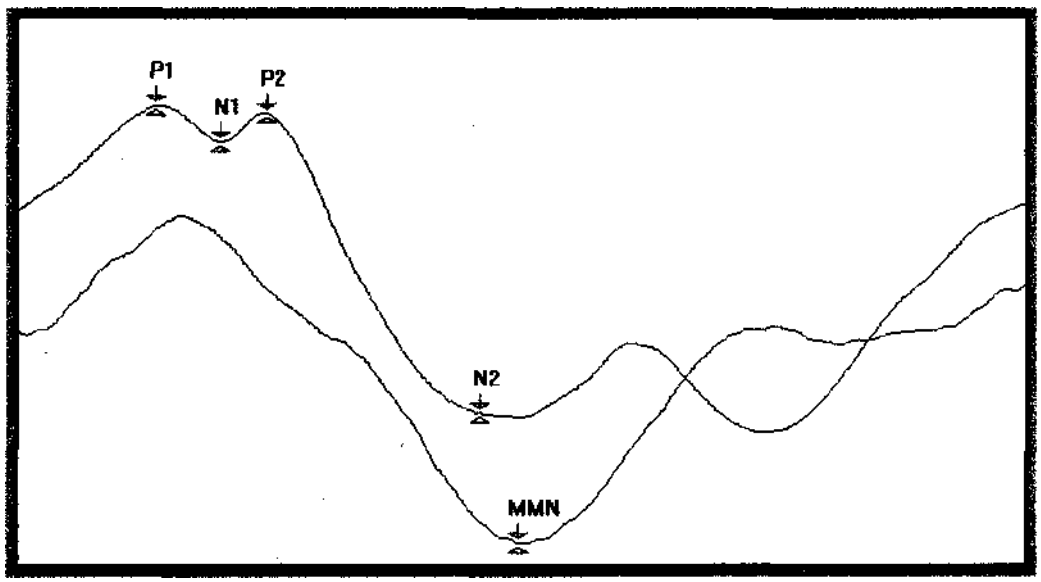
Auditory Long Latency Responses in Children with Learning Disability



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Introduction

Specific Learning disability (SLD) is a disorder in the psychological processes involved in understanding or using language, spoken or written, which may manifest in an imperfect ability to listen, think, speak, read, write, spell or do mathematical calculations. Exclusion from this group is based upon organic deficits including visual, hearing, motor, or economic disadvantage (Public Law-94, 1992). Thus SLD can be termed a syndrome possessing a cluster of symptoms and different deficits can underlie SLD. Prevalence estimates of this disability have been found to range from 3% to 10% (Snowling, 2000). Prevalence rates can vary across languages (Kujala & Naatanen, 2001). Prevalence rate in India varies from to 3% to 10% (Ramaa, 2000).

The causes of SLD are numerous and often poorly defined. The debate on the nature and origin of SLD as well as factors underlying it has been going on for decades resulting however in no clear agreement (Kujala & Naatanen, 2001). There are a wide variety of theories that attempt to account for dyslexia. Snowling (1998) classifies the theories that have received most attention into two general approaches. The first is domain specific view, which posits that the dyslexia arise from deficits in systems that are specifically linguistic. Here the deficits are traced to be present in phonological processing and memory. On the other hand, many claim that deficits in underlying nonlinguistic sensory mechanisms are the core deficits in the disorder. This could involve visual processing and/or auditory processing

The hypothesis that children with specific learning disability have auditory processing disorder has been experimentally investigated by many studies. But, whether these auditory deficits are seen only in association with the language disorder or as a causal factor is yet to be explored (Rosen, 2003). Though a majority of studies in the literature report that a subgroup of children with learning disability have auditory processing deficit, there is no consensus regarding the nature of the auditory processing disorder. Tallal (1980) described a deficit in dyslexics involving processing of brief, rapidly changing auditory stimuli. The findings that dyslexics are mainly impaired in processing stop_consonants which are characterized by brief and rapid spectral changes supports the role of temporal processing in speech perception deficit of dyslexics. Further, investigations revealed that the deficit is not limited to processing of rapidly changing stimuli, but encompasses other aspects of auditory

processing also. There is a controversy as to whether the deficits in auditory processing are limited to speech or it involves processing of both speech and non speech stimuli. Experimental studies carried out on dyslexics to probe the underlying auditory processing disorder reveal two schools of thought. One school of thought supports a dysfunction specific to the phonological system. They evidence this view through studies showing abnormal processing of speech stimuli whereas normal processing of tonal stimuli (Serniclaes, Sprenger, Charolles, Carre & Demonet, 2001). The second school of thought evidences for general auditory processing deficit (Rosen & Manganari, 2001).

Auditory processing of an individual can be assessed through either behavioral tests or electrophysiological tests. Behavioral tests mainly aim at cutting down the external redundancy and assess for the processing of modified auditory stimuli. Each of these tests assess one or more of auditory processes and are sensitive to cortical and/or brainstem lesions of the auditory pathway. On the other hand electrophysiological tests assess for the underlying neurophysiology. Auditory evoked potentials (AEPs) provide powerful objective methods of assessing the neural integrity of pathway from auditory nerve to the cortex (Hood, 1998). Using these techniques, it is possible to follow the course of brain's activity in time with the precision of tens of milliseconds and thus obtain knowledge not only of the end product of processing but also of the sequence, timing and stages of specific processes (Tapio, Leppanen & Lyytinen, 1997).

A majority of the electrcphysiological studies carried out on learning disordered population have used cortical potentials to understand the auditory processing. Prolonged latencies (Byring & Jaryilehto, 1985; Leppanen & Lyytinen, 1997; Guruprasad, 1999; Dawson, Finely, Philips & Lewy, 1989; Jirsa & Clontz, 1990; Arehole, 1995; Radhika, 1997) and reduced absolute amplitudes (Pinkerton, Watson & McClelland, 1989; Jirsa & Clontz, 1990) for P1, N1, P2 and N2 waves have been reported in children with learning disability. Long latency responses give information regarding the basic representation of the sound signal. Neurophyiological correlate of auditory discrimination can be studied using mismatch negativity (MMN), which is a modality specific cortical component of AEPs. It reflects automatic, pre attentive auditory discrimination, represented as a small negative deflection superimposed on N1P2 or P2N2 complex (Lang et al., 1995). Neural activity within MMN portrays the earliest representation stage of auditory input. Compared to other auditory

potentials, MMN appears to represent a correlate of language specific phonetic traces that serve as recognition models for speech sound during auditory perception (Naatanen, 2001).

It has been reported in the literature that MMN to certain speech and non speech contrasts are affected in children with learning disability (Schulte-Korne, Deimel, Bartling, & Remschmidt, 1999; Schulte-Korne, Deimel, Bartling & Remschmidt, 2001; Kraus, McGee, Carrel, Zecker, Nicol & Koch, 1996). MMN has also been used to monitor changes in neural plasticity related to auditory training in normal subjects as well as children with learning disability (Naatanen, Schroger, Karakas, Tervaniemi & Paavilainen, 1993; Kraus, McGee, Carrel, King, Tremblay & Nicol, 1995)

Need and aims of the study

It has been reported in literature that children with learning disability may have auditory processing disorder. Underlying cause of APD may include poor representation of acoustic signals and this can be studied by recording ALLR. A majority of the previous studies on children with LD have used tonal stimuli or only one speech stimuli to record ALLR. There is a need to compare the representation of non speech and speech stimuli as well as compare representation of different speech stimuli in children with LD.

Faulty representation of acoustic signal can lead to poor auditory discrimination and it has been documented that children with LD have poor discrimination. The auditory discrimination ability can be assessed objectively using MMN. A majority of the studies have investigated MMN in children with learning disability for speech stimuli that is deviant in place of articulation and manner of articulation. But there is dearth for studies comparing MMN for speech contrasts varying in place of articulation, manner of articulation, voicing and duration of the stimuli in children with learning disability. Also there is controversy as to whether these deficits in children with learning disability are limited only to speech stimuli or it occurs for both speech and non speech stimuli. Research in this area would throw light on usefulness of MMN in identification of auditory processing disorder in children with learning disability. If MMN can be used in assessment of auditory processing problem, it would help in early identification of children with learning disability or those who are at risk for learning disability. This would in turn enable early rehabilitation of children with

learning disability. As the neural plasticity is more in younger children, the benefits of rehabilitation will definitely be more if rehabilitation is started early in children.

A majority of the studies reported in literature have used synthetic speech in an attempt to carry out a controlled study to assess the fine discrimination ability. However, it is of paramount importance to investigate the perception of natural speech in these children to understand the difficulties faced in real life situations. Hence, the present study used natural speech, as spoken by a native Kannada speaker to elicit MMN. This research will also be helpful in understanding the neurophysiologic basis of speech discrimination in children with learning disability.

Research has also indicated that the effect of auditory learning can be documented using electrophysiological measures. Reduction in latency and increase in amplitude of long latency waves and MMN has been reported in individuals who have shown improvement in auditory processing of speech signal. However, there is dearth of such studies in children speaking Indian languages.

Hence the present study was designed to investigate the following aims:

1. To study the auditory long latency responses to speech and non speech stimuli in children with learning disability.
2. To study MMN for speech and non speech stimuli (tonal) in children with learning disability. Speech discrimination was studied for the following four deviances:
    a. place of articulation
    b. manner of articulation
    c. voicing and
    d. vowel duration

    Deviances used for tonal stimuli included frequency deviation and duration deviation
3. To study the effect of auditory learning on auditory long latency responses and MMN.

Review of literature

In the last few decades, there have been a number of extremely encouraging experimental studies in the area of Specific Learning Disability (SLD). A review of these studies reveals heterogeneity in the characteristics, causes and associated deficits. Though it is not known whether it is the cause or just an associated deficit, results of various investigations have revealed that there is a subgroup of children with learning disability having auditory processing deficit. In a recent review often studies the incidence of auditory processing disorder in children with dyslexics is estimated to be 40% (Ramus, 2003).

Jerger and Musiek (2000) defined auditory processing disorder (APD) as a deficit in the processing of information that is specific to auditory modality. The problem may be exacerbated in unfavorable conditions and may be associated with difficulties in speech understanding, language development and learning. It includes disability in subtle sound difference discrimination that interferes with accurate perception of individual words and leads to confusion of conversation, difficulty in auditory figure-ground (listening in noise) and auditory lags or delays in speech processing (Silver, 1993).

It has been well documented that, at the behavioral level a subgroup of children with dyslexia have primary disturbance in phonological processing (Adlard & Hazan, 1998). The deficit can be in any or all three types of phonological processing skills which include phonological awareness, phonological memory and rate of access for phonological information (Ray, DeMartino, Espesser & Habib, 2002). Studies have shown that children with dyslexia have poor speech discrimination ability that results in phonological processing deficit (Rosen & Manganari, 2001). Manis, et al. (1997) administered phonological awareness and phoneme identification tasks to dyslexic children and compared their performance with that of chronological as well as reading level matched controls. Results showed less sharply defined categorical perception of voice onset time difference in children with Dyslexia. Also, their performance was as good as that of reading level age matched children but significantly poorer than that of chronological age matched children. In all the children, phonological awareness was directly related to the phonemic identification performance. This was supported by Been and Zwartz (2003) who in their study found a

difference in phonemic perceptual boundaries of children at risk for dyslexia. Underlying neurophysiology was postulated to be less neuronal surface available and thus lowered amount of neurotransmission. On the other hand, Goswami, Thomson, Richardson, Stainthorp, Hughes, Rosen and Scott (2002) attributed the core difficulty to the deficit in accurate specification and neural representation of speech. They observed significant differences between dyslexic and normally reading children, in amplitude envelope onset detection. They propose that a likely perceptual cause of this difficulty is a deficit in the perceptual experience of rhythmic timing.

The auditory temporal deficit hypothesis suggests that at least a subgroup of children with reading disorder have a deficit in low level auditory temporal processing that affects the perception of short transitional acoustic elements that provide important acoustic cues for phonemic contrasts (Tallal, Miller & Fitch, 1993). Results of an investigation by Rey, De Martino, Espesser and Habib (2002) support the general temporal deficit theory of dyslexia. They investigated the impact of the temporal alteration and the impact of complex syllabic structure on consonant order judgments. Thirteen phonological dyslexic children and ten control subjects matched for chronological age were compared on a temporal order judgment task using the succession of two consonants (/p/ & /s/) within a cluster. In order to test the possible relevance of the temporal deficit hypothesis, the task also included two additional conditions where either the two stimuli were artificially slowed or two phonological structures were opposed (CCV and CVCV). It was observed that the temporal order judgment performance was significantly poorer in dyslexics than in controls. Moreover, in the "slowed speech" condition performance of dyslexics improved to reach that of the normal subjects, whereas manipulating the phonological structure complexity provided no significant improvement. Finally, performance of dyslexics especially on the slowed condition correlated with several tests of phonological processing.

Electrophysiological studies have been used to understand the neurophysiology of auditory processing in these children. Electrophysiological tests are also helpful in early identification of auditory processing problems. In this section a brief summary of the electrophysiological investigations on children with learning disability is presented.

Auditory Evoked Potentials in individuals with Learning Disability

Auditory evoked potentials such as auditory brainstem response, middle latency response and long latency responses can be used to study the basic representation of sound signals in the auditory nervous system. Neurophysiological representation of speech discrimination can be studied using mismatch negativity.

Early and Middle Latency Responses

Reports of early and middle latency responses in children with learning disability are equivocal. Normal auditory brainstem responses are obtained for click stimuli in a majority of the investigations (Tait, Roush & Johns, 1982). However a few investigators have reported abnormal auditory brainstem responses in children with learning disability. Abnormalities observed were absent waves (Greenblatt, Bar & Zappulla, 1983) and delayed waves (Sohmer & Student, 1978). Binaural auditory brainstem responses are reported to be more useful than monaural responses in identification of auditory processing disorders in these children as abnormal binaural interaction component is observed in these children (Gopal & Kowalski, 1999; Mason & Mellor, 1984). In a recent investigation, King, Warrier, Hayes and Kraus (2002) recorded early responses for transition of/da/ syllable and observed that responses to speech transition were abnormal in some of the children with learning disability, even though responses to clicks were normal. More studies on speech evoked ABR are warranted to confirm these findings.

Archole, Augustine and Simhadri (1995) recorded auditory middle latency responses to click stimuli from children with learning disability. Results showed prolonged latencies of P_a compared to normal especially for contralateral recording. On the other hand, Mason and Mellor (1984) reported normal latency for P_o, P_a and Pt,. Decrease in absolute amplitude has also been reported in children with learning disability (Kraus et al., 1985).

Auditory Long Latency Responses (ALLR)

A majority of the electrophysiological studies done on learning disordered population have used ALLR to understand the auditory processing. Initial investigators compared the latency and amplitude of the peaks in children with learning disability to those of age

matched controls for responses elicited using clicks or tone bursts. Results from a majority of studies revealed increased latencies (Satterfield, Schell, Backs & Hidaka, 1984; Byring & Jaryilehto, 1985; Leppamann & Lytinen, 1997; Guruprasad, 1999; Dawson, Finely, Philips & Lewy, 1989; Jirsa & Clontz 1990; Arehole, 1995; Radhika, 1997) and reduced absolute amplitudes (Pinkerton, Watson, & McClelland, 1989; Jirsa & Clontz, 1990; Leppamenn & Lyytinen, 1994) for P1, N1, P2 and N2 waves in this population. But refuting this, normal (Radhika, 1997) and decreased latencies (Mason & Mellor, 1984) have also been observed. Similarly Lincoln, Courehensne, Harms and Allen (1995) reported increased amplitude and Jirsa (1992) reported normal absolute amplitudes in children with learning disability. Purdy, Kelly and Davies (2002) reported earlier $P_{(}$ in children with learning disability.

There is dearth for studies investigating speech evoked ALLR in children with learning disability. Cunningham, Nicol, Zecker and Kraus (2000) evaluated the maturational progression of speech-evoked P1/N1/N2 cortical responses over the life span. They attempted to determine whether responses are distinctive in clinical populations experiencing learning problems and elucidate the functional significance of these responses. The results revealed that the maturational patterns in the group of children with learning problems did not differ from the normal group. However, P1/N1/N2 parameters were significantly correlated with standardized tests of spelling, auditory processing and listening comprehension in children with learning problem group.

Testing in noise may be more sensitive in identifying subtle abnormalities which may not be discovered during routine evaluation. Wible, Nicol and Kraus (2002) reported that cortical responses for speech stimuli (/da/ of 40 ms duration) were affected in presence of noise in a subgroup of children with learning problems and this performance correlated with behavioral measures of auditory processing and spelling. However ALLR responses in quiet were normal in these children. Thus a review of literature suggests that abnormal, asynchronous, auditory cortical encoding may underlie some language-based learning problems in a subgroup of children with learning problems. However these results does not indicate whether the cortical responses to all the stimuli are equally affected.

Mismatch Negativity (MMN)

In the recent past, MMN has become an emerging tool in studying the auditory processing in children with specific learning disability. MMN, originally described in 1975 by Naatanen and colleagues (Naatanen, Gaillard & Mantysala, 1978) is elicited by infrequent changes in a sequence of a repetitive auditory stimulus (Winkler, Tervaniemi & Naatanen, 1997). This negative potential is usually seen as an increased negativity in the latency region following the peak of N1 and during P2, usually peaking 100 to 300 ms following stimulus onset. It may be seen as an enlarged N1, a second negative peak or an attenuation of the P2 wave (Picton, 1990). The MMN reflects central code of stimulus change; its amplitude and latency are related to the degree to which deviant stimuli differ from the standard stimuli, not the absolute levels of the deviant/standard stimuli (Stapells, 2002). It can be elicited by frequency, intensity, duration, spatial or phonemic changes (Kraus, McGee, Micco, Sharma, Carrell & Nicol, 1993a). Generally, the larger the acoustic differences, the earlier and larger is the MMN although there may be ceiling effects in amplitude with large differences (Picton, Alain, Otten, Ritter & Archim, 2000). MMN can be recorded from newborns (Alho, Sainio, Sajaniemi, Reinikainen, & Naatanen, 1990) and pre-term newborns (Cheour-Luhtanen et al., 1996). Hence, it can help in the early identification of children who are at risk of APD.

The results of behavioral studies have demonstrated that children with specific learning disability often demonstrate difficulty discriminating rapid acoustic changes that occur in speech. Attempts have been made to investigate if there is a neurophysiologic deficit which can explain this behavioral deficit. Kraus, McGee, Carrell, Zecker, Nicol, and Koch (1996) compared the performance of normal children and children with learning problems in behavioral discrimination task (/ba/ Vs /wa/ and /da/ Vs /ga/) as well as MMN. Results showed that the children with learning problems had deficit in discrimination of /da/ and /ga/ but showed intact performance *in* discriminating /ba/ and /wa/. MMN recorded from these subjects correlated with the behavioral findings. MMN elicited with /da/ - /ga/ pair had diminished magnitude and prolonged latency whereas /ba/ - /wa/ pair elicited normal MMN. It was concluded that, these children have deficit in the preattentive processing in auditory pathway which leads to deficit in conscious perception of cues important for place of articulation. Further, Kraus et al. (1996) concluded that the perception of all spectro-

temporal changes might not be impaired to the same extent. This may be because, two different contrasts may tap into separate and distinct neural mechanisms or they may be processed at distinct locations along the auditory pathway. Similar findings were reported by Marie Cheour, Leppanen and Kraus (2000).

Maurer, Bucher, Brem and Brandeis (2003) investigated differences in frequency and phoneme mismatch negativity between kindergartners with and without risk for familial dyslexia. The results indicated that the mismatch response of children at risk was attenuated to frequency deviance and less left lateralised to phoneme deviance. Schulte-Korne, Deimel, Bartling and Remschmidt (1998) examined MMN for tone and speech stimuli in dyslexic and normal children. While there were no group differences for the tone stimuli, MMN was significantly attenuated in the dyslexic group for the speech stimuli. This finding lead to the conclusion that dyslexics have a specific speech processing deficit at the sensory level which could be used to identify children at risk at an early age. However, further investigations revealed that the deficit is not specific to speech stimuli. Schulte-Korne, Deimel, Bartling, and Remschmidt (1999) recorded MMN for a complex tonal pattern, where the difference between standard and deviant stimuli was the temporal, not the frequency structure. Dyslexics had a significantly smaller MMN in the time window of 225-600 ms. These results indicate that dyslexics have a significant pre-attentive deficit in processing of rapid temporal patterns suggesting that it may be the temporal information embedded in speech sounds, rather than phonetic information per se, that resulted in the attenuated MMN found in dyslexics in previous studies. In support of this, Schulte-Korne, Deimel, Bartling and Remschmidt (2001) found similar deficits in adult dyslexics. They found that the late component of the MMN elicited by passive speech perception was attenuated in dyslexic adults in comparison to a control group. But there was no group difference in MMN elicited for tonal stimuli.

Leppanen, Richardson, Pihko, Eklund, Guttorm, Aro and Lyytinen (2002) measured event-related brain responses to consonant duration changes embedded in pseudowords applying an oddball paradigm in 6-month-old infants with and without high risk of familial dyslexia. Pseudoword tokens with varying /t/ duration were presented with an interval of 610 msec between the stimuli. The results revealed that infants at risk due to a familial

background of reading problems process auditory temporal cues of speech sounds differently from infants without such risk, even before they learn to speak.

Bradlow et al. (1999) investigated behavioral discrimination of/da/ Vs /ga/ and its neurophysiologic correlate. It was observed that varying the formant transition duration from 40 ms to 80 ms did not result in improved behavioral response but there was enhancement of MMN response. These results suggest that, the presence of MMN does not indicate that the stimuli can be discriminated behaviorally. However, it is difficult to have normal behavioral discrimination in subjects with absent MMNs.

Thus a review of literature shows that MMN may be affected in children with learning disability. However there is dearth of information on MMN to natural speech stimuli and comparison of MMN to deviant stimuli that vary in terms of place of articulation, manner of articulation, voicing and durational cues. Also the results of the studies comparing MMN for speech and non speech stimuli have yielded equivocal results.

Electrophysiological tests in monitoring improvement in auditory skills

There is growing evidence in literature that children with APD improve with auditory training programs. Both behavioral and electrophysiological tests have been used to document the improvement in auditory skills. Tremblay, Kraus, McGee, Ponton and Otis (2001) studied the effect of auditory training on N 1 P2 complex on ten normal hearing young children in response to two synthetic speech variants of the syllable /ba/. Results showed that, as perception improved, N1P2 amplitude increased. These changes in waveform morphology are thought to reflect increases in neural synchrony as well as strengthened neural connections associated with improved speech perception. These findings suggest that the N1 P2 complex may have clinical applications as an objective physiologic correlate of speech sound representation associated with speech sound training.

Bischof, Gratzka, Strehlow, Haffner, Parzer and Resell (2002) investigated the effect of auditory discrimination training on reading and orthography performance in children with dyslexia. The training was done for discrimination of tonal and speech stimuli. In the results, they observed a significant difference between the pre and post training auditory

discrimination performance. They also observed a significant correlation between auditory discrimination, reading and orthography performance.

There is large amount of evidence that the MMN can serve as an index of learning-associated neural plasticity. Naatanen, Schroger, Karakas, Trevaniemi and Paavilainen (1993) showed that the MMN gradually emerged in those subjects who learned to discriminate complex spectrotemporal sound patterns, but not in those who did not learn to discriminate them. Kraus, McGee, Carrel, King, Tremblay and Nicol (1995) reported that behavioral training in the discrimination of speech stimuli resulted in a significant change in the duration and magnitude of MMN. Tremblay, Kraus and McGee (1998) investigated MMN in subjects learning discrimination of new speech contrasts and found that MMN emerged even before the subject was able to discriminate the contrasts. Since the MMN reflects neural plastic changes and behavioral studies have reported that the discrimination ability of learning disabled children improves with training, it holds promise in evaluating the efficacy of training in children with learning disability. Kujala and Naatanen (2001) reported that in dyslexic children, the change in the reading skill measures with training correlated with change in MMN amplitude.

Thus, it can be concluded that training improves auditory abilities of children with learning disability. Limited number of investigations have also revealed that the improvement with training can be monitored using LLR and MMN.

Method

Participants

Two groups of subjects were included in the study. Experimental group included fifteen children diagnosed as having learning disability by an experienced Speech and Language Pathologist / Clinical Psychologist. Children were in the age range of seven to twelve years and had no history of otological or neurological pathologies. Control group had thirty age matched children with a good scholastic performance. All the children had normal hearing sensitivity and normal middle ear functioning.

Instrumentation

The present study was conducted using the following instruments:

1. A two channel OB-922 diagnostic audiometer was used to carry out the pure tone and speech audiometry.
2. A calibrated GSI-33 immittance meter was used to examine the middle ear functioning.
3. Intelligent Hearing Systems (version 2. IX) was used to record and analyze the auditory cortical evoked potentials.
4. An audio CD Deck was used to play the material developed for auditory training.

Material for testing and training

Electrophysiological recording was done using both speech and tonal stimuli. Six syllables, /tfa/, /dza/, /sa/, /da/, /da/ and /da_s/ (/da/ with shorter vowel duration), spoken by a male native speaker were recorded into a computer with audiolab software using a unidirectional microphone kept at six inches distance from the mouth. The Stimuli were then edited to keep the duration constant (250msec) across all the syllabi except /da_s/. /da_s/ stimulus was of 175 msec duration. Ten normal hearing adults rated the quality of the stimuli and the stimuli were re-recorded if the quality was not rated as good. Stimuli were then loaded into Intelligent Hearing Systems (IHS) using Stimconv software. Tonal stimuli were generated using an inbuilt stimulus generator of IHS. Stimuli were 250 msec 1000 Hz, 250 msec 1100 Hz and 175 msec 1000 Hz puretones.

The material for auditory learning was prepared using "sound generator" software. Tones of different frequency, intensity and duration were generated. They were synthesized using audiolab software to make tonal pairs. They were then sequenced from pairs with largest difference to pairs with least difference. The material was then copied onto two compact discs.

Test procedure

Subjects were seated in a comfortable position to ensure a relaxed posture and minimum muscular artifacts. They were instructed not to pay attention to the stimuli. A silent cartoon movie was played to minimize the possibility of active attention. Three recordings of MMN were carried out using binaural stimulation. Among the three recordings, two used speech stimuli and one used tonal stimuli. Each recording consisted of presentation of one standard stimulus and two deviant stimuli. Thus, MMN was recorded for a total of six stimulus contrasts. MMN was differentially recorded from F_z, TL and TR with lower forehead as the ground and nose tip as the reference electrode sites. TL was located halfway between T3 and T5. TR was located half way between T4 and *T(*, electrode sites of 10-20 electrode system. The data was acquired after ensuring that the impedance at all the electrode sites were within permissible limits. The protocol used for recording is described in Table 1. Order of the recording was random across the subjects to avoid the order effect.

Table 1: Stimuli and recording parameters for eliciting MMN

Parameter	
Stimulus type	Recording 1 Standard :/tja/, Deviant 1: /dza/, Deviant 2: /sa/ Recording 2 Standard :/da/, Deviant 1: /$\underset{i}{d}$a/, Deviant 2: /da$_s$/ Recording 3 Standard: 250 msec, 1000 Hz tone Deviant 1: 250 msec 1100 Hz tone Deviant 2: 175 msec 1000 Hz tone

Intensity	70 dB SPL
Probability ratio	5 : 1
Repetition rate	1.9 / sec
Number of sweeps	100 (Total number of deviant stimuli)
No. of channels	3
Analysis time	500 msec
Gain	75000
Band pass filter	1 Hz - 30 Hz
Transducer	ER3A Insert phones

Data analysis

The responses were analyzed for latency and amplitude of ALLR and MMN. In the ALLR, P1, N1, P2 and N2 peaks were identified through visual inspection of the standard waveform. Peak latency, peak amplitude and relative amplitude of the waves were noted. MMN was determined by subtracting the averaged waveform for standard stimulus from the averaged waveform for deviant stimulus.

Auditory training

Children who obtained poor results in any one of the electrophysiological recordings were advised to attend auditory learning program. Therapy was initiated for five such children. However, only two children completed the training program. Two sessions of forty-five minutes each, were carried out in a week for a period of two months.

Prior to the initiation of program, an assessment was done for each child, to find out the just noticeable difference (JND) for frequency, intensity and duration of puretones. Children were trained to discriminate puretones differing either in frequency, intensity or duration. Training was carried out for all three parameters. In the initial two sessions, children were guided regarding the concept of pitch, loudness, duration and quality of the signal. The training involved discrimination task and started with tonal pairs having a difference above their JND. The difference was gradually reduced depending on the performance of the child. They had to discriminate a tonal pair with 75% accuracy before

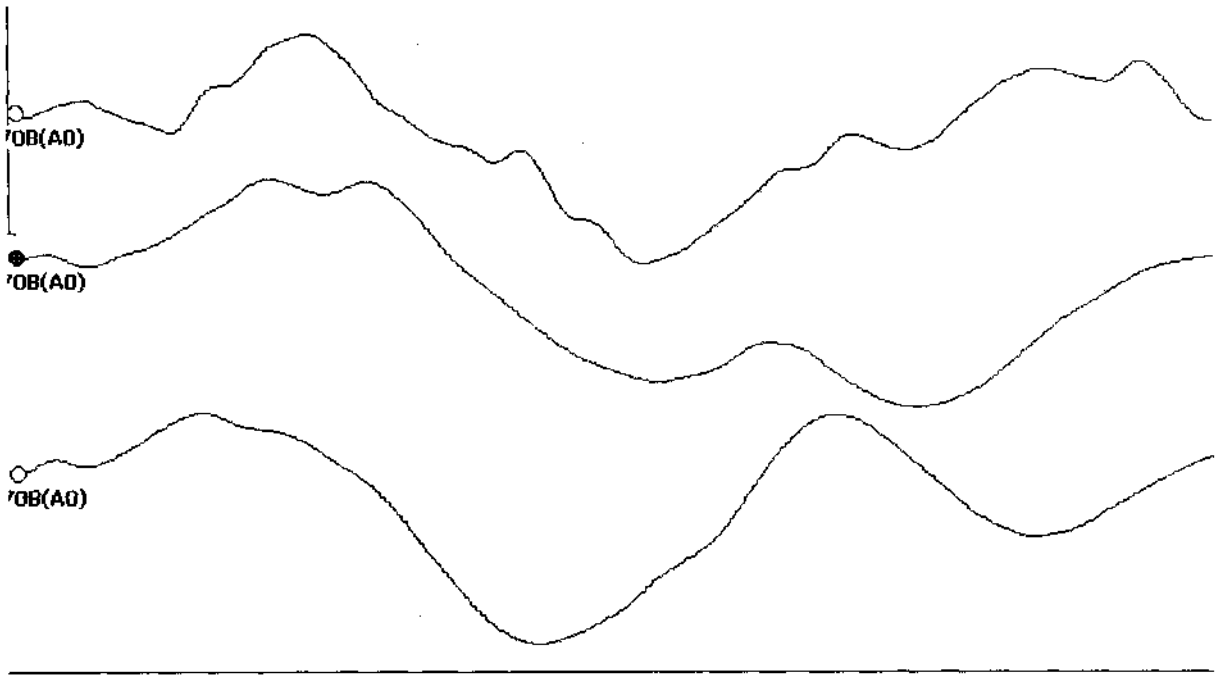
proceeding to the next finer level. The training continued until the children discriminated a difference of 50 Hz, 2 dBHL and 50 msec. The training was done both in quiet as well as in the presence of noise. Electrophysiological tests were repeated after two months to investigate the effect of auditory training on neurophysiological functioning.

Results

The data obtained from the experimental and control group were subjected to statistical analysis using SPSS (Version 10.0). In addition to descriptive statistics, mixed design ANOVA has been carried out to investigate the effect of stimuli, electrode site and group on latency and amplitude of ALLR and MMN. Pre and post therapy assessment could be carried out only for two children. Hence no statistical analysis was carried out and descriptive comparison of the two subjects has been carried out.

Results of ALLR

The grand average ALLR waveforms for normal children is shown in Fig 1. Table 2 shows the mean and standard deviation of latency of ALLR waves, Pi, Ni, P2 and N2, in the control group, for two speech stimuli /tja/, /da/ and for tonal stimuli (1000 Hz). Table 3 shows the results of ALLR in subjects with learning disability. It was observed that in the control group, the latencies of all the waves were comparable for tonal stimuli and /tja/ but /da/ elicited ALLR waves with prolonged latency. No such trend was seen in the experimental group. In the control group, the latency of ALLR waves were shorter at Fz when compared to those of TL and TR for /da/ and tonal stimuli whereas latencies were longer for responses picked up from $F_z$than for those picked up from T_L and TR for /tja/. In the experimental group the latency of the waves were shorter for responses picked up from F_z than those picked up from TL and TR for all the stimuli. Inspection of Table 2 and 3 shows that the mean latencies were longer for children with learning disability when compared to those of normal children for /t[a/ and tonal stimuli but there was not much difference in means obtained for /da/ stimuli. Figure 2 depicts the comparison of ALLR recorded at F_z for the three stimulus contrasts across two groups.



Figl. Grand average of ALLR elicited for syllables /tja/(top), /da/(middle) and tonal stimuli (bottom)

Table 2: Latency (in msec) of ALLR waves in control subjects

		P1		N1		P_2		N_2	
Stimuli	Site	Mean	SD	Mean	SD	Mean	SD	Mean	SD
/tja/	F_z	70.26	15.50	117.77	31.94	153.55	35.20	230.00	33.36
	T_L	60.81	19.53	108.03	28.16	148.42	34.13	215.10	48.55
	TR	61.32	18.41	104.94	28.08	151.58	34.59	218.00	48.14
/da/	F_z	92.43	12.01	132.40	49.45	163.93	31.35	249.00	34.18
	T_L	103.63	20.33	143.87	28.01	193.03	41.75	259.43	40.93
	TR	104.07	23.05	145.87	30.78	197.53	39.35	266.30	46.04
Tone	F_z	73.47	11.28	117.37	23.93	147.50	25.43	216.47	21.81
	T_L	71.20	16.49	117.93	23.84	161.93	29.59	232.27	36.43
	T_R	71.97	17.32	117.23	27.73	161.47	28.83	228.53	38.08

Table 3: Latency (in ms.) of ALLR waves in subjects with learning disorder

		PI		**N1**		P_2		N_2	
	Site	Mean	SD	Mean	SD	Mean	SD	Mean	SD
/tja/	F_z	81	16	134.83	49.45	179.08	55.18	255.92	53.23
	T_L	116.90	39.59	154.55	51.63	207.18	58.24	276.64	57.47
	TR	121.55	40.12	172.18	50.82	215.45	56.74	279.27	61.49
/da/	F_z	87.80	14.32	134.40	24.69	167.07	20.14	235.93	25.42
	T_L	103.00	27.92	149.07	32.79	202.80	42.94	274.07	51.01
	TR	106.00	25.87	154.13	36.68	205.93	49.55	270.53	53.17
Tone	F_z	78.13	14.73	125.00	30.58	170.33	37.88	230.33	50.54
	T_L	87.73	21.84	138.80	35.92	196.67	58.93	260.73	47.13
	TR	97.07	21.30	147.80	34.21	193.00	38.10	257.93	42.61

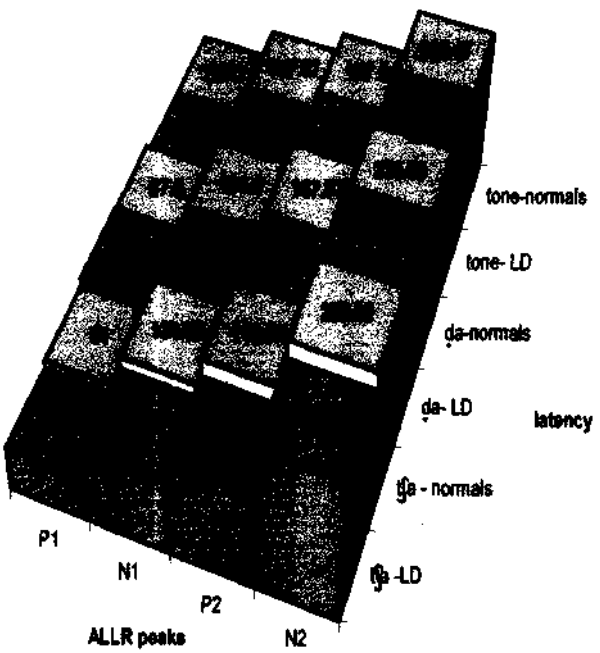


Fig. 2: Comparison of latencies of ALLR waves of LD and normal children

Mixed design AN OVA was carried out with latency of the waves as dependent variable, site of electrode and stimuli as within subject independent variable and group as between subject independent variable. Mauchly's test revealed that the assumption of sphericity was violated. Hence the F ratio was corrected using Greenhouse-Giesser estimate. The corrected F ratios are given in Table 4. It can be observed from the table that the stimuli, electrode site and group had a main effect on latency of all the waves. It was also observed that there was a significant two way interaction effect between group and electrode site as well as group and stimuli for all the waves. But there was no three way interaction (electrode site, stimuli and group) for any of the waves except P1.

Table 4: Results of AN OVA showing the main effect

Waves	Stimuli	Electrode site	Group	Group x Stimuli	Group x site	Grooup x stimuli x site
P1	19.89*	28.27*	38.89*	12.07*	26.28*	5.32*
N1	4.36*	10.4*	22.34.*	4.43*	9.58*	1.284
P_2	3.89**	27.6*	18.68*	3.96**	5.37*	0.79
N_2	6.27*	15.01*	13.27*	3.65**	8.73*	0.39

** significant at 0.01 level

* significant at 0.05 level

Post hoc Bonferroni test for effect of stimuli showed that the latency of all the peaks were significantly longer for /da/ than that for tonal stimuli but there was no significant difference between the latencies of the ALLR waves for /tja/ and tonal stimuli. The responses elicited for /da/ and /tja/ stimuli differed significantly for latency of Pi only.

Posthoc test to investigate the effect of electrode site on latency revealed that latency of Pi picked up from three electrode sites were significantly different from each other. The latency was shortest for responses picked up from forehead and longest for responses picked from right temporal region. For Ni, P2 and N2 waves latencies of the responses picked up from forehead were significantly shorter than those obtained from temporal regions. There was no significant difference between the latencies of Ni, P2 and N2 picked up from right and left temporal regions. Though not statistically significant, the mean latency of Ni was shorter

when responses were picked up from left temporal region when compared to that of right temporal region. Such difference was not observed for P2 and N_2 waves.

As there was a significant two way interaction, Independent t-test was carried out to compare the difference in means between the two groups separately for responses picked up from different electrode site for each stimulus. The results of Independent t test are shown in Table 5. It can be observed that for /tja/ stimuli, latencies of all the ALLR waves of the experimental group were significantly longer ($p<0.01$) than those of the normal group when responses were picked up from left and right temporal lobes. However latencies of the ALLR waves picked from forehead showed a significant difference only for P1. There was no significant difference between the mean latency of the two groups for all the waves except N_2 picked up from left temporal lobe when the stimuli used was /da/. For tonal stimuli, there was a significant difference ($p<0.01$) between the latencies of P1, N1 and P2 picked from left and right temporal lobes. Responses picked from forehead showed a significant difference only for latency of P2. There was no significant difference between the mean values of N2 for responses picked up form all the sites.

Table 5: 't' value of Independent Samples Test

Stimuli	Electrode site	**P1**	N,	P_2	**N_2**
/tja/	**F_z**	2.02**	1.34	1.80	1.92
	T_L	6.05*	3.73*	4.03*	3.44
	TR	6.69*	5.44*	4.41*	3.37*
/da/	**F_z**	-.68	.083	.25	-1.06
	T_L	1.53	1.64	1.71	2.27*
	TR	1.60	1.54	1.36	1.18
Tone	F_z	.93	.17	2.37*	1.03
	T_L	2.47*	2.64*	2.87*	1.75
	TR	4.30*	3.51*	3.22*	1.61

** Significant at 0.05 level

* Significant at 0.01 level

Amplitude of ALLR

Table 6 shows the mean amplitude of N1P2 and P2N2 complexes in control and experimental subjects. As expected the amplitude of P2 N2 complex was more than that of N1P2 complex. It can be observed from the table that, N1P2 complex had maximum amplitude when responses were picked up from left temporal region and minimum amplitude when responses were picked up from forehead. This was true for all the stimuli in both the groups. P2N2 complex had maximum amplitude when responses were picked up from forehead followed by those picked from left temporal region for all the stimuli in both the groups. A majority of the responses showed lower amplitude in the control group when compared to that of the experimental group. The standard deviation was high for all the responses in both the groups indicating the high variability.

Table 6: Mean and SD of amplitude of ALLR waves fin micro volts') in control and experimental groups

		NIP_2				P_2N_2			
		Control group		Experimental group		Control group		Experimental group	
Stimulus	site	Mean	SD	Mean	SD	Mean	SD	Mean	SD
/tja/	F_z	1.32	1.26	1.76	1.96	5.90	3.83	4.79	2.76
	T_L	3.2	2.44	3.59	2.68	3.13	1.99	4.02	2.38
	TR	2.88	2.30	2.07	2.19	3.09	1.85	3.29	2.13
/da/	F_z	1.71	1.50	1.61	1.40	7.49	3.33	4.86	2.24
	T_L	3.58	2.19	3.07	1.56	3.35	1.70	3.22	2.13
	TR	3.04	1.82	2.07	1.74	3.15	2.11	2.99	2.03
Tone	F_z	1.48	1.74	1.19	.97	6.48	4.60	3.41	3.77
	T_L	2.84	1.89	1.49	1.86	5.12	3.50	2.08	1.00
	TR	1.89	1.74	1.39	.86	3.94	2.76	2.46	1.62

Mixed design ANOVA revealed that there was a main effect of stimuli ($F=43$; $p<0.05$) and site ($F= 9.77$; pO.Ol) but not group ($F=0.01$; $p>0.05$) on the amplitude of NiP_2 complex. ForN2P2 complex, there was amain effect of electrode site ($F=18.83$; pO.Ol) and

group (F=6.2; p<0.01). However, stimuli used did not have a main effect (F-0.24; p>0.05) on the P_2N_2 complex. Analysis of amplitude of P_2N_2 complex, showed a significant two way interaction between group and stimuli (F=4.26; p<0.01) well as group and site (F=4.3; p<0.01). However, no significant two way interaction (F=0.91; p>0.05 for group x stimuli; F=0.30; p>0.05 for group x site) was found for N1P2 complex. Also there was no three way interaction (F=0.84; p>0.05 for $N1P_2$ complex; F=0.78; p>0.05 for P_2N_2 complex) for both the measures.

Post hoc Bonferroni test for effect of stimuli on $N1P_2$ complex showed no significant difference among all the stimuli used. Post hoc test for the effect of site showed that the amplitude of NiP_2 complex was significantly lesser for responses picked up from forehead when compared to the responses picked up from temporal regions whereas the amplitude of P_2N_2 complex was significantly higher for responses picked up from forehead when compared to responses picked up from temporal regions.

Results of Independent t test showed that there was no significant difference between the amplitude of the two groups for speech stimuli. Responses to tonal stimuli showed a significant difference for P_2N_2 complex picked up from all the sites and $N1P_2$ complex picked up from temporal regions (refer Table 7 for t values).

Table 7: t values for N1P2 and N2P2 complex

Stimuli	Electrode site	N1P2	P2N2
/tja/	F_z	-0.77	1.02
	T_L	-0.40	-1.21
	TR	1.01	0.28
/da/	F_z	0.18	2.79
	T_L	0.68	1.67
	TR	0.49	-6.4
Tone	F_z	-0.49	3.49*
	TL	2.91*	4.02*
	TR	2.5*	2.93*

** Significant at 0.05 level. * Significant at 0.01 level.

Results of MMN

Latency of MMN

The grand average MMN waveforms of normal children and for the stimulus contrasts are shown in Fig. 3, 4 and 5. Table 8 shows the mean and standard deviances of the peak latency of MMN obtained from control and experimental groups for the six deviances used in the study. Figure 6 depicts the comparison of latency recorded at F_z, across two groups. It can be observed that for all the deviances, the mean latency is longer for the experimental group when compared to the control group. This was true for MMN picked up from all the sites. It can also be observed that in the control group, for speech stimuli, the peak latency was longer for responses picked up from F_z followed by that from right and left temporal regions. Such clear trends were not seen for the experimental group. But for tonal stimuli, latency was shortest when responses are picked up from $F_z$both in control and experimental groups. Comparison of the latency of MMN for different stimuli shows that the latencies were shorter for tonal stimuli when compared to speech stimuli in both control and experimental group.

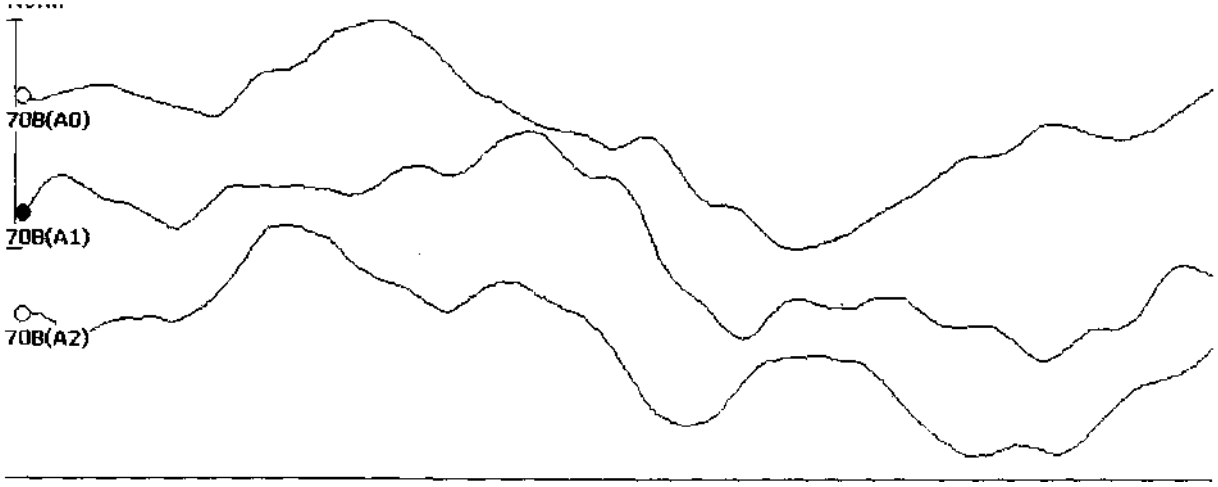


Fig. 3: Grand average of MMN elicited for deviance in voicing and manner of articulation in normal subjects

AO - Frequent stimuli (/tja/)

Al - Infrequent stimuli (/dza/)

A2 - Infrequent stimuli (/sa/)

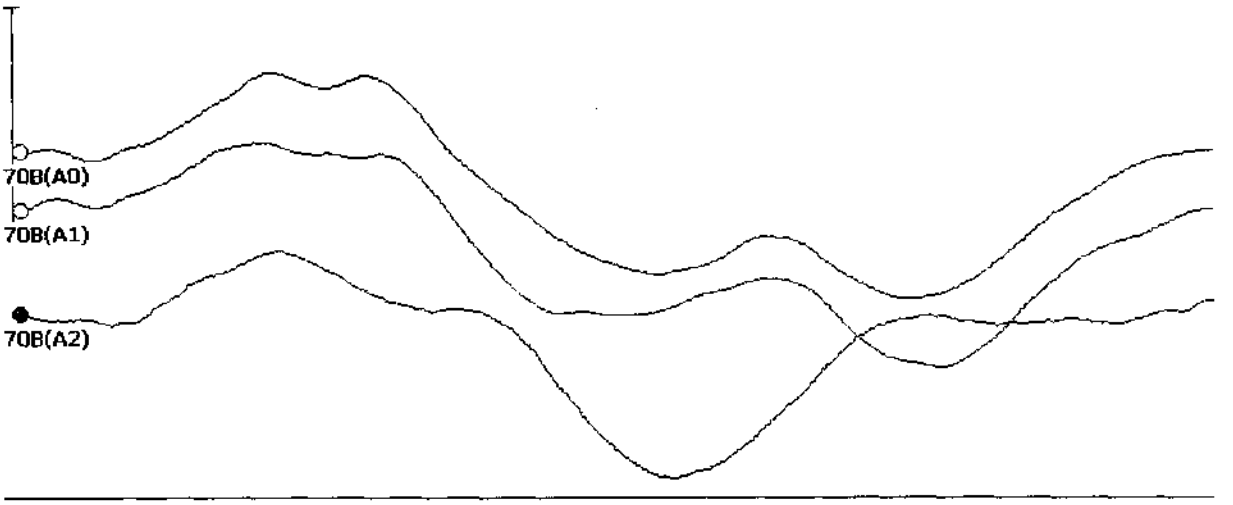


Fig. 4: Grand average of MMN elicited for deviance in place of articulation and vowel duration in normal subjects

AO - Frequent stimuli (/da/)

Al - Infrequent stimuli (/da/)

A2 - Infrequent stimuli (/da$_s$/)

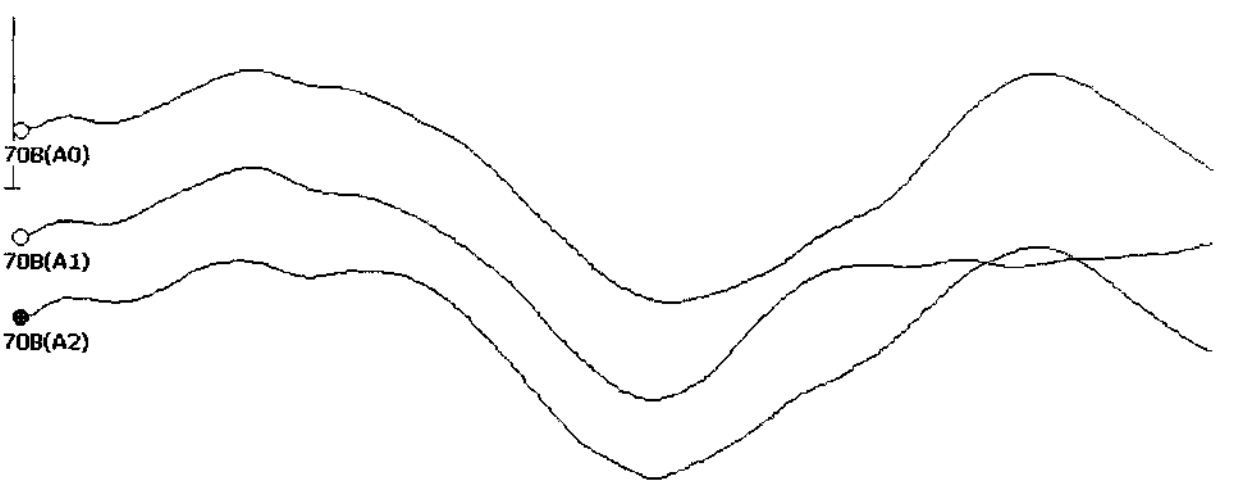


Figure 5: Grand average of MMN elicited for tonal stimuli in normal subjects

AO - Frequent stimuli (lOOOHz, 250 msec)

Al - Infrequent stimuli (11OOHz, 250 msec)

A2 - Infrequent stimuli (lOOOHz, 175 msec)

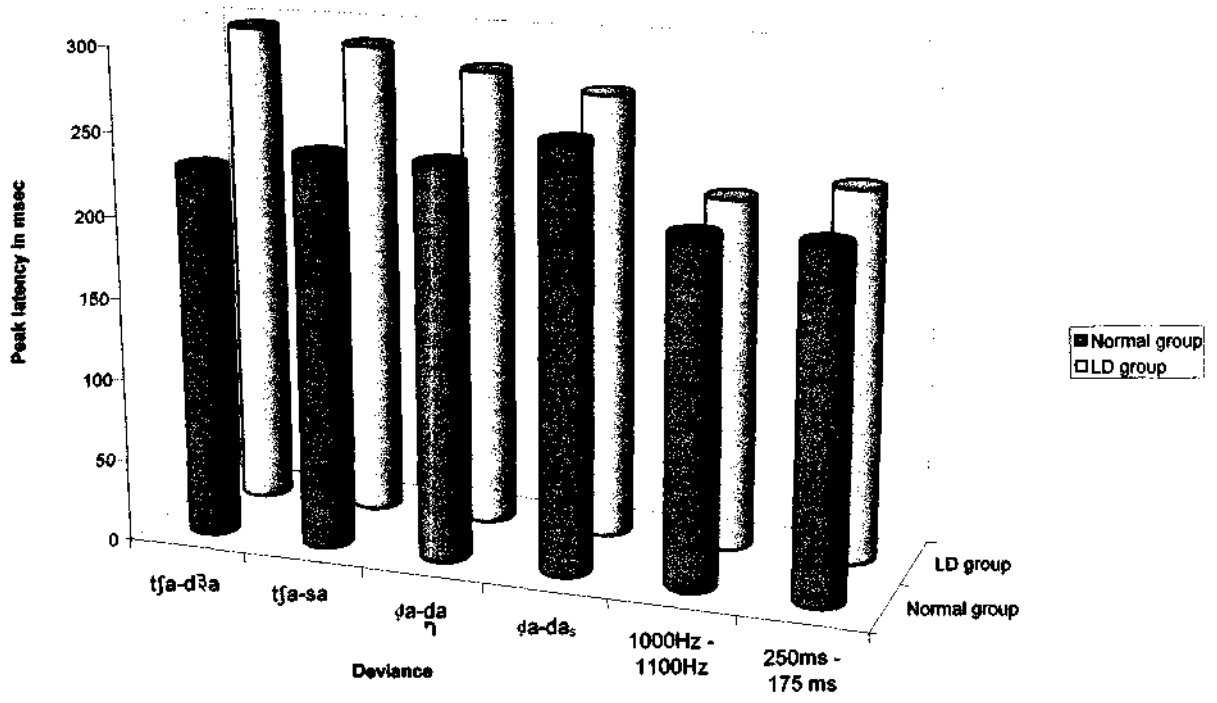


Fig. 6: Comparison of latency of MMN across LD and normal children.

Table 8: Mean and SD of peak latency (in msec) of MMN

Stimuli		Group	F_z		T_L		TR	
			Mean	SD	Mean	SD	Mean	SD
Speech	/tja/-/dza/	Control	225.83	37.23	216.58	48.66	221.64	47.52
		Experimental	296.43	46.36	304.78	47.23	309.0	51.38
	/tja/-/sa/	Control	237.86	48.25	219.06	52.27	224.80	48.20
		Experimental	288.75	90.65	293.4	34.46	274.25	28.00
	/da/-/da/	Control	237.47	24.63	212.6	35.62	213.90	38.23
		Experimental	277.5	41.03	290.4	45.29	293.56	41.56
	/da/-/das/	Control	254.87	20.67	218.67	50.17	225.6	52.58
		Experimental	267.85	38.25	281.0	40.56	284.85	41.73
Tone	lOOOHz-1100 Hz	Control	207.5	15.43	209.56	25.05	213.37	25.76
		Experimental	212.06	52.9	248.38	77.41	254.92	83.04
	250 msec 175 msec	Control	208.27	20.94	217.27	28.9	219.47	32.96
		Experimental	223.0	61.94	243.21	68.82	249.79	75.61

Mixed design ANOVA was carried out to investigate the effect of group, electrode site and the deviance used on latency of MMN. As the data violated assumption of sphericity, Greenhouse-Gieser effect was used to correct the results. Results revealed that there was a main effect of group ($F=31.65$; $p<0.01$) but repeated measures, electrode site ($F=0.81$; $p>0.05$) and stimuli ($F=0.65$; $p>0.05$) did not affect the results. There was a significant interaction between site and stimuli ($F=4.54$; $p<0.01$), site and group ($F=3.84$; $p<0.05$) as well as group, site and stimuli ($F=4.5$; $p<0.01$).

Independent t test was carried out to investigate the significant difference between groups for latency of MMN picked up from different sites for different stimuli. The results shown in Table 9, indicate that the latency of MMN was significantly longer in the experimental group when compared to that of control group, for responses picked up form all the sites when the /tʃa/-/dʒa/ and /ḍa/-/ɖa/ contrasts. For deviances, /tʃa/-/sa/ and /ḍa/-*/das/* contrasts as well as for frequency deviance, the experimental group had significantly longer latency for responses picked form temporal regions but there was no significant difference between the responses picked up from forehead. There was no significant difference between the two groups for duration deviance.

Table 9: t values for MMN for different deviances across two groups

Electrode site	/tʃa/-/dʒa/	/tʃa/-/sa/	/ḍa/-/ɖa/	/ḍa/-/daꜱ/	1000 Hz-1100 Hz	250 msec-175 msec
F_z	-4.29*	-1.77	-3.74*	-1.47	-.44	-1.19
T_L	-4.09*	-2.64*	-5.88*	-4.05*	-2.5*	-1.77
TR	-3.86*	-1.56	-5.38*	-3.7*	-2.51*	-1.87

* significant at 0.01 level

Amplitude of MMN

The mean and standard deviations for amplitude of MMN picked up from different electrode sites for different deviances for the two groups are shown in Table 10. Results of mixed design ANOVA indicate that there was no effect of group ($F= 0.18$; $p>0.05$), stimuli

(F=048; p>0.05), electrode site (F=2.44; p>0.05) on peak amplitude of MMN. As in the previous analysis, the data violated assumption of sphericity and Greenhoouse-Geiser estimate was used to correct the results. Two way interaction between the variables were not observed but there was a significant three way interaction among the effect of electrode site, stimuli and group (F=2.16; p<0.05).

Table 10: Peak amplitude of MMN (in microvolts)

Stimuli		Group	F_2		T_L		TR	
			Mean	SD	Mean	SD	Mean	SD
Speech	/tja/-/dZa/	Control	-2.2	1.67	-2.4	1.73	-2.78	1.93
		Experimental	-4.12	3.25	-4.31	1.77	-4.55	3.19
	/tja/-/sa/	Control	-3.44	2.5	-3.52	2.29	-3.42	2.33
		Experimental	-2.36	1.97	-3.97	1.68	-3.48	2.12
	/da/-/da/ *i*	Control	-2.77	2.13	-3.07	1.97	-2.82	2.11
		Experimental	-2.22	1.73	-3.15	1.54	-2.38	1.47
	/da/-/da_s/	Control	-5.54	3.05	-4.18	2.3	-3.29	2.43
		Experimental	-5.39	3.28	-4.01	1.77	-4.25	2.38
Tone	lOOOHz-1100 Hz	Control	-2.68	1.7	-2.89	2.29	-2.73	2.07
		Experimental	-4.08	2.96	-4.41	3.95	-2.77	5.41
	250 msec 175 msec	Control	-2.48	1.57	-2.98	2.17	-3.18	2.42
		Experimental	-4.85	2.77	-4.07	2.08	-3.9	2.5

Independent t test showed significant difference between the two groups (refer Table 11 for t values) only in the following conditions:

- For /tja/-/d^a/ contrast when responses were picked up from forehead and left temporal region.
- For frequency deviance when responses were picked up from forehead.
- For duration deviance when responses were picked up from forehead and right temporal region.

Table 11: t values for amplitude of MMN for deviant deviances

Electrode site	/tf/-/dZa/	/tja/-/sa/	/da/-/da/	/da/-/da_s/	1000 Hz-1100 Hz	250 msec-175 msec
F_z	2.24**	0.83	0.73	0.14	2.01**	3.69*
T_L	2.89*	0.38	0.13	0.24	1.59	1.57
TR	1.77	0.17	0.56	1.23	.04	2.2**

* significant at 0.01 level

** significant at 0.05 level

Analysis of individual data

The individual data from the experimental subjects were inspected to identify abnormalities in ALLR and MMN for different stimuli. The mean value + 1 SD was considered as the normal range and latencies or amplitudes beyond that value were considered as prolonged. Table 12 gives the number of children with abnormal ALLR and/or MMN responses. It can be observed from the table that the number of children with abnormal ALLR waves to /tja/ stimuli were more than that for /da/ and tonal stimuli. It was noticed that, all the children with abnormal ALLR waves also had abnormal MMN responses. More number of subjects showed abnormal MMN responses to speech stimuli than for tonal stimuli. The subjects with abnormal MMN responses for tonal stimuli also showed abnormal responses to speech stimuli but some of the subjects with abnormal responses to speech stimuli had normal MMN for tonal stimuli. Among the speech stimuli, more number of subjects showed abnormal MMN responses to /tja/ and /d^a/ contrasts.

Table 12: Table showing number of children with learning disability showing normal, prolonged and absent responses

Response	Stimuli	Normal	Prolonged	Absent
ALLR	/tja/	9	5	1
	/da/	14	-	**1**
	Tone	14	-	**1**
MMN - speech	/tja/-/dZa/		4	8
	/tja/-/sa/	5	5	5
	/da/-/da/	6	4	5
	/da/ - /da$_s$/	5	7	3
MMN-Tone	lOOOHz-HOOHz	9	4	2
	250 msec 175 msec	9	4	2

Pre and Post therapy comparison

MMN was recorded before and after auditory learning program was analyzed for two children. During the first evaluation, both the children had showed prolonged latency of ALLR waves for /tfa/ stimuli but normal responses to /da/ and tonal stimuli. Subject 'A' showed prolonged MMN responses to /tfsjf/d^a/ contrasts and /tfa/-/sa/ contrasts. MMN was absent for /da/-/da$_s$/ contrasts. Normal MMN responses were obtained for /da/-/da/ contrasts and tonal stimuli. Subject 'N' showed absent MMN to /tja/-/sa/ contrast and /da/-/da/ contrast. Peak latency of MMN was prolonged in rest of the recordings. Post therapy recording showed reduction in latency of all the ALLR waves in both subjects. MMN could be recorded for all the contrasts in subject 'A' and the latencies of the responses were shorter than those of the pre therapy recordings. In subject 'N', MMN recorded for speech stimuli showed shorter latencies than those of the pre-therapy recordings but MMN was absent for tonal stimuli.

Discussion

Controversy exists regarding the underlying cause of learning disability. Some investigators assume that a deficit in auditory processing is the source of the phonological disorder observed in children with learning disability. Others maintain that the phonological deficit in dyslexia is basically linguistic, not acoustic, in nature. The results of the present study indicate that there is a subgroup of children who have auditory processing problem though it cannot be ascertained whether the auditory processing problem is the causal factor for learning disability or it is just an associated factor.

Earlier investigations have indicated that many of the features distinguishing speech sounds, like voice onset time and formant transitions, require the detection of timing differences of complex auditory patterns in just a few milliseconds and this is affected in subjects with learning disability (Schulte-Korne, Deimel, Bartling & Remschmidt, 1999). There is a controversy as to whether the processing of both non speech and speech stimuli are affected or processing of only speech stimuli is affected. The main finding of this study was that, the children with learning disability show impaired auditory processing at the cortical level and the abnormality observed depends on the stimuli used. Processing of speech signal is affected more than the tonal stimuli. But discrimination of signals depends on the cues used for processing the signal and not on whether it is speech or non speech stimuli. The site of recording was also a factor influencing the results of the study. The latencies of the potentials were affected more than the amplitude suggesting that major dysfunction is in terms of time required for processing signals. Prolonged latencies of the ALLR waves and MMN suggest that a subgroup of children with learning disability require greater time for processing the signal when compared to that of normal subjects.

Auditory Long Latency Responses (ALLR)

It was observed in the study that the latencies of ALLR waves were prolonged in children with learning disability. A majority of the studies reported in literature also show prolonged latency of ALLR waves in children with learning disability (Satterfield, Schell, Backs & Hidaka, 1984; Byring & Jaryilehto, 1985; Leppanenn & Lytinnen, 1997). Results of investigations by Radhika (1997) and Guruprasad (2000) showed that children with

learning disability form a heterogeneous group as some children had normal ALLR responses and some had abnormal responses. The results of the present study support these findings. Though a group difference was observed in the present study, analysis of individual data revealed that nine out of fifteen children with learning disability had normal ALLR waves to all the stimuli.

Nl is related to selective attention and may be reflecting an early selective process. It compares the attended stimulus with the model stored in memory or on the other hand some active supervising process, which evaluates the arriving information or the access to memory (Bernal et al., 2000). Controversy exists about whether P2 is an exogenous or endogenous component. Some investigators view P2 as an exogenous potential which is sensitive to the physical attributes of the stimulus (Polich, Ladish, & Burns, 1990) whereas others report that P2 is modified by the attentional demands of the tasks performed (Johnson, 1989). It can be concluded from the results of the present and the earlier studies that these early representations of auditory signals are affected in some of the children with learning disability.

To address the controversy of processing of speech and non speech stimuli in children with learning disability, the results of ALLR to tonal stimuli were compared with that of speech stimuli. It was observed that the number of children with abnormal ALLR to speech stimuli were more than that for tonal stimuli. It was interesting to note that all the children who showed abnormal ALLR to tones also showed abnormal ALLR to speech stimuli but some of the children with abnormal ALLR to speech stimuli had normal ALLR to tonal stimuli. This shows that processing of complex signals such as speech is affected more in these children. The results also revealed that perception of all the speech stimuli are not affected equally. It was observed that the perception of/tja/ was more affected in children with learning disability when compared to perception of /da/. The reason for this discrepancy is not clear. It is possible that the perception of /tfa/ is affected more because it requires processing of more cues compared to that of/da/.

The results of the present study also suggests that the abnormality observed in children with learning disability is more when the responses are picked up from the temporal regions when compared to those picked up from frontal region. It can be inferred from this that the scalp distribution of the peaks is affected in children with learning disability.

The fact that the latencies of ALLR waves were affected, suggests that the perception of speech is affected in these children due to impaired processing of acoustic cues of speech and there is a neurophysiologic basis for this abnormality. Probably, ALLR can be used to identify auditory processing disorder in children with learning disorder even before they acquire speech. Molfese (2000) also reported that children diagnosed as having dyslexia at the age of 8 years had prolonged N1 latency during infancy. Leppanen, Pihko, Eklund and Lyytinen (1999) reported that infants at high risk for dyslexia had prolonged latency of P1. Children, who cannot detect, react to and process auditory information quickly will have learning problems. Thus, it can be concluded that a subgroup of children with learning disability have difficulty in basic processing of the auditory stimuli, as reflected in P1, N1, P2 and N2 of ALLR waves.

Results of MMN

MMN is an accurate index of preattentive cortical sound discrimination. The results of the study suggest that the preattentive cortical sound discrimination is affected in children with learning disability. Processing of both speech and non speech signals were affected but more number of children showed deficits for speech stimuli when compared to non speech stimuli. The results also varied depending on the type of deviance used in speech and non speech stimuli.

MMN for speech stimuli

The type of deviances used in the present study included deviances in terms of place of articulation, manner of articulation, voicing and vowel duration. Results of MMN indicated that the speech sound discrimination based on manner of articulation was affected in children with LD only when responses were picked up from left temporal region. But speech contrasts based on place of articulation and voicing elicited abnormal MMN from all the electrode sites. Duration deviance in speech elicited abnormal MMN recordings from temporal regions only.

Both behavioral as well as electrophysiological studies have earlier reported that processing of cues for perception of place of articulation is affected in children with LD

(Rosen, 1992; Kraus et al., 1996; Schulte-Kome et al., 1999). Kraus et al. (1996) reported that behavioral speech sound discrimination and MMN for /b/ - /w/ continuum was not affected in children with learning disability but /d/ - /g/ continuum was affected. Schulte-Korne et al (1999) observed that the MMN for /B/ - */d/* contrast was affected in children with learning disability. Rosen (1992) also observed that those features that signal place of articulation appear to be particularly vulnerable when auditory processing breaks down. The results of the present study reinforce these concepts.

Leppanen et al. (2002) reported that processing of durational cues is affected in infants at risk for learning disability due to a familial background of reading problems. They process auditory temporal cues of speech sounds differently from infants without such a risk even before they learn to speak. In the present study also, speech contrasts which differed in duration elicited abnormal MMN suggesting deficient perception of durational cues in a subgroup of children with learning disability.

There is dearth for information on MMN for voicing contrast in children with learning disability. However, behavioral studies have indicated that children as well as adults with learning disorder exhibit deficits in category labeling tasks involving contrasts based on voice onset time (Manis et al., 1997). Manis et al., (1997) reported that dyslexics with low phonemic awareness made poorer /b/-/p/ distinctions than both chronological age matched and reading level matched controls. They concluded that some dyslexic children have a perceptual deficit that may interfere with processing of phonological information. The results of the present study support these findings and also reveal that there is a neuophysiological basis for the abnormal voiced-voiceless perception observed in these children.

MMN for tonal stimuli

The results of the present study and a review of literature suggest that processing of speech is affected in children with learning disability. However, there is a controversy as to whether this type of deficit exists for non speech stimuli. The results of the present study show that these children have abnormal MMN for tonal stimuli also. But when tonal stimuli were used to record MMN, no group difference was obtained for MMN for duration

deviation whereas MMN for frequency deviance showed a group difference. Earlier investigation by Schulte-Korne et al. (1998) showed that MMN for frequency deviance of tone was not affected in dyslexics whereas MMN for speech stimuli was affected. They had concluded that dyslexics have a specific speech processing deficit at the sensory level which could be used to identify children at risk at an early age. Similar results have also been reported in adult dyslexics (Schulte-Korne et al, 2001). However, Baldeweg, Richardson, Watkins, Foale, and Griselier (1999) reported that MMN elicited for pitch deviances were smaller in dyslexics when the deviance used was 50 Hz. An investigation by Schulte-Korne et al. (1999) using complex tonal pattern that differed in temporal pattern but not frequency, showed that dyslexics have a significant pre-attentive deficit in processing of rapid temporal patterns. Renvall and Hari (2003) reported that electroencephalographic studies demonstrate smaller auditory responses to infrequent deviances of speech and non speech sounds in dyslexic than normal-reading subjects. The results of the present study reinforces the consensus that processing of both speech and non speech stimuli is affected in children with learning disorder but processing of all the cues are not equally affected. The present study also revealed a difference in the scalp distribution of LLR and MMN in children with LD.

Based on the results of the present study and earlier investigations, it can be inferred that it is the acoustic information embedded in speech sounds, rather than phonetic information per se, that resulted in the attenuated MMN found in dyslexics. Behavioral studies reported in literature also indicate that processing of both speech and non speech stimuli is affected in children with learning disability. Breier et al (2001) observed that children with reading disorder have a deficit in phoneme perception that was evident in inconsistent labeling of voice onset tokens (/ga/-/ka/) as well as in their labeling of tone onset tokens, supporting the hypothesis that deficits in speech perception in this group extend to non speech as well as speech stimuli containing similar acoustic cues. The duration of the period between the burst and the onset of voicing is a primary cue for voiced-voiceless differentiation. Thus it can be hypothesized that difficulty in voiced-voiceless differentiation observed in children with learning disorder is due to difficulty in discrimination of durational cues. The present findings support the hypothesis of a basic non linguistic auditory - information processing deficit in dyslexic individuals, which is also manifested in the preattentive analysis of acoustic features.

Discrimination of speech and non speech stimuli is affected in children with learning disorder and the basis of speech discrimination deficits may lie in deficits of neurophysiologic encoding along the auditory pathway. ALLR and MMN can be used to identify the subgroup of children who have auditory processing disorder. If auditory processing problems are detected early in infancy, there will be increased efficiency in rehabilitation of these children.

Effect of training

There is large amount of evidence that the MMN can serve as an index of learning-associated neural plasticity. Since the MMN reflects neural plastic changes and behavioral studies have reported that the discrimination ability of learning disabled children improves with training, it hold promise in evaluating the efficacy of training in children with learning disability. Though no conclusive statements can be made from the data of two subjects, it was observed that the latencies of ALLR and MMN after therapy were shorter than those recorded before therapy. This suggests that the time taken for processing the signals was reduced after therapy. It can be inferred from these results that probably children with learning disability can be trained in speech and non speech discrimination through auditory training programs. Earlier reports have also indicated that auditory training is helpful in children with learning disability. Naatanen, et al. (1993) showed that the MMN gradually emerged in those subjects who learned to discriminate complex spectrotemporal sound patterns, but not in those who did not learn to discriminate them. Kraus, et al. (1995) reported that behavioral training in the discrimination of speech stimuli resulted in a significant change in the duration and magnitude of MMN. Tremblay, et al. (1998) investigated MMN in subjects learning discrimination of new speech contrasts and found that MMN emerged even before the subject was able to discriminate the contrasts. Kujala and Naatanen, (2001) reported that in dyslexic children, the change in the reading skill measures with training correlated with change in MMN amplitude. The results of the present study are in consonance with these results.

The finding that the latency of ALLR and MMN responses decreases with training is consistent with the observation that training induces increased temporal coherence in the

cortical neural activity (Kraus et al, 1995). It has been hypothesized that an increase in the number of neurons firing at or near the same time would result in an earlier response.

Thus, it can be concluded that a subgroup of children with learning disabilities have abnormal ALLR and MMNs. It can also be concluded that they have abnormal processing of acoustic cues for both speech and non speech stimuli. Further, cues dependent on spectral aspects are more difficult to process than those that depend on temporal components. Electrophsyiological tests can be used to detect this processing deficit at an early age. This will facilitate early intervention programs. Early intervention programs would be more effective as the central nervous system is highly plastic at the early developmental stages (Singer, 1995). Electropysiological tests can also be used to monitor the improvement in auditory skills in children with learning disability.

Summary and Conclusions

Specific Learning disability (SLD) is a disorder in the psychological processes involved in understanding or using language, spoken or written, which may manifest in an imperfect ability to listen, think, speak, read, write, spell or do mathematical calculations. Exclusion from this group is based upon organic deficits including visual, hearing, motor, or economic disadvantage (Public Law-94, 1992). A review of literature reveals heterogeneity in the characteristics, causes and associated deficits of children with learning disability. Though, it is not known whether it is the cause or just an associated deficit, results of various investigations have revealed that there is a subgroup of children with learning disability having auditory processing deficit.

Auditory processing of an individual can be assessed through either behavioral tests or electrophysiological tests. Electrophysiological studies have revealed that the latency of the auditory evoked potentials is prolonged and the amplitude is decreased in children with learning disability. However there is dearth of information on speech evoked long latency responses in children with learning disability. MMN has been used to investigate auditory discrimination abilities in children with learning disability. A majority of the studies have investigated MMN in children with learning disability for speech stimuli that is deviant in place of articulation and manner of articulation. But there is dearth for studies comparing MMN for speech contrasts varying in place of articulation, manner of articulation, voicing and vowel duration in children with learning disability. Also there is controversy as to whether these deficits in children with learning disability are limited only to speech stimuli or it occurs for both speech and non speech stimuli. Research in this area will throw light on usefulness of MMN in identification of auditory processing disorder in children with learning disability. If MMN can be used in assessment of auditory processing problem, it would help in early identification of children with learning disability or those who are at risk for learning disability. This would in turn enable early rehabilitation of children with learning disability. As the neural plasticity is more in younger children, the benefits of rehabilitation will definitely be more if rehabilitation is started early in children.

A majority of the studies reported in literature have used synthetic speech in an attempt to carry out a controlled study to assess the fine discrimination ability. However, it is

of paramount importance to investigate the perception of natural speech in these children to understand the difficulties faced in real life situations. Hence the present study used natural speech, as spoken by a native Kannada speaker to elicit MMN. This research is also helpful in understanding the neurophysiological basis of speech discrimination in children with learning disability.

Research has indicated that the effect of auditory learning can be documented using electrophysiological measures. Reduction in latency and increase in amplitude of long latency waves and MMN has been reported in individuals who have shown improvement in auditory processing of speech signal. However, there is dearth of such studies in children speaking Indian languages.

The present study was designed to investigate the following aims:

1. To study the auditory long latency responses to speech and non speech stimuli in children with learning disability.
2. To study MMN for speech and non speech stimuli in children with learning disability. Speech discrimination was studied for the following four deviances:
    a. place of articulation
    b. manner of articulation
    c. voicing and
    d. vowel duration

    Deviances used for tonal stimuli included frequency deviation and duration deviation
3. To study the effect of auditory learning on auditory long latency responses and MMN.

Data was collected from fifteen children diagnosed as having learning disability by an experienced Speech and language pathologist/ Clinical psychologist and thirty age matched control subjects. Three recordings of MMN were carried out using binaural stimulation. Each recording consisted of presentation of one standard stimulus and two deviant stimuli. The stimuli used were as follows:

- Standard : /tja/, Deviant 1, /dza/, Deviant 2:/sa/
- Standard : /da/, Deviant 1: /da/, Deviant 2: /da$_s$/
- Standard: 250 msec, 1000 Hz tone, Deviant 1: 250 msec 1100 Hz tone,

Deviant 2: 175 msec 1000 Hz tone

MMN was differentially recorded from F_z, TL and TR with lower forehead as the ground and nose tip as the reference electrode site using IHS Smart evoked potential system. The responses were analyzed for latency and amplitude of ALLR waves and MMN. Children who obtained abnormal MMN recordings were advised to attend auditory training program and the electrophysiological testing was repeated after auditory training program.

Analysis of the data obtained revealed the following results:

- ALLR and MMN responses are abnormal in subgroup of children with learning disability suggesting the presence of auditory processing disorder
- Processing of both speech and non speech stimuli are affected in children with learning disability. However, the number of children showing abnormality for speech evoked responses is greater than that observed for tonal stimuli
- Among the different deviances used in the study for recording MMN, perception of place of articulation cues was maximally affected followed by voicing and durational cues. Perception of manner of articulation was least affected in these children.
- The latency of ALLR and MMN waves reduced after auditory training program.

Thus ALLR and MMN can be effective tools for the early identification of subclinical auditory processing disorder in children with SLD. They also are effective in monitoring the change in neural plasticity that can occur with the auditory training program.

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