MMN FOR DURATION DEVIANCE IN CHILDREN WITH S.L.I.

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Reg. No. MSHM2K07

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University of Mysore, Mysore.

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May - 2003

CERTIFICATE

This is to certify that this Dissertation entitled "MMN **FOR DURATION DEVIANCE IN CHILDREN WITH S.L.I."** is a bonafide work in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student (Register No. MSHM2K07).

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Certificate

This is to certify that this Dissertation entitled **"MMN FOR DURATION DEVIANCE IN CHILDREN WITH S.L.I."** has been prepared under my supervision and guidance. It is also certified that this Dissertation has not been submitted earlier in any other University for the award of any Diploma or Degree.

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DECLARATION

This Dissertation entitled **"MMN FOR DURATION DEVIANCE IN CHILDREN WITH S.L.I."** is the result of my own study under the guidance of **Dr. Asha Yathiraj,** Reader and H.O.D, Department of Audiology, All India Institute of Speech and Hearing, Mysore and not been submitted earlier in any other University for the award of any Diploma or Degree.

Mysore,

May, 2003 Reg. No. MSHM2K07

.... TO A VERY SPECIAL CHILD

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INTRODUCTION

Deficit of auditory processing have been implicated in developmental disorder of high-level cognition processing, such as language impairment (Tallal, Stark & Mallits, 1985). Specific language impairment (SLI) is the diagnostic term of choice in current practice in which language deficit or delay is found in the absence of frank neurological, sensorimotor, nonverbal cognitive or social emotional deficits (Leonard, 1997).

Several studies, directed at uncovering the basis for the disorder have shown that many of these children have particular difficulty in discriminating speech sound distinguished by rapidly changing acoustic spectra (Tallal & Piercy 1975; Tallal, Stark & Curtiss, 1976; Frumkin & Rapin, 1980). However, the nature of this problem and its relationship to impaired language development is controversial. According to one view, this apparent perceptual difficulty is simply one manifestation of high order failure in the development of linguistic abilities concerned with phonological and syntactic processing of speech and can thus be regarded as a language disorder (Cromer 1978, cited in Stefnatos, Green & Ratcliff, 1998; Leonard, 1982). An alternative view however, suggests that the problem is attributed to deficient auditory temporal processing since children with development dysphasia (then used term for SLI) are also impaired in perception of rapidly changing non speech sounds (Tallal & Piercy, 1974; Tallal et al., 1976). These children also have deficit in fine grained auditory discrimination (Elliot, Hammer & Scholl, 1989).

Language decoding impairment is supposed to be only one feature of more common and more general defect in reacting to auditory stimuli (Stefantos, Green & Ratcliff, 1989). Kraus, Mc'Gee, Littman, Nicol and King (1994); Kraus et al., (1996); Kraus (2001) hypothesized that children with learning problems have difficulty in perceiving fundamental acoustic parameters that originate from abnormalities in neurophysiological encoding of acoustic differences which occur after peripheral sensory encoding and before conscious perception. It has been argued that if such a basic auditory problem could underlie their language impairment by impeding the process by which linguistically important acoustic cues in speech signal are encoded, then this question has important implication for understanding and treatment of the disorder. Various studies have been done to prove the above hypothesis.

Elliot et al., (1989) in a study involving behavioural response, measured the just noticeable difference (JND) in normal and developmental dysphasic children and found that not only did the language impaired children have larger JND, but also, JND measure could correctly classify children as normal or language impaired with relatively high level of accuracy.

The neurophysiologic correlate of behavioural discrimination is mismatch negativity (MMN). MMN provides an index of neurophysiologic representation of acoustic contrast and thus provides a tool for exploring the processing of acoustic differences that underlie speech perception (Kraus, Mc'Gee, Carrell, Zecker, Nicol & Koch, 1996).

MMN is generated by the brain's automatic respone to auditory stimulation (Naatanen & Escara, 2000). It originates in the auditory thalamocortical pathway (Scherg & Picton, 1990, cited in Kraus, et al., 1996) and elicited when a physically deviant stimulus occur in a series of homogenous stimuli. It is sensitive to auditory differences (Naatanen & Gailliard, 1983, cited in Korpilahti, 1995; Naatanen, 1992, cited in Korpilahti, 1995), even when the deviance is near the perceptual discrimination threshold level (Lang, Nyrke & Naatanen, 1989).

The same cerebral mechanism that elicit attention independent MMN are suggested to be necessary, but not sufficient conditions for the conscious perception of auditory stimulus change (Naatanen, 1990).

Korpilahti and Lang (1994), Hollopaenen, Korpilahti, Lang and Slillanpaa (1994) used duration and frequency change MMN to investigate children with developmental dysphasia. They found that the peak amplitude of frequency change MMN (500 Vs 553) was significantly attenuated in dysphasic children as compared to their healthy controls. For duration deviance a significant between-group difference in MMN existed only when a large deviant-standard (50/500 msec) was used.

There is a relationship between the language impairment and long latency evoked response potentials (ERP). On the basis of the previous research (Jirsa, Kimberly & Clontz, 1990; Neville, Coffey, Holcomb & Tallal, 1993; Korpilahti & Lang, 1994) it is assumed that deficits of auditory processing would also be reflected in the LLR wave complexes.

In the present study cortical evoked potentials, namely, late latency response and MMN are investigated in children with SLI. MMN for three duration deviances are used to investigate the auditory discriminatory ability of children with SLI. The results are compared with age and sex matched control

Need for the study

Researchers hypothesized that language learning problem may arise from faulty neural representation of sound in central auditory system (Kraus et al., 1996). Language impaired children have difficulty with perception of certain fundamental acoustic differences (Tallal & Piercy, 1974; Elliot et al., 1989). By using MMN, which is an objective measure of automatic processing of auditory discrimination, it is intended to identify auditory processing deficits in children with specific language impairment.

There is a dearth of studies investigating MMN for duration deviances in language impaired children. Most of the studies already done have investigated MMN for larger duration deviance (> 50 msec of deviance) and still the result of these studies could differentiate the SLI group from the normal (Korpilahti & Lang, 1994). However, there is possibility that the use of large deviance could identify children with more severe language impairment and not those with subtle problems. With the use of smaller duration deviance it may be possible to detect children having subtle problem, thus making the investigation a more sensitive one.

If there is a difference in the duration perception in children with SLI when compared to the controls, it would throw light on the specific auditory perceptual

problem that these children may have. This information would be of further help in designing the line of treatment to improve their auditory perceptual problems.

Aim of the study

The aim of the present study was

To compare cortical evoked response potentials, namely, late latency response (LLR) and MMN for duration deviance in children with SLI with that obtained on age and sex matched controls,

REVIEW OF LITERATURE

"Mismatch Negatity, the brain's automatic change detection response in audition, usually peaks around at 100 - 250 msec in response to change in repetitive auditory stimulus. It is considered as an outcome of a comparison process between a new, deviant stimulus and memory trace formed by a standard stimulus in the auditory system (Cheour, Korpilahti, Martynova & Lang, 2001). Thus, an MMN is elicited when a sound discriminably changes in frequency, duration or intensity or when a phoneme is replaced by another phoneme.

MMN has also been considered as an index of involuntary attention switch to changes in auditory stimuli. This point has been suggested on the basis of its frontal lobe component. Naatanen and Michie (1979, cited in Alho, 1995) noted that MMN is mainly generated by the auditory cortex sources, but it also has a frontal generator, one usually stronger on the right than the left hemisphere. The activation of frontal generator might belong to the chain of processes leading to involuntary attention switch to sound change pre-perceptually detected in the auditory cortex (Giard, Perrin, Pernier & Boucher, 1990). This frontal activation indeed seems to occur a little later than the auditory cortex activation (Renne, Alho, Ilmoniemi, Sinkkoneni, Virtanen & Naatanen, 1999, cited in Naatanen & Escara, 2000) supporting the assumption that the change-detection signal generated by auditory cortex triggers the frontal mechanism of attention switch (Naatanen, 1990).

In addition to indexing short duration memory traces, MMN also probes the permanent auditory memory traces, such as those of phonemes of one's mother

tongue (Naatanen, 1995). This particular aspect of MMN is utilized in studying neurophysiology of language acquisition. So, MMN is in some way related to neurophysiology of short-term and long term memory besides its important role in perception of auditory discrimination.

Neurophysiology of MMN

The mismatch response has been recorded at multiple levels of auditory pathways which include the midbrain, thalamus and the cortex with distinct contributions by right and left sides, and non-primary and primary pathways. In humans, the generating source inferred from scalp recordings has included auditory cortex (Hari et al., 1984; Giard, Perrin, Premier & Boucher, 1990) and frontal cortex (Naatanen & Michie, 1979, cited in Alho, 1995; Giard, Perrin, Pernier & Bouchet, 1990).

Intracranial recordings in animals have revealed MMN in the hippocampus (Csepe, Karmos & Molnar, 1987) auditory midbrain (King, McGee, Rubel, Nicol & Kraus, 1995), auditory thalamus and the cortex (King et al., 1995; Kraus, McGee, Littman, Nicol & King, 1994).

The MMN appears to have a strong extralemniscal (non primary) pathway origin. Specifically, dipole source analysis is consistent with non-primary auditory cortex contributions (Scherg & Picton, 1990, cited in Alho, 1995). The role of non-primary pathways in auditory learning and CNS plasticity has been demonstrated by numerous single neuron experiments (Edeline & Weenberger, 1991, cited in Alho,

1995). The non-primary pathway origin of MMN is consistent with MMN changes that accompany perceptual learning.

Scalp distribution of MMN

There is a right versus left hemispherical specialization for audition which depends on the acoustic characteristics of the stimulus. MMN elicited by tones is larger over the right hemisphere in both adults and children irrespective of the ear being stimulated (Giard et al., 1990; Korpilahti & Lang, 1994; Csepe,1995). However in response to speech stimuli the MMN has been found to be symmetric (Aaltonen, Eerola, Lang, Uusipaikka & Tuomainen, 1994) or asymmetric (Csepe, 1995).

Csepe (1995) reported that MMN elicited by vowels were slightly larger over the right hemisphere while MMN to stop consonant has maximum amplitude over the left hemisphere.

There have been two opinions regarding the scalp distribution of neural generators of MMN for non-speech stimuli. One group of researchers believes that scalp topography of MMN varied as a function of the standard stimulus that used to elicit the MMN. Among such researchers are Giard et al., (1995) and Paavilainen Alho, Reinikainen, Sam and Naatanen, (1991) who studied the scalp distribution of MMN elicited by deviant sound differing from the standard in intensity, frequency and duration. They found different scalp topography for different stimuli.

Lavanen, Ahonen, Hari, McEvoy and Sams (1996) also observed MMN location differences for changes in tone frequency, duration and interstimulus interval.

Based on this they proposed that different MMN are derived from different neural population.

Frodle-Bauch, Kathmann, Moller, Hegari (1997, cited in Kraus & Cheour, 2000) found that equivalent dipole source underlying frequency MMN are significantly more anterior than the source underlying duration MMN.

These results indicate that the source underlying MMN may differ depending on the deviant parameter. However there is another group of researchers who speculate that the same set of neural generators may contribute to each of the different scalp recorded MMN, but the relative contribution of the generators may vary. For example, the generators may include one process specific to detecting deviance and another process specific to change in stimulus environment, regardless of the type of change. The two generators may have different scalp topographies as may, for example if a sensory MMN is generated in the temporal lobe and a stimulus independent MMN is generated in the frontal lobe. The different scalp distribution may then reflect different degrees of activation of sensory and stimulus independent generators. The activation of stimulus independent generators may depend on the size of mismatch (Picton, Alain, Otten, Ritter & Achim, 2000). If this is so, it is essential to compare scalp topographies across different types of deviances after adjusting for the discriminability of the deviant stimuli. Deouell and Bentin (1998) closely controlled the discrimination difficulty and were unable to find any significant differences in scalp topographies among MMN related to changes in pitch, intensity, interval and location. Paavilainen et al., (1991) found similar scalp topographies among frequency, intensity, and duration MMN but noted that the polarity inversion

at mastoid was less prominent for intensity MMN than for the others, indicating some differences in the source configuration underlying the different MMN.

In summary, the MMN has been used to further our understanding of neural representation of sounds in the brain. The representation of acoustic change is evident in the auditory pathway structures (midbrain, thalamus, cortex-especially nonprimary subdivisions) and nonauditory areas (frontal cortex and hippocampus). The involvement of a specific pathway depends on the acoustic and phonetic characteristics of the stimuli.

Factors Affecting MMN Responses

Although the classical paradigm of recording MMN involves presenting regular train of auditory 'standard' stimuli in which the occasional 'deviant' stimuli differs from the other in terms of physical attributes, there are a host of other factors that have been reported to affect MMN. The factors are discussed in the following section in terms of stimulus dependent factors and subject dependent factors.

Stimulus Dependent Factors:

Type of stimulus: MMN can be recorded to any suprathreshold physical change such as frequency, intensity and duration of tones as well as in response to more complex changes as in speech.

MMN for speech stimuli: The MMN elicited to speech stimuli can be symmetric (Aaltonen et al., 1994) or it can be asymmetric (Csepe, 1995)

Csepe (1995) reported that MMN elicited by vowel were slightly larger over the right hemisphere, while MMN to stop consonants had maximum amplitude over left hemisphere. Likewise Alho (1995) have shown that MMN elicited by syllables (standard /da/, deviant /de/and /ba/) were longer over the right hemisphere. Further, while MMN elicited by non-native speech syllables were initially symmetric, responses became especially enhanced over the left hemisphere following training (Tremblay, Kraus, Carrell & McGee, 1998). In addition, the MMN elicited by the syllable /da/ was longer over the left hemisphere when /da/ signalled a phonetic change but was symmetric when the same /da/ signalled a pitch change (Sharma, Kraus, McGee, Carrell & Nicol, 1993). Naatanen et al., (1997, cited in Kraus & Cheour, 2001) also found a left hemisphere enhancement to phonetically relevant native language prototype.

From the studies cited above it appears that linguistic or phonetic nature of speech stimuli as well as the specific acoustic phonetic sound structure influence the amplitude and hemispherical distribution of speech evoked MMN.

MMN for non speech stimuli

Frequency Deviance

Csepe (1995) studied the effect of frequency deviance from 25% to 5% in generation of MMN and found that the smaller the difference, the larger the latency range and larger the cortical area involved in participation of MMN. The amplitude however showed significant enhancement with increasing frequency deviation reaching a plateau at a deviance of 15% (Csepe & Molnar, 1997).

Duration Deviance

Pekonen, Rinne and Naataen, 1995, noted that the MMN was found to be larger for duration changes than for intensity and frequency changes. Joutsiniemi et al., (1998) studied duration change in MMN in healthy subjects in the age range of nine to eighty four years. Two duration deviances, 25 msec and 50 msec were presented randomly in the same block. The standard stimulus was 75 msec in duration. Results indicated that a deviance of 50 msec evoked clear responses in thirty-nine out of forty subjects whereas a deviance of 25 msec could elicit observable MMN in only 32 subjects. Thus a larger deviance elicits a more definite response.

Korpilahti and Lang (1994) investigated two duration deviances, 50 msec Vs 110 msec and 50 msec Vs 500 msec in normals and dysphasic children. It was observed that peak amplitude of duration MMN increased in both groups when the physical difference between the standard and deviant increased (50/100 msec Vs 50/500 msec). The scalp distribution for duration change MMN was recorded more centrally.

Sreevidya (2001) attempted to find the electrophysiological threshold for duration discrimination by using a 50 msec tone burst (1000 Hz) as standard. In adults, a deviance of 3 msec could elicit MMN in all the subjects, which is in accordance with the psychophysical correlate of duration difference reported by Abel, Krever and Alberti (1972). In children in the age rang of 8-12 years, the electrophysiological threshold was found to be 5 msec. In both adults and children amplitude increased and latency decreased with the increase in duration deviance.

Csepe, Karmos and Molnar (1989, cited in Cheour et al., 2001) observed that no MMN was elicited with frequency deviants of 5% and below. However, Paavilainen Jiang, Lavlkainen and Naatanen (1993, cited in Cheour et al., 2001) noted that the MMN could even be elicited by frequency increments of 5%. Retrospectively, it was concluded that difference in two findings was due to the difference in the stimulus duration used. Csepe et al., (1989, cited in Cheour et al., 2001) used duration of 5 msec, whereas Paavilainen et al., (1993, cited in Cheour et al., 2001) used duration of 30 msec. The latter author opined that interplay of frequency and duration influence the time needed for forming a better memory trace which inturn results in better generation of MMN.

Naataenen (1995) opine that for small frequency differences MMN amplitude is small and the S/N ratio is more. Lang et al., 1990 suggested that frequency deviance up to 10% are considered to produce relatively pure MMN, as determined on the basis of MMN latency and wave form. Another component that determines difference limen of frequency in MMN generation is the harmonic composition of the standard stimulus. Tervaniemi, Alho, Paavilainen, Sams, Naatanen, (1993 cited in Sinkkonen & Tervaniem, 2000) postulated that a rich harmonic sound structure facilitates better frequency discrimination and thus MMN elicited has larger amplitude and latencies earlier than the MMN to pure sinusoidal sounds with a corresponding frequency change. They also found that enhanced MMN amplitude and a shortened latency were also obtained when the spectrally rich sounds included the fundamental frequency and only its first two multiples.

The use of duration deviance to study MMN provides an advantage of it not being contaminated by response of additional neural elements, as is often the case with frequency deviance. However for duration, a border condition emerges when very short stimuli are used: a decrement of a duration less than 200 msec causes a perception of both duration and loudness decrement (Hawkins & Presson, 1986, cited in Sinkkonen & Tervaniemi, 2000).

Intensity Deviance

When there is a very small difference in intensity of the two stimuli, MMN amplitude is low and the S/N ratio is poor (Naatanen, 1995). On the other hand amplitude increases and latency decreases with the increase in intensity deviance (Csepe, 1995; Naatanen, Paavilainen, Alho, Reinikainen & Sam, 1987, cited in Lang et al., 1995; Jose, 1999). Jose (1999) also found that the total MMN duration increases with the increase in deviance. He found attenuated amplitude, latency, total duration and magnitude in an unattended condition like reading. Naatanen, Jiang, Lavikainen, Reinikainen and Paavilainen (1993) also reported that MMN to intensity deviance is clearly attenuated in the absence of attention, suggesting that the intensity MMN to be vulnerable to attention.

Magnitude of Deviance

When the physical difference between the standard and deviant stimuli is small, it is easier for the subject to ignore the test stimuli (Lang et al., 1995; Naatanen, 1995). With a small difference, however the MMN amplitude is low and the signal-to-noise ratio is poor. (Lang et al., 1995). When the deviance exceeds a certain critical limit, the highly deviant obtrusive stimulus causes a passive switch of attention

(Naatanen, 1995). In that situation, a large P_3 component is superimposed on the deviant waveform. Deviances up to 10% are considered to produce relative pure MMN, as determined on the basis of MMN latency and wave shape (Lang et al., 1995).

Rate of Stimulus Presentation

Naatanen et al., (1987, cited in Lang et al., 1995) have reported that if simple stimuli are used, MMN amplitude increases when the inter stimulus interval (ISI) shortened, provided that intervals between the deviants are of same duration. A possible explanation of this phenomenon is that when the repetition rate of the standard stimuli increases, the memory trace evoked by it becomes more intense. This in turn strengthens the MMN response generated by a comparison process (Naatanen, 1995). There are however, two problems with a short ISI. First, if the decay speed of the memory trace contains any essential information, this information may be lost. Second, in the case of long latency response, the 'tail' of the response may be lost, including P_{3a} wave which signals the obtrusiveness of the stimuli. Changing ISI is a powerful way to dissociate the MMN from N_1 response. The shorter the interval from the preceding stimulus, the smaller the N_1 wave, as distinguished from the MMN which changes little. In practice, an ISI of 300 msec has been used with adults, however; with paediatric population an ISI of 400-450 msec has been advised (Lang et al., 1995).

There is a complex interaplay of stimulus parameters affecting MMN. In general it has been found that MMN measures are more for duration deviances then

for the frequency and intensity deviances. However, the parameters selected for recording of the MMN should be based on the objectives of the study.

Subject dependent factors

Age and Maturation

Although not enough MMN studies have been conducted in all the age groups, such as in infancy or pre school age, to fully understand the maturation of this negativity, most child MMN studies indicate that MMN is developmentally quite stable in terms of both latency and amplitude (Csepe, 1995; Lang et al., 1995). Nevertheless, in infants and young children, the MMN latency tends to be longer than in adults (Cheour-Luhtanen, Alho, Kujala, Saino, Reinikainen, Renland, Aaltonen & Naatanen, 1995) the amplitude greater (Csepe, 1995; Sharma, Kraus, McGee, & Nicol, 1993; Cheur et al., 1999) and the overall duration larger than in adults (Kraus et al., 1993).

In neonates and infants the MMN response to tones peaked approximately between 300-500 msec (Cheour, Ceponiene, Hukki, Haapanen, Naatanen & Alho, 1999). For speech evoked MMN the peaking was observed between 200-250 msec (Cheour-Luhtanen et al., 1995). The scalp distribution of MMN in infants was maximum at centroparietal sites, rather than the frontocentral sites as seen in adults (Cheour et al., 1997).

Morr, Shafer, Kreuzer and Kurtzberg (2002) studied maturation of mismatch negativity in typically developing infants and children. They used two oddball paradigm of frequency deviance (1000/1200 Hz) and (1000/2000 Hz). They found a

found a MMN like negativity in the time frame of 140-200 msec in 3-44 month old infants and preschoolers for the larger tonal difference (1000/2000 Hz). Significant negative correlation was observed between age and latency but not for age and amplitude. For the first tonal deviance (1000 /1200 Hz) majority of subjects tested, failed to exhibit MMN like component, particularly those below 24 months of age. Their data suggested that an adult like MMN could not be reliably elicited to a 1000/1200 Hz difference until 4 years of age.

In an earlier study Shafer, Morr, Judith, Kreuzer and Kurtzberg (2000) investigated maturation of auditory processing in school age children. The results showed that MMN was found to decrease with latency by 11 msec/year from 4 to 10 years of age. No development change in MMN amplitude was seen.

Thus it may be inferred that it is possible to record MMN like potential in infants with slightly larger deviances. The MMN elicited has longer latency and larger amplitude and wider spread of the negativity.

Subject's Attention,

The use of MMN as an objective measure of auditory function is to a large extent based on the assumed full or partial independence of the MMN from attention. Several studies have shown that the MMN elicited by deviant stimuli when they are targets of attended sound sequence or when the sounds are ignored are of very similar amplitude (Naatanen, Simpson & Loveless, 1982). Comparison with the Magnetic counterparts of MMN (MMN_m) has yielded very similar amplitudes in attended and unattended conditions (Alho, 1995). Other documented studies claim a difference in

MMN elicited in attended and unattended conditions. Sams and Alho (1986, cited in Alho, 1995; Naalanen et al., 1983, cited in Alho, 1995) found that deviants in attended condition have larger negativity than when the auditory stimuli are ignored. Later, they reasoned that this difference is due to the superimposition of the N_2b component on the MMN.

Aaltonen et al., (1994) reported that MMN for phonetic change *(*/b/-/d/ or vice versa) were larger in amplitude when phonetic stimuli were attended than when they are ignored.

The role of attention for frequency deviance was studied by Naatanen et al., (1993). They found that although the frequency MMN is elicited even in complete absence of attention, its amplitude might some time be attenuated. Thus, the threshold of frequency MMN is not affected by attention whereas the amplitude is affected.

In contrast, the MMN for intensity reduction is strongly attenuated though not fully eliminated, in the absence of attention (Woldorff, Hackley, Hillyard, 1991; Naataanen et al., 1993).

Naatanen et al., (1993) postulated that the intensity MMN is very much vulnerable to attention and clearly attenuated in the absence of attention. Nevertheless, no data demonstrated a total disappearance of the MMN in the absence of attention.

Naatanen (1990) therefore suggested that the sensory analysis resulting in neural sound representation is not affected, but that the excitability of the MMN process triggered by deviant stimulus might be dampened in absence of attention. However, the same author in 1995 suggested that a passive condition is preferred to avoid mixed waveform caused by N_2/P_3 waves, typically of active condition. Ignoring can be achieved by focussing the subject attention away from the test stimuli like watching video movie or reading a book.

To conclude, a complicated interaction seems to exist between the stimulus parameters and the individuals variables (age, attention and individual variability) to influence the MMN parameters.

Clinical Application of MMN

The MMN has been recorded in many different clinical contexts. Its advantage over the sensory evoked potential is that it reflects discrimination and memory in addition to sensation. Its advantage over later endogenous evoked potential is that it occurs automatically and does not require the patient's involvement in any task. MMN has been used to evaluate the perceptual and mnemonic processing that occurs in a group of patients with particular disorder, which helps us to understand what is going wrong in that disorder. MMN has also been used to diagnose a particular abnormality of processing or a particular clinical disorder in an individual patient. However, MMN obtained or individual measurements are not yet sufficiently reliable, sensitive or specific.

MMN in the Paediatric Population

The attention independent, passive elicitation of MMN is of considerable advantage for its application in studying developmental/childhood disorders. MMN has been studied in children with developmental dysphasia (SLI), learning disability, autism, cleft lip palate and also in newborns with the family history of learning disorders. In the forth coming section, studies related to developmental language disorders and learning disabilities have been reviewed.

MMN in developmental dysphasia

Deficient auditory perception and impaired processing of rapidly presented sequential information are often connected with developmental dysphasia (Tallal & Piercy, 1974, 1975; Stark & Tallal ,1988, cited in Korpilahti, 1995). MMN has been used as an objective measure to investigate the processing abilities in dysphasic children. At the same time MMN studies have been used to investigate the mechanism underlying perceptual deficits in dysphasic children.

Downsen, Finley, Phillips and Lewy (1989) compared hemispheric asymmetries of speech related brain potentials of autistic, dysphasic and normal children in a situation where they attended to the signal. The stimuli consisted of clicks, speech and musical piano chords. In the developmental dysphasic an inherent dysfunction in the left hemisphere language area was found.

Korpilahti (1995) combined MMN and behavioural tests to examine auditory discrimination and memory function in school age children with SLI (developmental dysphasia). In the SLI group, the frequency MMN was weaker than the control

group. The tests of auditory short-term memory and rhythm were most problematic for the SLI children and these deficits correlated with the attenuated MMN in SLI children.

Korpilahti and Lang (1994) examined MMN in 14 dysphasic children and 12 normal children in the age range of 7 - 13 years. A frequency deviance of 500/553 Hz and two duration deviance of 50/110 msec and 50/500 msec were used to elicit MMN. In the dysphasic group the peak amplitude of the frequency MMN was significantly attenuated. The duration MMN showed a significant difference between the two groups only for stimuli with highly contrasting values (50/500 msec). No negative correlation between the peak latency and age was found in the dysphasic group as was observed for the control group. The MMN for frequency deviance lateralization to the right hemisphere in the normal subjects. Whereas in the dysphasic group the individual maximum values were often recorded in the left hemisphere. For duration deviance, in duration (50/110 msec), no hemispherical asymmetry was found in the control as well as dysphasic group. In duration II condition (50 Vs.500 msec), 50% of normal and 36% of dysphasic showed a trend to right hemisphere dominance. The left hemisphere dominance in duration II condition was 25% for normal subjects and 14% for dysphasic. Holopainen (1997, cited in Cheour et al., 2001) replicated these results in 3-7 years old dysphasic children. They also found that the amplitude of the frequency change MMN (500 Hz Vs 533 Hz) was significantly attenuated in dysphasic children as compared to their healthy controls.

From the above studies it can be concluded that 'MMN' holds a promise in identifying the auditory processing problem in children with SLI. The MMN for

frequency deviance has repeatedly been reported to differentiate children with SLI as a group from the normal group. The measure of amplitude of MMN is found to be of more diagnostic value than the latency measure of MMN.

MMN in children with learning disability (LD)

Almost 10% of children exhibit learning and reading problems (Torgeson, 1991, cited in Kraus, 2001). In the literature there are many studies demonstrating that children with LD have auditory and visual processing problems (Larsen, Foger & Sowell, 1976; Kraus & McGee, 1994, cited in Kraus, 2001). MMN has been used to investigate the auditory processing problems, usually reported to be associated with LD.

Kraus et al., (1996) found significantly reduced MMN duration in fourteen learning disabled children as compared to controls. They did not find any differences in the latency component.

Sreevidya (2001) also reported reduced MMN duration in three out the eight dyslexic children. In the remaining five subjects MMN could not be elicited.

Radhika (1998) investigated MMN for frequency deviance in dysplexic children. She reported absent MMN in three out of the twelve LDs that she evaluated. Also, the duration of MMN in LDs was found to be drastically reduced to 26-50 msec while it was 60-75 msec in normals. The reduced MMN duration was attributed to the incomplete attention processes. In her study the general morphology revealed a clear reduction in amplitude in dyslexics.

Radhika (1998), Guruprasd (2000) and Srividya (2001) reported prolonged latencies in the proportion of LDs subject in which MMN could be elicited.

From the above studies, it is notable that a significant proportion of children with learning disability do no elicit MMN, and those who do, have a reduced amplitudes at later latencies.

MMN in adult aphasics

A number of studies suggest that the presence of MMN may be related to an individual's ability to discriminate auditory stimulus differences as well as be related to their language abilities.

Auther, Wertz, Miller and Kirshner (2000) evaluated MMN response to speech stimuli in aphasic adults to determine the relationship among aphasic patients auditory comprehension, site of lesions and the presence or absence of MMN. The results showed that presence of MMN response was significantly related to auditory comprehension performance. Poor comprehension and absence of MMN was related to a lesion in the temporal lobe, while good comprehension and the presence of MMN were related to lesion that spared the temporal lobe. Though the magnitude of this relationship was not perfect, it was concluded by the authors that MMN can be an index of auditory comprehension and its relationship to the site of lesion.

Wertz, Auther, Burch-Sims, Abou-Khalil, Krishner and Duncan (1998) evaluated the MMN in normal and aphasic adults to tone and speech stimuli to determine auditory discrimination and relationship between MMN measures and

severity of aphasia. MMN were present in 89% of the normal subjects and 79% of the aphasic subjects to tone stimuli. However, MMN were present in 100% of normal subjects and only in 54% of the aphasic subject to speech stimuli. The duration of MMN to speech stimuli was significantly related to the severity of aphasia.

Csepe, Osman-Sagi, Molnar and Gosy (2001) investigated MMN elicited by tones, vowels, voicing stop contrast and place of articulation contrast in four aphasic patients and matched controls. An extensive Neuro physiological investigation was performed to highlight the assumed dissociation and possible interaction between impaired acoustic/phonetic perception and deficient comprehension in aphasic. The MMN elicited by consonant contrasts was the most vulnerable in aphasics and was correlated with patient's performance shown in behavioural phonemic discrimination task.

The above-cited studies imply that the MMN techniques are a promising method for investigating automatic sensory processes underlying auditory perception in aphasics.

MMN as a measure of memory trace

A part of clinically oriented MMN research is based on the fact that the MMN is the outcome of comparison of incoming signal against the memory trace built up by the frequently repeated stimuli (Csepe & Molnar, 1997).

The correlative measure of the MMN appearance, magnitude and strength of memory trace gave a big impetus to those studies in which patients whose sensory

memory was assumed to be impaired. Pekkonen, Jousmdki, Partanen and Karhu (1993) measured pitch and duration MMN at different Inter Stimulus Interval (ISI) as a function of age. They concluded that while the automatic stimulus comparison process was not affected by aging, the functional limits of the trace were influenced. They hypothesised that this may lead to processing problems such as involuntary attention switching and becoming less sensitive with age.

In another study by Woods (1992) significant changes in MMN distribution were found in ageing subjects when compared to young subjects which probably was due to deterioration in their memory capacity.

MMN in Parkinson's disease

In Parkinson's disease, an impaired change detection was supposed in general. A study done by Pekkonen, Rinne and Naatanen (1995) revealed that the pitch MMN was smaller in non-demented patients with Parkinson's disease than in age-matched controls. In this study, the MMN area was measured and the MMN attenuation was interpreted as a consequence of dopamine deficiency in these patients.

In an another study by Karayanidis, Andrews, Ward and Midice (1995, cited in Jose, 1999) showed that MMN amplitude reduces in Parkinson's disease. They found that MMN, among other components such as P_{3a}, P_{3b} and N_{2b} showed a conspicuous amplitude reduction with age and further attenuation due to Parkinson's disease. They also noted that the later, the late part of MMN, 'called Nd' showed a significant increase. These results are in agreement with the result of Vieregge, Verlegner, Wascher, Stuven and Kompp (1994, cited in Jose, 1999) which provided

evidence for a distinctive impairment of controlled processing, that is a disturbed auditory selective attention, as revealed by a significantly smaller processing negativity and unchanged P_{300}.

MMN in Schizophrenia

To understand the neurophysiological deficits in schizophrenia MMN was used to find whether the neuro cognitive function is so pervasive that it extends even to the level of perceptual processing of auditory events. (Csepe & Molnar, 1997).

In a study by Javitt, Doneshka, Grochowski and Ketler (1995) on medicated and nonmedicated schizophrenics, MMN and P300 were measured in passive and active modes. It was found that the MMN was severely impaired in both the groups of schizophrenics. The amplitude reduction of MMN was similar in medicated and non-medicated groups. Oades, Zerbin, Dittman-Balecr (1996) studies scalp distribution of pitch deviation elicited MMN in paranoid schizophrenics, non-paranoid schizophrenics and found that in paranoid patients, MMN was preferentially distributed on fronto central sites, whereas in non paranoids the distribution site was more towards the partial lobe. O'Donell et al., (1994) did not find any differences in pitch deviation elicited MMN between normal subjects and medicated schizophrenia patients.

Though these studies are unequivocal, it may be concluded that impairment or lack of MMN generation in schizophrenics may contribute to the observed disturbance in shifting attention towards novel stimuli or inadequate processing of relevant versus irrelevant stimuli (Csepe & Molnar, 1997). However, it is not fully

clear yet which components of processing reflected by MMN are assumed to be characteristics of schizophrenia.

MMN in Cochlear Implant Users

In recent years ERPs studies are being done to investigate the auditory perception changes brought about by the direct stimulation of the auditory nerve through cochlear implants. Though the mode of stimulation in cochlear implantees has been the electrical pulses, the resultant stimulation is hypothesized to be the same as that of acoustic stimuli. Trautwein, Ponton, Wong, Waring (1998, cited in Ponton et al 2000) found that the neurophysiological MMN threshold and psychophysical differential thresholds in adult cochlear implant users are comparable.

Ponton, et al., (2000) compared the late obligatory Auditory Evoked Potentials (AEPs) and MMN in children with cochlear implants with normal hearing children. The result showed that although the morphology of the obligatory AEPs was substantially altered by the absence of N_1 peak, the MMN was robustly present in the group of implanted children who had good spoken language perception through their device. However, differences were found in scalp distribution of MMN between implanted and hearing children.

These findings suggest that MMN is a good measure of basic auditory processes necessary for the development of spoken skills in profoundly deaf adult and children who use cochlear implant.

To summarise, it is evident that the MMN has been useful in furthering scientific knowledge about the biological processes underlying auditory perception. It is however important to realise that MMN and behaviour responses to the same signal present different aspect of signal processing. The former is preattentive and neurologic while the latter involves conscious integration of perceptual information. Where the processes underlying the MMN and conscious perception intersects is not yet clear. It is presently unclear whether the MMN will be sufficiently reliable to be used in the clinical application involving individual subjects.

METHOD

The present study was undertaken to investigate the cortical evoked response potentials (ERPs), namely, LLR and MMN in children with SLI.

Subjects

Two groups of subjects were evaluated in the present study, an experimental group and a control group.

Experimental Group : Ten diagnosed cases of SLI in the age range of 3-8 years were selected for the present study. They were diagnosed by qualified speech and language pathologist. The distribution of children in terms of age and sex is given in table 1.

Table 1: Age and sex wise distribution of the experimental and the control group

Age in years	Males	Females
3 - 4	2	0
4 - 5	2	1
5 - 6	2	1
6 - 7	1	0
7 - 8	0	1
Total	7	3

The criteria for selection of the experimental group was:

IQ within the range of 85-100 as tested by a psychologist.

No evidence of neuromuscular disability.

No evidence of emotional disturbance.

No complaint of hearing problem and history of ear discharge.

No structural anomalies in the head and neck regions.

Predominantly right hand user.

Pure tone air conduction threshold within 15 dBHL at octaves between 250Hz-8kHz in both ears.

Immittance audiometry results as 'A' type tympnogram for probe-tone of 226 Hz and ART (Acoustic reflex threshold) at 80 dB and below for .5, 1.2 & 4kHz.

Control group : Age and sex matched normal developing children were selected from neighbouring preschool and primary school.

Criteria for selection of control group was:

Language level at par with age as reported by the teacher.

Average or above average performance in class, as reported by their respective teacher.

No difficulty in reading and writing as reported by the teacher.

IQ within normal range.

No evidences of neuromuscular disability, emotional disturbances, hearing problem and structural anomalies in the head and neck region.

Predominantly right hand user.

Pure tone A.C threshold within 15 dBHL in both ears at octaves in the frequency range of 250 Hz - 8 kHz.

Normal immittance audiometry results in both ears, as described for the experimental group.

Instrument

The following instruments were used for the study.

Madsen OB 922 audiometer calibrated to ANSI (1996) standard was used for hearing screening .

GSI -33, version- 2 immittance audiometer calibrated to ANSI (1996) was used to rule out middle ear pathology.

Nicolet Bravo (Version II) with P-300 software was used for MMN recording.

Procedure

Pure tone audiometry followed by immitance audiometry was done in a sound treated room with the ambient noise levels within permissible limits (ANSI, 1991). After screening the children for clinical normal hearing and normal middle ear status, evoked potential recordings were done. MMN recording was done in a sound attenuated, electrically shielded room. TDH 39 earphones with MX-41/AR ear cushions were used to deliver the stimulus. Silver coated disc type electrodes were used for recording electrical evoked potentials. The following protocol was used for MMN recordings:

Electrode montage 3-channel recording was done.

Cz, C_3, C_4	Non inverting. (10-20 international recording system, Jasper, 1958)
M_1 & M_2 (Linked)	Inverting
Fz	Common

Stimulus parameters

Type	Tone burst
Polarity	Alternating
Frequency	1000 Hz
Intensity	60 dB
Rate	1.9/sec
Stimulus	Gated
Trigger	Inter
Ear	Right ear stimulation (for both standard and deviant)
Sweeps	500
Standard stimulus	50 msec
Plateau	30 msec
Ramp (rise/fall time)	10 msec
Deviant Stimulus	55 msec. 60 msec. 65 msec (in three different paradigms)
Plateau	35, 40 & 45 msec (for three different paradigms)
Ramp	10 msec (Constant)
Probability of deviant stimuli	20%

Acquisition Parameters

Analysis time	350 msec
Delay	50 msec (Prestimulus baseline)
Sns	100
Filter setting	0.1 - 30 Hz

Premeasurement procedure

All the subjects were seated comfortably in a reclining chair. They were instructed not to make gross body or head movements. They were also instructed not to pay attention to the auditory stimuli. A silent animated video was played for them to be awake and alert during the testing session, which lasted for 30 to 45 minutes.

Recording procedure

Electrode sites were prepared by cleaning the predetermined site with cotton dipped rectified spirit. Skin abrasive gel was gently scrubbed to increase the conductivity of the surface. Silver coated disc electrodes smeared with water-soluble conducting gel were placed on the prepared site. The electrodes were secured with adhesive tape, recommended for medical use. The absolute electrode impedance and inter electrode impedance was maintained below 10,000 ohms and 3,000 ohms respectively. The earphones were placed carefully not to dislodge the electrodes. The electrodes lead and electrode box was kept away from earphone to minimise artefacts. The stimuli were rooted to the right earphone. Three recordings of 500 sweeps each were obtained on every child for the duration deviance of 5, 10 and 15 msec. The standard stimulus had a duration of 50 msec. The deviant stimuli in the three different paradigm had a duration of 55, 60 and 65 msec respectively. The deviance was in terms of the duration of plateau i.e. 35, 40 and 45 msec respectively, while the ramp was kept constant (10msec).

Analysis of waveform

For each channel two waveform were obtained, one for the frequent and another for the infrequent stimuli.

The waveforms were analysed through visual inspection. Peaks and peak complexes of P_{100} and N_{250} were identified in both frequent and infrequent waveforms.

The MMN was obtained by subtracting the response of frequent (standard) stimulus from the response of infrequent (deviant) stimulus. The amplitude of the subtracted waveform (MMN peak amplitude) was obtained with reference to prestimulus voltage reference line. The following parameters of frequent, infrequent and subtracted waveforms were used for group comparison :

Wave morphology of LLR waveform

Latency of peaks of P_{100} and N_{250} in the frequent and the infrequent **waveforms.**

Amplitude of peak of P_{100} - N_{250} complex in the frequent and the infrequent waveform.

Peak latency of MMN.

Absolute onset latency of MMN

Peak amplitude of MMN with references to the prestimulus reference voltage line.

Total duration of MMN. This was calculated from the onset of MMN to its offset in the following positive peak.

Within group comparison was also done to determine the effect of magnitude of deviance, site of recording and the interaction of the two on the following MMN measures :

Peak latency of MMN

Absolute onset latency of MMN

Peak amplitude of MMN

RESULTS AND DISCUSSION

In the present study cortical potentials, namely, LLR and MMN for duration deviance were investigated in children with SLI. Late latency responses (LLR) and MMN responses obtained in children with SLI were compared with age and sex matched controls.

ERP wave forms were obtained using an odd ball paradigm for three duration deviances. Mean (M) and standard deviation (SD) of parameters of LLR wave forms of standards and deviant stimuli were compared using independent two tail 't' test. MMN obtained by substracting standard response from deviant was also compared for its peak amplitudes, peak latency, absolute onset latency and the total duration. Within group comparison were done to determine the effect of magnitude of deviance and site of recording. The scalp topography of MMN was also investigated within the groups.

The results are discussed under the following headings:

I. Comparison of LLR wave form between two groups: (analyzed using independent t-test)

Ia. Comparison of wave morphology.

Ib. Comparison of peak latency of P_{100} and N_{250}.

Ic. Comparison of peak amplitude of P_{100} - N_{250} wave complex.

II. Within group comparison of MMN : (analyzed using 2 way ANOVA)

IIa. MMN peak latency for three magnitude of deviance and site of recording,

IIb. Absolute onset latency of MMN for three magnitude of deviance and site of recording.

IIc. MMN peak amplitude for three magnitude of deviance and site of recording.

III. Comparison of MMN between two groups : (analyzed using dependent t-test)

IIIa. Comparison of peal latency.

IIIb. Comparison of absolute onset latency of MMN

IIIc. Comparison of total duration of MMN

IIId. Comparison of peak amplitude.

I. COMPARISON OF LLR WAVE FORM BETWEEN GROUPS

Ia. Comparison of Wave Morphology

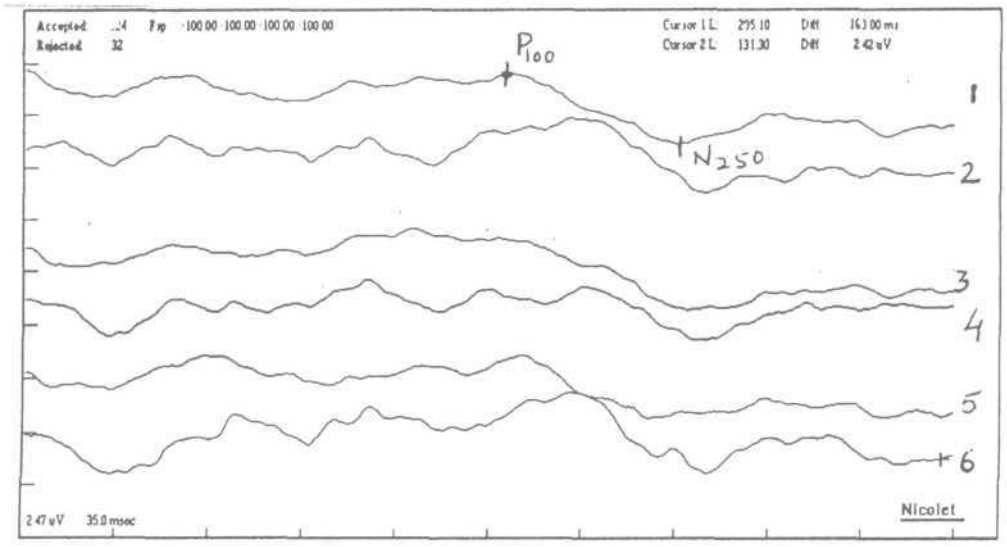
The wave morphology of LLR in the language impaired group was found to be poor as shown in figure 1 as compared to that of the control group (figure 2). P_{100} & N_{250} peaks, though present in all the SLI subjects, were not readily identifiable in majority of these subjects. Only three subjects elicited a clear LLR wave form. A group of researches believe that learning disability and SLI are part of the same continum (Leonard, 1997; Bishop & Adams, 1990; Catts, 1993). Leonard (1997) postulated that SLI and reading disorder are spoken and written manifestation of the same impairment, on the bases that in both the disorders the primary deficit lies in the higher order processing. Hence, in the present study findings on learning disabled population are used to draw inference about the SLI group.

Radhika (1998) reported getting poor wave morphology in seven out of twelve learning disabled children she tested.This poor morphology of LLR wave reflects a clearer maturational lag of the supra temporal region, reticular formation, hippocampus region, Heschel's gyrus and associated areas which are found to be the major generator site of LLR potentials (Ritter, Simon & Vaughan, 1983). A poor morphology indicates poor neural representation of the stimuli (Kraus, 2001).

The wave morphology in three children, where it was clearer was similar to that obtained in control group which had a large positivity peaking at about 100 msec (range 98-148 msec). This positivity was followed by a prominent negativity peaking between 160-260 msec, as shown in figure2. Korpilahti and Lang (1994), Shafer et al., (2000) and Ponton et al., (1998) also support getting such LLR in peadiatric group. An adult like frontocentral N_1 is not consistently present until after 9 years of age (Ponton et al., 2000). Korpilahti and Lang (1994) suggested that the N_1 wave is known to fade quickly when the ISI is shortened. This applies especially to non specific component of N_1 and also to its superatemporal component (Naatanen, 1988.

Korpilahti & Lang (1994) reported that a short ISI elicits in children quite a strong potentials. A positive component with the latency of 100 msec and a negativity peaking at 250 msec, seemed to 'invert' the polarity of the ERP wave form as compared to adults. They further suggested that it is possible that such ERP wave form results from prolonged latency and slower habiluation of N| in children. Csepe, Dieckmann, Hoke & Ross (1992, as cited in Shafer et al., 2000) found that a mature ERP wave structure occurred only in children over 10 years. No Ni wave was recordable in children under 8 years.

Figure 1 : Representative LLR wave forms for the deviance of 5 msec at three electrode site in the SLI group.

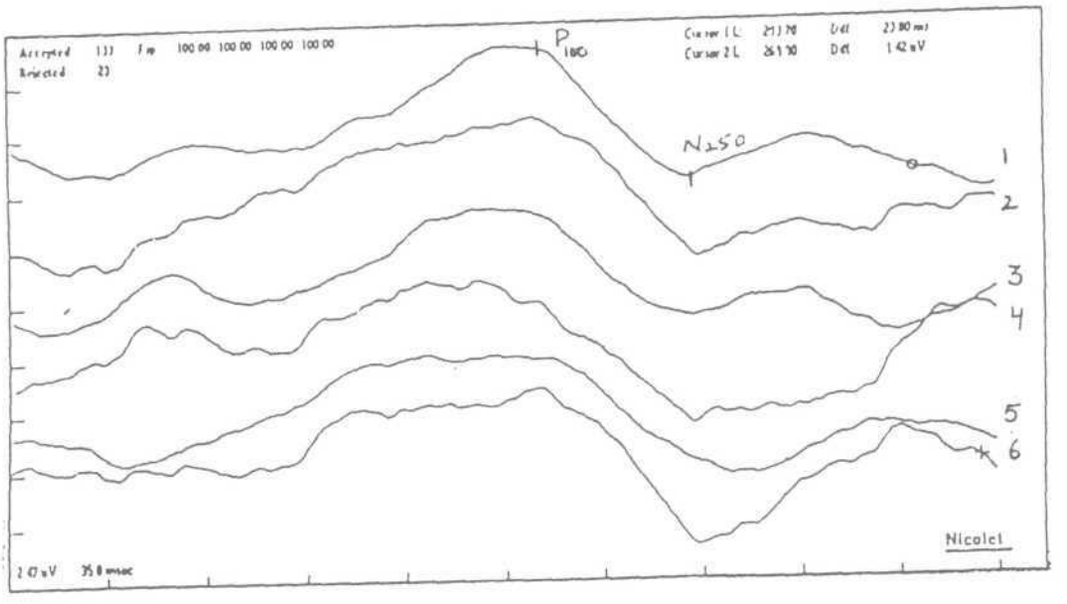


Cz
- 1. Standard wave
- 2. Deviant Wave

C3
- 3. Standard wave
- 4. Deviant Wave

C4
- 5. Standard wave
- 6. Deviant Wave

Figure 2 : Representative LLR wave forms for the deviance of 5 msec at three electrode site in control group.



Cz
1. Standard wave
2. Deviant Wave

C3
3. Standard wave
4. Deviant Wave

C4
5. Standard wave
6. Deviant Wave

Ib. Comparison of Peak Latency of P_{100}

Table 2 depicts the mean, SD and significance of difference of the P_{100} peak latency between the normals and SLI group. P_{100} latency was compared for both the standard and deviant wave forms. As can be seen, at none of the sites and for none of deviances, there exists a significant difference between the two groups. This implies that latency measure of P_{100} did not differentiate the two groups. Similar findings were also reported by Korpilahti and Lang (1994) who used a larger duration deviance and reported that P_{100} did not differentiate the SLI group from normals.

Arehole (1995) also found no significant difference in P_{100} measure between normals and learning disabled group. Korpilahti and Lang (1994) considered P_{100} as an exogenous potential and this could have been the reason that no group difference for the latency component of P_{100} could be found.

Comparison of Peak Latency of N_{250}

Table 3 indicates the comparison of the peak latency of N_{250} in control and SLI group. Both standard an deviant wave forms were compared. Although 90% of the times the latency of N_{250} of standard as well as deviant were longer in the SLI group, the difference was not statistically significant. Korpilahti and Lang (1994) found that N250 peak latency for the standard stimuli was significantly longer in the dysphasic children for the larger duration deviance (50 msec/500 msec). This difference disappeared for shorter duration deviances (50 m.sec/110msec). In the present study, perhaps, the difference has not been noticed because the deviance was much smaller than that used by the above authors.

Radhika (1998) observed that latency of N_2 (here referred to as N_{250} wave was normal in ten LD children and delayed in two of the children as compared to the control group. According to Ritter, Deacon, Gomes, Javitt and Vaughan (1995) N_2 response is associated with processing of stimuli irrespective of its complexity. However N_2 is influenced by the physical dimension of stimuli related to automatic processing. The prolonged latency of the N_{250} indicated slower processing in the central auditory pathways of the language impaired children, possibly explained by the their delayed maturation.

Table 2 : Comparison of latency of P_{100} in msec between the control and the SLI group at the three recording sites for three deviances.

Deviance	Group	C_2 site						C_3 site						C_4 site					
		Standard			Deviant			Standard			Deviant			Standard			Deviant		
		Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'
5 msec, 50 Vs 55	Control	122	13.9	0.048 NS	128	16	0.098 NS	126	33	-.427 NS	134	22	0.865 NS	130	26	0.641 NS	130	22	0.463 NS
	SLI	122	13.8		127	21		132	19		125	23		123	15		126	20	
10 msec, 50 Vs 60	Control	126	12	-0.910 NS	129	12	-.198 NS	124	22	-1.129 NS	123	16	-1.813 NS	125	17	-.731 NS	125	15	-1.703 NS
	SLI	133	20		131	12		135	20		136	15		132	17		138	19	
15 msec, 50 Vs 65	Control	120	21	0.766 NS	116	17	-1.28 NS	123	19	-0.117 NS	118	21	-1.620 NS	124	20	0.754 NS	129	30	-0.247 NS
	SLI	114	11		127	21		124	12		133	19		117	20		132	25	

NS = Not significant

Table 3 : Comparison of latency of N_{250} in msec between the control and the SLI group at three recording sites for three deviances.

Deviance	Group	C_2 site						C_3 site						C_4 site					
		Standard			Deviant			Standard			Deviant			Standard			Deviant		
		Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'
5 msec, 50 Vs 55	Control	230	21	-0.062 NS	225	26	-0.495 NS	223	49	-0.193 NS	226	23	-0.597 NS	223	52	-0.355 NS	226	20	-0.959 NS
	SLI	231	34		231	23		226	28		232	28		230	32		235	21	
10 msec, 50 Vs 60	Control	230	33	0.147 NS	237	35	0.247 NS	239	27	1.256 NS	232	33	-0.165 NS	238	23	1.068 NS	232	32	-0.303 NS
	SLI	232	33		233	30		241	36		234	36		234	35		237	32	
15 msec, 50 Vs 65	Control	232	19	1.929 NS	222	15	-0.841 NS	225	21	0.665 NS	228	17	0.174 NS	220	21	0.452 NS	227	19	-0.641 NS
	SLI	235	20		228	17		228	23		229	19		225	23		232	20	

NS - Not significant

Comparison of P_{100}-N_{250} Complex Amplitude:

From table 4, it is seen that the amplitude of P_{100} - N_{250}, complex is significantly attenuated in the SLI group. The attenuation is seen in both the standard and the deviant wave forms. This implies that the amplitude measure of P_{100} - N_{250} complex is a reliable parameter to differentiate the two groups.

Several studies have reported decreased absolute amplitude of LLR peaks in the learning disorder population. Mason and Mallor (1984), Scatterfiled, et al., (1984), Brunswick and Rappen (1994), Duncan, Ramsey, Wilkins, Hamburger and Odou-Potkin (1994), Radhika (1998) and Gurprasad (2000) reported attenuated amplitude of N_{100} and N_{250} . The reduced amplitude of P_{100} - N_{250} complex indicate poor auditory processing owing to diminished neural synchrony and weakened neural connections in the auditory cortex (Kraus et al., 1994 ; Kraus, 2001).

Table 4 : Comparison of amplitude of P_{100} - N_{250} complex in µV between the control and the SLI group at three recording sites for three deviancies.

Deviance	Group	C_2 site						C_3 site						C_4 site					
		Standard			Deviant			Standard			Deviant			Standard			Deviant		
		Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'	Mean	SD	't'
5 msec, 50 Vs 55	Control	4.2	1.2	2.87**	6.3	2.1	3.6**	5.4	2.0	2.87**	5.2	1.4	3.6**	5.4	2.0	2.61**	4.5	1.0	2.19*
	SLI	2.9	0.8		3.6	1.2		3.1	1.6		3.3	1.0		3.4	1.3		3.7	3.7	
10 msec, 50 Vs 60	Control	5.2	1.8	2.87**	7.8	2.1	3.89**	5.3	1.4	4.84**	7.5	1.9	3.79**	4.6	1.7	2.75**	7.1	2.0	3.41**
	SLI	3.2	1.0		4.8	1.5		2.8	0.7		4.6	1.4		2.9	0.8		2.9	1.3	
15 msec, 50 Vs 65	Control	6.2	1.8	3.15**	8.0	2.2	3.36**	7.2	1.8	1.73*	7.8	1.7	3.23*	5.1	1.5	2.11*	7.4	1.3	3.3*
	SLI	4.1	1.2		5.3	1.2		4.0	1.2		5.4	1.4		3.7	0.8		5.4	1.3	

* Significant at 0.05 level

** Significant at 0.01 level

II. WITH IN GROUP COMPARISON OF MMN

*II*a. MMN Peak Latency

Two way ANOVA was conducted on both the groups separately to determine the interaction of maginitute of deviance and site of recording on peak latency of MMN. It was found that in both the SLI and control groups, recording site, magnitude of deviance and the interaction of the two did not have any significant effect on peak latency. However, it was noticed that in both the groups the peak latency was comparatively shorter for the deviance of 15 msec than that for the 5 msec.

IIb. Absolute Onset Latency of MMN

Two way ANOVA was conducted to analyse the effect of magnitude of deviance and site of recording on the onset latency of MMN. As with the MMN peak latency, it was found that in both the SLI and the control group the two independent variable and their interaction did not have any significant effect on the absolute onset latency of MMN.

IIc. MMN Peak Amplitude

Two way ANOVA (Table 5) was conducted in both the groups separately to determine the interaction of recording sites and magnitude of deviance on the MMN peak amplitude. In both the groups it was observed that peak amplitude was significantly affected by electrode site, magnitude of deviance and the interaction of the two.

Table 5 : Two way ANOVA for interaction of deviance and recording site on MMN peak amplitude.

Independent variables	Control group		SLI group	
	F ratio	P value	F ratio	P value
Deviance	34.09	0.00	22.80	0.00
Recording site	13.28	0.00	12.27	0.00
Deviance X site	16.55	0.00	10.55	0.00

Table 6 : Duncan's post hoc analysis, effect of magnitude of deviance on peak amplitude in the control and the SLI group.

Group	Deviance	Peak amplitude
Control	5	-0.39
	10	-2.2
	15	-2.9
SLI	5	-0.057
	10	-1.3
	15	-1.8

Effect of magnitude of deviance on peak amplitude

Duncan's Post hoc anlaysis (Table 6) revealed that in both the groups the deviance of 15 msec, followed by 10 msec and 5 msec affected the amplitude in the given order. It is evident that there was a significant decrease in peak amplitude with the decrease in magnitude of deviance (Table 6). Sreevidya (2001) also reported a positive correlation between the magnitude of deviance and the peak amplitude.

Picton et al., (2000) noted that generally, the larger the acoustic differences, the larger is the MMN, although there may be ceiling effect in amplitude with large differences. In the present study comparatively smaller deviances were used and hence a positive relation between the magnitude of deviance and the MMN amplitude was observed.

Effect of electrode site on peak amplitude

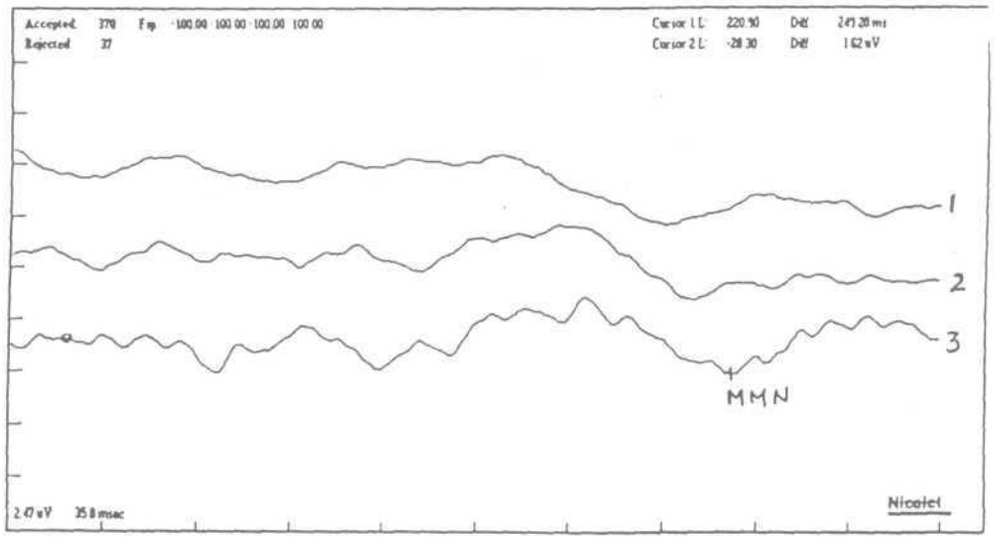
In the control group, results of the post hoc analysis revealed that the amplitude was maximum at the site C_3 followed by Cz. The amplitude was the least at the C_4 site. The same order of affect was noticed for the SLI group. This implies that MMN lateralized towards left side in the control as well as in the SLI group.

These findings are contradictary to the one reported by Korpilahti and Lang (1994) where in the normals and SLI group, the MMN for duration deviance was recorded more centrally. Downson et al., (1989) reported a right sided assymetry in dysphasics for speech evoked MMN which was reverse of what was characteristric of normal group.

Research dealing with scalp topography of MMN suggests that MMN elicited by tone is larger over the right hemisphere (Korpilahti & Lang , 1994; Csepe, 1994). In the present study it was found that in the control as well as in SLI group, MMN for change in duration of tones was distributed more towards the left hemisphere.

In the present study left side preference for processing of duration change in pure tones can be expalined on the basis of scalp-current density (SCD) maps reported by Giard et al, (1990). These authors recorded MMN to frequency changes and the SCD analysis suggested that the activity of auditory cortex in the hemisphere contralateral to stimulated ear contributed more strongly to the MMN than did the ipsilateral auditory cortex activity. In the present study the stimuli were rooted to the right ear and therefore it is possible that contralateral activity contributed more to the MMN amplitude and hence the left hemispherical dominance was seen (i.e. higher amplitude at C3). Further, no hemispherical difference between the normal and language impaired group was seen.

Figure 3 : Representative wave forms depicting MMN for deviance of 5 msec at Cz in the SLI group

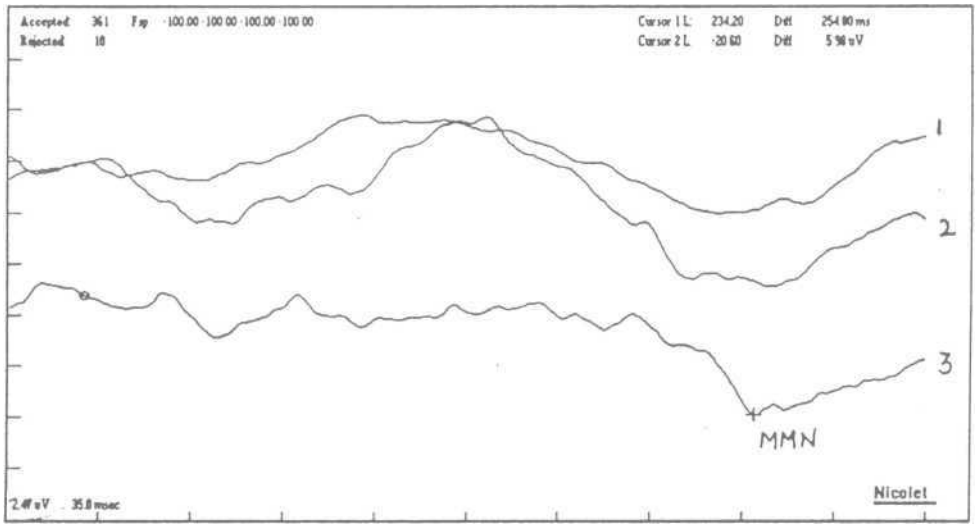


1. Standard wave

2. Deviant wave

3. Difference wave

Figure 4 : Representative wave forms depicting MMN for deviance of 5 msec at Cz in the control group



1. Standard wave
2. Deviant wave
3. Difference wave

III. COMPARISON OF MMN BETWEEN THE TWO GROUPS

The responses of standard wave form were subtracted from the responses of deviant to get the MMN. The peak latency, absolute onset latency, peak amplitude and duration of MMN were compared in control and SLI groups.

Table 7 : Comparison of MMN peak latency in msec between in the control and the SLI group at three recording sites for three deviances.

Deviance	Group	C_2			C_3			C_4		
		Mean	SD	't'	Mean	SD	't'	Mean	SD	't'
5 msec, 50 Vs 65	Control	236	35	0.124 NS .	224	30	0.375 NS	230	32	0.184 NS
	SLI	238	50		216	46		234	47	
10 msec, 50 Vs 60	Control	239	28	0.042 NS	235	35	0.043 NS	232	33	0.015 NS
	SLI	239	56		234	45		239	43	
15 msec, 50 Vs 65	Control	230	34	0.198 NS	218	34	-0.244 NS	228	33	0.1.5 INS
	SLI	227	53		222	39		235	56	

III a. Comparison Of Peak Latency Of MMN

Table 7 depicts the comparison of peak latency of MMN in control and the SLI group. No significant difference between the two groups was found. The SLI group had a higher SD which probably resulted in overlap in the latency of the two groups leading to no significance difference between the groups.

Korpilahti and Lang (1994), Korpilahti (1995) and Holopainen (1997, cited in Cheour, 2001) reported that peak latency measure of MMN did not differentiate the language impaired children from normal. Korpilahti (1995) postulated that MMN latencies are suggested to yield information about the mental speed of auditory encoding. From the findings of MMN studies it can concluded that the speed of encoding in SLI group is usually not different from the normal group. However it

should be remembered that MMN is a preattentive neurophysiological response and it does not involve conscious integration of perceptual information. It might be possible that in children with SLL, the preattentive processes involved in speed of mental encoding are normal but there is deficit in the processes involved at conscious level which often reflect in their behaviroural responses.

Table 8 : Comparison of absolute onset latency of MMN in msec between the control and the SLI group at three recording site for three deviances

Deviance	Group	C_2			C_3			C_4		
		Mean	SD	't'	Mean	SD	't'	Mean	SD	't'
5 msec, 50 Vs 55	Control	179	36	-1.707 (NS)	182	28	-1.073 (NS)	187	31	-0.579 (NS)
	SLI	199	25		198	27		197	31	
10 msec, 50 Vs 60	Control	174	32	-2.02 (NS)	180	35	-2.27 (NS)	180	35	-2.64 (NS)
	SLI	197	29		198	31		190	35	
15 msec, 50 Vs 65	Control	165	27	-1.387 (NS)	180	29	-0.791 (NS)	177	36	-0.006 (NS)
	SLI	195	36		192	37		182	42	

IIIb. Comparison of absolute onset latency of MMN

The comparison of two groups revealed that the absolute onset latency of MMN in SLI group was always later than the control group. The differences were however not statistically significant (table 8). Sreevidya (2001) also did not find any difference in onset latency of MMN between LD and the control groups.

IIIc. Comparison of total duration of MMN

Total duration of MMN is the time span from its onset in the preceeding peak to its offset in the following positive peak. In the SLI group a clear offset of MMN could be seen but in the control group the spread of MMN continued beyond the

analysis window (Fig. 4). Therefore, the two groups could not be compared statistically. However, this clearly indicates that the total duration of the MMN was larger in the control group than compared to the SLI group.

Sreevidya (2001), Radhika (1998) also reported decreased total duration of MMN in LD children.

Korpilahti and Lang, 1991 as cited in Radhika 1998 suggested that MMN thought to be measuring subliminal attention and its reduction shows a poor or low subliminal attention in SLI group.

In the present study, because of the time constraints the analysis window had to be restricted for the duration of 350 msec (50 msec prestimulus). A longer analysis window would have resulted in decrease in rate of stimulus presentation, thereby increasing the time of each recording. In that case maintaining child's attention till the completion of three recordings would had been a difficult task.

Table 9 : Comparison of MMN peak amplitude in µv between in the control and the SLI group at three recording sites for three deviances.

Deviance	Group	C_2			C_3			C_4		
		Mean	SD	't'	Mean	SD	't'	Mean	SD	't'
5 msec, 50 Vs 55	Control	-2.3	0.56	-4.133*	-1.4	0.79	-0.015 NS	-2.5	2.3	-1.21 NS
	SLI	-1.2	0.36		-1.4	0.47		-1.2	0.1	
10 msec, 50 Vs 60	Control	-2.3	1.0	-2.87**	-2.06	0.83	-1.537 NS	-2.2	1.2	-2.3 NS
	SLI	-1.2	0.6		-1.5	0.79		-1.1	0.7	
15 msec, 50 Vs 65	Control	-2.9	1.0	-2.62**	-2.7	0.83	-2.68**	-3.2	1.2	-2.57**
	SLI	-1.8	0.75		-1.8	0.63		-2.0	0.56	

NS - Not significant
* - Significant at 0.05 level
** - Significant at 0.01 level

IIIc. Comparison of Peak Amplitude of MMN.

From table5 and figure 3 and 4, it is seen that MMN peak amplitude is significantly attenuated in the SLI group. The difference was consistently significant at the Cz site whereas for C_3 and C_4 site it is significant for larger magnitude of deviance.

Korpilahti and Lang (1994) reported statistically significant difference in MMN amplitude between the language impaired group and normals for larger deviance in duration (50 msec Vs 500 msec). The smaller deviance (50 msec Vs 110 msec) could not differentiate the two groups. Sreevidya (2001) reported significant difference in MMN amplitude between learning disorder children and normals for duration deviances of 5 & 10 msec.

Researchers have given three explanations which may account for the underlying problems which result in an attenuated amplitude and reduced duration in the MMN. These explanation are :

Weak automatic echoing memory **:** The basic assumption behind elicitation of MMN is that a frequently repeating stimulus forms a memory trace and a stimulus that does not match the memory trace evokes an extra activity. This extra activity is called MMN (Naatanen, 1990). Stronger the memory trace, larger is the MMN amplitude and shorter is its latency (Ritter et al., 1995).

In children with SLI, it is assumed that either the memory traces are not strong enough or they fade away quickly (Korpilahti and Lang 1994). With longer ISIs the memory trace tends to fade away and therefore MMN decreases when ISI is

increased (Mantysalo & Naatanen 1987, cited in Korpilahti & Lang, 1994; Naatanen et al., 1987, cited in Korpilahti & Lang, 1994). In the present study the effect of the ISI (450 msec) along with the relatively small temporal deviance together might have caused attenuation of MMN in the SLI group.

Deficit in temporal processing **:** Tallal and Piercy (1974, 1975) hypothesized that language impairment in developmental dysphasics is due to deficit in temporal processing. Children with SLI have problem processing fast occurring/changing events (Tallal et al., 1985). If the presentation rate of the stimulus is rapid, the preattentive auditory processing is impaired.

Poor listening skills **:** Another hypothesis proposed by Korpilahti and Lang (1994) is that the children with SLI may have poor listening skills i.e. decreased attention to auditory stimuli. This could be on account of underdevelopment of the automatic detection of a noval stimuli and / or due to poor passive attention switching. According to this view, it is assumed that the frontal subgenerators of MMN are also contributing to the reduced MMN in the SLI group.

In the present study the reduced duration of MMN in the SLI group probably could be due to one or all the above three reasons. The findings of the study indicate that they may have a poor automatic echoic memory, poor temporal processing as well as poor listening skills.

From the results of the present study it can be concluded that:

The latency measures of P_{100} and N_{250} in children with SLI were not significantly different from that in children with normal language development.

The amplitude of P_{100} - N_{250} wave complex was significantly attenuated in children with SLI.

The MMN peak amplitude was significantly reduced in children with SLI.

The total duration of MMN was reduced in the SLI group.

No significant difference in peak latency of MMN was found between the SLI and control group.

No difference in hemispherical lateralization of MMN was found between the two groups. In both the groups maximum amplitude of MMN was found on C3 site, implying that the MMN for duration deviance was lateralized towards the left hemisphere in both the groups.

SUMMARY AND CONCLUSION

Deficient auditory perception have been implicated in specific language impairment (Tallal, Stark & Mallets, 1985). Kraus et al., (1996) suggested that children with learning problems have difficulty in perceiving fundamental acoustic differences that originate from abnormalities in neurophysiological encoding of acoustic differences that occur after peripheral sensory encoding and before conscious perception. There is dearth of studies investigating MMN for duration deviance in SLI children and those which have been done have used larger duration deviances. It was assumed that studying MMN for smaller duration deviance would make a sensitive tool to differentiate children with SLI from normal children. Hence the study was undertaken.

In the present study cortical evoked potentials, namely, late latency response (LLR) and MMN were investigated in children with SLI. MMN for three duration deviances was used to investigate the auditory discriminatory ability of children with SLI. The results were compared with age and sex matched controls.

Ten children in the age range of 3-8 years who had been diagnosed as having SLI constituted the experimental group. Ten age and sex matched subject comprised the control group. Both the groups were screened for normal hearing sensitivity and middle ear function. Nicolet Bravo (Version II) with P-300 software was used for ERP recording. ERP wave forms were obtained using an 'odd ball' paradigm for three duration deviances of 5, 10 and 15 msec. The standard stimulus had a duration of 50

msec. The deviant stimuli in three different paradigm had a duration of 55, 60 and 65 msec respectively.

The LLR wave forms obtained for standard and deviant stimuli were compared between the groups for the following parameters:

Wave morphology of LLR

Latency of P_{100} and N_{250} in standard and deviant wave.

Amplitude of P_{100} - N_{250} wave complex in standard and deviant wave.

The MMN was obtained by subtracting the response of standard from the response of deviant stimuli. The SLI and control group were compared for the following parameters of MMN.

Peak latency of MMN

Peak amplitude of MMN

Total duration of MMN

Hemispherical lateralization of MMN

Within group comparison were also done to determine the affect of the magnitude of deviance and the recording site on MMN parameters.

The following conclusion are made based on the finding of the present study :

(i) The latency of P_{100} and N_{250} were not significantly different between the two groups,

(ii) The amplitude of P_{100} - N_{250} wave complex, in both standard and deviant LLR waves, was significantly attenuated in the SLI group.

(iii) Peak latency of MMN was not significantly different between the two groups,

(iv) Peak amplitude of MMN was significantly reduced in the SLI group,

(v) In both the groups MMN was lateralized towards the left hemisphere.

(vi) In both the groups the MMN amplitude was significantly affected by the magnitude of duration deviance. As the magnitude of deviance increased, the amplitude also increased.

Hence in the present study it was found that amplitude of P_{100} - N_{250} complex and MMN peak amplitude were significantly reduced in children with SLI. In clinical practice the diagnosis of SLI is done through subjective assessment tools. It is suggested that amplitude measures of P_{100} - N_{250} complex and MMN be used to predict problems due to auditory processing deficits. The findings of the present study may offer a new diagnostic method for early identification of SLI. Once the child is diagnosed as having auditory processing deficits, the course of his/her therapy can be designed accordingly.

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