dB SNR in comparison to ALLR amplitude for a control group under the same conditions.

Cunningham et al. (2001) reported no difference in latency of N1 and P2 in the presence of noise for children with and without learning problem. They compared the neurophysiological responses to speech in noise for children with learning disability (LP) to those with no learning problems (NL). For the children with no learning problem they found that the P1-to-N1 and P2-to-N2 amplitude for /da/ and /ga/ stimulus was reduced, where as there was no significant difference in the latency. However, the P2-to-N2 response in noise demonstrated a significantly larger amplitude reduction in LP children compared to normal children. The earlier waveforms (P1 and N1) showed no group differences in latency and amplitude in noise.

Purdy, Kelly, & Davies, (2002) found in children being diagnosed as having learning disability, who were also suspected as having (C) APD based on the SCAN and

SSW test, had Wave Na of the MLR was later and Nb was smaller as compared to children having o learning problem. The main differences in cortical responses were that P1 was earlier and P3 was later and smaller in the LD group.

Wible, Nicol, & Kraus, (2004) evaluated the timing deficits and training related improvements in auditory cortical responses to speech in noise. They compared the cortical responses to speech stimuli in quiet and in presence of noise, for both children having a learning problem, and children with no learning problem. They used BBN at 0 dB SNR. They found that for children with no learning problem, there was a reduction in the amplitude of the peaks of cortical responses in presence of noise as compared to the responses in quiet.

Based on the several studies reviewed here it's easy to observe that Learning disability (LD) is a very heterogeneous group; it has a lot of subgroups. Some children having LD may exhibit auditory processing deficits, while some may not exhibit auditory processing. In order to test for the auditory processing disorder in these children various tests can be administered. The problem with these tests is that they don't account for subject variables such as attention span of the child or the motivation.

It has been frequently cited in the literature the sensitivity of the neurophysiological responses that is, the auditory evoked potentials, in detecting the presence of an auditory processing disorder in children. Also, it is a known fact that children with learning disability are at risk of having an auditory processing disorder (Jerger & Musiek, 2000). Most of the electrophysiological studies in children with a learning disability although have been done in a quiet situation. The problem with these

studies is that they don’t strain the auditory system enough to reveal the presence of an auditory processing disorder. In order to reveal the deficit(s) studies need to be done in adverse listening situations.

The speech perception in noise in individuals with learning disability is more severely affected than in individuals with no learning problem. Hence, there is a need to compare and correlate the speech perception abilities and the AEP results in the learning disabled.

3. Method

The main aim of the study was to compare the AEP's recorded from children with learning disability with the AEP's recorded from the children with no learning impairment having normal hearing sensitivity. An attempt was also made to correlate the behavioral performance in SPIN test to the AEP's, which would help in predicting the presence of auditory processing disorder in children with learning disability. Two groups of subjects were taken to achieve the objectives.

Subjects:

A total of 20 subjects were taken for the study. They were divided into two groups. Group I consists of children with learning disability who served as the clinical group; and group II consists of children with no learning disability who served as the control group.

Clinical Group: A total of 20 ears from '10' children in the age range of 7 to 15 years, who were diagnosed as having learning disability by an experienced speech language pathologist; and psychologist was taken. All the children had normal hearing sensitivity.

Selection Criteria:

Subjects who met the following criteria were taken:

- All the subjects had pure tone thresholds within 15 dB HL at octave frequencies from 250 Hz to 8000 Hz for air conduction and between 250 Hz and 4000 Hz for bone conduction.

- All the subjects had good Speech Identification Scores (above 90%) in quiet.
- All of them had ‘A’ type tympanogram with acoustic reflex threshold within normal limits, indicating a normal middle ear function.
- No relevant otologic history was reported by the subjects.
- No history of any observable medical or neurological impairment.
- All the subjects were diagnosed as having learning disability by an experienced speech and language pathologist, based on the Early Reading Skills test results (by Loomba, 1995), and a psychologist.

Control Group: A total of 10 ears from ‘10’ children in the age range of 7 to 15 years, whose language skill was adequate to their age were taken. All of them had normal hearing sensitivity.

Selection Criteria:

- All the subjects had pure tone thresholds within 15 dB HL at octave frequencies from 250 Hz to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction.
- All the subjects had Speech in noise (SPIN) scores 70% and above at 0 dB SNR.
- ‘A’ type tympanogram with acoustic reflex threshold within normal limits was obtained from all the subjects, indicating a normal middle ear function.
- No relevant otologic history was reported by the subjects.
- No history of any observable medical or neurological impairment was noticed.

- A checklist developed by WHO (1999, cited in Singhi, Kumar, Malhi, & Kumar, 2007) was administered on all the children to rule out the presence of any learning impairment.

Equipments:

i) A calibrated diagnostic audiometer, Grandson Sandler-61 (GSI-61) was used for pure tone and speech audiometry. The pure tone audiometry and speech audiometry was carried using TDH-39 ear phone for air conduction and speech test, whereas B-71 bone vibrator for bone conduction testing.

ii) A calibrated immittance meter (GSI Tympstar) was used to test for acoustic reflexes and status of the middle ear via tympanometry.

iii) Intelligent Hearing System (IHS smart EP Version 4.0) was used to record auditory brainstem responses and auditory late latency responses for speech as the stimulus, in the presence of ipsilateral noise and without noise. Both the stimulus and the noise were presented through an insert ear phone, ER-3A.

Environment:

The testing was administered in a sound treated room. The ambient noise conditions were within the permissible limits (ANSI 1991; S3.1).

Procedure:

Initially a detailed case history was taken enquiring about the presence of any medical, neurological and, otological problem. Detailed informations were also gathered

about the presence of any signs or symptoms which could be indicative of an audiological problem.

A checklist developed by WHO in 1999 was administered to rule out any age inappropriate language deficits for the control group. For the clinical group detailed language testing was done to check for their language level. Early Reading Test developed by Loomba (in 1995) which consists of alphabet testing, visual and auditory discrimination test, phoneme-grapheme correspondence, structural analysis, close and oral reading test, was administered by a speech language pathologist and/or a psychologist to diagnose the children as having learning disability.

Both the groups underwent a routine audiological evaluation which consisted of:

i) *Pure tone audiometry:* The behavioral pure tone thresholds at octave frequencies from 250 Hz to 8000 Hz for air conduction and 250 Hz to 4000 Hz for bone conduction were obtained. The thresholds were tracked using the modified Hughson and Westlake method (Carhart & Jerger, 1959).

ii) *Speech identification scores (SIS):* SIS were obtained at 40 dB above the SRT for each ear individually. Monosyllabic word list developed by Mayadevi in 1978 was used for the same.

iii) *Speech in noise scores (SPIN):* SPIN test was done in two different conditions having speech stimulus at 40 dB above the SRT level. The type of noise used was the speech noise. A standardized word list developed by Vandana (1998) was used as the stimulus.

The conditions in which SPIN scores obtained were:

- ✓ SIS at 0 dB SNR
- ✓ SIS at +3 dB SNR

iv) *Immitance Measurement:* Tympanometry and reflex-metry was done using 226 Hz probe tone. The acoustic reflex thresholds were checked at 500 Hz, 1 kHz, 2 kHz and 4 kHz both ipsilaterally and contralaterally for both the ears to rule out presence of any middle ear pathology.

Those who fulfilled the selection criteria either for the control group or the clinical group underwent AEP recording. Details of the AEP recording are given below.

Procedure to obtain data:

AEP recording: Both the ABR and LLR were recorded in a single session. The subjects were made to sit comfortably on a reclining chair. They were asked to relax and avoid extraneous movements of head, neck, and limbs in order to avoid muscle artifacts.

Electrode placement:

Each electrode sites were first cleaned by scrubbing with cotton soaked in skin preparing paste. The electrodes were then dipped in to skin conduction paste and fixed on the scalp sites using a surgical tape. It was ensured that independent electrode impedance was less than 5 kΩ and inter electrode impedance was within 3 kΩ. Three silver chloride disc type electrodes were used for AEP recordings. The parameters used to record ABR and LLR can be seen in the Table 3.1 and 3.2 respectively.

Table 3.1: Parameters used to record ABR

STIMULUS PARAMETERS		**ACQUISITION PARAMETERS**	
Stimulus type	Speech stimulus (/da/)	*Mode*	Monaural stimulation
Stimulus duration	40 msec	*Electrode type*	Disc electrode
Stimulus rate	9.1/sec	*No of channels*	Single channel
Polarity	Alternating	*Analysis window*	60 ms
Number of Sweeps	1500	*Filter settings*	100 Hz – 3000 Hz
Intensity	65 dB SPL for both the subject groups	*Notch Filter*	On
Transducer	ER-3A insert receiver	*Replicability*	Twice for all the 3 conditions
Ipsilateral masking	i) without noise ii) with 65 dB SPL WBN (0 dB SNR) iii) with 62 dB SPL WBN (+3 dB SNR)	*Electrode montage*	Ground: non test ear mastoid (M_i) Inverting: test ear mastoid (M_i) Non inverting:forehead (Fpz)
		Gain	1,00,000 times
		Artifact rejection	40 µV

Table 3.2: Parameters used to record ALLR

STIMULUS PARAMETERS		**ACQUISITION PARAMETERS**	
Stimulus type	Speech stimulus (/da/)	*Mode*	Monaural stimulation
Stimulus duration	40 msec	*Electrode type*	Disc electrode
Stimulus rate	Speech: 1.1/sec	*No of channels*	Single channel
Polarity	Alternating	*Analysis window*	500 ms with pre stimulus recording of 50 ms
Number of Sweeps	300	*Filter settings*	1 Hz – 30 Hz
Intensity	65 dB SPL for both the subject groups	*Notch Filter*	Off
Transducer	ER-3A insert receiver	*Replicability*	Twice at all the 3 conditions
Ipsilateral masking	i) without noise ii) with 65 dB SPL WBN (0 dB SNR) iii) with 62 dB SPL WBN (+3 dB SNR)	*Electrode montage*	Ground: non test ear mastoid (M_i) Inverting : test ear mastoid (M_i) Non inverting : forehead (Fpz)
		Gain	50,000 times
		Artifact rejection	80 µV

ABR and LLR were recorded at 65 dB SPL. This level was chosen because according to Olsen, (1998) normal conversational level is between 55-65 dB SPL.

For ABR the Wave V latency was noted. For the LLR the N1, P2 latency and N1-P2 complex' amplitude were identified and marked. More emphasis was given on these two components of the LLR waveform as the N1 amplitude was found to be more affected by noise (Putter-Katz et.al, 2005).

Waveform Analysis

Both the ABR and LLR waveforms were stored for further analysis. Later the waveforms were recalled and analyzed. The waveforms were shown to three experienced audiologists. Their task was to identify the presence or absence of a response for both ABR and LLR for all the stimuli conditions. When there was an agreement regarding presence of response between the three audiologists the latencies of Wave V of ABR, N1, P2 of LLR and the amplitude of N1-P2 complex for LLR were noted. The prominent peaks of the response were then correlated to the behavioral SPIN results in both the groups.

The following parameters of the responses were calculated:

i) For the ABR the wave V latency was compared between the conditions and within the groups. It was also compared between the groups for the same condition. This was done to check for significant difference between the conditions and also across the groups.

ii) For ALLR also the latencies of N1 and P2, and the amplitude of N1-P2 complex were compared between the three stimulus conditions and across the two groups. For the same stimulus condition group wise comparisons were made. Also within the group comparisons were made to see which condition was significantly different from the other.

iii) The latency and amplitude of both the AEPs was compared with the SPIN scores within the three stimulus conditions in both the groups separately. This was done to check if there was any correlation between the SPIN scores and either of the AEPs.

4. Results

The SIS scores and the AEP data obtained for the different conditions and from both the control (children with no learning problem) and the clinical (children been diagnosed as having learning disability) groups were tabulated. They were then compared to check if there was any statistically significant difference in the data between the two groups. The data was also analyzed to compare the differences amongst the three stimulus conditions in each group separately. The final part of the analysis was to correlate the SIS scores with the AEP results for each of the three conditions separately. The details of the statistical analysis are given below.

4.1 Auditory brainstem Responses

The latency of the wave V was noted for both the groups in all the three conditions. The mean and standard deviation (SD) values for the same is given in the table 4.1. The table also shows the Z-values and the level of significance from the result of Man Whitney test.

Table 4.1

Mean and SD for Wave V latency for both the groups along with Z-value and significant level between the groups for the three conditions

Condition	Group	Mean (in msec)	SD	Z -Value	Sig. Level
ABRw	*Normals*	6.81 (N=7)	.54	1.43	0.15
	LD	7.07 (N=11)	.44		
ABR0	*Normals*	7.58 (N=7)	.92	0.70	0.48
	LD	7.73 (N=11)	.67		
ABR3	*Normals*	7.27 (N=7)	.70	1.62	0.10
	LD	7.57 (N=11)	.45		

Note: ABRw: ABR without noise; ABR0: ABR at 0 dB SNR; ABR3: ABR at +3 dB SNR; Sig. Level: Significance Level; same for the later tables also.

Mixed ANOVA's (condition 3 x groups 2) were done to examine the effects of the three stimulus conditions on the wave V latency of both the groups. The result of the mixed ANOVA revealed that there was a significant difference in the Wave V latency across the three conditions [$F(2, 32) = 21.605$, $p<0.001$]. Further analysis was done for pair wise comparison to see which conditions were significantly different from each other using Bonferroni's Post Hoc Test. The results of the test revealed that there was a significant difference in the latency in without noise condition with that of 0 and +3 dB SNR. Within the 0 and + 3 dB SNR condition there was no significant difference.

Mixed ANOVA results did not show any significant difference between the groups [$F(1, 16) = 1.155$, $p>0.05$]. It also did not reveal any significant interaction between the group and within the condition on the wave V latency [$F(2, 32) = 0.27$, $p>0.05$].

4.1.1 Auditory Brainstem Response in the control group

The latency of the wave V was noted for all the three conditions. The waveform recorded from one of the subject is shown in figure the 4.1, and the mean and SD are given in the figure 4.2.

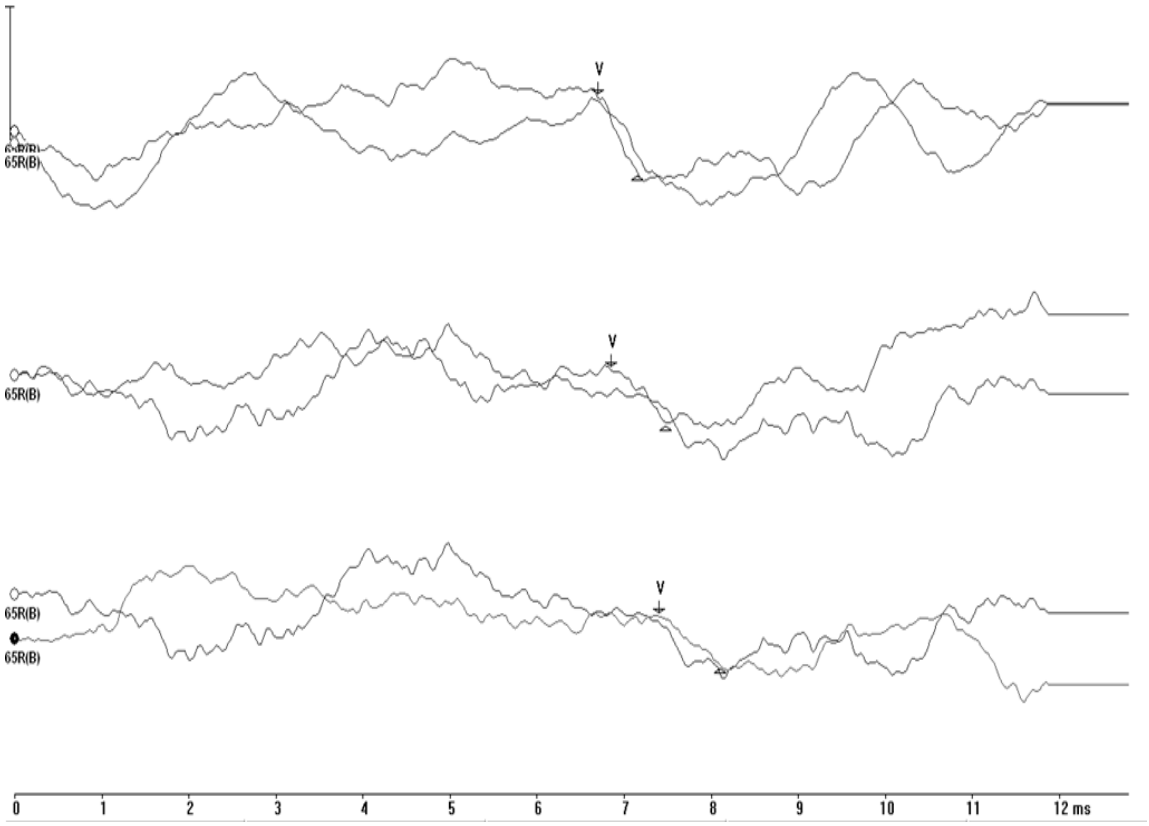

Figure 4.1: ABR waveform recorded from the right ear for all the conditions for one of the subjects in the control group.

The first pair of waveforms recorded in the condition with no ipsilateral noise, followed by a pair of waveforms recorded at 0 dB SNR condition, and final pair is at the +3 dB SNR condition.

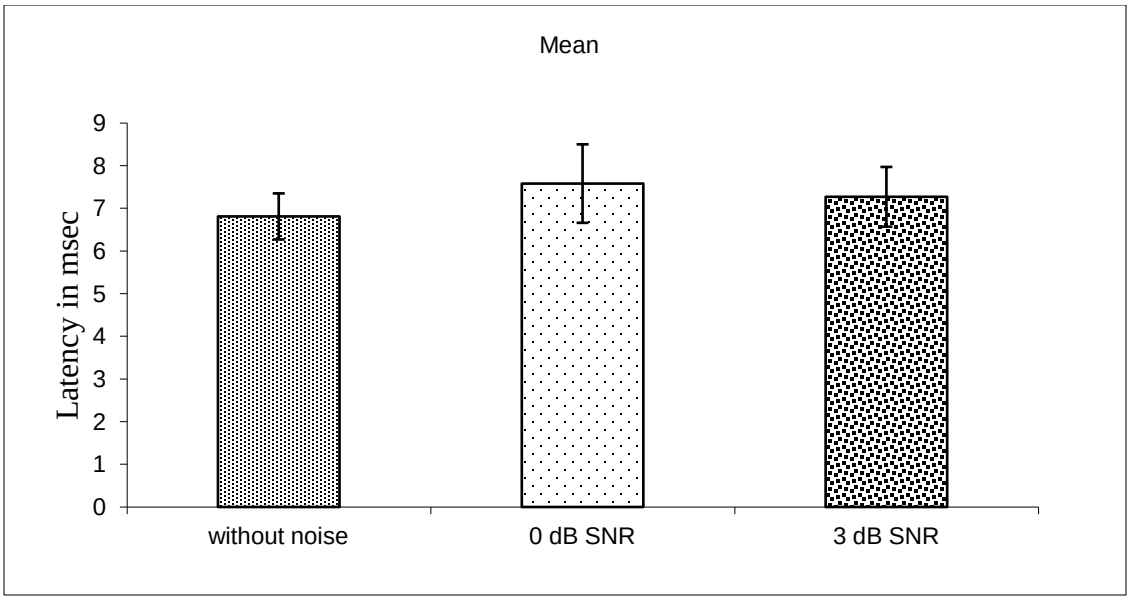

Figure 4.2: Mean and SD for Wave V latency for the control group.

It can be seen from the figure 4.2 that for the stimulus condition with 0dB SNR the latency was maximum. The wave V latency was the shortest when ABR was recorded without ipsilateral noise.

The latencies were compared across the stimulus conditions using Friedman's test for the control group. Friedman's test revealed a significant difference in the latency across the three stimulus conditions [χ^2 (2) = 10.571, $p<0.005$]. To find out pair wise difference wilcoxon's test was done. The results of the Wilcoxon's test are depicted in the table 4.2.

Table 4.2

Depicts the Z-values and significance level for the ABR Wave V latency between the three stimulus conditions obtained in the control group

Condition	**Z -Value**	**Sig Level**
ABRw - ABRs0	2.66	***.008***
ABRw - ABRs3	2.37	***.018***
ABRs0 - ABRs3	1.183	.237

The wilcoxon's test revealed that there was a statistically significant difference in the wave V latency in the condition with no noise as compared to the conditions with noise (both 0 and 3 dB SNR). The comparison of the latencies in the two conditions with noise revealed no significant difference.

4.1.2 Auditory Brainstem Response in the clinical group

The ABR wave V was noted for all the three stimulus conditions. The waveform recorded from the right ear for one of the subject is shown in the figure 4.3, and the mean and SD values are given in the figure 4.4.

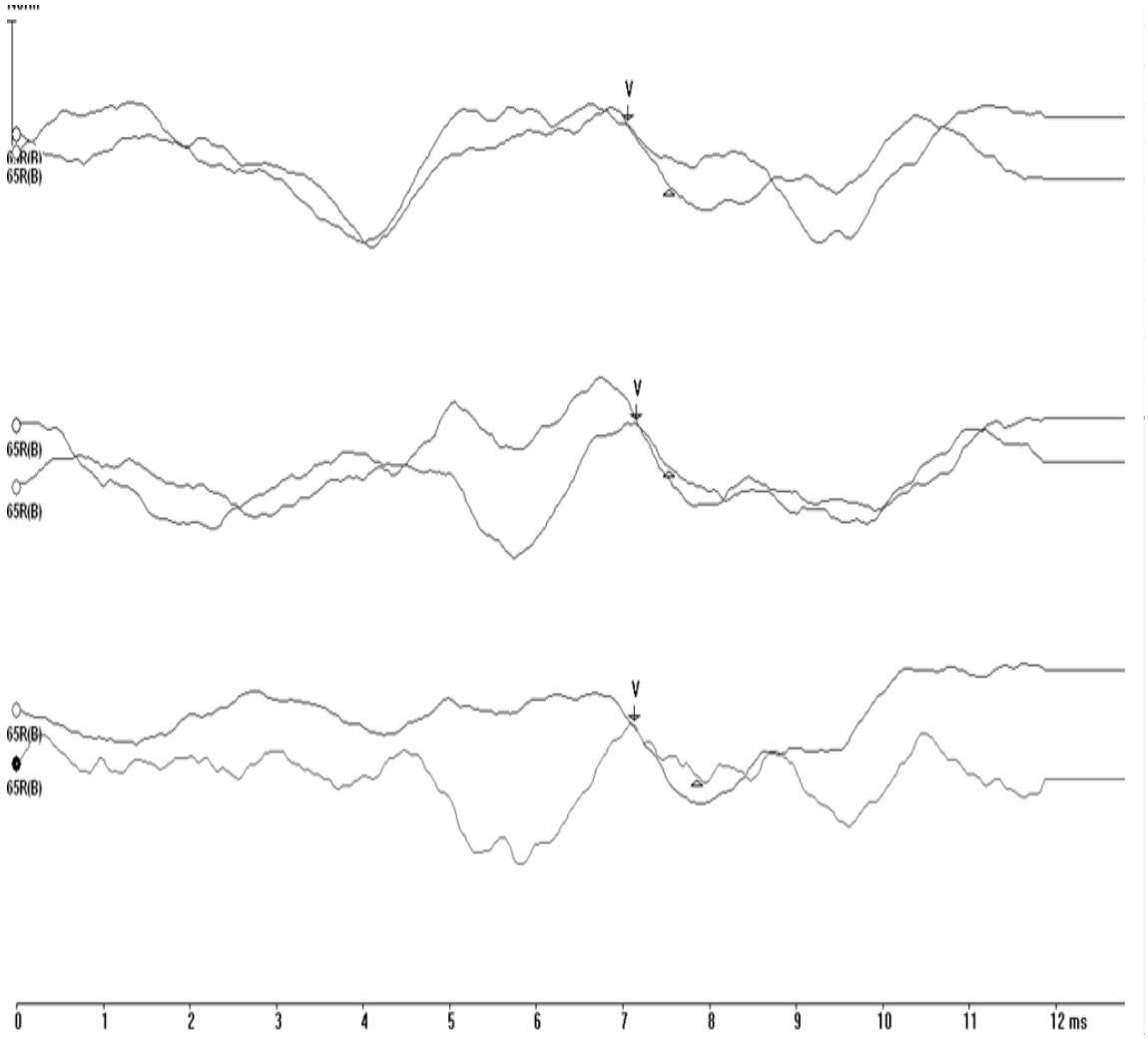

Figure 4.3: ABR waveform recorded from the right ear for all the conditions for one of the subjects in the clinical group.

The first pair of waveforms recorded in the condition with no ipsilateral noise, followed by a pair of waveforms recorded at 0 dB SNR condition, and final pair is at the +3 dB SNR condition.

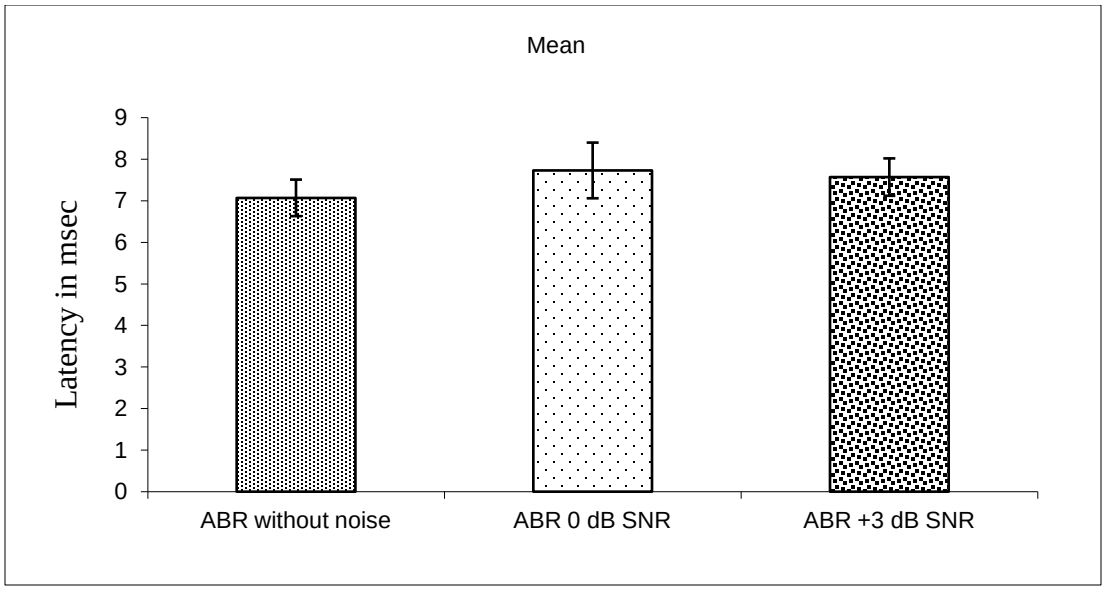

Figure 4.4: Mean and SD for Wave V latency for the clinical group.

The figure 4.4 shows that the wave V latency in the clinical group also shows a longer latency for the condition with 0 dB SNR. In the condition where ABR was recorded without ipsilateral noise, the wave V latency was the least.

The repeated measure ANOVA was administered to see the significant difference in the wave V latency between the three conditions. It revealed that there was a significant difference in the Wave V latency across the three conditions [$F(2, 20) = 12.085$, $p<0.001$]. Bonferroni's post hoc test was done for further pair wise comparison. The results of the test revealed that there was a significant difference in the latency in without noise condition with that of 0 and +3 dB SNR. Whereas within the 0 and + 3 dB SNR condition there was no significant difference.

4.1.3 Auditory Brainstem Response in the control vs. the clinical group

As the sample size was small in the control group non parametric statistical (Man Whitney Test) analysis was done to compare the latencies of Wave V across the two groups. The Z-values and the significance level is given in the table 4.1.

It can be seen in the table that the wave V latency was shorter for control group than the clinical group for all the conditions. However, they have failed to reach a statistically significant level. The amount of Wave V latency shift is same for both the groups in all the three conditions. Maximum shift however occurred at 0 dB SNR.

4.2 Auditory Late Latency Responses

The latency of N1 and P2, and the amplitude of the N1-P2 complex were obtained. The mean and the SD values of all the three parameters are given in the table 4.3.

Table 4.3

Mean and SD values for N1, P2 latency and N1-P2 amplitude of ALLR obtained in both the groups

		Control Group			**Clinical Group**		
	Condition	**Without noise**	**0 dB SNR**	**+ 3 dB SNR**	**Without noise**	**0 dB SNR**	**+ 3 dB SNR**
N1 Latency	*Mean*	153.85	145.71	152.57	191.12	204.43	195.75
	SD	39.50	35.10	28.48	35.51	35.94	29.17
P2 Latency	*Mean*	233.33	236.33	215.66	266.57	279.42	276.85
	SD	42.95	52.72	37.16	43.89	34.92	42.96
N1-P2 Amplitude	*Mean*	7.265	6.00	4.90	5.60	5.48	2.23
	SD	2.38	2.62	6.01	3.23	1.68	3.95

Mixed ANOVA's (condition 3 x groups 2) were also done for each of the ALLR components; N1, P2 latency and N1-P2 amplitude independently; to check for the main effect across the three stimuli conditions, and between the groups. The results of the ANOVA indicated that there was a significant difference in the latencies of both N1 and P2 between the two groups [$F(1, 21) = 13.077$, $p<0.05$] and [$F(1, 18) = 7.723$, $p<0.05$] respectively. There was no significant difference in the latencies of N1 and P2 across the three stimulus conditions [$F(2, 42) = 0.063$, $p>0.05$] and [$F(2, 36) = 0.642$, $p>0.05$] respectively. Also there was no interaction between the groups and the conditions for both N1 and P2 latency [$F(2, 42) = 1.131$, $p>0.05$] and [$F(2, 36) = 0.915$, $p>0.05$] respectively. For N1-P2 amplitude there was no significant difference between the groups

[F (1, 19) = 0.148, p>0.05], or across the three stimulus conditions [F (2, 38) = 0.575, p>0.05]. Along with this interaction effect was also absent between groups and stimulus condition [F (2, 38) = 1.112, p>0.05].

4.2.1 Late Latency Response in the control group

The mean and SD of N1, P2 latency and N1-P2 amplitude was calculated. The waveform recorded from the left ear for one of the subject is shown in figure 4.5. The mean and SD have been depicted in the figure 4.6 and 4.7 respectively.

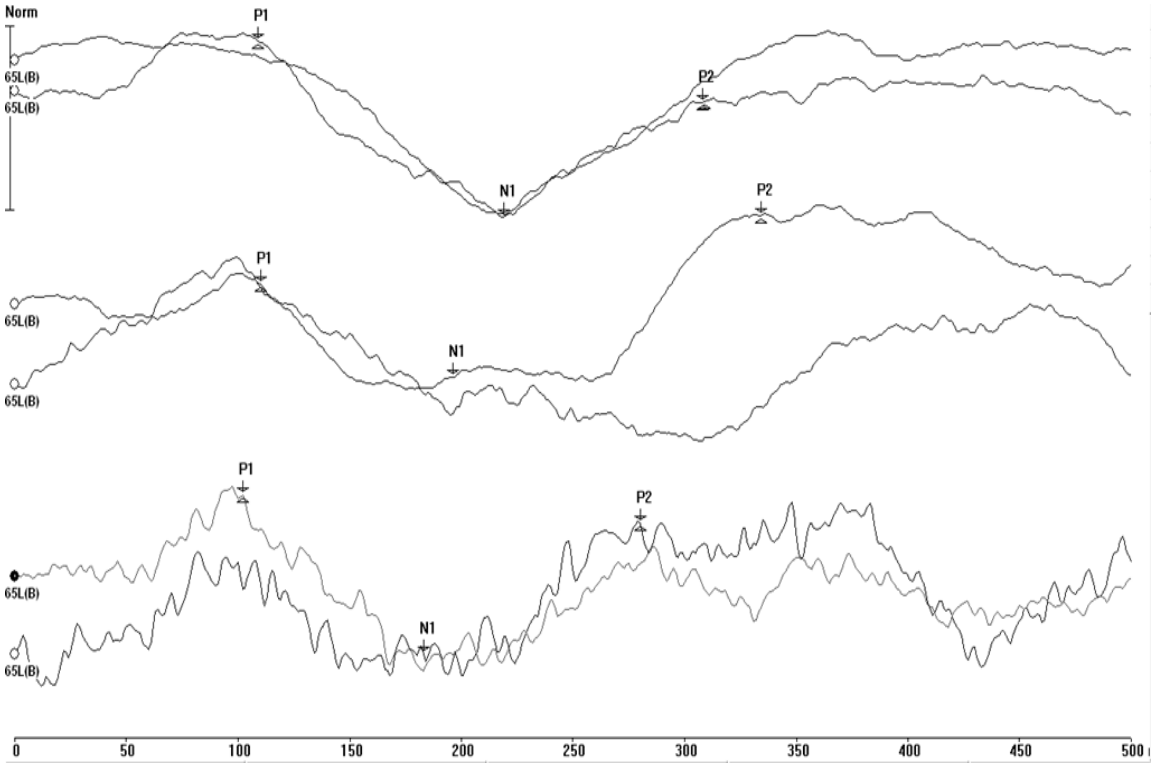

Figure 4.5: ALLR waveform recorded from the left ear for all the conditions for one of the subjects in the control group.

The first pair of waveforms recorded in the condition with no ipsilateral noise, followed by a pair of waveforms recorded at 0 dB SNR condition, and final pair is at the +3 dB SNR condition.

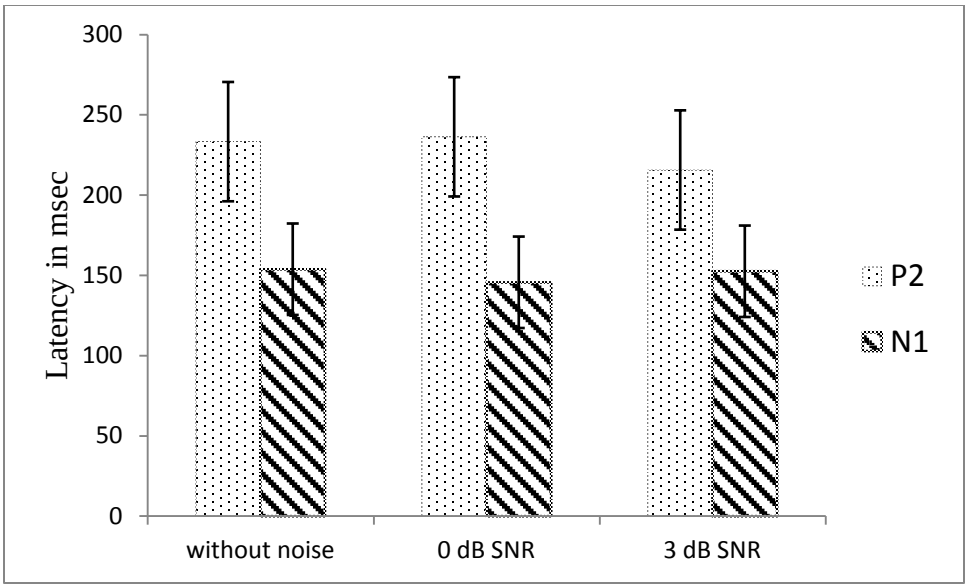

Figure 4.6: Mean and SD values for N1, P2 latency for the control group.

It can be seen from the figure 4.6 that the N1 and P2 latency was almost similar in all the three stimulus conditions. The figure 4.6 shows that the amplitude however was maximum in the condition without any noise and minimum for 3 dB SNR condition.

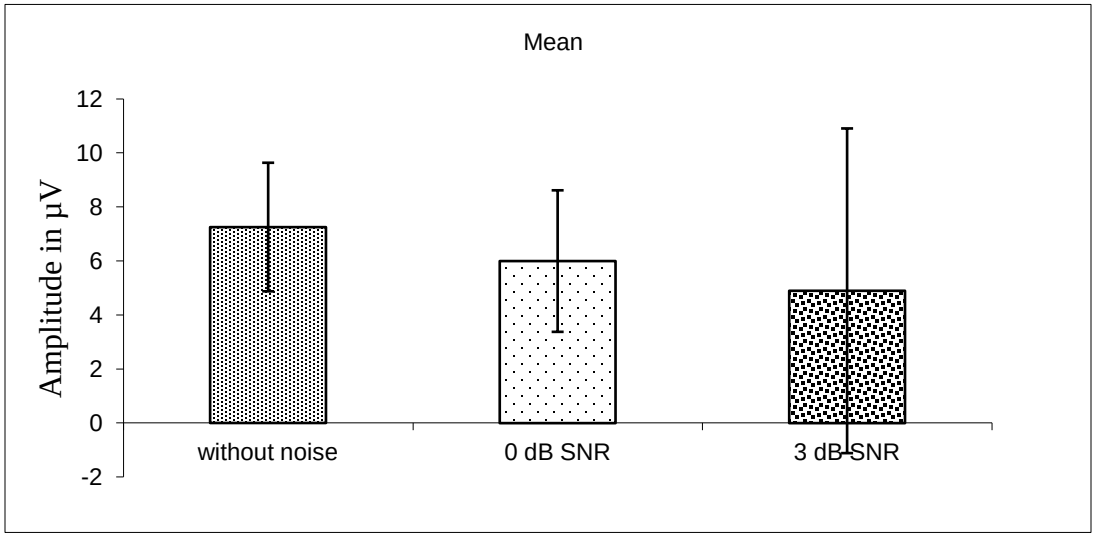

Figure 4.7: Mean and SD values for N1-P2 amplitude for the control group.

Friedman's test was done to check for significance difference, if any, in the latencies of N1, P2 and the N1-P2 amplitude of ALLR across the three different stimulus conditions. The result obtained is given in the table 4.4.

Table 4.4

Chi Square values, degrees of freedom along with significance level for ALLR components across the three stimulus conditions obtained in the control group

Parameter	**Chi Square Value**	**Degree of Freedom**	**Sig. Level**
N1 Latency	2.00	2	0.36
P2 Latency	4.33	2	0.11
N1-P2 Amplitude	4.33	2	0.11

It can be seen from the table 4.4 that there is no statistically significant difference in the N1 and P2 latency across the stimulus conditions. A similar result was also observed for the N1-P2 amplitude across the three conditions.

4.2.2 Late Latency Response in the clinical group

The latency of N1, P2 was noted along with the N1-P2 amplitude. The waveform recorded from the right ear for one of the subject is shown in the figure 4.8. The mean and SD for the latency and amplitude are given in the figure 4.9 and 4.10 respectively.

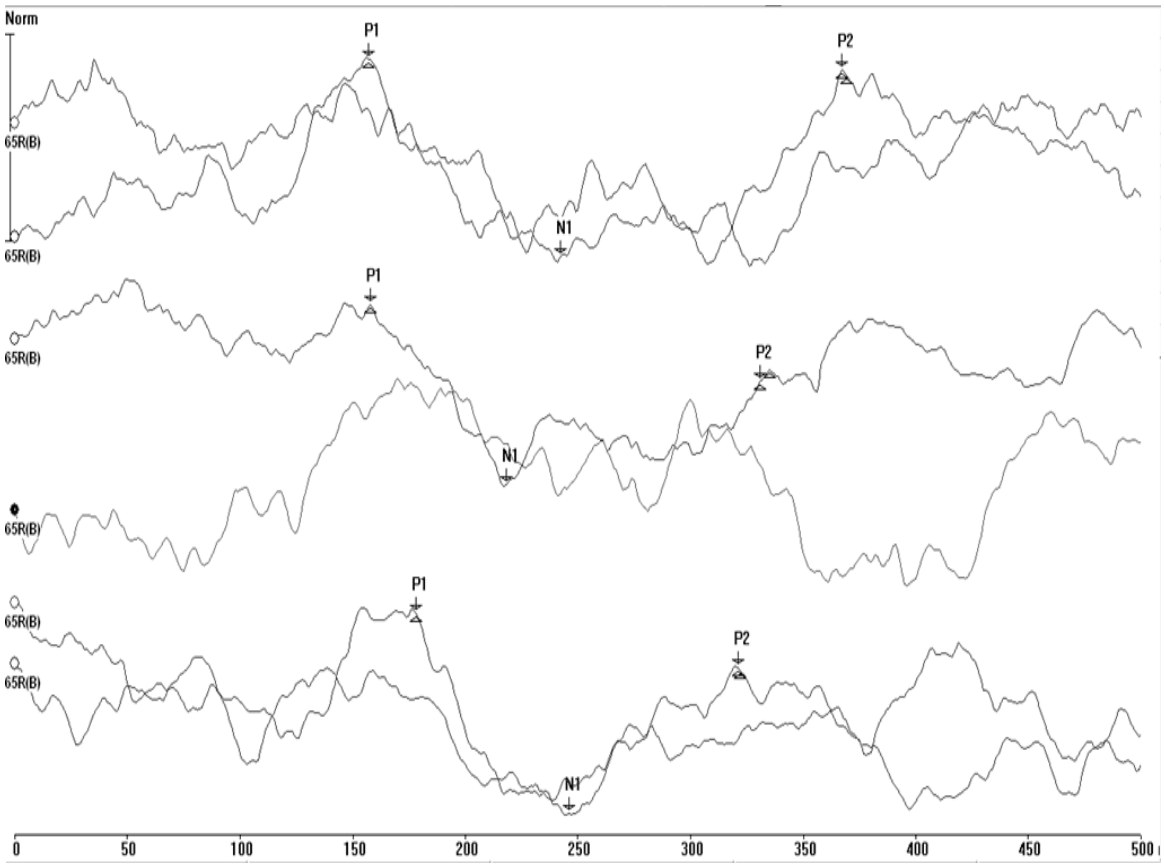

Figure 4.8: ALLR waveform recorded from the right ear for all the conditions for one of the subjects in the clinical group.

The first pair of waveforms recorded at the condition with no ipsilateral noise, followed by a pair of waveforms recorded at 0 dB SNR condition, and final pair is at the +3 dB SNR condition.

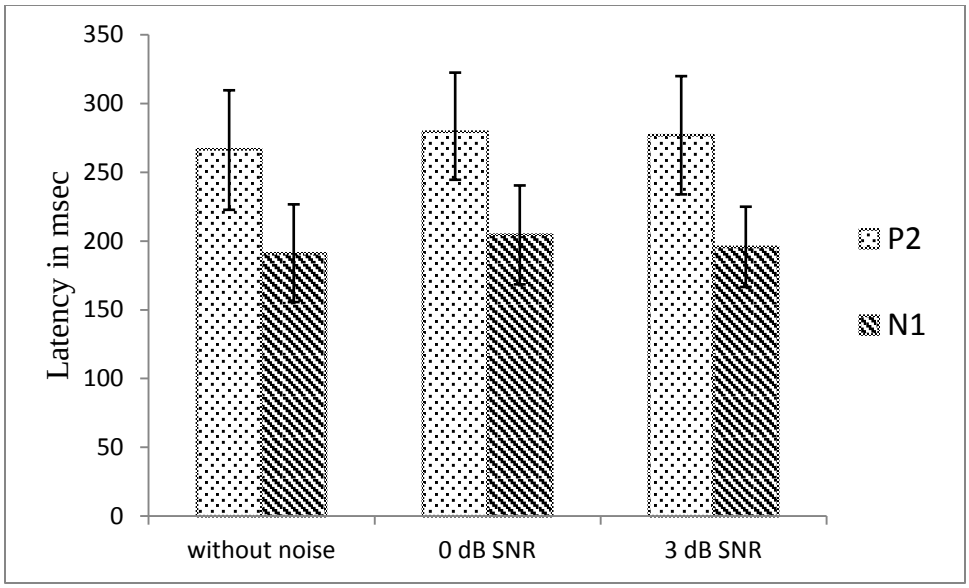

Figure 4.9: Mean and SD values for N1 and P2 latency in the clinical group.

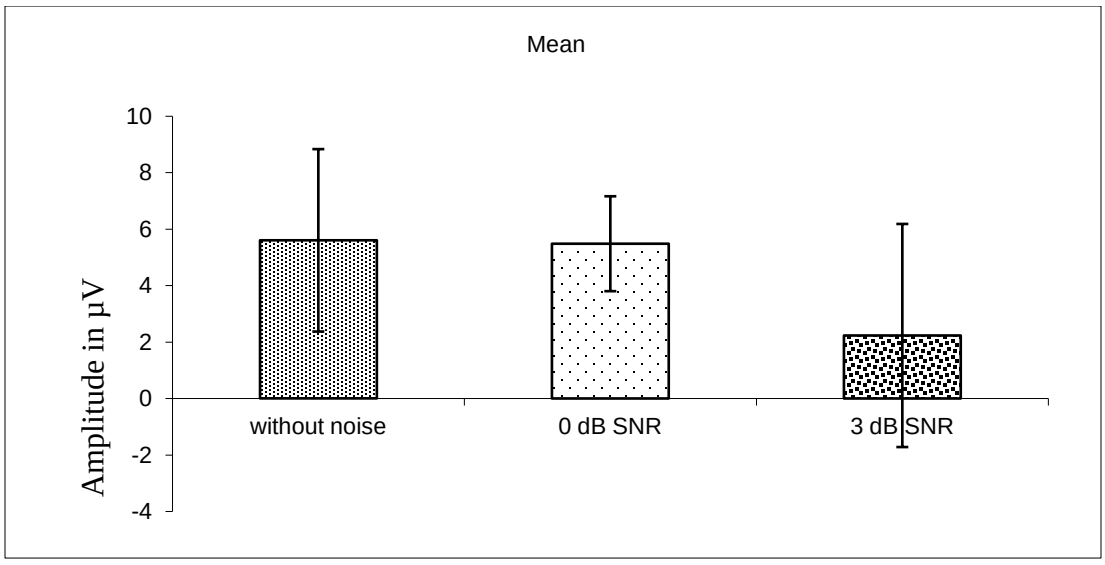

Figure 4.10: Mean and SD values for N1-P2 amplitude in the clinical group.

From the figure 4.9 it can be seen that there is a slight difference in the latency of N1 and P2 across the three conditions. The latency is least in the condition with no noise and is maximum for the 0 dB SNR condition. From the figure 4.10 it can be seen that the

amplitude of the N1-P2 complex was maximum in the absence of noise and least at 3 dB SNR.

The results of the repeated measure ANOVA showed that there is no significant effect in the N1 [F (2, 30 = 1.153, p>0.05], P2 [F (2, 26 = 0.582, p>0.05] latency and N1-P2 [F (2, 28 = 0.135, p>0.05] amplitude across the three stimulus condition. Thus the presence or absence of noise had no significant effect on any one of the ALLR components.

4.2.3 Late Latency Response in the control vs. the clinical group

Man Whitney test was done to compare the ALLR components between the two groups, across the three stimulus conditions. The Z-values and the significance level are given in the table 4.5.

Table 4.5

Z-values and significance level for ALLR components between the clinical and the control group at three stimulus conditions

Parameters		**Without noise**	**0 dB SNR**	**+ 3 dB SNR**
N1 Latency	**Z-value**	2.28	3.02	3.32
	Sig. Level	***0.022***	***0.002***	***0.001***
P2 Latency	**Z-value**	1.56	1.94	3.06
	Sig. Level	0.11	***0.052***	***0.002***
N1-P2 Amplitude	**Z-value**	0.75	1.36	0.05
	Sig. Level	0.45	0.17	0.95

It can be seen from the table that there is a significant difference in the N1 latency between the groups in the three stimulus conditions, whereas for P2 latency there is a significant difference in presence of noise at both 0 and + 3 dB SNR, but not without noise. However, the amplitude of N1-P2 complex did not differ statistically between the groups in anyone of the stimulus conditions.

4.3 Speech Identification Scores (SIS)

SIS scores were obtained in both the groups and the values were noted. The mean and SD values were calculated and are given in the table 4.6.

Table 4.6

Mean and SD values for SIS for the two groups across the three stimulus conditions

Condition	**Group**	**Mean (In %)**	**SD**
SISw	*Normals*	100.00 (N=10)	0.00
	LD	99.50 (N=20)	1.53
SIS0	*Normals*	85.20 (N=10)	6.54
	LD	66.85 (N=20)	15.35
SIS3	*Normals*	95.60 (N=10)	6.38
	LD	82.65 (N=20)	9.88

Note: SISw: SIS without noise; SIS0: SIS at 0 dB SNR; SIS3: SIS at +3 dB SNR

It can be observed from the table 4.6 that the speech identification scores obtained without ipsilateral noise is 100% in the control group. The clinical group had 99.5 % scores in the absence of ipsilateral noise. In the presence of noise the SIS deteriorates for

both the groups. However, SIS obtained in the clinical group was more severely affected than the control group.

Mixed ANOVA (condition3 x groups 2) was done to check for the main effect of the stimulus conditions in the two groups on SIS. According to the mixed ANOVA results there was a significant difference [$F(2, 56) = 63.867$, $p<0.001$] in the SIS scores across the three conditions. Bonferroni's test was carried out further for pair wise comparison between the conditions. The results showed that the SIS obtained in all the stimulus conditions was significantly different from each other.

There was a significant difference in the SIS scores between the two groups [$F(1, 28) = 16.421$, $p<0.001$], where the scores were higher for the control group than the clinical group. An interaction effect was also found between the SIS obtained in three stimulus conditions and, the two groups, which was statistically significant [$F(2, 56) = 9.474$, $p<0.001$].

4.3.1. Correlation of Auditory Brainstem Response to SIS scores

All the subjects in the control group had 100 % speech perception scores without ipsilateral noise, because of which it was not possible to do a statistical test to find the correlation between ABR Wave V and the SIS scores. For the clinical group Spearman's correlation test was done. The results of the test are shown in the table 4.7.

Table 4.7

r-values along with significance level between the ABR and SIS obtained in three stimulus conditions in the clinical group

	r-values	**Significance level**
ABRw - SISw	-0.11 (N=20)	0.62
ABR0 - SIS0	0.10 (N=13)	0.74
ABR3 - SIS3	-0.14 (N=15)	0.61

The table 4.7 shows that there is no particular trend followed by the wave V latency and the SIS scores in different condition. Hence, it can be interpreted from the table that there is no correlation between the ABR Wave V latency and the SIS scores observed across the stimulus conditions in the clinical group.

4.3.2 Correlation of Late Latency Response to SIS scores

Spearman's correlation test was administered to observe the relationship between the ALLR parameters and SIS scores obtained in the three stimulus conditions. The result of which is seen in the table 4.8.

Table 4.8

r-values along with the significance level between the ALLR components and SIS obtained in the clinical group for different conditions

	r-values	**Significance level**
N1 Lat. w – SIS w	.344 (N=19)	.149
N1 Lat. 0 – SIS 0	-.094 (N=18)	.710
N1 Lat. 3 – SIS 3	.086 (N=19)	.727
P2 Lat. w – SIS w	.387 (N=19)	.101
P2 Lat. 0 – SIS 0	-.097 (N=18)	.703
P2 Lat. 3 – SIS 3	.093 (N=16)	.732
N1-P2 amp w – SIS w	-.043 (N=19)	.861
N1-P2 amp 0 – SIS 0	.191 (N=18)	.447
N1-P2 amp 3 – SIS 3	.050 (N=17)	.848

Note: N1 lat w: N1 latency without noise; P2 lat w: P2 latency without noise; N1-P2 amp w: N1-P2 amplitude without noise; N1 lat 0: N1 latency 0 dB SNR; P2 lat 0: P2 latency 0 dB SNR; N1-P2 amp 0: N1-P2 amplitude 0 dB SNR; N1 lat 3: N1 latency 3 dB SNR; P2 lat 3: P2 latency 3 dB SNR; N1-P2 amp 3: N1-P2 amplitude 3 dB SNR.

The results of the spearman's test showed that there was no correlation between the ALLR parameters (N1, P2 latency and the amplitude of N1-P2 complex) with the SIS obtained in any of the three conditions.

From the results the following points can be concluded:

- In the control group, there was a significant difference in the Wave V latency for the conditions with and without noise observed for the ABR. Within the two conditions of noise (0 and +3dB SNR) a difference in latency was there but it failed to reach a significant level. The latency was shortest in the condition with no ipsilateral noise followed by + 3dB SNR condition and the 0 dB SNR condition. For the ALLR parameters there was no significant difference in the N1, P2 latency and N1-P2 amplitude across the three stimulus conditions. No correlation was seen between the wave V latency or the parameters of ALLR and the SIS scores in any of the three conditions.

- In the clinical group also, there was a significant difference in the Wave V latency in the presence and absence of ipsilateral noise observed for the ABR. The condition with no ipsilateral noise was significantly different from those with noise, but, within the two conditions of noise no significant difference was found. They had shortest latency for the condition with no ipsilateral noise and the maximum for the 0 dB SNR condition. In all the ALLR parameters no significant difference was observed in any of the stimulus conditions. Wave V and the SIS scores did not correlate in any of the stimulus conditions, and neither did any of the ALLR parameters showed any correlation.

- No group difference was revealed in the wave V latency for any of the three stimulus condition. The ALLR parameters showed a variable result. The N1

latency was significantly different between the two groups for all the three stimulus conditions. The P2 latency was significantly different between the groups for both the conditions with ipsilateral noise (0 and +3 dB SNR). In the conditions with no ipsilateral noise there was a difference in the latency, but it failed to reach a significant level. The amplitude of the N1-P2 complex did not differ significantly between the groups for any of the three stimulus conditions.

Based on this we can conclude that ABR does not give enough information to differentiate children with normal language skills from those having a learning disability. In the ALLR, N1 and P2 latency are more sensitive in differentiating between the children with a learning problem with those having no learning problem indicating having auditory processing disorder. According to the present study the amplitude of the N1-P2 complex however is not a sensitive parameter of ALLR to identify children with learning disability having auditory processing disorder.

5. Discussion

The main aim of the study was to correlate the SIS scores with the AEPs recorded in different stimulus conditions. Also, a group wise comparison was made between the AEPs recorded from the two groups. The results obtained from both the groups are discussed below.

5.1 Auditory brainstem Responses (ABR)

The wave V latency was prolonged for the control group in the condition with ipsilateral noise. Within the two conditions of noise however there was no significant difference.

Similar results have been quoted by Cunningham et al. (2001), who found that in the presence of background noise the latency of wave V increases for children with no learning problem. Russo et al. (2004) found that in the presence of background noise, brainstem encoding of speech is disrupted. In particular, noise interferes with the onset response. In the majority of normal subjects they evaluated the onset response was severely degraded, while in 40% of subjects it was completely abolished. They concluded that the onset portion of the response is more susceptible to degradation in the presence of noise rather than the sustained portion.

The results obtained in the present study add on to the existing studies which reveal that in the presence of noise the ABR wave V latency is affected. The lack of difference in the wave V latency between the two condition of noise; 0 and + 3 dB SNR; can be attributed to the fact that the noise degrade the onset response hence resulting in almost equal shift in wave V.

The clinical group also had significant difference in the latency in the presence and absence of ipsilateral noise. Within the two noise conditions (0 and + 3 dB SNR) however, no significant difference was observed

Johnson, Nicol, & Kraus, (2005) reported similar findings where they found that the children with learning problem exhibited delayed peak latency of the wave V indicating poor synchrony to transient events. They also report that environmental stresses such as noise and rapidly presented stimuli further negatively influence the neural encoding of linguistic information in children with learning problem.

Wible, Nicol, & Kraus, (2004) report that although most of the children with learning problem had wave V latencies that fell in the normal range it tended to fall in the lower end of the continuum. They conclude that the brainstem processing of speech sound rather than being completely different for children with a learning problem, is to some extent similar in both children with and without a learning problem.

The possible reason for the deficits observed for the clinical group in the current study cannot be attributed to an overall deficit in neural synchrony. The prolongation of the wave V latency seen for the clinical group is comparable, although to a higher degree, to what has been observed for the control group under stressful situations. Hence, it can be said that the deficits which are seen for the clinical group can also be observed in the control group under stressful environmental conditions such as in the presence of background noise. Based on this it can be concluded that the children with learning disability rather than having a deficit in neural synchrony have abnormal representation of specific neural activity (Johnson, Nicol, Zecker, & Kraus, 2007).

Group wise comparison revealed no significance difference in the wave V latency for both the groups for all the conditions although; the clinical group had longer wave V latency in all the conditions. The trend followed by both the groups were similar having shortest latency for the condition without ipsilateral noise and longest for condition with 0 dB SNR. These results are in support of the previous studies done (Johnson, Nicol, & Kraus, 2005; Johnson et al. 2007), which also reported that although the wave V latency is prolonged in children with learning disability as compared to children with no learning problem, the difference is not statistically significant.

The lack of any statistical difference between the two groups can be because of a smaller number of samples collected. Another reason can be the heterogeneity of the LD group. Thus, it can be concluded that ABR for speech stimulus, with or without noise ipsilaterally, may not be efficient to identify abnormal auditory processing in individuals with learning disability.

5.2 Auditory Late Latency Responses (ALLR)

There was no significant difference in the N1 and P2 latency across the three stimulus conditions in the control group.

The results are similar to those stated by Cunningham et al. (2001); Wible, Nicol, & Kraus, 2002; and Wible, Nicol, & Kraus, (2004) who report that in the presence of noise there is no change in the latency of the ALLR peaks in children having no learning problem. However, the SNRs used in the studies were different where the Cunningham et al. (2001) and Wible, Nicol, & Kraus, (2004) used 0 dB SNR, while Wible, Nicol, & Kraus, (2002) used +15 dB SNR.

Contradictory studies have also been reported in the literatures. It has been reported that with the addition of noise there is an increase in the latency of the ALLR components (Whiting, Martin, & Stapells, 1998; & Martin, Kurtzberg, & Stapells, 1999).

It is possible that the noise does not affect the firing of the neurons in the cortex to the extent it effects the firing of neurons at the level of brainstem. Another reason can be the fact that the cortical response requires lesser degree of synchronous firing than the brainstem response, and the presence of background noise does not compromise the synchronous firing to that great an extent (Cunningham et al, 2001).

In the clinical group also, no significant difference was observed in the N1 and P2 latency across the three stimulus conditions. These results are similar to those stated by Cunningham et al. (2001); Wible, Nicol, & Kraus, (2002, 2004) who report that in the presence of noise there is no change in the latency of the ALLR peaks in children with learning problem. The reason for the insignificant difference in the latencies could be the same as that mentioned for the control group.

Comparison between the two groups revealed that there was a significant difference in the N1 latency for all the three stimulus conditions, and P2 latency for the condition with 0 and +3 dB SNR noises. The clinical group had prolonged latency of N1 and P2 for all the conditions.

The results of the present study are in contradiction to those done by Cunningham et al, in 2001, who reported no difference in the latency of any of the ALLR components for the two groups in the presence and absence of noise.

The reason for the differences in the findings of the present study with that of the previous authors can be because of the fact that the learning disabled group is a heterogeneous one (Cunningham et al, 2001; Wible, Nicol, & Kraus, (2002, 2004). Some of them show results which are similar to those observed in children with no learning problem whereas, the others show a significance deviance. It is possible that the children taken for the present study were those who fall under the second category thereby varying the latency significantly.

The amplitude of the N1-P2 complex in the control group was different across the three stimulus conditions, but failed to reach a significant level.

Similar results were showed by Cunningham et al. (2001) and Wible, Nicol, & Kraus, (2004) who found that there is a reduction in the amplitude at 0dB SNR compared to no noise condition for children having no learning problem. However, Wible, Nicol, & Kraus, (2002) found a significant reduction in the amplitude at +15dB SNR compared to no noise condition for children having no learning problem.

In the present study no significant difference in the amplitude of N1-P2 complex was found although there was a reduction in the response amplitude in the conditions with ipsilateral noise. The lack of significance can be because of a smaller sample size. Another reason can be the way in which the amplitude was measured. The above reported studies all measure the RMS amplitude of the cortical response, whereas in the present study the peak-to-trough amplitude of the N1-P2 complex was taken.

In the clinical group there was a reduction in the amplitude of the N1-P2 complex in the conditions with ipsilateral noise (0 and +3dB SNR). However, it failed to reach a significant level.

Cunningham et al. (2001); and Wible, Nicol, & Kraus, (2002, 2004) have also shown similar results for children with a learning problem. They found a reduction in the amplitude of the N1-P2 complex in condition with ipsilateral noise when compared to no noise condition.

The possible reason for the reduction in the amplitude of the N1-P2 complex can be attributed to asynchronous firing of the neurons responsible for the generation of cortical responses in stressful conditions such as presence of background noise (Wible, Nicol, & Kraus, (2004).

The amplitude of the N1-P2 complex showed no significant difference between the two groups across either of the three stimulus conditions. This is in consonance with the previous findings of (Wible, Nicol, & Kraus, (2004). They found that the introduction of background noise had a similar effect of reduction in the response amplitude for children with and without a learning problem.

It can be suggested that in the children with a learning problem, the poor cortical representation of speech sounds in the presence of noise cannot be attributed to an abnormal decrease in overall response activity. Rather, it is possible that the activity associated with the neural encoding of speech sounds is being distributed differently over time across the responses recorded in noise in the children with learning problem (Wible, Nicol, & Kraus, (2004).

It can be concluded from the above discussion that the N1, P2 latency of ALLR can be used to identify auditory processing disorder in children with learning disability. However, amplitude is not a sensitive parameter for both with and without ipsilateral noise to identify an auditory processing disorder.

5.3 Speech Identification Scores (SIS)

The control group had 100 % scores in quiet. The control group had hearing sensitivity within normal limits and did not have any other abnormality, which resulted in good SIS in quiet.

The presence of noise reduced the SIS scores for the control groups. Within the two conditions of noise, 0 and + 3 dB SNR, the scores were more severely degraded in the 0 dB SNR condition. This supports the literature that in adverse listening situations even children with normal language skills perform poorly (Mills, 1975 and Elliot, 1979). Bradlow, Kraus, & Hayes, (2003) stated that as the listening condition becomes more adverse (from -4 to -8 dB SNR) the speech perception deteriorates even further.

The reason for reduction in SIS scores in 0dB condition than +3 dB can be because the poor SNR affects the speech processing to a greater extent than a higher SNR.

In the condition with no ipsilateral noise the clinical group also had higher SIS scores than in the conditions with noise. The scores were significantly poorer in the condition with ipsilateral noise (0 and +3 dB SNR). This is consistent with the previous studies which report that children with learning disability have poorer speech perception abilities Chermak, Vonhof, & Bendel, (1989).

The poorer performance for the clinical group in the conditions with ipsilateral noise as compared to no noise condition can be because of a similar phenomenon as that seen for the control group. That is, a poorer SNR affects speech intelligibility more than a higher SNR.

On comparing the performance between the two groups it was found that the control group had significantly higher scores in all the conditions as compared to the clinical group. In the presence of noise the SIS scores reduced significantly more for the clinical group. Within the two conditions of noise, 0 and + 3 dB SNR, the scores were more severely degraded in the 0 dB SNR condition for both the groups. Similar results have been quoted by Mills, 1975 and Bradlow, Kraus, & Hayes, (2003).

There is considerable literature reporting that children with learning disability perform poorer than the children with no learning problem in the presence of background noise (Brady, Shankweiler, & Mann, 1983; Chermak, Vonhof, & Bendel, 1989; Stollman, Kapteyn, & Sleeswijk, 1994; Bradlow, Kraus, & Hayes, 2003) and the findings of the present study are analogous to them.

Cunningham et al. (2001) and Johnson, Nicol, & Kraus, (2005) have shown that in quiet there in no significant difference in the speech perception of children with and without a learning disability. The reason they report is that the quiet condition is an ideal listening situation which does not strain the auditory system. Hence, it is not possible to detect the subtle auditory deficits present in the children with learning disability in quiet conditions.

5.4 Correlation between the AEPs and Speech Identification Scores (SIS)

In order to categorize the cause of the learning disability an attempt was made to correlate the AEPs measured in the present study with the SIS scores obtained in each condition. No correlation was found between the ABR wave V latency and the SIS scores in one any of the condition for either of the groups.

The control group also showed no correlation between the ABR wave V latency and the SIS scores in the three conditions. There is dearth of information regarding the correlation between the SIS scores with that of wave V latency.

The reason could be that the wave V is not very sensitive to the differences in the processing of speech sounds in the control and the clinical group. Another reason can be that the synthetic speech stimulus does not accurately represent the brainstem processing for speech.

The clinical group showed no correlation between the ALLR response parameters and the SIS scores for the three conditions. Very less information is available to support or contradict the current findings. However, literature is available measuring the JNDs (Cunningham et al, 2001) and its correlation with the cortical responses. They found that children with poorer JND had more reduction in the RMS amplitude of the response as compared to children with better JNDs. It is also possible that the brief duration of the stimulus (40 msec /da/) used here is not sufficient to assess the cortical response adequately.

6. Summary and Conclusion

Many studies have reported the usefulness of using AEPs over behavioral measures to identify (C) APD in children with a learning problem. Overall the results of the studies can be summarized as follows. In the brainstem measure it is the latency of wave V and amplitude of wave V slope which is a sensitive measure for differentiating children with learning problem with those having no learning problem. In the auditory late latency response it is the amplitude of N1-P2 complex which is a sensitive measure.

Among the AEPs, the ABR is the most likely to be normal in children with APD leading to the view that sub cortical structures in the ascending auditory pathway are not involved in APD. Hall and Mueller (1997) recorded the ABR, AMLR and P300 in a series of over 200 children evaluated for APD. Fewer than 10% had abnormal ABR findings, whereas majority of the children had abnormal findings for cortical AEPs. Abnormal ABR consisted of modest delays in inter-peak latencies.

Song et al. (2006) reported that although click ABR in children with auditory processing disorder are typically within clinical norms, they tend to be delayed. It is therefore possible that scalp-measured ABR are not sensitive enough to document this minute effect and that deficits are therefore observed only in response to more complex stimuli. The speech-evoked ABR may be conceptualized as the neural code of speech syllable (Johnson, Nicol & Kraus, 2005).

One of the well recognized challenges encountered by the children with (C) APD is to comprehend speech in the presence of background noise and competing speech signals (Jerger & Musiek, 2000). Wible, Nicol, & Kraus, (2004) found smaller amplitude

of the P2 and N2 wave complex recorded for children with auditory learning problems when speech signal /ga/ was presented at 0 dB SNR in comparison to ALR amplitude for a control group under the same conditions.

Thus, in the present study an attempt was made to correlate the SIS scores with the AEPs recorded in three different stimulus conditions between two groups of children. It also focused on the differences in the parameters of the AEPs recorded between the two groups for the different stimulus conditions.

In order to accomplish these objectives two groups of subjects were taken, with ten subjects in each group. The control group consisted of children with no learning problem based on a screening checklist developed by WHO (1999, cited in Singhi, et al. 2007). The clinical group included children who were diagnosed as having learning disability based on Early Reading Skills (Loomba, 1995) administered by a speech language and pathologist. The children in both the groups underwent routine audiological testing constituting of pure-tone, speech audiometry and immitance testing. All the children had normal hearing sensitivity and middle ear function.

ABR and ALLR were recorded from all the subjects in three different stimulus conditions; absence of ipsilateral noise, 0 and +3 dB SNR. SIS scores were also found for both the groups in all the three stimulus conditions.

The mean and SD for the SIS scores, ABR wave V, ALLR N1 and P2 latency, and amplitude of N1-P2 complex was found for all the three conditions from both the groups. Mixed ANOVAs were done to find the differences in the AEPs parameters for the three stimulus conditions for both the groups separately. For the control group

Friedman's test was done to find the difference in parameters of the AEPs in the three conditions. Wilcoxon's post hoc test was also administered where ever needed. For the clinical group repeated measure ANOVA was done to find the differences in AEP parameters between the three conditions. The post hoc test administered was Bonferroni's test to observe the difference in AEP parameters between any two conditions. To compare the AEP parameters between the groups Man Whitney test was done. Finally Spearman's correlation test was done to find the correlation between the SIS scores and the AEP parameters for all the three conditions for both the groups separately.

The results obtained are as follows:

- There was a significant difference in the latency of wave V in the condition with no ipsilateral noise when compared to the conditions with ipsilateral noise (0 and +3dB SNR), with the latency in 0 dB SNR condition being maximum. Within the two conditions of noise (0 and +3dB SNR) no significant difference was found. These results held true for both the control and the clinical group.
- Comparison of the wave V latency showed delayed wave V for the clinical group in all the conditions when compared with the control group. This difference however, failed to reach a significant level.
- The latency of N1 and P2 did not differ significantly between the three stimulus conditions for both the control and the clinical group.
- Group wise comparison showed that N1 latency was significantly different in all the three conditions between the two groups. The P2 latency differed significantly only in the two condition of noise (0 and +3dB SNR) between the two groups.

- The amplitude of N1-P2 complex for both the groups was reduced in the conditions with ipsilateral noise (0 and +3dB SNR) when compared with the condition of no ipsilateral noise. However, it has failed to reach a significant level.
- The amplitude of the N1-P2 complex did not differ significantly between the two groups for any of the conditions.
- The SIS scores were different significantly in the three conditions for both the groups.
- Group wise comparison also revealed significant difference between the scores for all the three conditions.
- No correlation was observed in the SIS scores and the ABR or ALLR parameters for both the groups.

The differences observed in the latency of wave V in the three stimulus conditions can be attributed to the fact that in the presence of noise the neural synchrony is affected which delays the brainstem response. This delay is longer for the clinical group as compared to the control group, although the pattern for the delay is similar in both the groups. From this it can be concluded that group differences observed for the wave V latency are not because of a deficit in brainstem response generators, rather it is because of deficit in neural synchrony. Moreover, the deficits observed for the clinical group are similar to those observed in the control group under stressful environmental conditions.

The lack of difference in the latency of N1 and P2 for the two groups individually for the three stimulus conditions can be attributed to the fact that ALLR is a cortical response and requires lesser degree of synchronous firing to generate a response. The presence of ipsilateral noise does not affect the timing of the neural firing to such as extent so as to degrade the response latency. It is possible that even in the clinical group the synchronous firing is not majorly affected to affect the latency of N1 and P2. Group wise difference in the latency can be attributed to the fact that the learning disabled group is a heterogeneous group. In one subset of children the results for the ALLR is similar to that obtained for children with no learning problem, even when the stimulus conditions are less than ideal. The other subset consists of children with learning problem who show deficits in the ALLR parameters when compared to children with no learning problem, even for ideal listening situations. It can be said that the presence of noise affect the cortical response in one group of children with learning disability and it is possible that the cause for their learning problem is auditory based deficits. On the other hand, in children with learning disability in whom the addition of noise did not degrade the cortical response can have reasons other than auditory deficits for their learning problems.

The reduction in amplitude of the N1-P2 complex in the conditions with ipsilateral noise (0 and +3dB SNR) when compared to condition with no noise for both the groups can be attributed to the fact that in the presence of competing signal the timing of response firing is affected such that there is a reduction in the amplitude of the cortical

response being measured. Lack of group difference may be because the noise affects the firing timing equally for both the groups.

The SIS scores were worst in the 0 dB SNR condition than in other conditions for both the groups. This can be because it is the most stressful condition putting maximum strain on the auditory system. Also, the poorer scores for the clinical group in comparison to the control group in all the conditions show that speech perception is poorer in children with learning problem. Also, adverse listening conditions have more detrimental effect on the clinical group than the control group.

Conclusion

It can be concluded form the present study that AEPs are sensitive measure to differentiate between children with a learning problem from those without a learning problem, especially in conditions with background noise. Although, the ABR wave V latency is not a sensitive measure, the latency of N1 and P2 of ALLR are sensitive measures. Hence, the N1 and P2 latencies are useful in identifying auditory processing deficits in children with learning disability. These parameters are sensitive to auditory processing disorders in both conditions with and without background noise. Also, when testing in adverse listening situations both 0 and +3 dB SNR are equally sensitive in identifying an auditory processing disorder. The results of the study also suggest that there is need not be a one-to-one relation between the AEP findings and SIS at different SNRs.

Implication of the Study

1) The current study would help to find out appropriate stimulus conditions for AEPs to assess auditory processing deficits.
2) A comparison between ABR and ALLR can help us in knowing which of the two is more sensitive in identifying processing disorders in learning disabled population.
3) AEPs in quite vs. noise may be a sensitive objective test for identifying auditory processing deficits in learning disabled population, as the subjective test results could be affected by attention.
4) AEPs may help in differentiating between different subgroups of learning disability.
5) AEPs can help in early identification of learning disability in a child.

7. References

Alain, C., Woods, D. L., & Covarrubias, D. (1997). Activation of duration sensitive auditory cortical fields in human. *Electroencephalography & Clinical Neurophysiology, 104*, 531-539.

Alexander, J., & Polich, J. (1997). Handedness & P300 from auditory stimuli. *Brain & Cognition, 35*, 259-270.

Allison, T., Wood, C. C., & Goff, W. R. (1983). Brain stem auditory, pattern-reversal visual, and short-latency somatosensory evoked potentials: Latencies in relation to age, sex and brain and body size. *Electroencephalography & Clinical Neurophysiology, 55*, 619-636.

Amenedo, E., & Diaz, F. (1998). Effects of aging on middle latency auditory evoked potentials: A cross sectional study. *Biological Psychiatry, 43*, 210-219.

American National Standards Institute (1991). *Maximum permissible ambient noise levels for audiometric test rooms (ANSI S3.1-1991).* New York: Acoustical Society of America.

American National Standards Institute. (1996). *Specifications for Audiometers. (ANSI S3.6-1996).* New York: ANSI.

Banai K., Nicol T., Zecker S. G., & Kraus N. (2005) Brainstem timing: implications for cortical processing and literacy. *Journal of Neuroscience;, 25,* 9850–9857.

Barnet, A. B., Ohlrich, E. S., Weiss, I. P., & Shanks, B. (1975). Auditory evoked potential during sleep in normal children, from ten days to three years of age. *Electro-encephalography & clinical neurophysiology, 39*, 29-41.

Beagley, H. A., & Knight, J. J. (1967). Changes in auditory evoked response with intensity. *Journal of laryngology & otology*, *81,* 861-873.

Beattie, R. C., & Boyd, R. (1984). Effects of click duration on the latency of the early evoked response. *Journal of Speech and Hearing Research, 27,* 70-76.

Bradlow, A. R., Kraus, N., & Hayes, E. (2003). Speaking clearly for children with learning disabilities: sentence perception in noise. *Journal of Speech, Language and Hearing Research, 46*, 80–97.

Brady, S., Shankweiler, D., & Mann, V. (1983). Speech perception and memory coding in relation to reading ability. *Journal of Experimental Child Psychology, 35*(2), 345–367.

Bruneau, N., Roux, S., Guerin, P., Barthelemy, C., & Lelord, G. (1997). Temporal prominence of auditory evoked potentials (N1 wave) in 4 to 8 year old children. *Psychophysiology, 34,* 32-38.

Cacace, A. T., Shy, M., & Satya-Murthi, S. (1980). Brainstem auditory evoked potentials: A comparison of two high frequency filter settings. *Neurology, 30,* 765-767.

Callaway, E. (1975). *Brain electrical potentials and individual psychological differences.* Crune's and stratten. New York.

Cambell, K. B., Bell, J., & Bastein, C. (1992). *Evoked potential measures of information processing during natural sleep*. In R.J. Broughton & R.D. Ogilvie,. (Eds). Sleep, arousal and performance: A tribute to Bob Wilkinson. (Pp.88-110).

Carhart, R., & Jerger, J. F. (1959). Preferred method for clinical determination of pure-tone thresholds. *Journal of Speech and Hearing Disorders, 24,* 330-345.

Ceponiene, R., Alku, P., Westerfield, M., Torki, M., & Townsend, J. (2005). ERPs differentiate syllable and non phonetic sound processing in children and adults. *Psychophysiology, 59,* 256-278.

Ceponiene, R., Shestakova, A., Balan, P., Alku, P., Yaguchi, K. & Naatanen, R. (2001). Children's auditory event related potentials index stimulus complexity and "speechness". *International journal of Neuroscience, 109,* 245-260.

Chermak, G. D., Vonhof, M. R., & Bendel, R. B. (1989). Word identification performance in the presence of competing speech and noise in learning disabled adults. *Ear & Hearing, 10(2), 90-3.*

Clements, S. (1966). *Minimal brain dysfunction of in children: Terminology and Identification.* Phase one of the Three phase Project, Washington, D.C.: US Department of the Health, Education and Welfare.

Coats, A. C. & Martin, J. L. (1977). Human auditory nerve action potentials and brainstem evoked responses: Effect of audiogram shape and lesion location. *Archives of otolaryngology, 103,* 605-622.

Cody, D. T. R., Jacobson, J. L., Walker, J. C., & Bickford, R. G. (1964). Averaged evoked myogenic and cortical potentials to sound in man. *Annals of Otology Rhinology and Laryngology, 73,* 763-777.

Colrain, I.M., Diparsia, P., & Gora, J. (2000). The impact of pre-stimulus EEG frequency on auditory evoked potentials during sleep. *Journal of experimental psychology, 54,* 243- 254.

Cranford, J. L., Rothernel, A. K., Walker, L., Stuart, A., & Elargovan, S. (2004). Effect of discrimination task difficulty on N1 and P2 components of late auditory evoked potential. *Journal of American Academy of Audiology, 15,* 456-461.

Cunningham, J., Nicol, T., 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.

Davis, H., & Zerlin, S. (1966). Acoustic relations of the human vertex potential. *Journal of the Acoustical Society of America*, *39,* 109-116.

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.

Don, M., Allen, A. R., & Starr, A. (1977). Effect of click rate on latency of the auditory brainstem responses in humans. *Annals of Otology, Rhinology & Laryngology, 86,* 186-195.

Don, M., Ponton, C., Eggermont, J., & Masuda, A. (1994). Auditory brainstem response (ABR) peak amplitude variability reflects individual differences in cochlear response times. *Journal of the Acoustical Society of America*, *96,* 3476-3491.

Elliot, L. L. (1979). Performance of children aged 9 to 17 on a test of speech intelligibility in noise using sentence material with controlled word predictability. *Journal of the Acoustical Society of America*, *66*, 651–653.

Emerson, R. G., Brooks, E. B., Parker, S. W., & Chiappa, K. H. (1982). Effect of click polarity on brainstem auditory evoked potentials in normal subjects and patients: Unexpected sensitivity of wave V. *Annals of the New York Academy of Science, 388,* 710-721.

Fisher, C., Morlet, D., & Giard, M. (2000). Mismatch negativity and N100 in comatose patients. *Audiology and Neuro-otology, 5,* 192-197.

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

Grimes, C. T., & Fiedman, A. (1971). Evoked response thresholds for long and short duration tones. *Audiology*, *10*, 358-363.

Hall, W. J. (2007). *New Handbook for Auditory Evoked Responses*. Allyn and Bacon publisher, Florida.

Hall, J. W., & Mueller, H. G. (1997). *Audiologists' desk reference: Diagnostic audiology principles and procedures.* San Diego, CA: Singular.

Halliday, A. M., & Mason, A. A. (1964). The effect of hypnotic anesthesia on cortical responses. *Journals of neurology, Neuro Surgery and Psychiatry, 27*, 300-312.

Hecox, K., & Galambos, R. (1974). Brainstem auditory evoked responses in human infants and adults. *Archives of otolaryngology*, *99*, 433-416.

Hymel, M. R., Cranford, J. L., & Stuart, A. (1998). Effects of contra lateral speech competition on auditory event related potentials recorded from elderly listeners: Brain map study. *Journal of American Academy of Audiology, 9*, 30-33.

Jerger, J., & Hall, J. W., III. (1980). Effects of age and sex on auditory brainstem response (ABR). *Archives of otolaryngology*, *106*, 387-391.

Jerger, J. & Jerger, S. (1970). Evoked response to intensity and Frequency change. *Archives of otolaryngology, 91*, 433-416.

Jerger, S., Martin, R. C., Jerger, J. (1987). Specific auditory perceptual dysfunction in a learning disabled child. *Ear and Hearing*, *8*, 78–86.

Jerger, J., & Musiek, F. (2000). Report of the Consensus Conference in the diagnosis of auditory processing disorders in school-aged children. *Journal of American Academy of Audiology, 11,* 467-474.

Jewett, D. L., & Williston, J. S. (1971). Auditory evoked far fields averaged from the scalp of humans. *Brain, 4,* 681-696.

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

Johnson, K. T., Nicol, T. G., Zecker, S. G., & Kraus, N. (2007). Auditory Brainstem Correlates of Perceptual Timing Deficits. *Journal of Cognitive Neuroscience, 19(3),* 376–385.

Johnson, K. L., Nicol, T. G., Zecker, S. G., Wright, B. A., & Kraus, N. (2004). Brainstem timing in learning impaired children with excessive auditory backward masking. *Association for Research in Otolaryngology, 27,* 118.

Krishnan, A. (2002) Human frequency-following responses: representation of steady-state synthetic vowels. *Hearing Research, 166,* 192–201.

Lader, M. (1977). Effects of psychotropic drugs on, auditory evoked potentials in men. Auditory correlates of evoked potentials. In. Desmedt, J.E. (Ed.). Basel, Switzerland. Karger.

Loomba, M. (1995). Descriptive analysis of the sequential progression of early reading skills among Indian children. *Unpublished Master's dissertation.* University of Mysore.

Martin, B. A., Kurtzberg, D., & Stapells, D. R. (1999). The effects of decreased audibility produced by high-pass noise masking on N1 and the mismatch negativity to speech sounds /ba/ and /da/. *Journal of Speech Language and Hearing Research, 42,* 271–286.

Mayadevi. (1978). Development and standardization of a common speech discrimination test for Indians. Unpublished master's dissertation. *University of Mysore.*

McAnally, K. I. & Stein, J. F. (1997). Scalp potentials evoked by amplitude modulated tones in dyslexia. *Journal of Speech Language and Hearing Research, 40,* 939-945.

McPherson, D. L., & Starr, A. (1993). *Auditory evoked potentials in the clinic. Evoked potentials in clinical testing.* In Halliday, A.M. (Ed.), Edinburgh: Chruchil livingstone.

McPherson, D. L., Tures, C., & Starr, A. (1989). Binaural interaction of the auditory brain stem potentials and middle latency auditory evoked potentials in infants and adults. *Electroencephalography and Clinical Neurophysiology, 74,* 124-130.

Mendel, M. J., Hasick, E. C., Davis, W. J., Hirsh, S. K., & Dinges, D. F. (1975). Audiometric comparison of middle and late components of the adult auditory evoked potentials during awake and asleep. *Electroencephalography and Clinical Neurophysiology, 38,* 27-33.

Mills, J. H. (1975). Noise and children: a review of literature. *Journal of the Acoustical Society of America, 58*, 767–779.

Murata, K., Araki, S., Tanigawa, T., & Uchida, E. (1992). Acute effects of alcohol on cognitive and central nervous system assessed by auditory event related potentials. *Nippon Eiseigaka Zasshi, 47,* 958-964.

National Joint Committee on Learning Disabilities. (1990). *Learning disabilities: Issues on definition. Collective perspectives on issues affecting learning disabilities:* Position papers and statements. Austin, TX: Pro-Ed.

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

Oku, T., & Hasegewa, M. (1997). The influence of aging on auditory brainstem response and electrocochleography in the elderly. *Journal for Otorhinolaryngology & its Related Specialties, 59*, 141-146.

Olsen, O. W. (1998). Average Speech Levels and Spectra in Various Speaking/Listening Conditions: A Summary of the Pearson, Bennett, & Fidell (1977) Report. *American Journal of Audiology, 7*, 21-25.

Onishi, S., & Davis, H. (1968). Effects of duration an rise time of tone bursts on evoked V potentials. *Journal of the Acoustical Society of America, 44*, 582-591.

Picton, T. W., & Hillyard, S. A. (1974). Human, Auditory evoked potentials: II effects of attention. *Electroencephalography and clinical neurophysiology, 36*, 191-199.

Picton, T. W., Hillyard, S. A., Kravsz, H. I., & Galambos, R. (1974). Human auditory evoked potential: I Evaluation of the components. *Electroencephalography and Clinical Neurophysiology, 36,* 129-190.

Picton, T. W., Woods, D. L., Baribear, B.J., & Healey, T. M. G. (1977). Evoked potential audiometry. *The Journal of Otolaryngology, 6*, 90-119.

Picton, T. W., Wood, D. L, Stuss, D., & Compbell, K. (1978). *Methodology and meaning of human evoked potential: Scalp distribution studies. Multidisciplinary perspective is event related brain potential research.* In D.A. Otto (ed.),. Washington, DC: US. Environmental Potential Agency.

Ponton, C. W., Eggermont, J. J., Don, M., Waring, M. D., Kwong, B., Cunnighan, J., & Trutwein, D. (2000). Maturation of the mismatch negativity effects of profound deafness and cochlear implant use. *Audiology and Neuro otology, 5,* 167-185.

Porjeez, B., & Begliter, H. (1981). Human evoked brain potentials and Alcoholism. *Clinical and Experimental Research, 5,* 304-317.

Purdy, S. C., Kelly, A. S., & Davies, M. G. (2002). Auditory brainstem response, middle latency response, and late cortical evoked potentials in children with learning disabilities. *Journal of American Academy of Audiology, 13(7),* 367-82.

Putter-Katz, H., Kishon-Rabin, L., Sachartov, E., Shabtai, E. L., Sadeh, M., Weiz, R., Gadoth, N., & Pratt, H. (2005). cortical activity of children with dyslexia during natural speech processing: evidence of auditory processing deficiency. *Journal of Basic Clinical Physiology and Pharmacology, 16(2-3),* 157-71

Rapin, I., Schimmel, H., & Cohen, M. M. (1972). Reliability in detecting the auditory evoked response for sleeping subjects. *Electroencephalography and Clinical Neurophysiology, 32,* 521-528.

Rapin, I., Schinnel, H., Tourk, L. M., Krasneger, N. A., & Pollak, C. (1966). Evoked response to clicks and tones of varying intensity in waking adults. *Electro Encephalography and clinical Neurophysiology, 21,* 335-344.

Roth, W. T., Ford, J. M., Lewis, S. J., & Kopell, B. S. (1976). Effects of stimulus probability and task relevance on event related potential. *Psychophysiology, 13,* 311-312.

Rothman, H. H. (1970). Effects of high frequencies and inter subject variability on the auditory evoked cortical response. *Journal of the Acoustical society of America, 47,* 569-573.

Rothman, H. H., Davis, H., & Hay, I. S. (1970). Slow evoked cortical potential and temporal features of stimulation. *Electroencephalography and Clinical Neurophysiology, 47,* 569-573.

Ruhm, H. B. (1971). Directional sensitivity and laterality of electro encephalic responses evoked by acoustic sweep frequencies. *Journal of Auditory Research, 11,* 9-16.

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

Salt, A. N., & Thornton, A. R. D., (1984). The effects of stimulus rise time and polarity on the auditory brainstem responses. *Scandinavian Audiology, 13,* 119-127.

Sanders, R. A., Duncan, P. G., & McCullough, D. W. (1979). Clinical experience with brainstem audiometry performed under general anesthesia. *Journal of Otolaryngology, 8,* 24-31.

Sayers, B., Beagley, H. A., & Henshall, W. R. (1974). Mechanism of auditory evoked response. *Nature, 247,* 481-483.

Sharma, A., Kraus, N., McGee, T. & Nicol, T. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant vowel syllables. *Electroencephalography and Clinical Neurophysiology, 104,* 540-545.

Shucard, J. L., Shucard, D. W. & Cummins, K. R. (1981). Auditory evoked potentials and sex related differences in brain development. *Brain and language, 13(1),* 91-102.

Singhi, P., Kumar, M., Malhi, P., & Kumar, R. (2007). Utility of the WHO Ten Questions Screen for Disability Detection in a Rural Community—the North Indian Experience. *Journal of Tropical Pediatrics, 53 (6),* 383-387.

Skinner, D., & Shinota, J. (1975). A comparison of the effects of sedatives on the auditory evoked cortical response. *Journal of the American Audiology Society, 1*, 71-78.

Song, J. H., Banai, K., & Kraus, N. (2008): Brainstem Timing Deficits in Children with Learning Impairment May Result from Corticofugal Origins. *Audiology Neuro-otology, 13,* 335-344.

Song, J. H., Banai, K., Russo, N. M., & Kraus, N. (2006). On the relationship between speech- and non-speech evoked auditory brainstem responses. *Audiology Neuro-otology, 11*, 233–241.,

Spink, U., Johannsen, H. S., & Pirsig, W. (1979). Acoustically evoked potential perpendance upon age. *Scandinavian Audiology, 8,* 11-14.

Starr, A., Amlie, R. N., Martin, W. H., & Sanders, S. (1977). Development of auditory function in newborn infants revealed by auditory brainstem potentials. *Pediatrics, 60,* 831-839.

Stockard, J. E., Stockard, J. J., Westmoreland, B. F., & Corfits, J. L. (1979). Brainstem auditory evoked responses: Normal variation as a function of stimulus and subjective characteristics. *Archives in Neurology, 36,* 823-831.

Stollman, M., Kapteyn, T., & Sleeswijk, B. (1994). Effect of time-scale modification of speech on the speech recognition threshold in noise for hearing-impaired and language-impaired children. *Scandinavian Audiology, 23*, 39–46.

Suzuki, T., & Horiuchi, K. (1981). Rise time pure-tone stimuli in brainstem response audiometry. *Audiology, 20*, 101-112.

Suzuki, T., & Taguchi, K. (1968). Cerebral evoked response to auditory stimuli is waking man. *Annals of Otology, Rhinology, Laryngology, 74*, 102-110.

Sugg, M. J., & Polich, J. (1995). P300 from auditory stimuli: Intensity and frequency effects. *Biological Psychology, 41,* 255-269.

Tanguchi, K., Picton, T. W., Orpin, J., & Goodman, J. T. (1969). Evoked response audiometry in new born infants. *Acta Oto Laryngologica (Stockhom suppl), 252,* 5-17.

Teas, D. C. (1965). Analysis of evoked and ongoing electrical activity at the scalp of human subjects. *Journal of Speech and Hearing Research, 8,* 371-387.

Vandana, S. (1998). Speech test in Kannada. *Unpublished master's dissertation.* University of Mysore.

Varghan, H. G., & Ritter, W. (1970). The sources of auditing evoked response recorded from the human scalp. *Electro Encephalography and Clinical Neurophysiology, 28,* 360- 367.

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 and Hearing, 19,* 218–231.

Wible, B., Nicol, T., & Kraus, N. (2005). Correlation between brainstem and cortical auditory processes in normal and language-impaired children. *Brain, 128,* 417-23.

Wible, B., Nicol, T., & Kraus, N. (2002). Abnormal neural encoding of repeated speech stimuli in noise in children with learning problems. *Clinical Neurophysiology, 113 (4),* 459-61.

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.

Witelson, S. F. (1991). Neural sexual Mosaicism: Sexual differentiation of the human temporo potential region for functional asymmetry. *Psychology Neurology and Endocrinology*, *16*, 131-153.

Wolpaw, J. R., & Penry, J. K. (1978). Effects of Ethanol, Caffeine and Placebo on auditory evoked response. *Electroencephalography and Clinical Neuro physiology*, *44*, 568- 574.

Yamamoto, K., Sakabe, N., & Kaiho, I. (1979). Power spectral analysis of auditory evoked response. *Journal of the Acoustical Society of America, 5,* 107-111.