AUDITORY EVOKED POTENTIALS IN CHILDREN WITH HISTORY OF OTITIS MEDIA WITH EFFUSION

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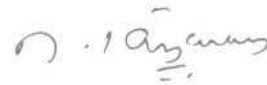MANASAGANGOTHRI, MYSORE - 570 006

MAY - 2002

DEDICATION

To My
Dearest Grandpa
& Brother

CERTIFICATE

This is to certify that the dissertation entitled **"AUDITORY EVOKED POTENTIALS IN CHILDREN WITH HISTORY OF OTITIS MEDIA WITH EFFUSION"** is the bonafide work in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student (Register No. M2K12).

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CERTIFICATE

This is to certify that the dissertation entitled **"AUDITORY EVOKED POTENTIALS IN CHILDREN WITH HISTORY OF OTITIS MEDIA WITH EFFUSION"** has been prepared under my supervision and guidance. It is also certified that this 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 **"AUDITORY EVOKED POTENTIALS IN CHILDREN WITH HISTORY OF OTITIS MEDIA WITH EFFUSION"** is the result of my own study under the guidance of **Dr. C.S. Vanaja,** Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any other University for the award of any Diploma or Degree.

Mysore,
May, 2002   Reg. No. M2K12

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INTRODUCTION

One area where modality - specific auditory perceptual deficits might be expected is in those conditions that produce auditory deprivation in early life, such as otitis media with effusion (OME). Otitis media with effusion is an inflammatory condition of the middle ear and mastoid air cell system characterized by accumulation of fluid in the middle ear without signs or symptoms of acute infection (Handler and Magardino, 2000). Hearing level associated with OME ranges from no threshold elevation to moderate degree of impairment (Bess, 1986, cited in Gravel & Ruben, 1996; Gravel, 1989, cited in Gravel & Ruben, 1996). While the configuration of the hearing level is typically characterized as equal (flat) across the speech frequency range, varying audiometric patterns may occur. Restricted auditory input during early life results in disordered emerging communication (expressive and comprehensive), higher order central auditory processing deficits, attention deficits, behavioral problem and ultimately below average academic achievements (Feagans, 1986, cited in Gravel & Ruben 1996). Experimental work, in both animals and humans, has shown anatomical abnormalities and electrophysiologic deficits in auditory processing as sequelae of OME in early life (Gravel and Ruben, 1996). Gravel and Ellis (1995, cited in Cacace and McFarland, 1998) putforth a hypothesis that chronic otitis media in humans could lead to verbally based learning disorders as

a result of reduced hearing sensitivity and /or from more complex deficits in auditory perception.

Although auditory- specific deficits may be anticipated, deficits observed in these children may not be restricted to a single sensory modality and may also manifest in gross-motor skills (Orlin, Effgen and Handler, 1997, cited in Cacace & McFarland, 1998). Zinkers Gottlieb and Schapiro (1978, cited in Cacace & McFarland, 1998) found significant deficits in a severe otitis media group with verbal tasks requiring mental arithmetic concept formation and auditory - sequential memory. However, they also found significant deficits in non-verbal tasks requiring visual sequencing and visual - motor coordination.

Electrophysiologic studies of children with and without histories of OME support an association between reduced auditory input in the early years and abnormal auditory brainstem development. The results of investigation of the auditory brainstem response (ABR) in such children are consistent with the interpretation that a history of early conductive hearing loss may be associated with some sort of abnormalities in auditory brainstem processing. Similarly, there can be possible abnormalities in the cortical structures due to deficient auditory input during early life. Such deficiencies in higher canters may lead to central auditory processing disorders (CAPD), which can be evaluated using higher potentials such as middle latency response (MLR), late latency response (LLR), mismatch negativity (MMN) and P300 Jerger (1992, cited in Cacace &

McFarland 1998) considers the area of CAPD in school aged children as a "very large terra incognita...". Although many tools are available to assess CAPD, very few are satisfactory for effective diagnosis.

Need for the study

It is evident from the review of literature that auditory deprivation in early developmental period due to OME may lead to CAPD. These children are shown to have abnormalities at the brainstem level as reflected by ABR and are also suspected to have higher level dysfunction. However, there is a dearth of studies evaluating both brainstem and cortical functioning in children with history of OME.

Aim of the study

The present study was designed to investigate CAPD in children with history of OME using ABR, LLR and MMN for intensity deviation.

REVIEW OF LITERATURE

There has been considerable speculation that children suffering from recurrent OME may be at risk of abnormal central auditory development because of sound deprivation. The hearing impairment due to conductive pathology need not be profound to effect a measurable central disability, since mild to moderate degree of hypacusis may be adequate to alter the higher auditory functional activities (Welsh, Welsh & Healy, 1983). Otitis media can have an adverse effect on the development of auditory processing abilities (Holm & Kunze, 1969; Jerger, Jerger, Alford, & Abrams, 1983; Teele, Klein, Chase, Menyuk & Rosener, 1990; Friel-Patti & Finitzo,1990). The age of onset, number of episodes and duration of otitis media has a direct relation to central auditory processing abilities (Teele, Klein & Rosner, 1984). Holm and Kunze (1969) found that auditory skills were significantly depressed in those children with a significant history of otitis media in early life. In a prospective study, Schilder and Colleagues (1994) found that children with a history of persistent otitis media at a preschool age showed only slight effects on their ability to discriminate speech in noise. However, the subjects, in this study received ventilation tubes on a routine basis if the episode of otitis media persisted for more than 2-3 months. A prolonged middle ear disease either unilateral or bilateral interferes with the normal acquisition of auditory skills (Welsh, Welsh & Healy, 1983).

The time of onset of hearing impairment also has a profound effect on the development of auditory behavior. During development, a period appears during which normal auditory input is crucial for a later development of optimal auditory function. The time occurring before the complete maturation of the organism has been termed a "Critical" or a "Sensitive" period for normal anatomical, physiological, and behavioral auditory development (Gravel & Ruben, 1996). In humans, the first three years of life are important for language development (Menyuk, 1996, cited in, Gravel & Ruben, 1996). Similarly various auditory processes have been reported to develop till early adulthood. The latency of ABR has been shown to stabilize by 18 months of age (Fria & Doyle, 1984). The MLR continues to mature till 12 years (Kraus, Smith, Reed, Stein & Cartee, 1985, cited in Kraus, McGee & Stein, 1994). The N1, P2 and N2 thought to stabilize by about 10-15 years of age (Kileny, 1985, cited in Hyde, 1997; Mason & Mellor, 1984,cited in Tonnquist-Uhien, 1996). Korpilahti and Lang (1994, cited in Shafer, Morr, Kreuzer, & Kurtzberg, 2000) found a significant negative correlation between MMN latency and age in children found 8 to 13 years of age. The P300 component shows a decrease in latency and an increase in amplitude from the age 5 through 16 (Goodin, Squires, & Henderson, 1978, cited in Tonnquist-Uhlen, 1996; Polich & Howard and Starr, 1985, cited in Jirsa and Clontz, 1990).

Although a direct relationship between OME and development of auditory/linguistic behavior has not been established unequivocally (Ventry, 1980; Paradise, 1983, cited in Pillsbury, Grose, & Hall, 1991), the results of a wide range of studies support some association between deficits in speech-language development, cognitive skill/learning abilities or auditory attention with recurrent OME. Welsh et al. (1983) concluded that there is a central disturbance of auditory perception in some children who have had recurrent middle ear in early childhood. Abnormalities in auditory processing persisted in their subjects even after hearing thresholds had returned to normal by middle ear surgery. Hence evaluation of central auditory processing disorders become crucial during early developmental stage. A number of behavioral and physiological test procedures are available for evaluation of central auditory nervous system (CANS). A brief review of these tests and their application in assessing children with OME is presented here.

Behavioral Tests

Behavioral tests are based on observation of overt responses to controlled auditory signals. These tests involve active participation for the subject during the assessment procedure.

These assessment produces employed towards identification of underlying deficit in central auditory processing must be able to tap a variety of central auditory mechanisms. The following section provides a comprehensive

summary of most commonly used behavioral test employed in identification of CAPD.

1. Monaural low-redundancy Tests

Monaural presentation involves the presentation of stimuli to a single ear at a time. In monaural low-redundancy test, the extrinsic redundancy is reduced by filtering and by time alterations such as compression or interruption.

Table. A : Summery of commonly used monoaural low- redundancy tests for the assessment of CAPD.

Test	Condition	Results
1. Low pass filtered speech (LPFS) test		
a. Bocca & Calearo (1963).	Temporal lobe tumor	Diminished clarity for speech contralateral to tumor.
b. Welsh, Welsh & Healy(1980).	Dyslexia	50-70% scores obtained.
c. Lynn & Gilroy (1977)	Temporal & parietal lobe lesions	Sensitivity & specificity of 74%
d. Karlsson & Rosenhall (1995)	1. Brainstem lesion 2. Brainstem vascular lesion 3. Temporal lobe lesions.	Sensitivity of 62%, 49% & 62%, respectively.
2. Synthetic sentence identification- Ipsilateral competing message (SSI-ICM)		
a. Jerger & Jerger (1974)	Intra-axial brainstem lesion	100% sensitivity
b. Jerger & Jerger (1975)	Brainstem involvement	40% deficit in ear contralateral to lesion side.

3. Compressed Speech		
a) Baran, Verkest, Gollegly & Kibb-Michael (1985),cited in Baran& Musiek(1991).	CANS involvement	67% hit rates
b) Karlsson & Rosenhall (1995).	Brainstem, brainstem vascular & temporal lobe lesions.	Hit rates of 64%, 47% and 80%, respectively.
4. Speech-in-noise		
a) Morales-Garcia & Poole(1972)	1. Brainstem lesions.	1. 14 of 15 patients had abnormal scores i.e. mean falling 20% below normal.
	2. Temporal lobe lesions	2. Depressed contralateral ear scores in 10 patients. Mean ear difference scores of 41%, 47% & 43%, respectively.
b) Olsen, Noffsinger &Kurdziel(1975)	Meniere's disease, **VIII** nerve pathology & temporal lobe lesion.	

2. Dichotic Tests

Dichotic testing refers to stimulation of both ears with difference stimuli, that is, the signals presented to the left ear after from the signals presented simultaneously to the right ear.

Table B: Summary of commonly used dichotic tests for the assessment of CAPD.

Test	Condition	Results
1. Dichotic consonant-vowel *(CV)* Test.		
a. Zurif & Ramier (1972), cited in Baran & Musiek (1991)	Left & right hemisphere damage.	Contralateral ear deficit in right brain damage subjects & similar sources for both ear in left brain damage subjects.
b. Speaks, Gray, Miller & Rubens (1975)	CANS Pathology	Depressed contralateral ear scores in all subjects.
c. Olsen(1997),cited in Baran& Musiek(1991).	Temporal lobectomy	Depressed scores for one/both ears in 31 of 40 patients.
d. Berlin, Cullen, Berlin, Tobey 7 Mouncey (1975),cited in Baran & Musiek (1991).	Right MGB lesion	Depressed left ear scores with normal right ear results
2. Dichotic digit test (DDT)		
a. Musiek (1983)	Intracranial lesions.	Abnormal performance in one/both ears in 17 of 21 patients.
b. Sedge, Mueller & Dillon (1982), cited in Baran & Musiek (1991)	Left temporal lobe lesions.	Bilaterally depressed scores
c. Muller, sedge & Salazar, (1985)	Right temporal lobe lesions.	Contralateral ear deficit
d. Stephens & Thornton (1976)	Brainstem lesion.	5 of 13 subjects showed abnormal results.

3. SSI-CCM		
a. Jerger & Jerger (1975)	Temporal lobe lesion	Depressed scores in contralateral ear.
b. Fifer, Jerger, Berlin, Tobey & Compbell(1983)	CANS lesions	5 of 6 subjects showed abnormal results in one ear
4. Competing sentences		
a. Lynm & Gilroy (1972),cited in Baran & Musiek(1991)	Posterior temporal lobe pathology	100% scores in ipsilateral & 0% in contra ear
b. Welsh, Welsh & Healy(1980)	Dyslexia	Approx. 80-80% scores.

3. Binaural Interaction Test

These test encompass those CANS tests that require an interaction of both ears in order to effect closure for dichotic signals that are separated by time, frequency, or intensity factors between the two ears.

Table C: Summary of commonly used tests of binaural interaction for the assessment of CAPD.

Test	Condition	Results
1. Rapidly alternating speech perception (RASP)		
a. Lynn & Gilory (1977)	Brainstem lesion	Only 6 of 47 patients had positive result
b. Musiek(1983)	Brainstem involvement	5 of 10 subjects had abnormal performance.
c. Welsh et al. (1980)	Learning disability	CAPD could be identified only in 10% subject.

	Test	Condition	Results
2.	Binaural fusion test		
	a. Welsh, Welsh, Healy Cooper (1982)	Dyslexia	Abnormal resynthesis performance.
	b. Smith and Resnick(1972)	Temporal lobe pathology, brainstem lesions.	Abnormal scores with brainstem lesion only.
3.	Masking level difference		
	a. Olsen, Noffsinger &Carhart(1976).	Widespread brainstem lesions.	Hit rate of 50% was obtained.
	b. Lynn, Gilroy, Taylor & Leiser (1981).	Brainstem and cortical lesions	Little/no release from masking in low brainstem involvement.
	c. Karlsson & Rosenhall (1995).	Brainstem lesions.	Sensitivity of 69% obtained.

4. Temporal Ordering Tests

Theses tasks requires the listener to make discriminations based on the temporal order or sequence of auditory stimuli.

Table D : Summary of commonly used temporal ordering tests for the assessment of CAPD

	Test	Condition	Results
1.	Pitch pattern sequence test		
	a. Pinheiero(1977)	Learning disability	Performance ranging form 17.5% to 33.3%
	b. Musiek, Pineiro & Wilson (1980)	Split - brain patients	Unable to respond verbally, only hummed response.
	c. Musiek & Pinheiro (1987)	Cerebral, brainstem & cochlear lesions	Sensitivity of 83% (cerebral lesions) & 45% (brainstem lesions). Specificity was 88%

2.	Temporal sequence test		
	a. Karaseva (1972) cited in Baren & Musiek (1991)	Unilateral auditory projection area lesion	Impairment in the contralateral ear.
	b. Carmon & Nachshon (1971)	Left & right hemisphere damage	Impaired performance for left hemisphere damage

Central auditory processing tests in children with history of OME

Masking level difference (MLD) is the most commonly used behavioral test in OME population. Many investigators have reported reduced MLD in children who have history of OME (Pillsbury, Grose, Joseph & Hall, 1991; Moore, Hutchings & Meyer, 1991, Hall & Grose 1993; Hall, Grose & Pillsbury, 1995). Pillsbury et al. (1991) measured MLD before and after the placement of pressure-equalizing tubes. Subjects were 30 children in the age range 5.1 to 13 years. MLDs were often abnormally small in the OME group before surgery and continued to be significantly reduced after the treatment in some cases even though hearing was within normal limits. The average MLDs for the normal hearing children was approx. 14.2 dB whereas children with OME showed an average MLD of 10.6 dB & 11.3 dB (1- & - 3 month post treatment, respectively). It was seen that more number of subjects who had experienced asymmetric loss of hearing showed abnormal MLD post-surgically.

Moore et al. (1991) determined the binaural MLD in children with history of otitis media. Testing was conducted on 35 children with median age 8.3

years. Results indicated that the mean MLD for the children with history of OME was significantly lower than that of the controls. Hall & Gross (1993) obtained similar results who investigated the MLD in a group of children with a history of OME had significantly reduced MLDs.

In 1995, Hall et al. conducted a study to determine long-term effect of OME on binaural hearing in children with history of OME over a 4-year period following insertion of tympanic membrane grommets. Although results indicted a significant improvement in the MLD with increasing time after middle ear surgery, the MLD remained significantly reduced even 2 years after the correction of hearing thresholds. In general, the results of this study suggest a slow recovery of binaural function in children with OME after restoration of normal hearing thresholds.

Welsh, Welsh & Healy (1983) reported test results from a central auditory battery on children with a history of OME. Results showed more than 75% of the children with history of OME failed one or more of the test components. Analysis of data from 35 OME subjects showed that 23% of subjects with OME history had poor performance on competing sentences test. Rest 77% subjects scored comfortably within the normal range. For filtered speech test, 20 (57%) scored in excess of twice standard deviation. Of these, 11 (55%) failed in one ear while 9 had bilateral impairment. On binaural fusion test, the laterality scores were identified by the low frequency presentation i.e. when the left ear received

the low frequency component and the right the high band, the score is arbitrarily recorded for the left ear. Twenty-seven of thirty-five (77%) subjects were unable to reproduce the test stimulus in a normal fashion. Ten (29%) failed in one ear; 6 on the right ear and the remaining 4 on the left. Those failing bilaterally did so in a range extending from 15% to 50%, with a mean score of approx. 40%. Only three (9%) subjects, failed on RASP and only 1 had a moderate variance from the normal (R; 60, L; 70); the remaining 2 were unilateral (80%).

An analysis of the composite scores suggested that relative failure rate was high for filtered speech test (57%) and binaural fusion (77%), while competing sentences and rapidly alternating speech test were completed with relatively high degree of success and can be regarded as providing little substantive information. Based on the data, Welsh et al. (1983) suggested that long term middle ear disease should be corrected to prevent the defect of a central auditory perceptual disorders.

It has been noted by a number of investigators (Keith, Rudy, Donahue & Katbamna, 1989; Willeford & Burleigh, 1985) that there is much variability inherent in the behavioral tests commonly used for the assessment of central auditory function. Although this variability may be attributed to technical factors such as electronic recording and playback techniques (Shee & Raffin, 1983). Other variabilities may result form the inappropriate use and selection of the central test battery (Keith et al., 1989). Much of the inconsistency in behavioral

tests results probably occurs because of the extreme heterogenecity found within this population (Duffy, 1986, cited in Jirsa, 1992; Willeford & Burleigh, 1985). In addition, linguistic behavioral tests using speech material, becomes language specific and thus difficult to use in multilingual country like India. Lastly, there are practical difficulties in administrating behavioral tests to children, wherein, the interpretation of results become highly subjective. Thus, there is a need for more objective and language non-specific tests like event-related potentials, for the accurate assessment of central auditory processing in young population.

Electrophysiologic Tests

In an effort to enhance the objectivity in the assessment of central processing difficulties, interest has focused in the use of electrophysiologic measures (Musiek & Baran, 1987). The auditory brainstem response (ABR) is very sensitive to VIII nerve and lower brainstem lesions but its usefulness in the assessment of upper brainstem and cortical lesions is limited (Musiek & Baran, 1987). It is well known for its higher test-retest efficiency.

The most frequently used diagnostic feature of ABR is an abnormal latency of different peaks. House and Brackmann (1979) report that 15 of 20 patients (75%) with a variety of extra-axial turmors of the low pons had abnormal ABRs. In another study, Musiek (1986) analysed ABRs from 23 patients with a variety of brainstem lesions, 17 demonstrated abnormal ABRs.

ABR have been extensively used to evaluate patients with demyelinating disorders of the brainstem, such as multiple sclerosis (MS). Stockard, Stockard & Sharbrough (1977, cited in Hall, 1992) studied ABR changes in patients with MS with confirmed brainstem dysfunction. ABR was found to be abnormal in 93% of patients. In another study, Chiappa, Harrison, Brooks &Young (1980, cited in Silman & Silverman, 1991) evaluated 202 MS patients with ABR. They found abnormal ABR in only 32% of their patients. In those with signs of brainstem involvement, ABRs were abnormal in 57%, while in those without brainstem symptoms, ABR were abnormal in only 19%. ABR can, in some instances, provided additional information regarding the site and extension of the tumor within the brainstem. Stockard & Rossiter (1977, cited in Chermak & Musiek, 1997) correlated ABR with clinical findings in over 100 patients with neurological disorders and showed a close relationship between the location of the pathology and certain ABR abnormalists. ABR has proven to be very useful in the evaluation of patients in coma. Starr and Achor (1975, cited in Chermak & Musiek, 1997) tested 51 patients who were comatose due to intoxication, metabolic disorders, or trauma. They found that the structural changes which occurred in the auditory pathways of the brainstem as a result of trauma or reduced circulation caused abnormal amplitudes and latencies of the ABR, whereas in patients in coma induced by intoxication or metabolic disturbances, the ABR was normal.

Middle Latency Response

The use of MLR in subject with central auditory involvement in a new and partially experimental procedure. There are very few sensitivity and specificity data on the MLR. In one of the early studies Kraus, Ozdamar, Hier & Stein (1982, cited in Chermak & Musiek, 1997) reported 24 patients with temporal lobe lesions had normal MLR. They found that a unilateral temporal lobe lesion diminished the amplitude of Pa without affecting the latency. Pa was more significantly reduced or absent, however, over the involved hemisphere. Woods, Clayworth and Knight (1985, cited in Chermak & Musiek, 1997) tested nine patients each with unilateral cortical lesions, and patients with subcortical lesions. Six out of nine patients with cortical lesions and showed abnormal ear or electrode effect. Another study on children with learning disability (LD) showed differences in the latency of the MLR when compared to children with no learning problems (Arehole, Augustine & Simhadri, 1995). This study revealed extended latency of the Pa wave in 5 of 11 children with LD, whereas only 1 child out of 11 in the control group demonstrated an increased Pa latency.

Late Latency Response

The LLRs are mainly comparised of Nl and P2 as most prominent components. The N1-P2 is a transient scalp potential complex evolved by any change in the perceived auditory environment that is sufficiently abrupt. The other two components namely N2 and P3 are potentials, elicited primarily by a

deviating stimulus (Naatanen, Simpson & Loveless, 1982, cited in Tonnquist-Uhlen, 2000; Picton, 1995) and represent the event-related, endogenous response. The Nl component is considered to be generated from the supra-temporal primary auditory cortex and associated areas (Elberling, Bak, Kofoed, Lebech, & Saemark, 1980; Scherg & Von Cramon, 1989, cited in Tonnquist-Uhlen, 2000). The P2 is also considered to be generated mainly in the vicinity of the auditory cortex within the temporal lobe (Elberling et al, 1980; Perrault & Picton, 1984, cited in Tonnquist-Ulhen, 2000). The generators of N2 include sub-cortical and limbic areas, or activity in the frontal lobes that may represent switch of attention (Naatamen & Picton, 1986, cited in Tonnquist-Ulhen, 2000, McCarthy, Wood, Williamson & Spencer, 1989, cited Tonnquist-Ulhen, 2000).

Used appropriately, N1-P2 complex can provided valid and accurate information about the underlying deficit of central auditory nervous system. Knight, Hillyard, Woods and Neville (1980, cited in Chermak & Musiek, 1997) showed that Nl is more sensitive than P2 to focal brain lesions in the tempro-parietal region. In case of bitemporal lesions, Nl and P2 peaks have been essentially absent or the latency was extended and amplitudes decreased (Musiek, 1991; Woods, Clayworth, Knight, Simpson & Naeser, 1987, cited in Chermak & Musiek, 1997). Knight et al. (1988) studied six patients with inferior parietal lesions and 9 with superior temporal gyrus lesions. Results showed significantly reduced amplitude of Nl for the superior temporal gurus group and

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inferior parietal lesions groups. No significant difference was found between these groups for P2 amplitude and latency characteristics.

Jirsa and Clontz (1990) compared a normal group of children with a group of children classified as CAPD. The result showed that inter subject variability was greater in the CAPD groups than in the normal group. The latency of Nl and P2 components were significantly reduced in the CAPD groups whereas there was no difference in the amplitude of the Nl & P2 of the two groups.

Mismatch negativity

In recent years, MMN has emerged as a potential tool to study central auditory processing. It is a relatively new and less frequently used clinical tool. It is highly variable but responses can usually be obtained in normal subjects without undue difficulty. The MMN is acquired using an oddball paradigm (Naatanen et al., 1982, cited in Tonnquist-Uhlen, 2000). Where the subject need not pay any attention to the stimuli.

Nataanen et al., (1982, cited in Tonnquist-Uhlen, 2000) reported that if the difference between the standard/frequent and deviant/rare stimuli is large, the peak latency of MMN becomes short and the MMN overlaps and add to the Nl wave. When the difference is small, the latency becomes longer and there is little, if any, change in the Nl wave. Sams, Paavilainen, Alho, and Naatanen (1985, cited in Hyde, 1997) showed that MMN was present when the deviant

stimuli were just discriminable from the standard stimuli, but not when the difference was not perceptible.

The MMN can demonstrate a difference in the standard and deviant waveform for stimuli that vary as little as 8 Hz in frequency (Naatanen, 1992, cited in Lang, Eerola, Korpilahti, Holopainen, Salo & Aaltonen, 1995) and 2 dB in intensity (Iyengar, 1999). Because of its differential sensitivity, the MMN is an ideal procedure for measuring various types of auditory discrimination.

Kraus, McGee, Ferre, Hoeprer, Carrel, Sharma & Nicol (1993) have shown deficits correlated to an abnormal MMN in patients with CAPD and specific auditory discrimination problems. In another study on patients with frontal cortex lesions, Alho, Woods, Algazi, Knight & Naatanen (1994, cited in Chermak & Musiek, 1997) found that MMN amplitude was diminished, most notably over the lesioned hemisphere and from the ear ipsilateral to the lesioned hemisphere.

Studies have shown a reduction in amplitude and duration of MMN in children with learning disability. But reports on latency of MMN are equivocal. Korpilahti and Lang (1994, cited in Leppanen & Lyytinen, 1997) found difference in the MMN component between 14 development language disorder children and 12 control children. The MMN response was significantly reduced in the developmental language disorder children. Similar findings were reported

by Guruprasad (1999) who found reduced MMN magnitude in 10 children with LD. The MMN was also found to be abnormal on latency parameter with increased onset, offset and peak latency.

P300

This is another late potential, an event related potential that can be used for evaluating the integrity of the central auditory nervous system in children with CAPD. Holcomb, Ackerman & Dykman (1985), cited in Leppanen & Lyytinen (1997) observed prolonged latency in the range of 900 - 1150 ms in LD children. A significant decrease in P300 amplitude in LD children have been reported by various investigators (Holcomb et al., 1985; Jirsa & Clontz, 1990; Radhika, 1998). Jirsa & Clontz (1990) investigated the P300 in children with confirmed CAPD and compared their result to age matched normal subjects. The CAPD subjects had significantly longer latencies and smaller amplitude than the control groups. Dawson, Finely, Philips, Galpert and Lewy (1989) recorded P300 from autistic children using phonemes. They observed greater reduction of amplitude of P300 over the left hemisphere than over the vertex or the right hemisphere. Courchesne and Young-Courchesne (1988, cited in Leppanen & Lyytinen, 1997) reported that auditory P300 was always smaller in the autistic children, regardless of the rate of stimulus presentation, whereas it was depressed in the developmental language disorder subjects with rapid stimulus presentation.

Barret (1993) cited in Mcpherson (1996) argued that reduced amplitude of P300 reflects "either a less or inefficient processing of the stimuli".

Electrophysiologic Studies in subjects with history of OME

The ABR has been studied extensively in the evaluation of neurological conditions affecting brainstem segment of central auditory nervous system, but fewer studies have been undertaken for the evaluation of brainstem functioning in individuals with history of OME. Most of the available information indicates that the vast majority of children with OME have abnormal ABRs (Folsom, Weber & Thompson, 1983; Hall & Grose, 1993). Folsom et al. (1983) conducted a study on children with a history of OME in the range of 6 to 10 years. Results indicated that the children having a history of OME, but normal hearing at the time of testing, showed abnormally long interwave latency difference (Wave I-III and Wave I-V), with normal latency of Wave I. Similar results were reported by Lenhardt, Shaia, & Abedi (1985). Anteby, Hafner, Pratt & Uri (1986, cited in Hall & Grose, 1993) reported significantly prolonged III-V and I-V interwave interval for children with OME history, whereas Chambers, Rowan, Mathies & Novak (1989) found that the I-III interval was significantly delayed but not the interval between I - V or III - V. Gunnerson and Finitzo (1991) recorded ABR for children in the age groups 5-7 years with a history of OME during early infancy. They found delays in both wave III and V. They also found that the binaural interaction response was often abnormal in children with

OME history. Hall & Grose (1993) studied 14 children in the age range of 5.8 to 9.2 years with a history of OME. They found significantly prolonged wave III and V and I-III and I-V interweave intervals. Although there is some variability among the results of these studies, a history of OME does appear to be dissociated with small but significantly prolonged either wave III or wave V (or both waves).

Thus, a review of literature shows brainstem dysfunction in children with history of OME. The brainstem changes are reflected by ABR abnormalities seen in these subjects. On the same line, temporary auditory deprivation caused due to a fluctuating conductive loss may lead to dysfunction in higher cortical centers. These cortical dysfunctions can be evaluated using higher auditory evoked potentials. Hence the presented study aimed at investigating cortical as well as brainstem functioning in children with OME using ABR, LLR and MMN.

METHOD

The present study aimed at investigation of central auditory processing in children with history of OME using electrophysiologic tests.

A total of eight children comprised the subject population for this study. All the subjects were in the age of 6-13 years. These subjects had recurrent bouts of OME (minimum two in a year) with initial bout occurring at or prior to 10 years of age. All the subjects had conductive hearing loss as indicted by pure tone here audiometric results and immittance evaluation. The ears were dry at the time of testing. No subjects had any neurological or psychological problem in the past and also at the time of testing.

Instrumentation

The following equipment were used for the study:

a. **Pure tone audiometer** : A calibrated 2-Channel clinical audiometer was used to establish the behavioural pure tone thresholds for all the subjects.

b. **Immittance Meter** : A calibrated immittance meter was used to assess middle ear function in all the subjects.

c. Electrophysiologic **Unit** : Bio-logic Auditory Evoked Potentials Systems (Navigator) version 5.44 with EP317 software was used for recording auditory evoked potentials.

Test Environment

All the testing was conducted in an acoustically treated room.

Test Procedure

Pure tone audiometery was carried out for all the subjects using double channel clinical audiometer. Middle ear functioning was tested using calibrated immittance meter. A detailed case history which included the following information collected from the subjects.

- Age/Sex

- Age of first attack of OME

- Frequency of attacks (per month)

- Duration of each attack

- Previous medical / audiological evaluation

- Associated problems / symptoms and ear pain, headache, etc.

- Family history of otitis media.

Patient Set-up

The subjects were seated on a reclining chair in a comfortable posture with the head fully supported to ensure noise free recording.

Electrode Placement

i) For ABR

Four electrodes were used for recording ABR. The electrode sites for the two channel mapping recording were selected with Cz as non-inverting, FPz as common and M1, M2 as inverting.

ii) For MMN

The electrode sites for the two-channel mapping recording were selected with Cz, Pz as non-inverting, FPz as common and M2, Ml as inverting. Silver chloride disc electrodes were fixed at the above mentioned sites after a thorough skin surfaces clearing with surgical spirit and a skin preparing solutions. Standard EEG paste was used to increase the conductivity

The impedance of the electrodes was measured with reference to the common electrode for both the channels. The impedance values were less than 5kΩ for each electroed and the inter-electrode impedance difference was less than 3 kΩ. If the electrode impedance was more than 5 kΩ, the electrode site was cleaned and the electrode was secured again, while recording MMN, the inverting electrodes M2 and M1 were linked together by means of a jumper.

Procedures for recording

The earphones were placed on the subject's ear being careful not to dislodge any electrode. Each ear was stimulated separately. Table M-I describes the protocol used for recording auditory evoked potentials.

Table M-I: Test protocol used for recording ABR and MMN

SI. No.	Parameters	ABR	MMN
1.	Stimulus type	Rare, clicks	Alt, TB
2.	Frequency : Frequent / Infrequent	-	lOOOHz/lOOOHz
3.	Intensity : Frequent / Infrequent	50dBSL	50/55dBSL
4.	Repetition time (clicks / sec.)	11.1,60.1,90.1	3.1
5.	Rise time (ms)	-	10
6.	Fall time (ms)	-	10
7.	Plateau (ms)	-	30
8.	Filter	100-3000 Hz	0.1-30 Hz
9.	Max. stimuli	2000	500
10.	Gain	1,00,000	50,000
11.	Notch filter	Off	Off
12.	Ratio : Frequent / Infrequent	-	5:1
13.	Transducer	Headphone	Headphone

Analysis

The following parameters were analyzed from the recorded waveform.

1. ABR

    a. Absolute latency of wave I, III & V.

    b. Inter-peak latency difference for I-III, III-V & I-V

    c. Amplitude ratio V/I

2. LLR : This was analysed from frequent waveform in Cz recording of MMN. The following parameters were analysed:

   a. Absolute peak latency of PI, N1, P2 & N2.

   b. Amplitude of N1-P2.

   c. Morphology of waveform.

3. MMN : This was obtained by subtraction of frequent waveform from infrequent waveform. The following parameters were analysed.

   a. Peak latency of MMN.

   b. Amplitude of MMN

   c. Duration of MMN

RESULTS

In the section, the results of hearing evaluation of each subject is described. The results include the following

- History of the subject

- Results of pure-tone audiometry

- Results of immittance evaluation

- Results of auditory evoked potentials

As it was a small sample and a heterogenous group, no statistical analysis was carried out. Pure tone audiogram was described based on the classification given by Clark (1981, cited in Harrell, 2000). Classification given by Liden (1974) was used to classify the tympanograms obtained for 226Hz probe tone. Latency and amplitude of ABR waves for clicks presented with a repetition rate was compared with the normative data given by Guruprasad (2000). The long latency response was compared with the normative data given by Shankar (1997) and Radhika (1998). Norms developed by Iyengar (2000) were used to classify MMN as normal or abnormal. The mean values given by the corresponding investigators ± 2 SD was considered as normal for all the potentials. For ABR to clicks presented at higher repetition rate, a shift of 0.1msec / 10 Hz increase in the repetition rate with variability of 0.2msec around the final value was considered as normal (Musiek & Gollegly, 1985).

Subject: 1

a. **History** :   A 13-year old female reported with a history of middle ear effusion in the left only, since the age of 6-7 years. She was diagnosed as having chronic suppurative otitis media (CSOM) by an otolaryngologist. The subject underwent medication at the age of about 11 years. Till this time the middle ear effusion was intermittent. During this age period, frequency of otitis media was once / twice once in four months. At the time of testing the subject complained of ear pain in both the ears. No ear discharge was present at the time of testing. There was no family history of OME. Reports of previous audiological and medical evaluation were no available.

b. **Results of audiometric and immittance evaluation results** :   Pure   tone audiometry indicated minimal hearing loss in the right ear with mild hearing loss in the left ear (fig. 1-1). Bone conduction thresholds were normal limits in both the ears. Immittance evaluation showed bilateral 'As' type tympanogram with
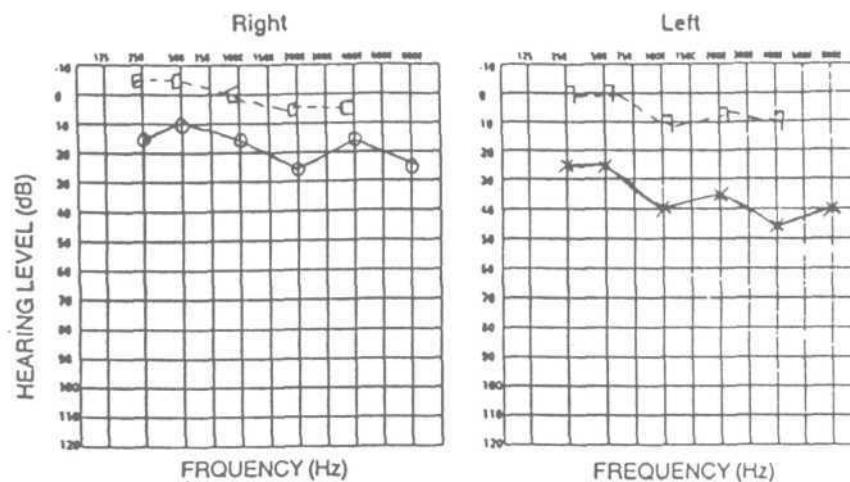
fig. 1-1 Audiogram showing pure-tone thresholds of subject 1.

c. Results of ABR : The ABR results showed that the absolute latency as well as interpeak latency interval were normal for clicks presented at repetition rate of 11.1/sec in the right ear. In the left ear, wave I latency was prolonged for clicks presented with a repetition rate of 11.1 /sec whereas latency of III & V peaks was within the normal limits. The I-III and I-V interpeak latency (IPL), was shorter than normal value whereas III-V IPL was normal. The shift in latency of wave V was normal when repetition rate were increased to 60.1/sec and 90.1/sec in both the ears. The ABR waveforms for clicks presented at different rates are shown in fig 1-2.
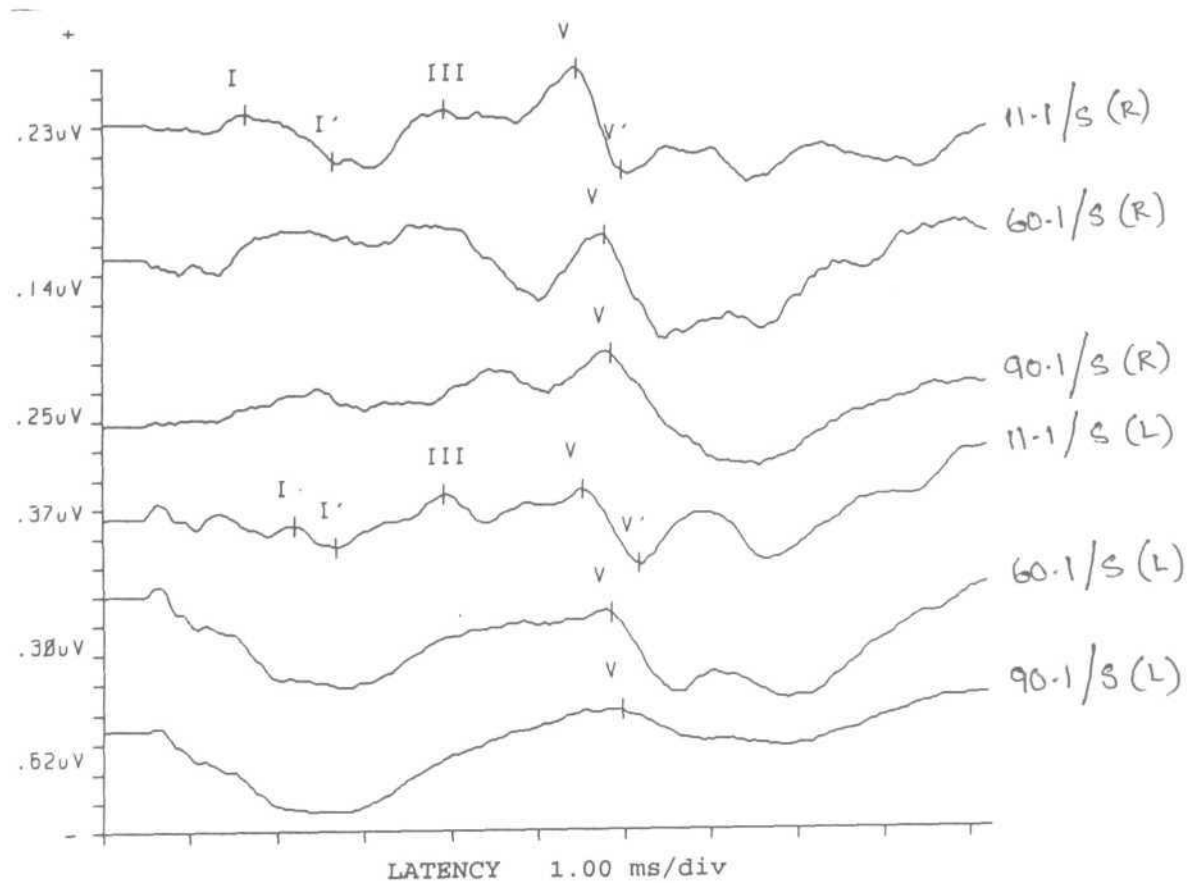
fig. 1-2. ABR waveforms recorded from subject 1.

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d. LLR results: The LLR recording showed prolonged PI and Nl waves in the right ear whereas P2 and N2 were recorded at normal latency. Similarly, both PI & Nl were prolonged in the left ear with normal latency value of P2 but N2 could not be identified. The N1-P2 amplitude, which could be measured only in right ear was within normal limits. The latency of various LLR components is shown in fig 1 -3 and 1 -4 for the right and the left ear, respectively.

e. MMN results: MMN was present in both the ears. The latency of MMN in both Cz and Pz recording was normal in the right ear whereas it was below the lower limit of normal range in the left ear. The amplitude was normal in both the ears for both the recordings. The duration of MMN was found to be longer in the right ear while it was within normal limits in the left ear. The MMN waveforms
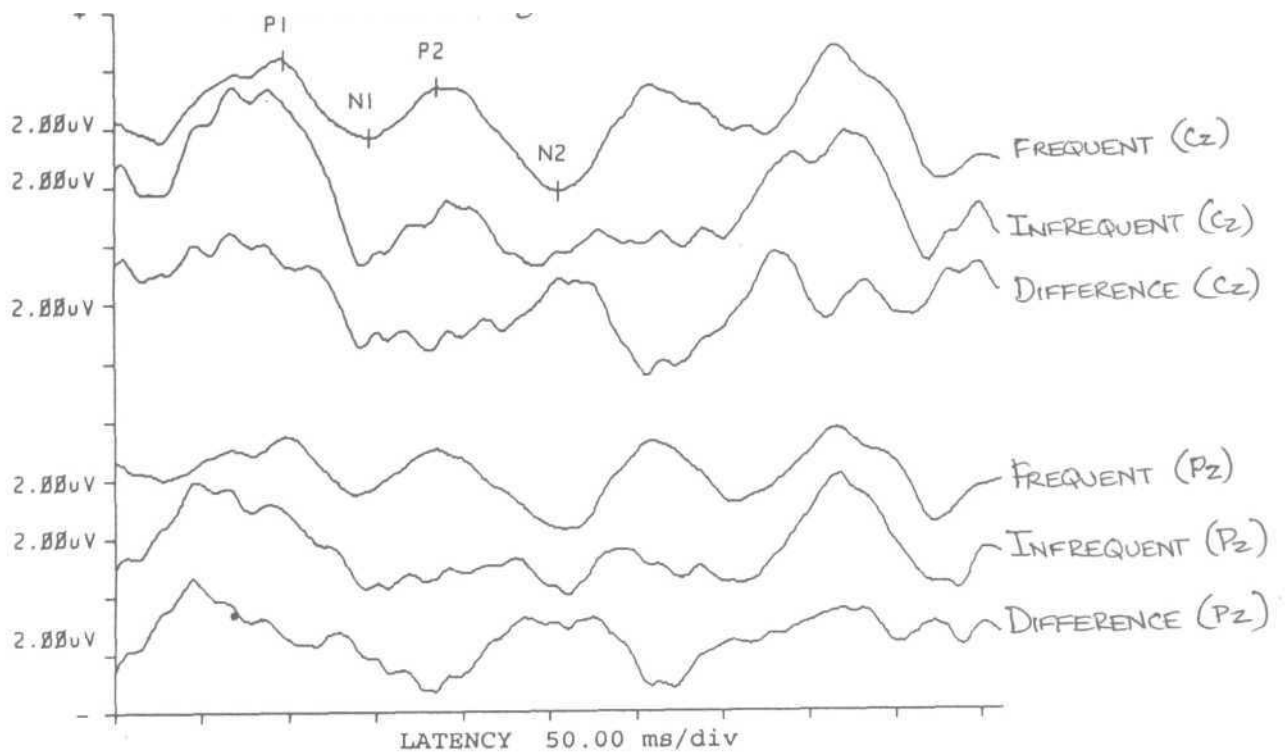
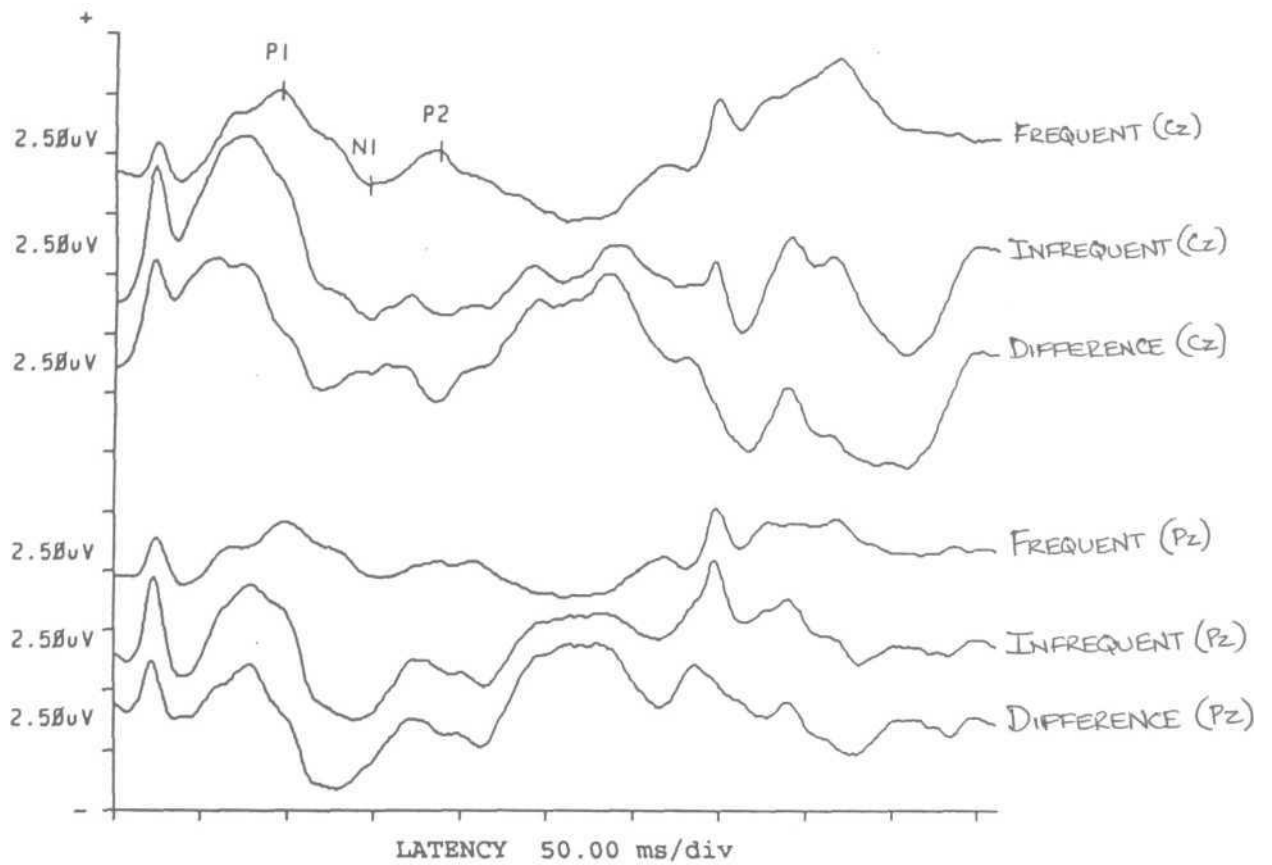**fig. 1-3. LLR** & **MMN waveforms of subject 1 for the right ear stimulation**

fig. 1-4. LLR & MMN waveforms of subject 1 for the left ear stimulation

Subject: 2

a. History : This subject was a 10 year-old-male with prolonged and persistent history of otitis media with effusion. The initial attack of otitis media was reported to have occurred at the age of 1 year. Discharge has been reported to be present in both the ears since the time of its onset. The subject had taken medication for the treatment of otitis media but ear discharge did not stop for more than 2-3 days after medication. Attacks of otitis media had been occurring once in every six months, persisting for about 12-15 days every time it occurred. At the time of testing, subject was under medication since 10 days. He was

diagnosed, as having bilateral CSOM (tubotympanic) by an otolaryngologist. The ears were dry at the time of testing.

b. Audiometric and immittance evaluation results: Audiometric results revealed a mild conductive hearing loss in the right ear and a minimal conductive hearing loss in the left ear (fig 2-1). Immittance evaluation showed 'B' type tympanogram indicating conductive hearing loss in both the ears.
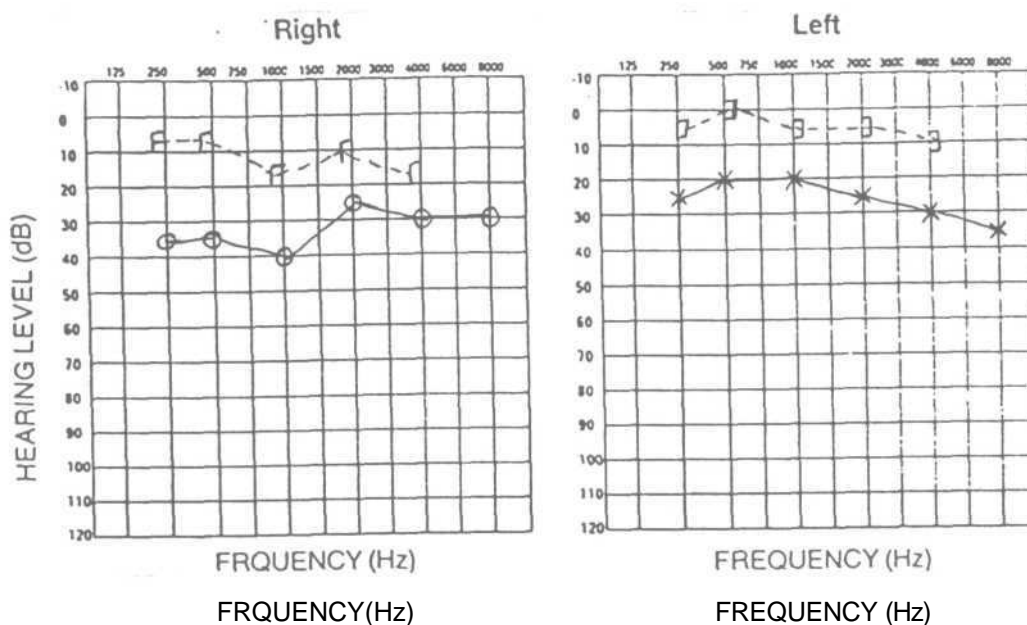

fig. 2-1. Audiogram showing Pure-tone thresholds of subjects 2

c. ABR results : For clicks presented at a repetition rate of 11.1/sec, the absolute latency of all the three major peaks was prolonged but the interpeak latency was normal in both the ears except for wave I-III IPL in the left ear which was slightly prolonged. The wave V/I amplitude ratio was normal in both the ears. The shift in the latency of wave V was also found to be within normal limits when the rate was increased from 60.1/sec to 90.1/sec. The ABR waveform at different repetition rates is shown in fig. 2-2.
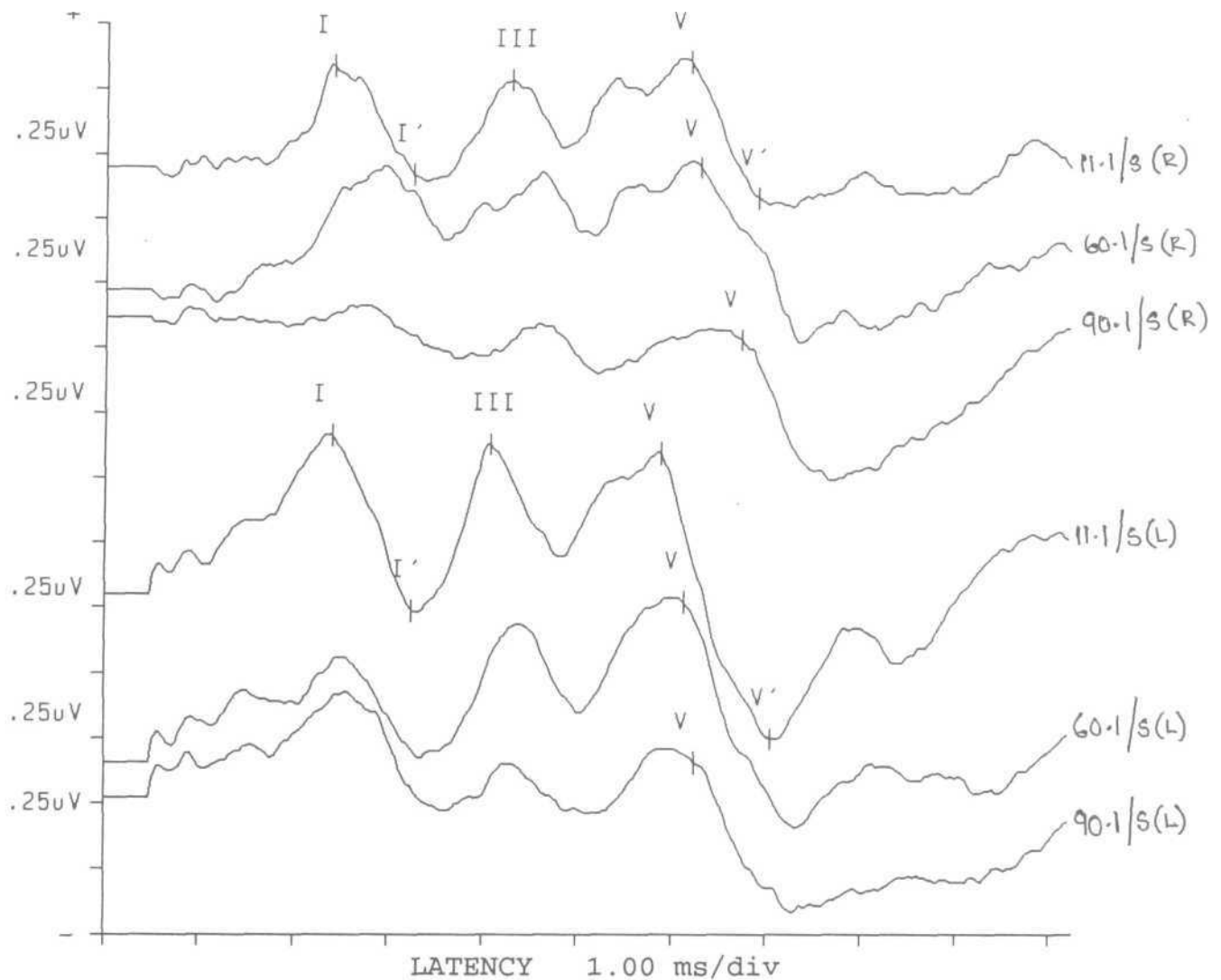
fig. 2-2. ABR waveforms recorded from subject 2

d. LLR results : The LLR waveform was very noisy in both the ears. On right ear stimulation, only PI and Nl could be identified. The P1 was followed by a broad Nl which could be due to absence of a well defined P2. Both P1 and Nl were prolonged in the right ear. In the left ear P1, Nl and P2 could be identified but latency of all these waves was prolonged. The Nl was a broad negative wave with latency ranging from 165-315 msee. The maximum negativity was at about 315 msee. The Nl - P2 amplitude was measured to be within normal limits in the

left ear. This could not be measured in the right ear due to absent P2 wave. The LLR waveforms are shown in fig. 2-3 and 2-4.

d. MMN results: The MMN waveform had poor morphology in this subject. The MMN could not be identified in the right ear as there were multiple peaks and poor waveform morphology in Pz recording. In Cz recording, MMN had normal duration but peak latency was prolonged. In the left ear, the peak latency as well as duration of MMN were prolonged for both Cz and Pz recording. The amplitude of MMN was, however, normal for both the ears. The MMN waveforms are shown in fig 2-3 and 2-4.
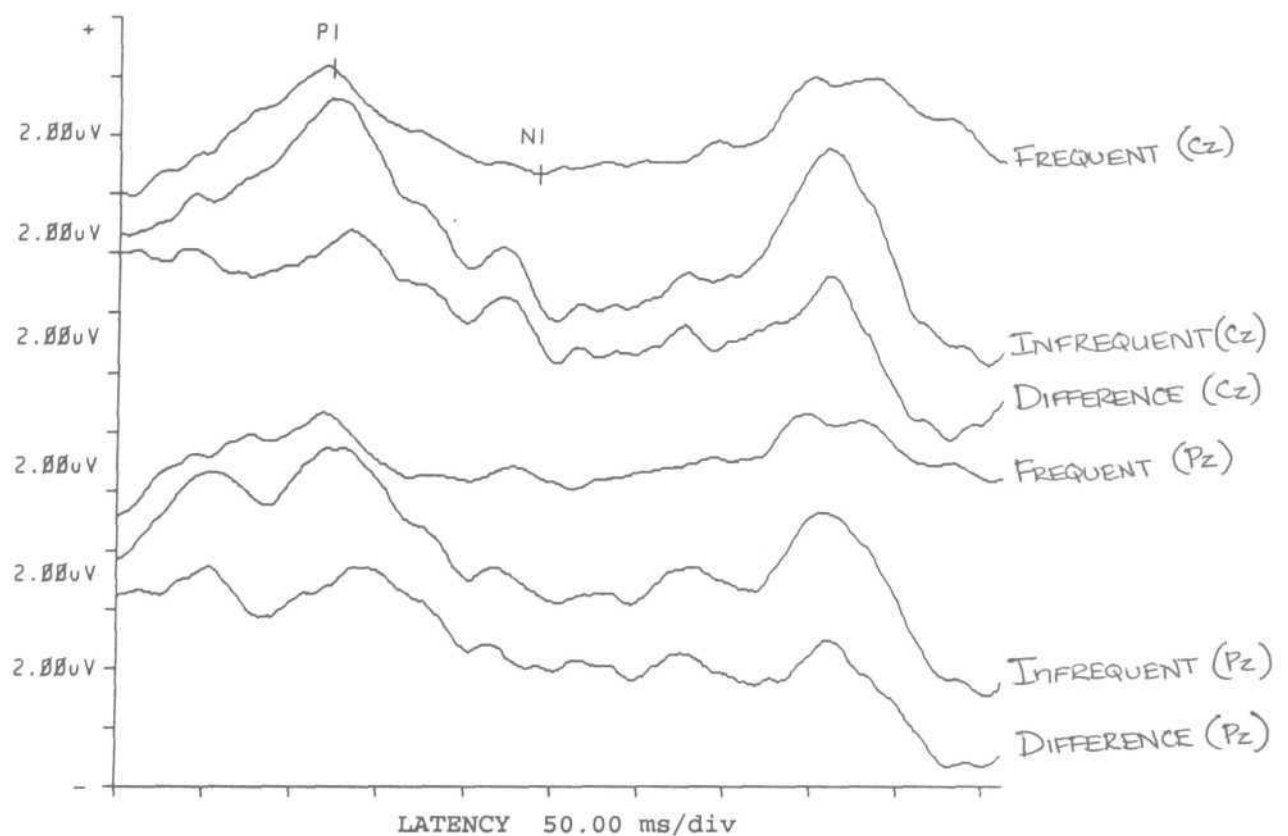

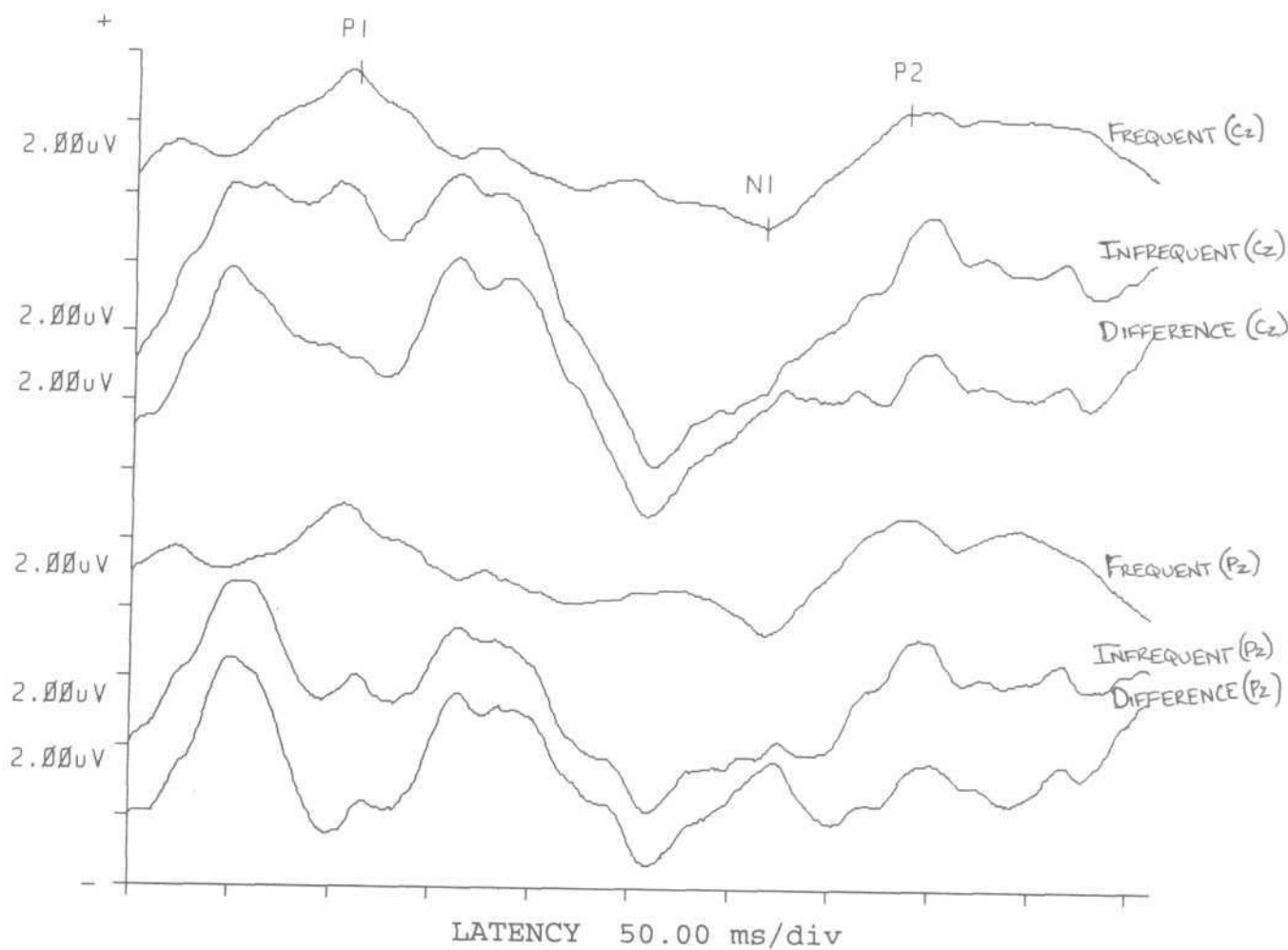**fig. 2-3 LLR & MMN waveforms of subject 2 for the right ear stimulation**

fig. 2-4 LLR & MMN waveforms of subject 2 for the left ear stimulation

Subject: 3

a. History :   A 10-year-old female reported with the history of OME in the right ear since the age of 5-6 years. Otitis media was reported to occur 3-4 times per month. Ear discharge resolved after medication, which was prescribed 15 days prior to testing. Ear discharge was also reported to the present in the left ear since 6 months. The subject had not undergone any medication prior to the present ongoing treatment. At the time of testing both the ears were clean and dry.

b. Audiometric and immittance evaluation results: As shown by the audiometric results in fig 3-1, hearing sensitivity was within normal limits in the right ear whereas a moderate conductive hearing loss was present in the left ear. Immittance evaluation showed 'As' type tympanogram in the right ear and 'B' type in the left ear. Reflexs were absent in both the ears.
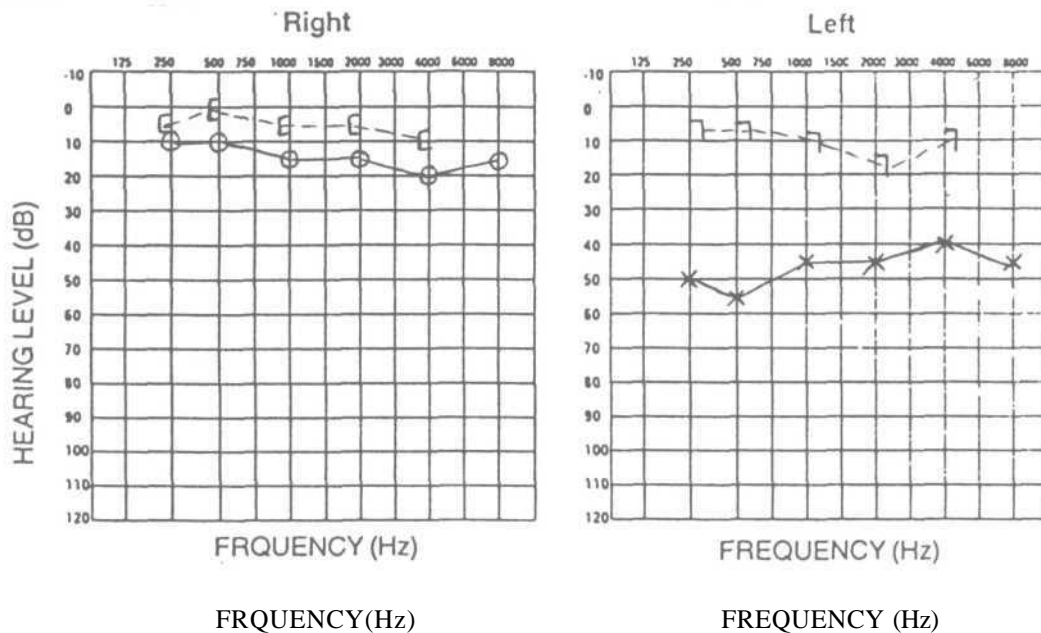

fig. 3-1 Audiogram showing pure-tone thresholds of subject 3

c. ABR results: The ABR results indicated prolonged absolute latency for all the peaks for clicks presented at 11.1 /sec repetition rate in both the ears. The I-III IPL was normal in both the ears whereas III-V IPL was normal only in the left ear. The III-V IPL in the right ear and I-V IPL in both the ears were found to be shorter than the normal value. The V/I amplitude ratio was normal for both the ears. With the increase in repetition rate no abnormal shift in wave V latency was seen in both the ears. The ABR waveforms for different click rates are shown in fig. 3-2.
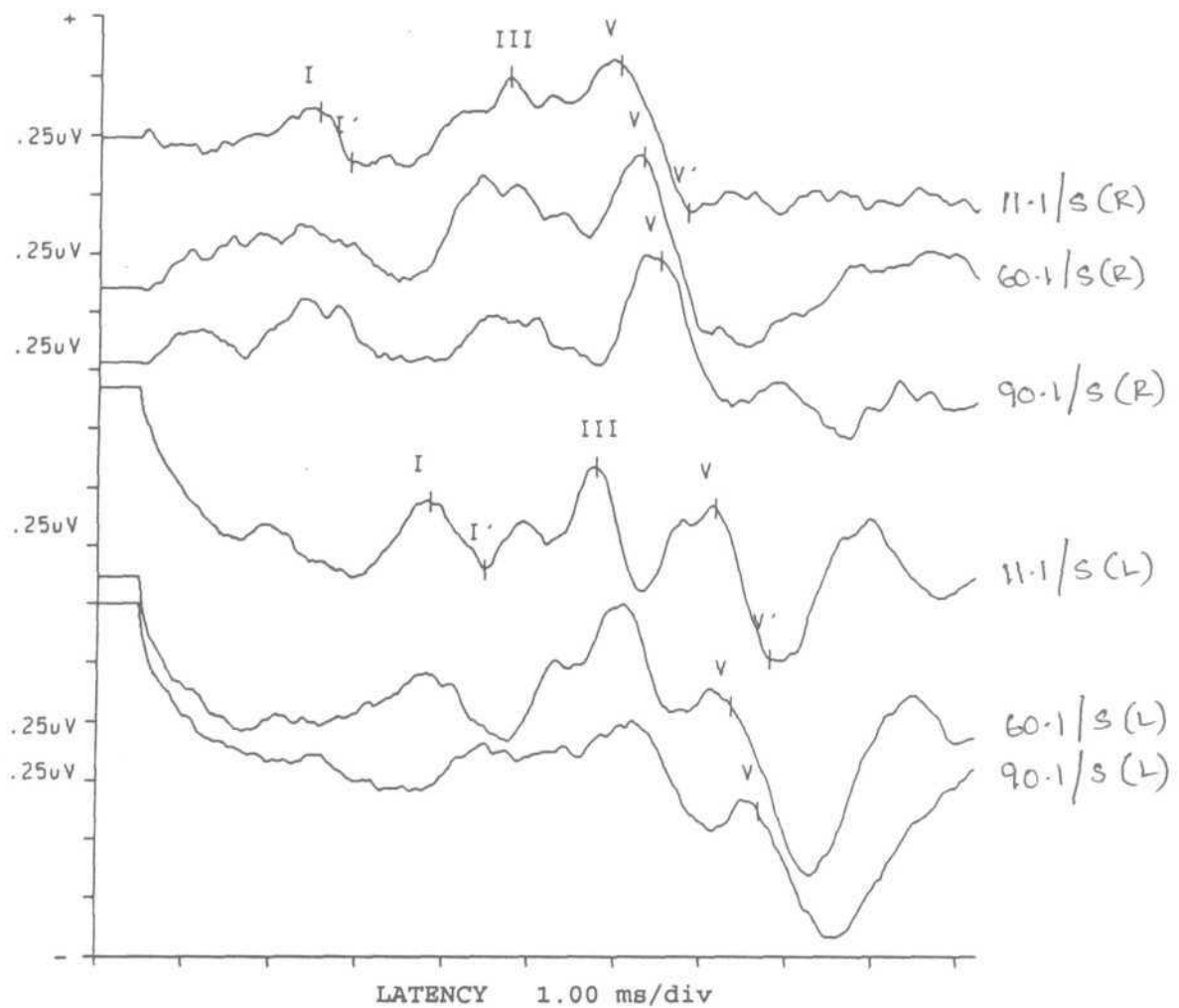
fig. 3-2. ABR waveforms recorded from subject 3

d. LLR results: The LLR was present in both the ears as shown in fig. 3-3 & 3-4. PI, Nl could be identified in both the ears. P2 through present, was not prominent in the waveform recorded from right ear. All the three waves were prolonged in both the ears with comparable latencies between the ears for the corresponding peaks. The N1-P2 amplitude was within the normal limits.

e. **MMN results:** A well defined MMN was present on stimulation of each ear separately. The peak latency of MMN was prolonged, though it was comparable in both the ears. The amplitude of MMN was slightly higher than normal in the

right ear whereas it was within normal range in the left ear in Cz recording. However, left ear also had higher MMN amplitude in Pz recording. The duration of MMN was found to be prolonged in both the ears. It was, however, comparatively longer in the right ear for Cz recording. The MMN waveforms for the right and left ear stimulation are shown in fig. 3-3 and 3-4, respectively.
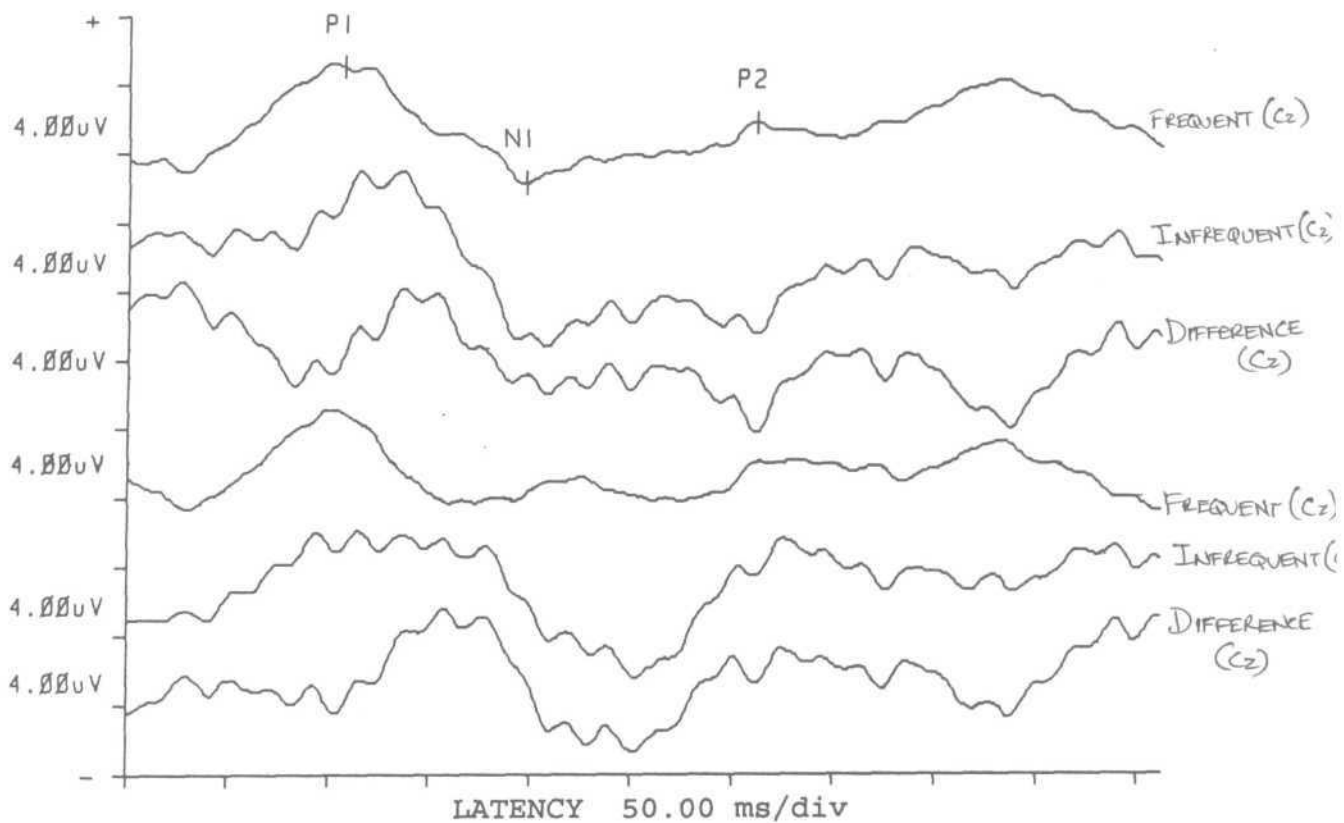

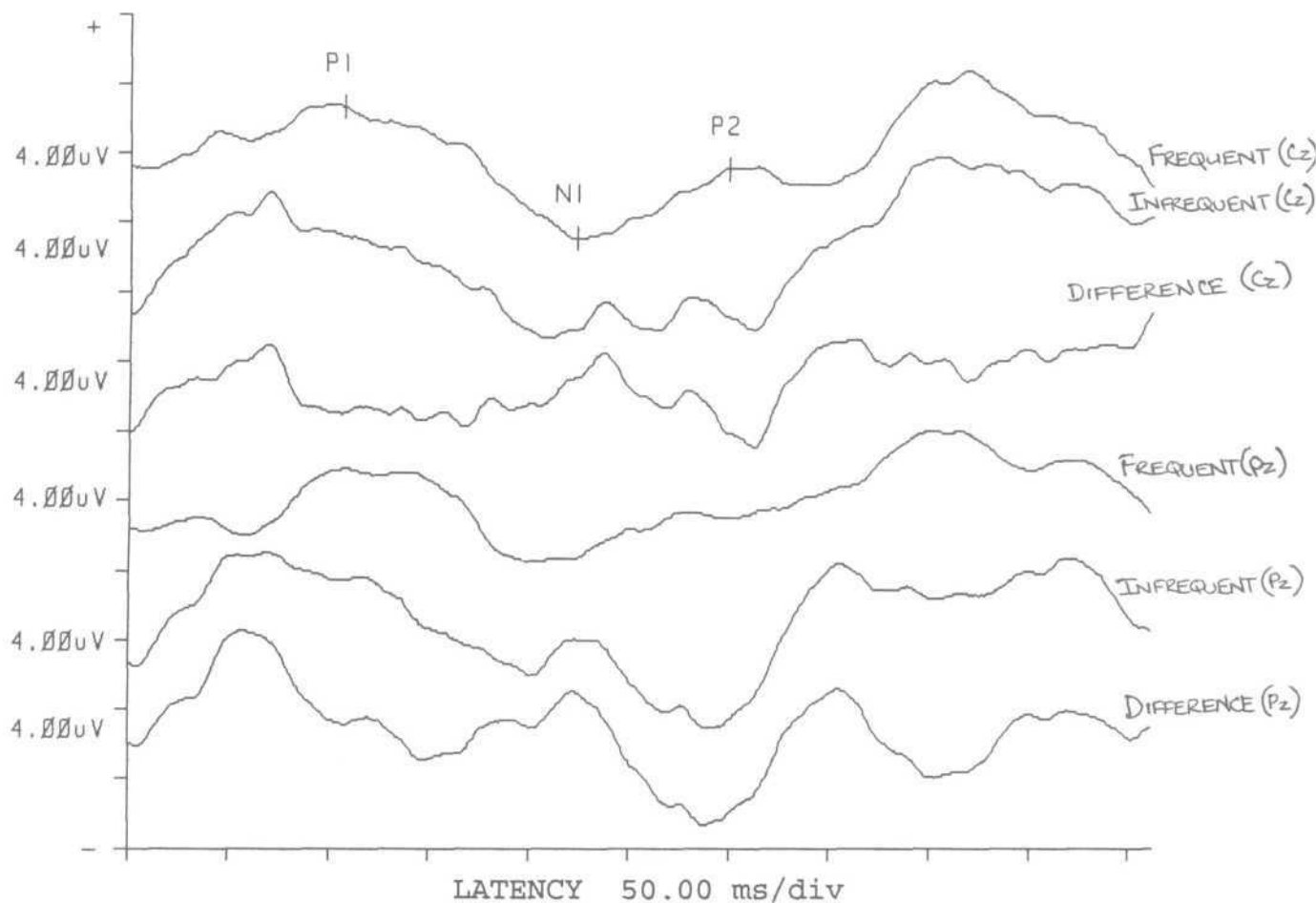fig. 3-3. LLR & MMN waveforms of subject 3 for the right ear stimulation

fig. 3-4. LLR & MMN waveforms of subject 3 for the left ear stimulation

Subject 4 :

This subject was a 10-year-old male who had a history of middle ear discharge since the age of 5 years. Attacks of ear discharge had been occurring with a frequency of one or two attacks per months since the time of its onset. The subject was disgnosed as having bilateral CSOM with chronic adenoiditis by an otolanyngologist. The subject was under medication at the time of testing. No medial treatment was sought for the middle ear discharge before the present

evaluation. Reports of previous audiological / medical evaluation were not available.

Audiometric and immittance evaluation results: A mild low frequency conductive hearing loss was seen in the right ear on pure tone testing. The left ear had mild conductive hearing loss across frequencies results of immittance evaluation showed 'B' type tympanogram in both the ears. Audiometric results are shown in fig. 4-1.
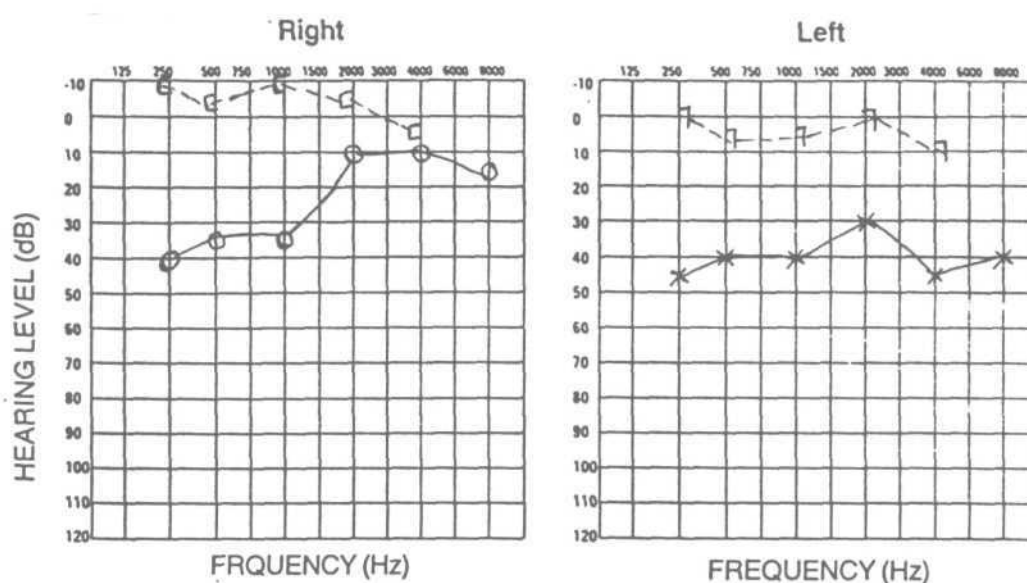

fig 4-1. Audiogram showing pure tone thresholds of subjects 4

ABR results: The absolute latency of wave I, III and V were prolonged in the right ear for clicks presented at 11.1/sec repetition rate. In left ear, only wave I was prolonged. The III-V and I-V IPLs were within normal limits in both the ears. The I-III IPL was slightly shorter for the both the ears. When the repetition rate was increased to 60.1/sec and 90.1/sec, the shift in wave V latency was well

within the normal limits. The ABR waveform at various click repetition rates are shown in fig 4-2.
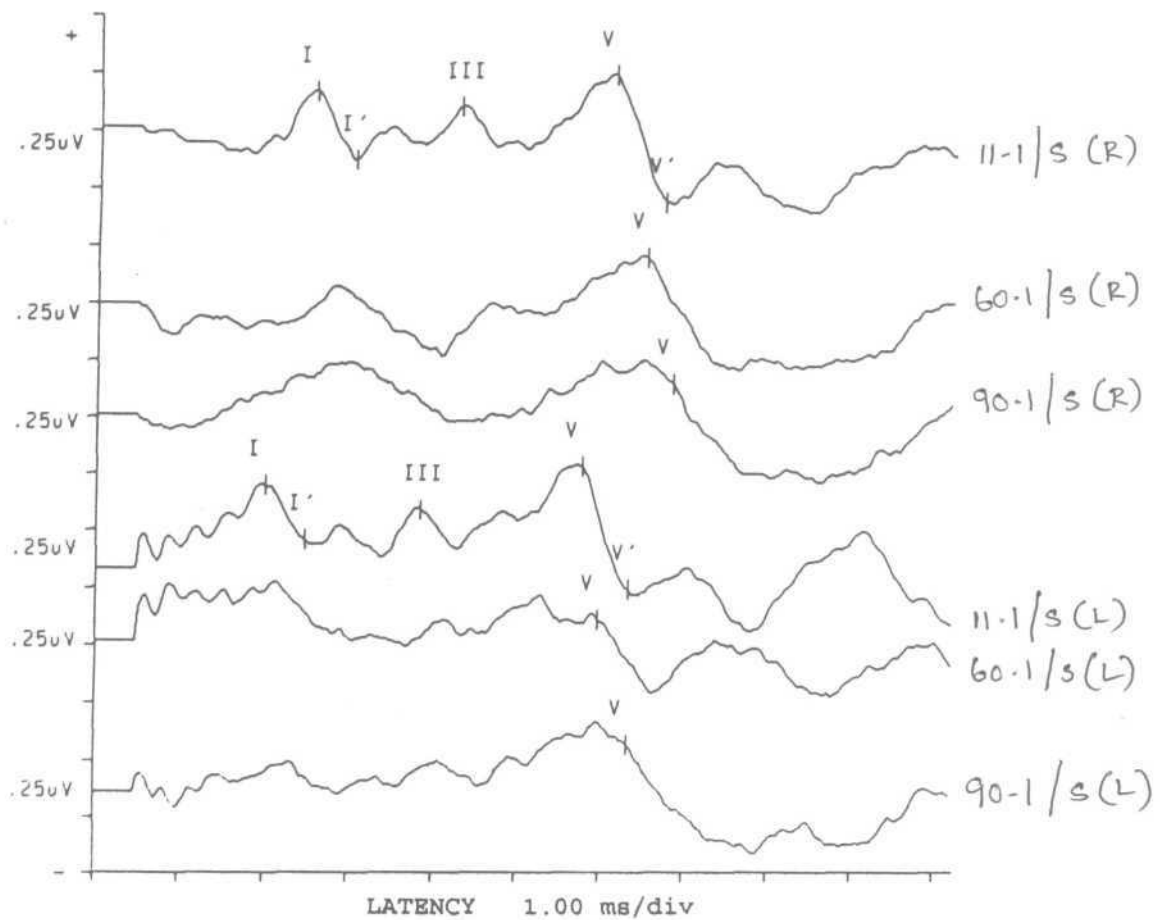

fig. 4-2. ABR waveforms recorded from subject 4.

LLR results: The LLR had poor morphology in this subject. P1 and N1 were the only two identifiable peaks. These peaks were found to be prolonged in recordings from stimulation of both the ears separately. Though the peaks were prolonged, latency of Nl was comparatively more delayed than P1 in both the ears. Other LLR peaks could not be identified due to poor waveform

morphology. The LLR waveform from both the ears are shown in fig 4-3 and 4-4.

MMN results: MMN was present in recordings for both the ears. Latency and the amplitude of MMN was normal in both the ears. In the Cz recording, the duration of MMN was normal in the right ear whereas it was prolonged in the left ear and vice-versa was seen in Pz recording. These MMN waveforms are shown in fig 4-3 and **4-4.**
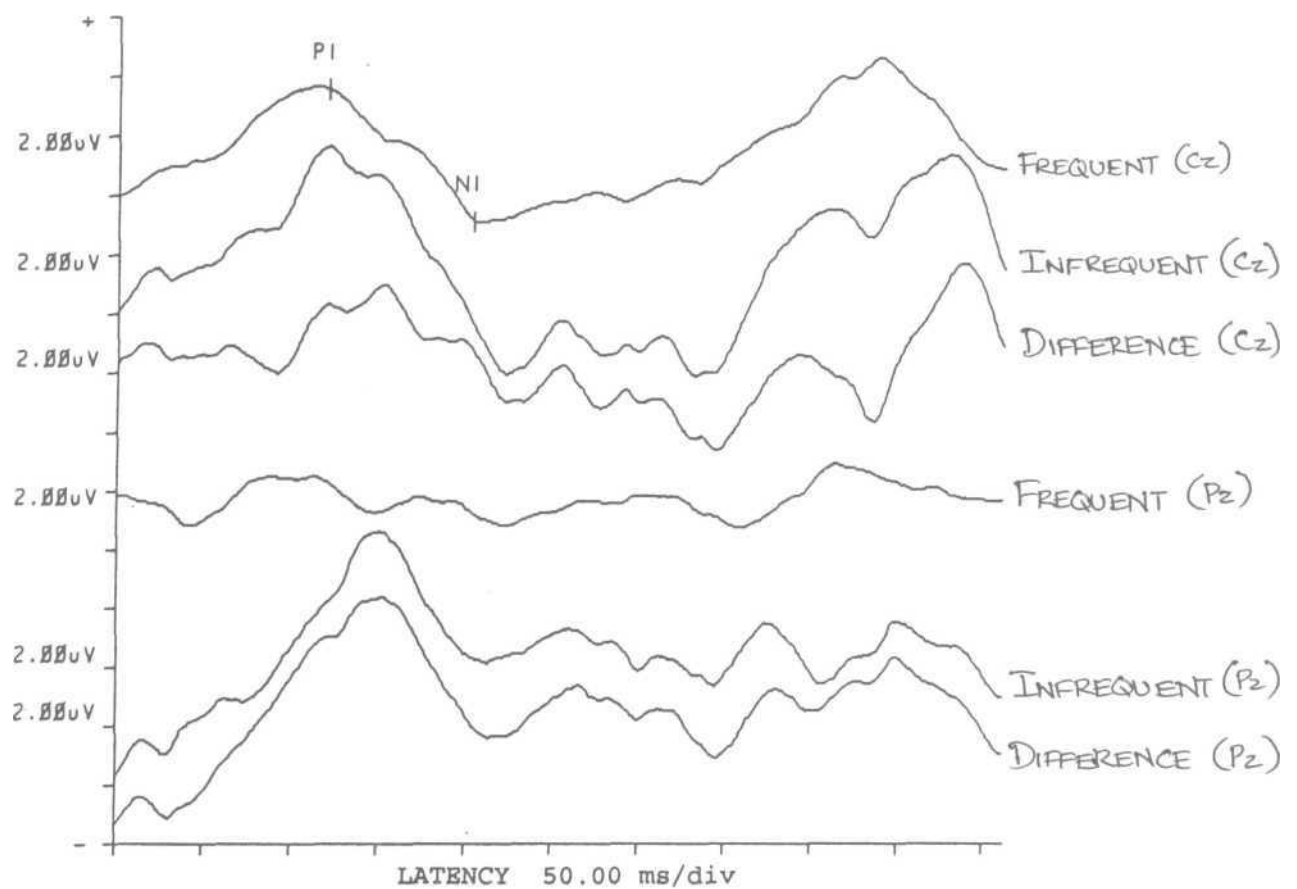

fig. 4-3. LLR and MMN waveforms of subject 4 for right ear stimulation
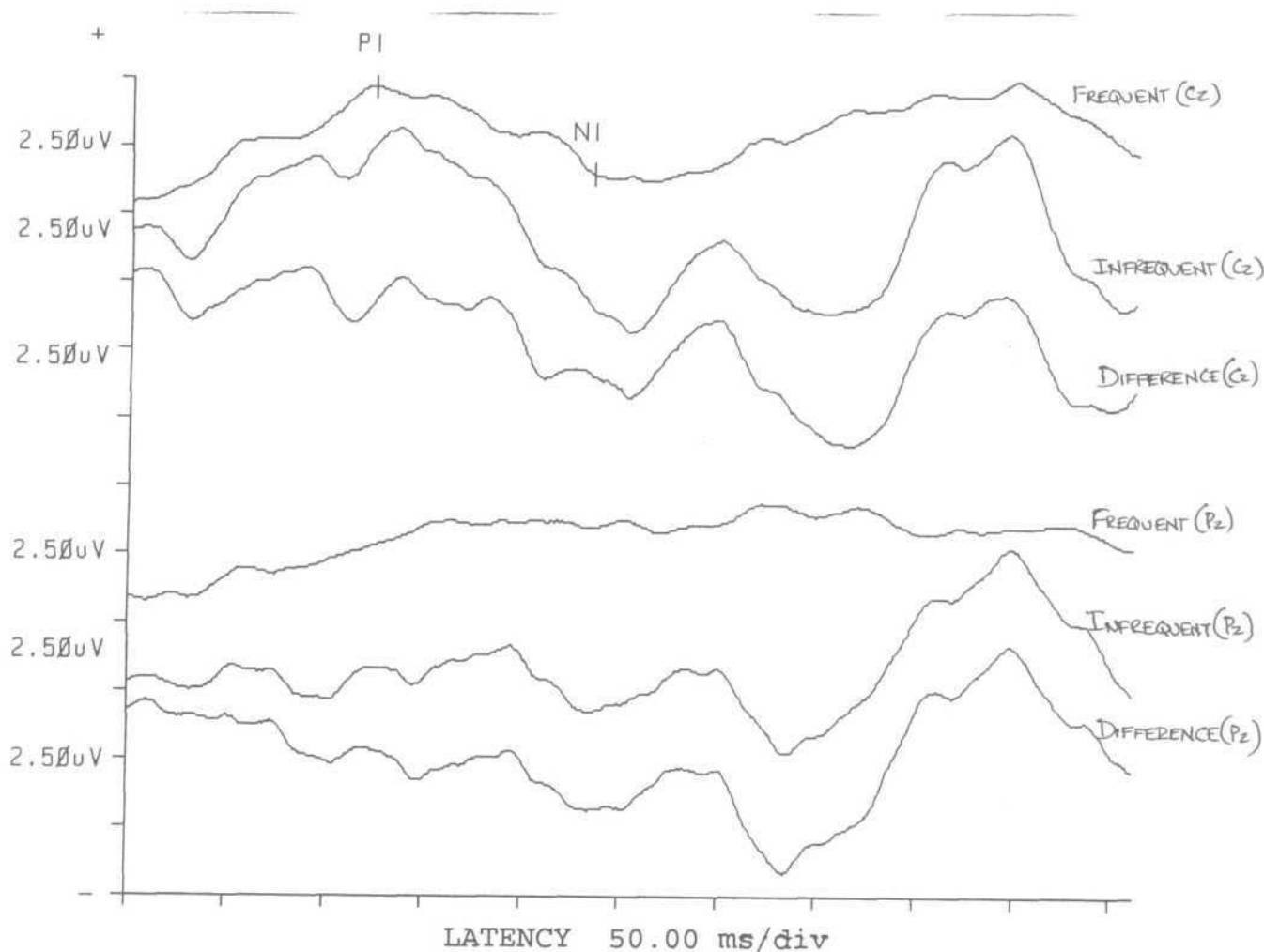
fig. 4-4. LLR and MMN waveforms of subject 4 for left ear stimulation

Subject: 5

a. History: This was a 6-year-old female with a history of middle ear discharge since the age of 4 years. No medical or audiological evaluation was done in these two years. Ear discharge was reported to be present since one year in both the ears. The ears were dry at the time of the testing. The child was prescribed medicine for the ear discharge on the day of testing.

b. Audiometric and immittance evaluation results: Pure tone audiometric results indicated near normal hearing sensitivity in both the ears (fig 5-1). Bone conduction threshold were also within the normal limits. But air bone gap indicated a conductive component in both the ears. These findings were supported by immittance evaluation showing 'B' type tympanogram in both the ears.
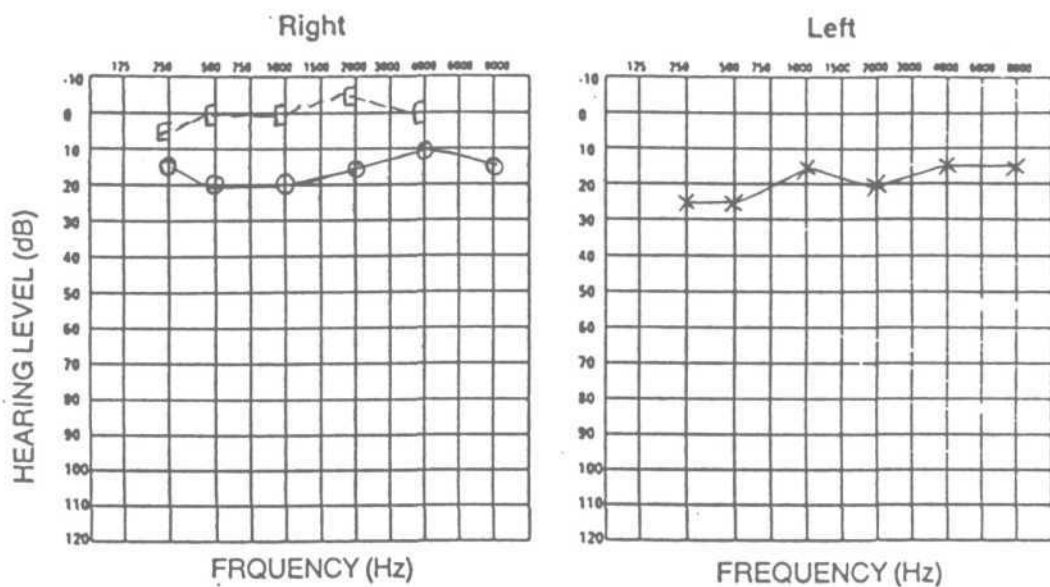
fig. 5-1. Audiogram showing pure-tone thresholds of subject 5

c. ABR result: For the clicks presented at the repetition rate of 11.1/sec, absolute latency of wave I, III and V was found to be prolonged. But the IPLs were within the normal range. The wave V/I amplitude ratio was also found to be normal in both ears. The shift in latency of wave V was normal when repetition rate was increased from 11.1/sec to 60.1/sec and 90.1/sec. The ABR waveforms for the right and the left ear are shown in fig 5-2.
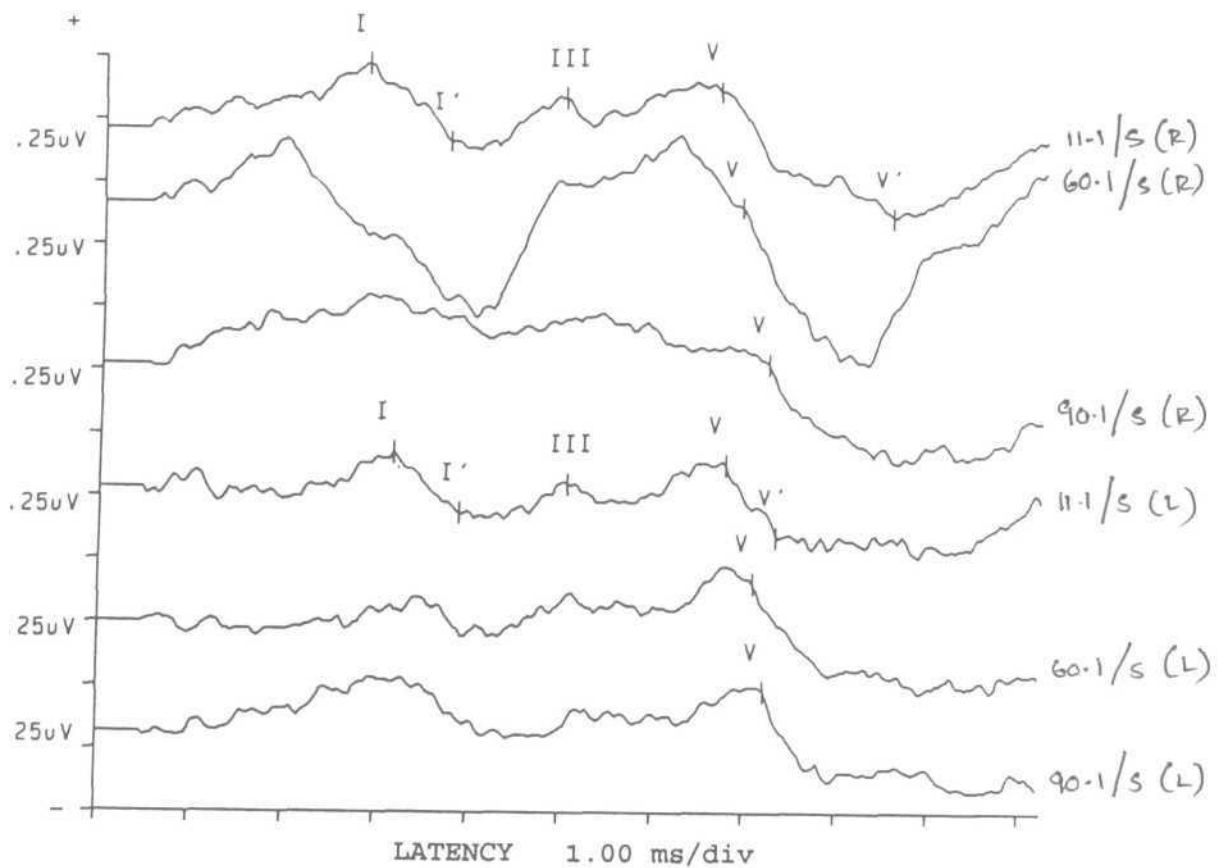
fig. 5-2 ABR waveforms recorded from subject 5

d. LLR results

In LLR waveform, only P1 and Nl could be identified for the right ear stimulation, whereas for the left ear PlI, Nl and P2 were present. When compared with normative data, latency of all the peaks was found to be prolonged in both the ears. The amplitude of N1-P2, which could be measured only in left ear, was within normal limits. The LLR waveforms for the right and the left ear are shown in fig 5-3 and fig 5-4, respectively.

e. MMN results: The latency and amplitude of MMN was normal for both the ears (fig 5-3 and 5-4). The duration of MMN, when compared to normals, was

found to be within normal range in the right ear for both Cz and Pz recording whereas it was within normal limits for Cz recording and was prolonged for Pz recording in the left ear.
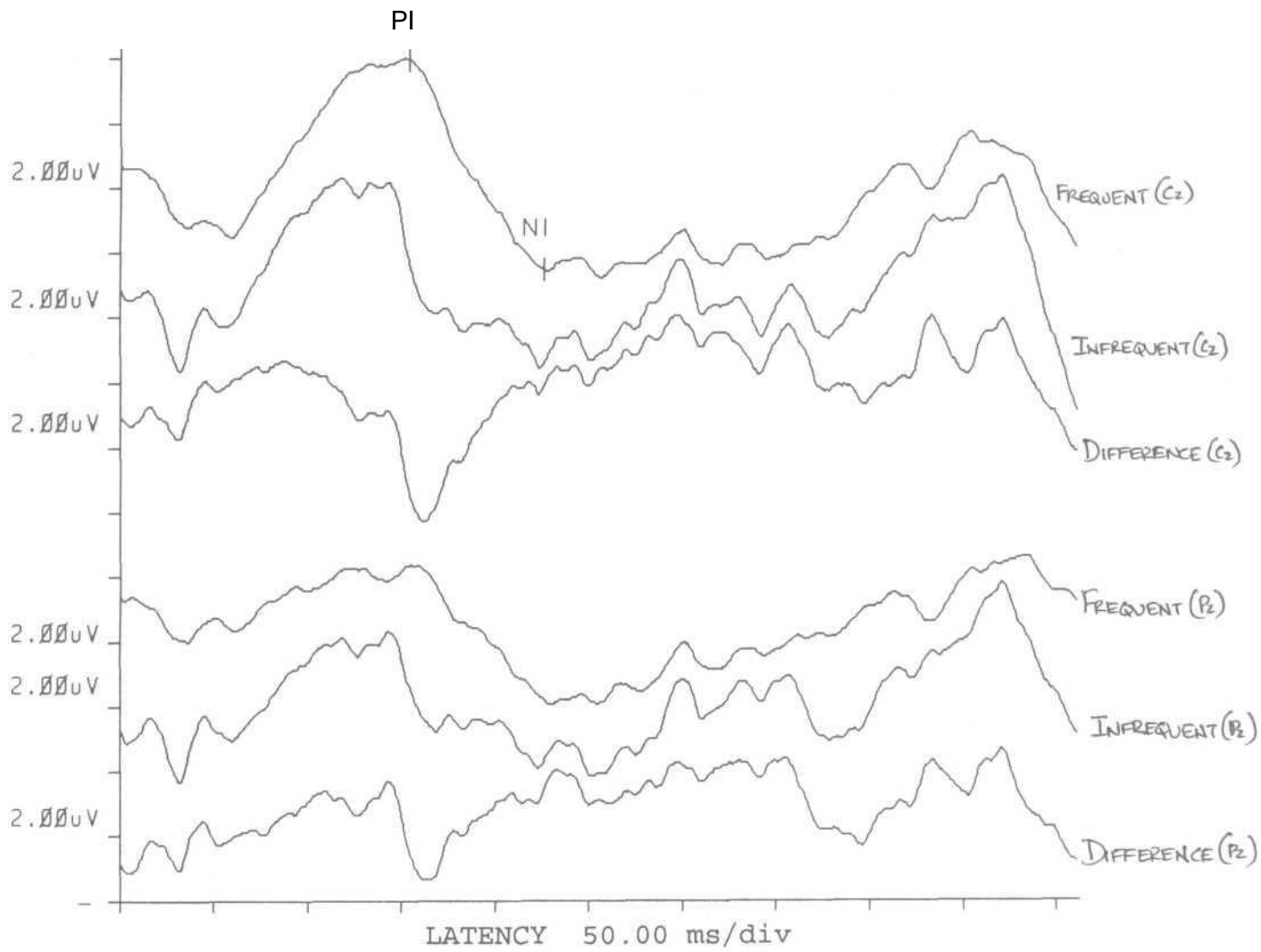
fig 5-3. LLR & MMN waveforms of subject 5 for the right ear stimulation
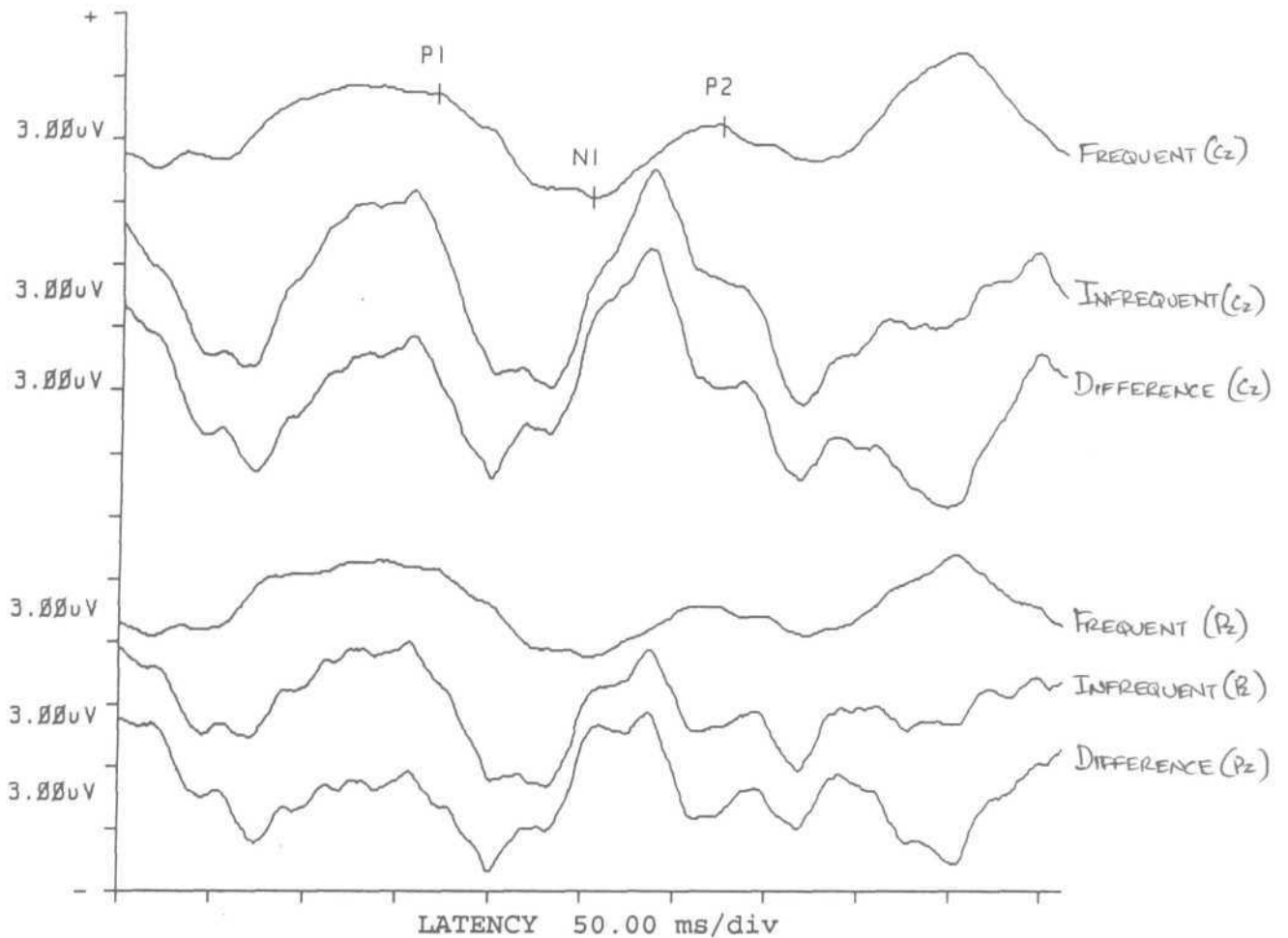
Fig. 5-4. LLR & MMN waveforms of subject 5 for the left ear stimulation

Subject 6

a. **History:** A 9-year-old-male reported with a history of OME in both the ears since childhood. As reported, the onset of otits media was around the age of 3-4 years. There was no family history of OME. At the age of 7 years, the child took medication for otitis media. Before medication, ear discharge occurred once in three months. The child was diagnosed as having bilateral

CSOM (inactive) with bilateral tympanic membrane perforation by an otolaryngologist. The ears were clean and dry during the testing.

b. **Audiometric and immittance evaluation results:** Both the ears had mild conductive hearing loss as shown by pure-tone audiometric results. The immittance evaluation showed 'B' type tympanogram in both the ears. The pure tone test results are shown in fig. 6-1.
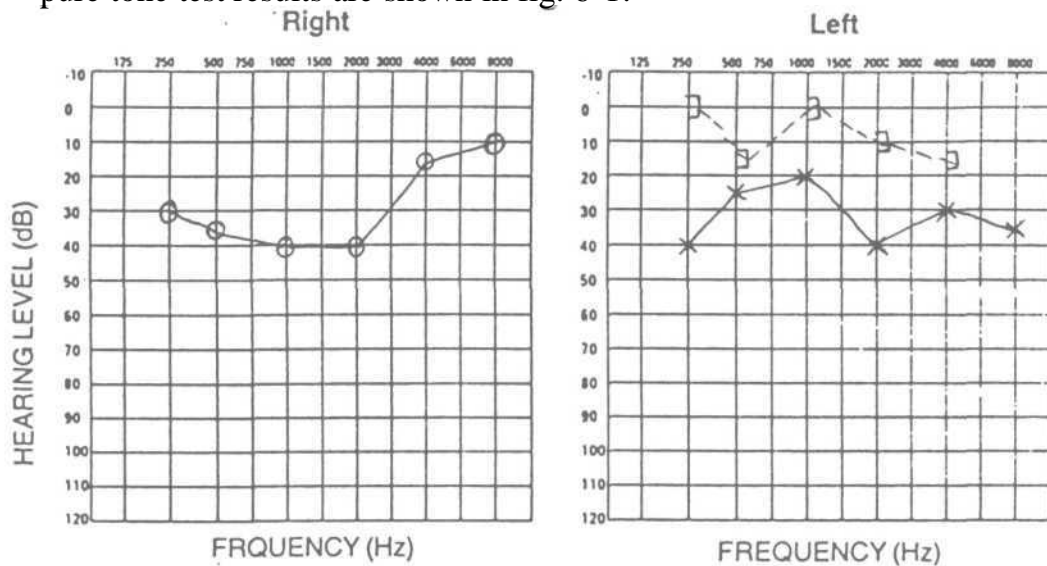
fig. 6-1 Audiogram showing pure-tone thresholds of subject 6

c. **ABR results:** The ABR results showed prolonged absolute latency of wave I, III and V for click presented with a repetition rate of 11.1/sec in both the ears except for wave III in the left ear. The I-III IPL in the right ear and I-V IPL in the left ear was slightly short. The III-V IPL was significantly prolonged in the right ear whereas in the left ear, III-V IPL was slightly prolonged. The I-V IPL was normal in the right ear but slightly shorter in the left ear. The wave V/I amplitude ratio was normal in both the ears. At higher repetition rate of 60.1/sec and

90.1/sec, shift in wave V latency was within normal limits in the right ear. In the left ear, wave V could not be identified due to poor waveform morphology. The ABR waveforms at different click rates are shown in fig 6-2.
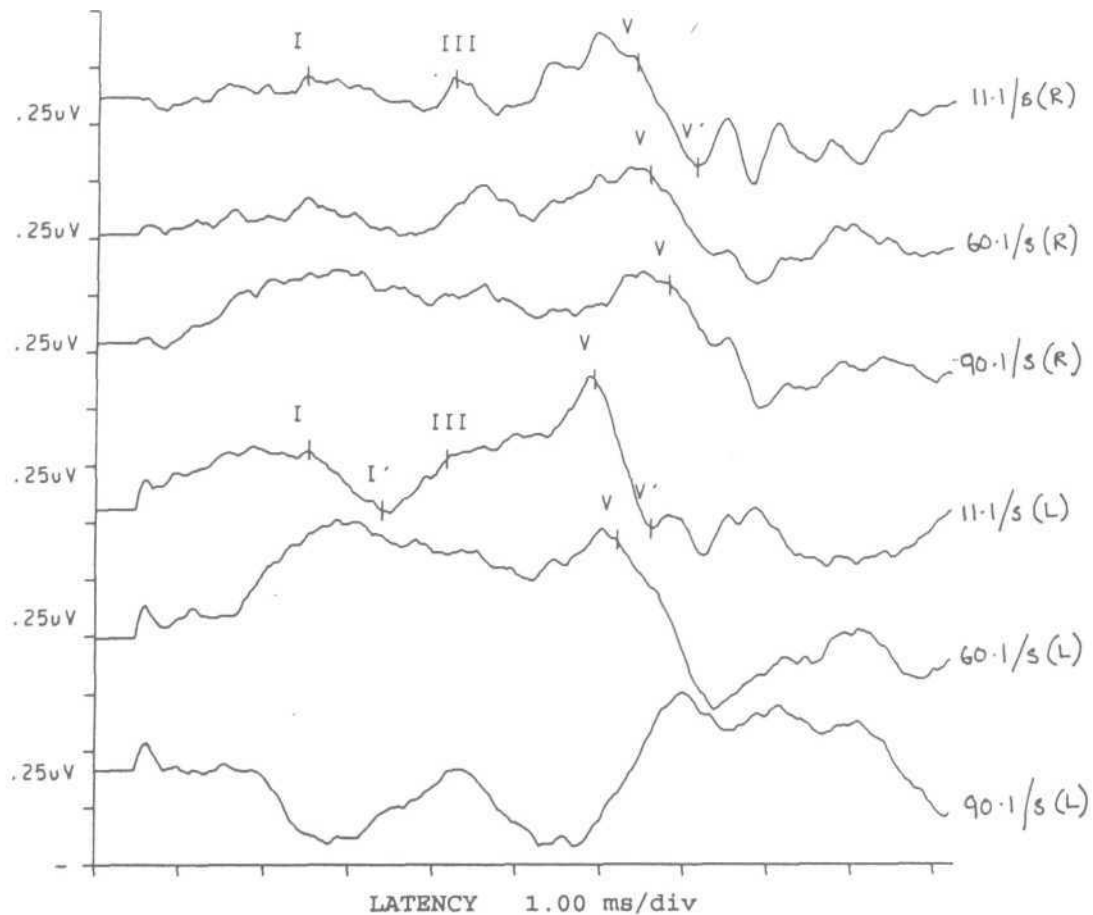
fig. 6-2. ABR waveforms recorded from subject 6

d. LLR results: All the LLR waves were found to be prolonged in both the ears. Only three LLR waves namely, PI, Nl and P2 could be identified in both the ears. The interaural comparison showed more delayed peaks in the left ear than the right ear. On the other hand,amplitude of Nl - P2 complex was larger in the

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left ear than the right ear. The LLR waveforms for the right and the left ear are shown in fig 6-3 and 6-4.

e. MMN results: The MMN recorded from the right ear stimulation showed prolonged peak latency whereas it was normal in the left ear (fig. 6-3 & 6-4). The duration of MMN was prolonged in the left ear. In the right ear, it was normal only for Pz recording. Amplitude of MMN was normal in both the ears.
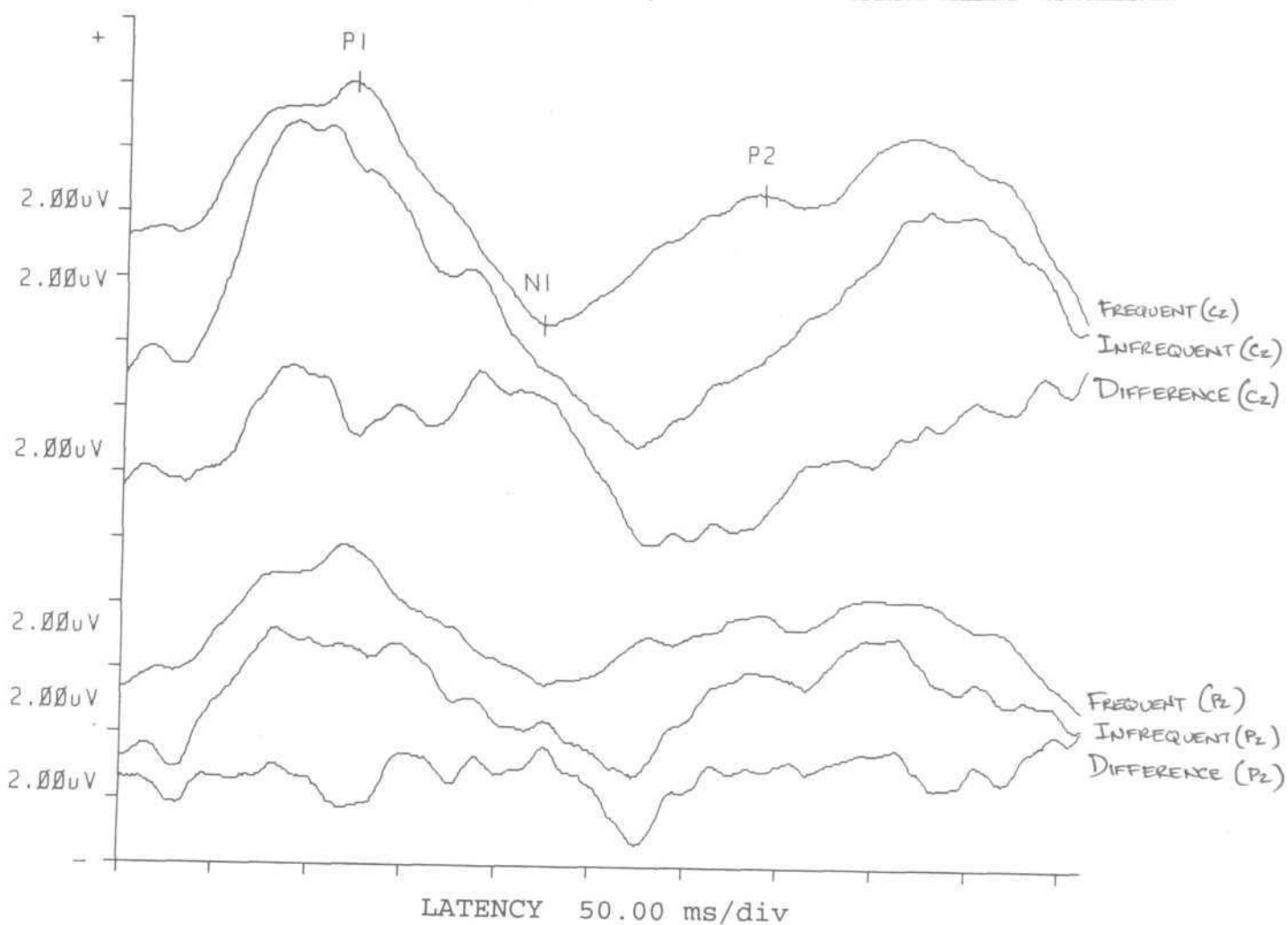
fig. 6-3. LLR & MMN waveforms of subject 6 for the right ear stimulation
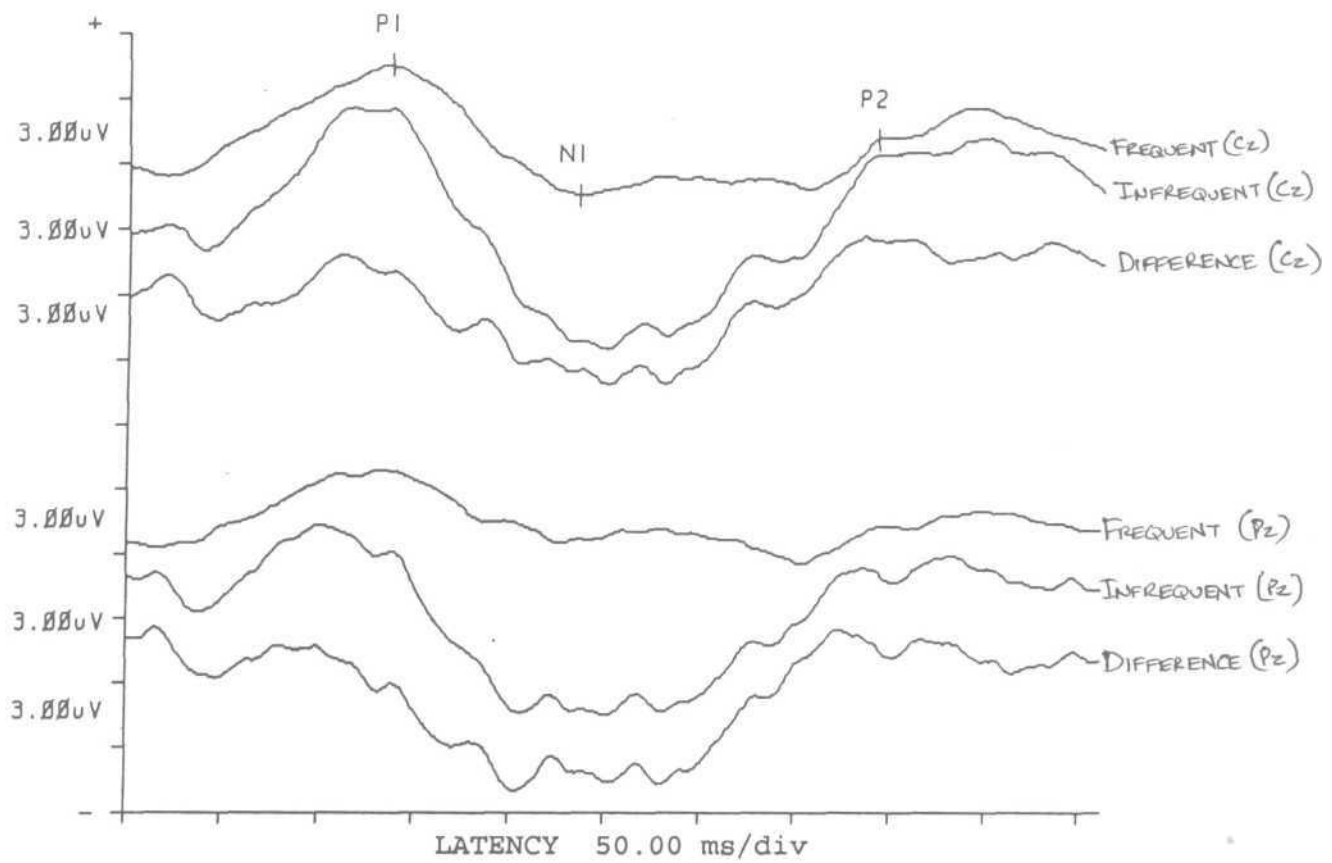
fig. 6-4. LLR & MMN waveforms of subject 6 for the left ear stimulation

Subject 7

a. History: This 6-year-old male subject came with the complaint of ear pain in both the ears. The ear pain was first noticed in the left ear one year back. Medicines were prescribed for 1 week after which, the pain was reported to the intermittent in both the ears. At the time to testing, the subject was under medication for ear discharge. He was diagnosed as having acute otitis media with unresolved suppurative otitis media in both ears by an otolaryngologist. He was recommended to undergo grommet insertion in both the ears with

adenotonsillectomy. Previous medical or audiological reports were not available at the time of testing.

b. Audiometric and immittance evaluation results: Pure tone audiometric results indicated a mild hearing loss in the right ear and a minimal hearing loss in the left ear. Bone conduction sensitivity was within normal limits. Middle ear evaluation revealed type 'B' tympanogram in both the ears. The results of pure tone audiometry are shown in fie. 7-1.
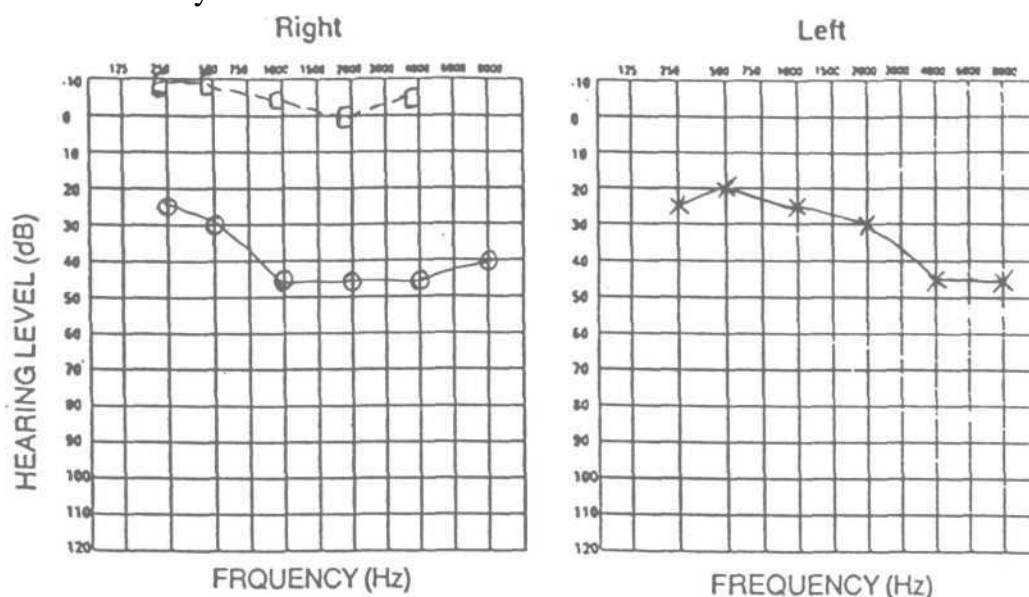
fig. 7-1. Audiogram showing pure-tone thresholds of subject 7

c. ABR results: Analysis was not possible in the right ear due to poor waveform morphology. This could be a result of large number of artifacts present in the recording. In left ear ABR waveform for the click rete of 11.1/sec, absolute latency of wave I, III and V were prolonged with normal IPLs. The amplitude ratio of wave V/I was normal. The shift in wave V latency was normal at

60.1/sec and 90.1/sec repetition rate. The ABR waveforms at different click rates are shown in fig. 7-2.
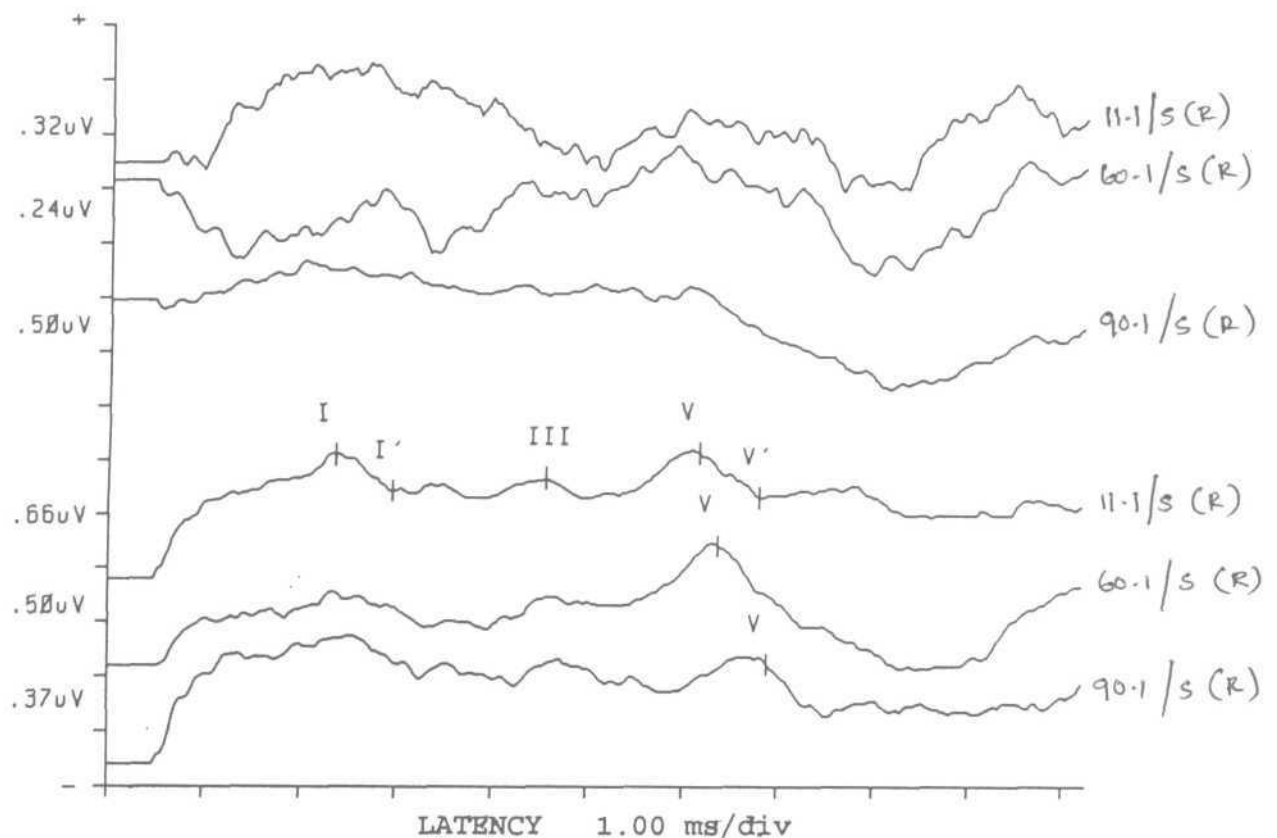
fig. 7-2. ABR waveforms recorded from subject 7

d. LLR results: The analysis of LLR waveform showed identifiable P1, N1, P2 and N2 in the right ear whereas the left ear waveform had only P1 and Nl waves (fig 7-3 & 7-4). Although the latency of all the waves was prolonged significantly, a close inspection revealed more prolongation in the left ear LLR components than the right ear. The Nl - P2 amplitude, which could be measured only in the right ear, was near normal value.

e. MMN results: The peak latency of MMN was within normal limits in both the ears. Amplitude of MMN was also found to be within normal range for both the ears. Discrepancy was observed on duration of MMN in Cz and Pz recordings. The MMN duration was normal in Cz recording but was abnormal in Pz recording of both the ears. The MMN waveforms for the right and the left ears are shown in fig. 7-3 and 7-4.
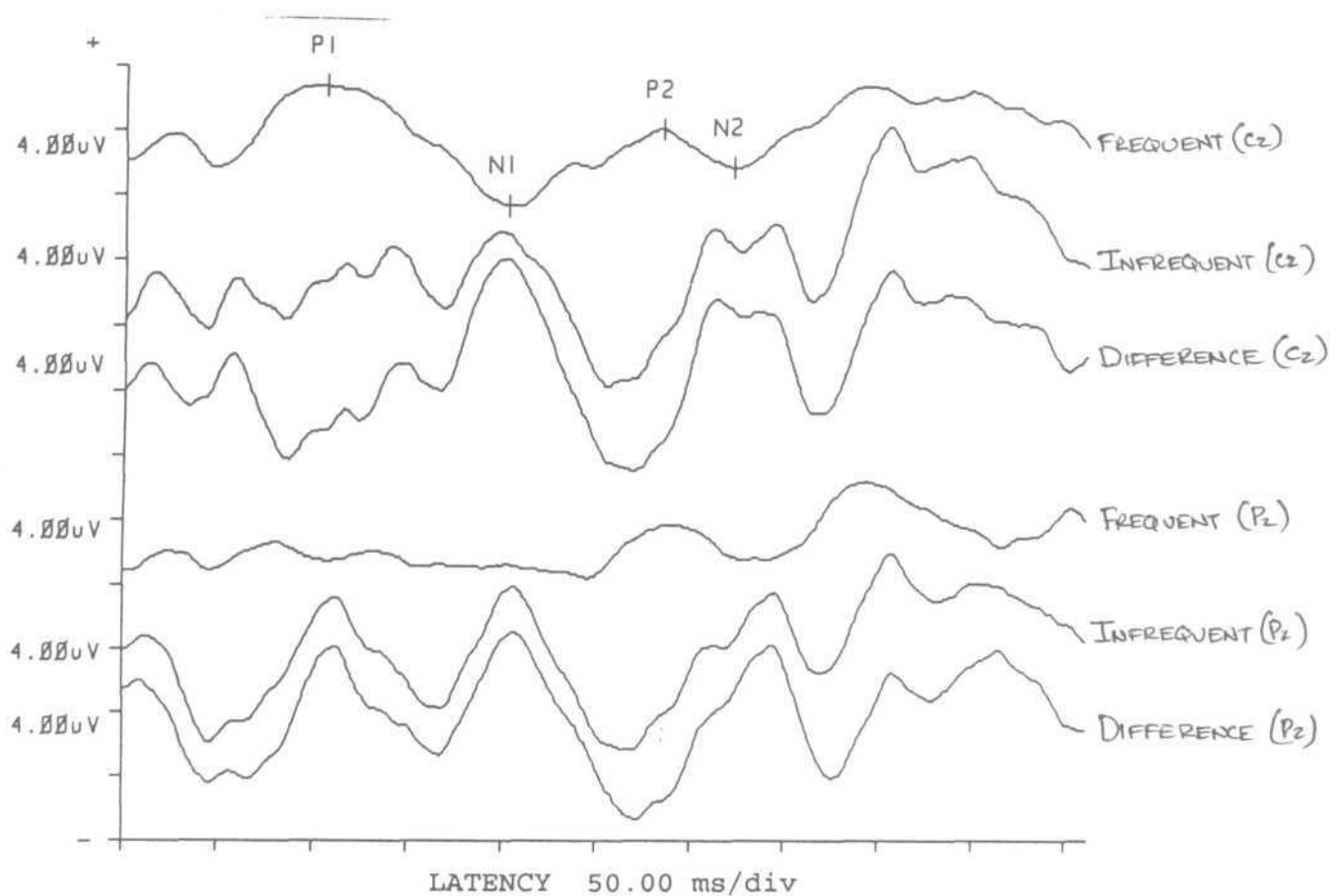
fig. 7-3. LLR and MMN waveforms of subject 7 for the right ear stimulation

fig. 7-4. LLR & MMN waveforms of subject 7 for the left ear stimulation

Subject 8

a. History: This subject was a 13-year-old male with the complaint of a hearing loss in both the ears since 2 years. He had a history of fall from the tree subsequent to which he was unconcious for half an hour. Later, there was bleeding from both the ears with giddiness, ear pain and tinnitus. He was under medication for 2 months for the same problem. At the time of testing, patient complaint of hearing loss in the left ear which was diagnosed chronic suppurative otitis media by an otoloryngologist.

b. Audiometric and immittance evaluation results: On pure tone testing the right ear showed moderate and the left ear showed a mild hearing loss. Bone conduction sensitivity was normal in both the ears. Immittance evaluation revealed 'B' type tympanogram in both the ears. Pure tone test results are shown in fig. 8-1.
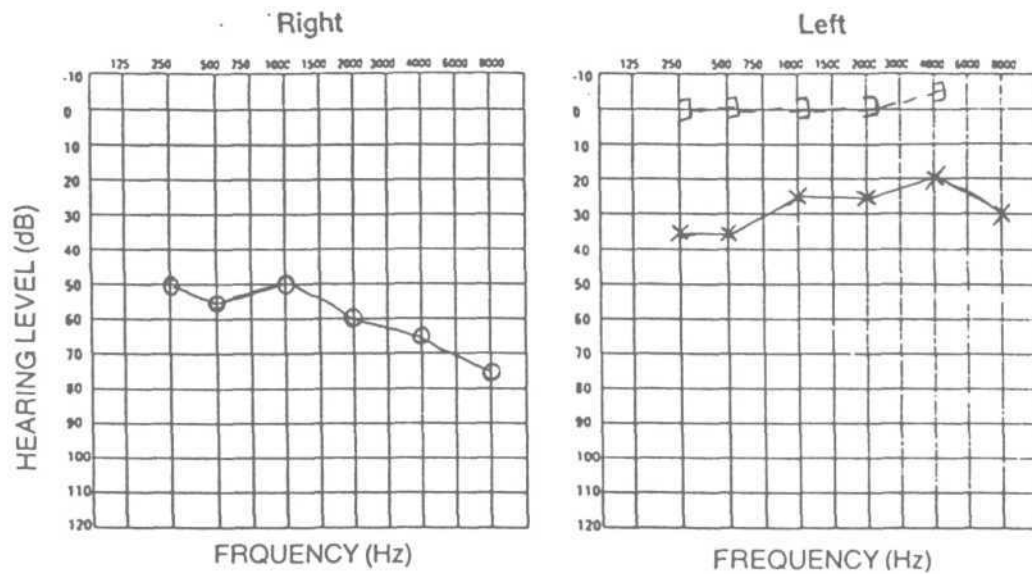
fig. 8-1. Audiogram showing pure-tone thresholds of subject 8

c. ABR results: When the clicks were presented at the repetition rate of 11.1/sec, only wave V could be identified in the right ear at latency of 7.28 ms. In the left ear, wave I and wave V were prolonged but wave III latency was normal. The amplitude of wave I was very small leading to a very high V/I amplitude ratio. When the rate was increased to 60.1/sec and 90.1/sec, the latency shift in wave V was within normal limits in the left ear but wave V could not be identified in the right ear. The ABR waveforms for different click rates are shown in fig 8-2.
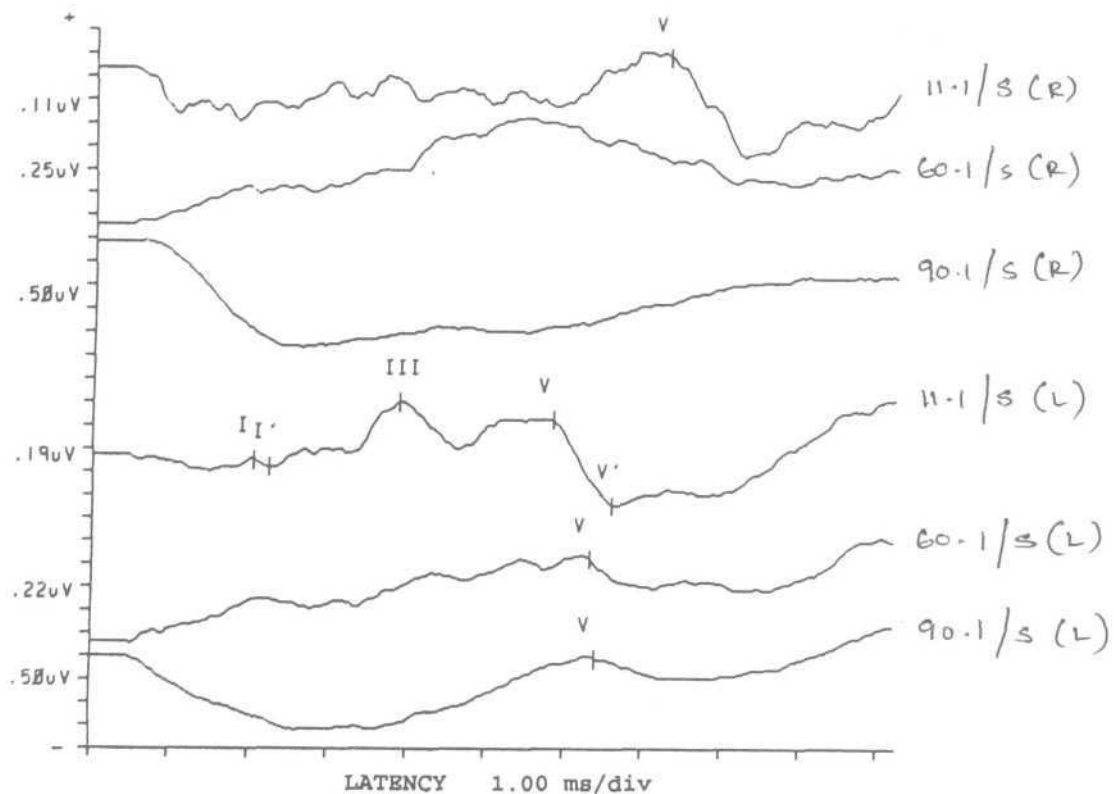
fig. 8-2. ABR waveforms recorded from subject 8

d. LLR results: P1 latency was within normal range in both the ears (fig. 8-3 &
8-4) but latency of Nl wave was abnormally prolonged for both the ears. The P2
was also prolonged in the right ear whereas it was absent in the left ear. The
amplitude of N1-P2 complex was normal in the right ear.

e. **MMN results:** The MMN was abnormal in the right ear with increased latency
in the Cz recording whereas it was absent in the Pz recording. The latency of
MMN in the left ear was within normal limits. The amplitude of MMN was
normal in the both ears. The duration MMN was prolonged in the left ear but was
found to normal in the right ear. The MMN waveforms for the right and the left
ear stimulation are shown in fig. 8-3 and 8-4.

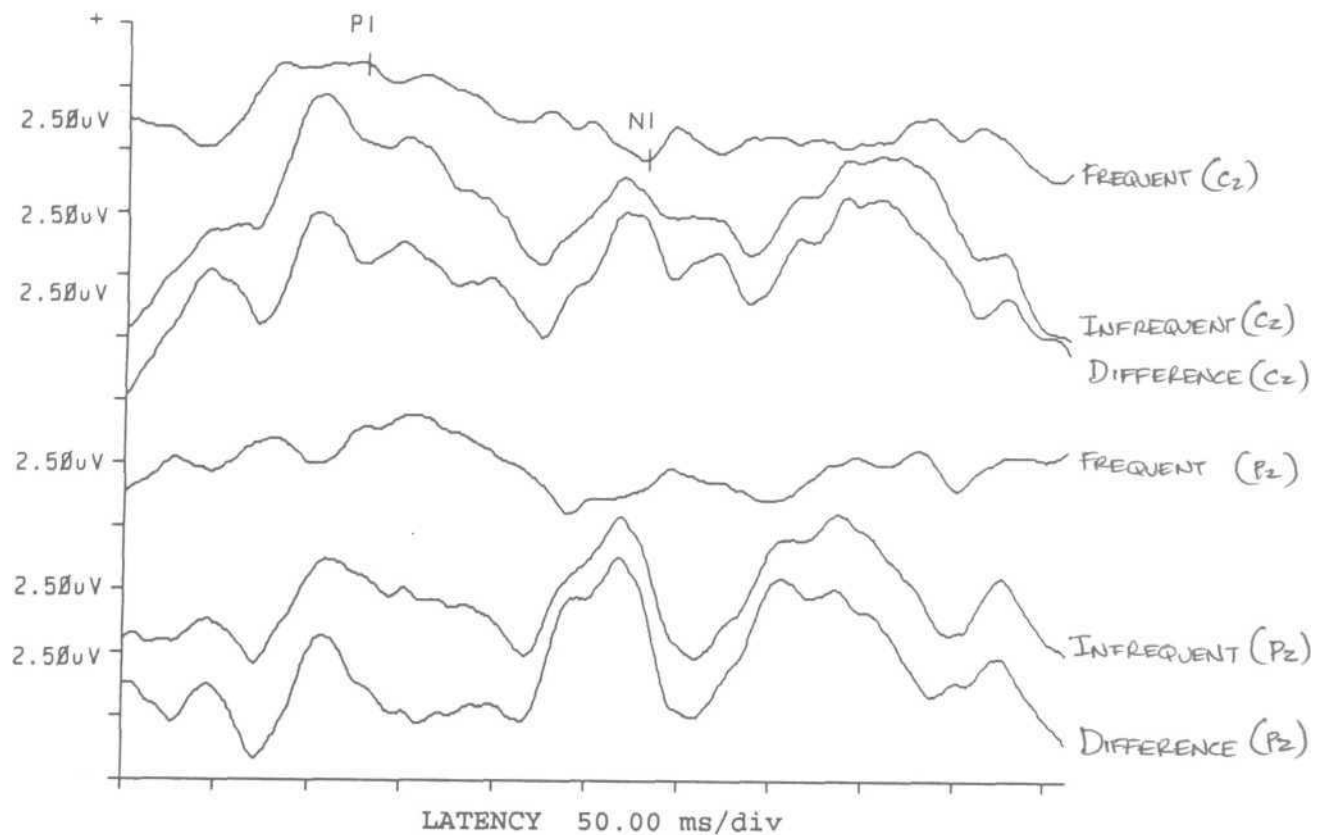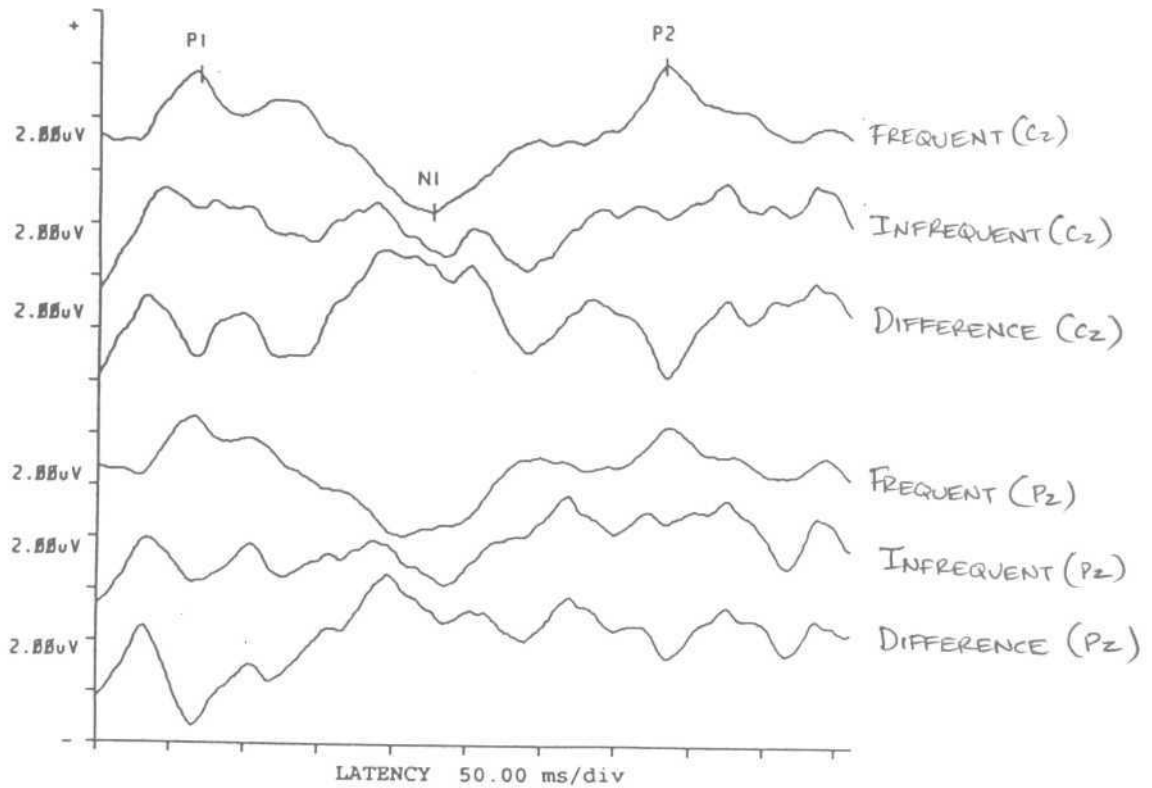fig. 8-3. LLR & MMN waveforms of subject 8 for the right ear stimulation.
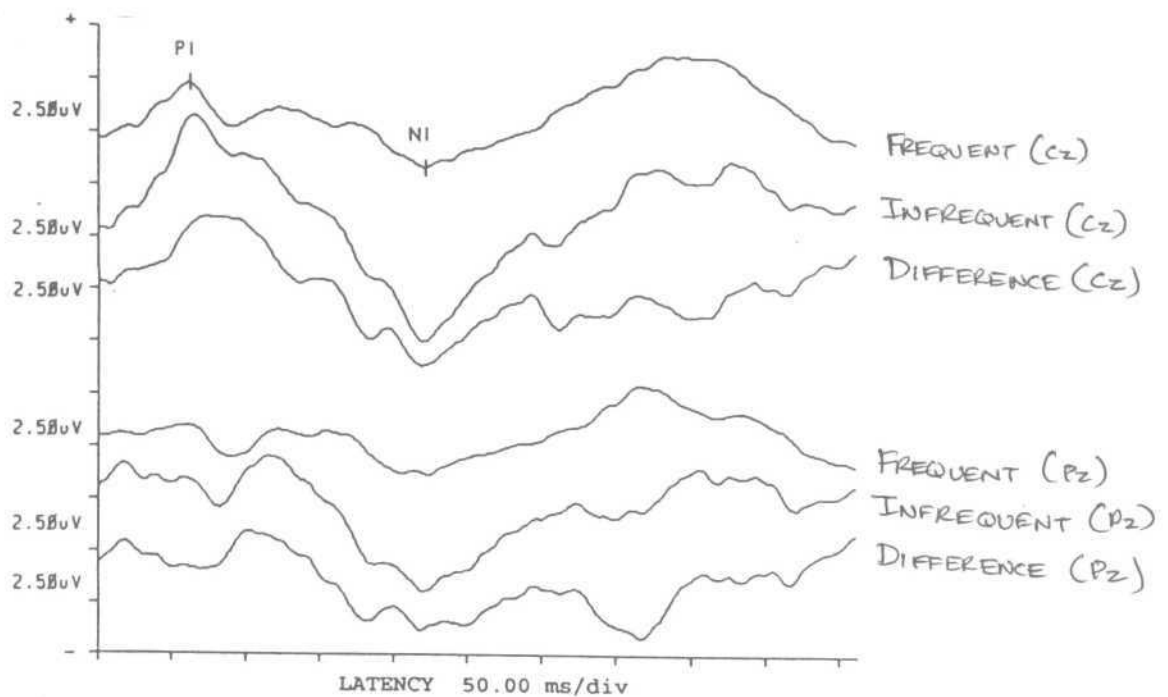
fig. 8-4. LLR & MMN waveforms for subject 8 for the left ear stimulation

DISCUSSION

The present study investigated the possible effects of fluctuating conductive hearing loss on central auditory processing by recording ABR, LLR and MMN. The study group was a small sample of children with history of unilateral / bilateral OME. Results were compared with the data reported from normal - hearing subjects (Guruprasad, 2000; Iyengar, 1999; Radhika, 1998 and Shankar, 1997).

ABR in children with history of otitis media with effusion

The various measures of ABR were recorded for clicks at different repetition rates viz. 11.1/sec, 60.1/sec and 90.1/sec. The results indicated prolonged absolute latencies of all the three waves (wave I, III and V) for clicks presented with a repetition rate of 11.1/sec for all the subjects. This finding can be attributed to the conductive loss present in the ear at the time of testing. According to Chisin, Gapany-Gapanavicius, Gafni and Sohmer (1983) a conductive pathology attenuates the sound reaching the cochlea, producing significant latency shifts and waveform changes in the ABR that are consistent with the effects of decreasing stimulus intensity level in normal subjects. The results of IPL measure were variable across the subjects. For subjects 2, 4 and 6, a paradoxically short III-V, I-V and I-III IPLs were found. This can be result of greater dispersion of absolute latency values around the mean. For eg : absolute

latency value of wave I at the upper limit of mean value and that of wave III towards the lower end of the range will result in shorter IPLs. But such findings has not been reported in literature. Out of 8 subjects, two had prolonged IPLs. Subject 2 had delayed I-III IPL in the left ear and subject 6 had delayed III-V IPL in both the ears. It may be noted that both these subjects had a long standing history of OME with onset at an early age. Subject 2 had onset of OME at 1 year of age. Subject 6 had early onset of otitis media around the age of 3-4 years as reported by the parents. There is a possibility of onset of otitis media even at an earlier age itself which might have gone unnoticed by the family members. These changes in IPLs, hence may reflect brainstem changes occurred due to auditory deprivation caused by OME. These results support the earlier studies which have indicated that children with history of OME may have significantly prolonged I-III and I-V IPLs (Folsom et al., 1983; Gunnesson and Finitzo, 1991; Hall and Grose, 1991). All these studies describe a small but significant delay in the I-III interval. In the study by Folsom et al. (1983) a significant delay in the III-V interval has also been reported. The results of the present study are in good agreement with these studies. Recently, Stollman, Snik, Schilder and Broek (1996) could not demonstrate any marked long term binaural auditory processing deficits induced by early asymmetric OME in man. These results support the normal findings on ABR found in a majority of subjects,

For clicks present with a repetition rate of 60.1/sec and 90.1/sec, the latency shift was normal for all the subjects except for subject 6 and 8. No wave V could be identified at 60.1/sec and 90.1/sec in left ear of subject 6. This rate effect can be a result of underlying neuronal abnormality due to a long history of OME. As discussed earlier the onset of otitis media was earlier in subject 6 when compared to other children. He also had abnormal IPL indicating brainstem dysfunction. Thus poor response to higher repetition rate may also be due to brainstem dysfunction. Absence of V wave in subject 8 may be due to a large conductive component. Even when the clicks were presenting at a repetition rate of 11.1/sec, latency of V peak was delayed in subject 6.

In general, these findings show that although the absolute latency of ABR waves were prolonged, the IPLs were within normal range in a majority of the subjects. The prolonged absolute latency of ABR waves can be due to presence of conductive loss at the time of testing. Several investigators in humans have indicated that early OME may affect auditory brainstem development and lead to changes in ABR (Lenhardt et al., 1985). If maturation of the auditory brainstem is most extensive during the first 1.5 years of life, then the time of onset of the OME would be of great importance. In the present study only subject 2, who had onset of otits media around 1 year of age and he had prolonged IPL. The results of previous studies showing abnormal ABR can be attributed to the fact that the children had their first bout of OME before 12 months of age. The abnormal

findings of ABR, delayed I-III and III-V IPLs, implicate wave III as a site of brainstem involvement. The cochlear nucleus contributes to wave III (Mollar, Jannetta and Bennett, 1981, cited in Lenhardt et al., 1985), and this nucleus is shown to have reduced neural volume when the ipsilateral ear had a conductive loss (Webster, 1983, cited in Lenhardt et al., 1985). A reduction in the neural cross-sectional areas or a change in physiologic response might be a consequence of partial auditory deprivation association with a conductive loss.

LLR and MMN in children with history of otitis media with effusion

Now considering the higher potentials, the morphology of the LLR waveforms showed great variability in the children. Latency of all the waves was prolonged when compared with the mean ± 2SD value for normal hearing subjects. On close inspection, of Nl wave was found to have maximum relative amplitude in a majority of the subjects followed by PI component.

The morphology of these obligatory auditory cortical potentials (Pl-Nl-P2) may indicate the effect of conductive loss on stimulus parameters. When the middle ear in filled with fluid, auditory sensitivity is reduced, since the transformer action is compromised. Otitis media can theoretically influence normal basilar membrane function. The membrane, which act as a filter, does not produce an instantaneous output, but rather has a rise time inversely related to its band width. If the sound energy transformed by the middle ear is consistently detected just above threshold (elevated by otitis media), then the prolonged rise

time could produce a time delay that would result in a neural delay reaching the first synapse in the brainstem (Lenhardt et al., 1985). These changes in the stimulus character may influence the morphology of LLR as well as ABR components depending on the site of dysfunction and make it difficult to compare with normative data collected on children with normal middle ear.

Another aspect which needs to be discussed in this context is the maturation of LLR components. In the present study, the P_1 and N_1 were the most consistently identified wave in all LLR recordings. Ponton, Don, Kwong, Waring and Eggermont (1998, cited in Ponton, Moore & Eggermont, 1999) showed that P1 and Nl potentials are the first to be present during early years of life and continue to mature until 5-10 years of life. Hence their presence should be expected with decreasing latency and increasing amplitude in normals as the age of the subjects increases. But latency and amplitude were abnormal in the present study indicating a deficit in the underlying mechanism responsible for LLR generation. At the same time, it should be borne in mind that subjects presented with different amount of air - bone gap at the time of testing, which can significantly alter the stimulus characteristics leading to abnormalities in these exogenous potentials.

It has been reported that N2 component is an endogenous response, and is an index of attention and discrimination tasks (Naatanen and Picton, 1986, cited in Tonnquist-Uhlen, 1996). The generators of N2 include sub-cortical and limbic

areas, or activity in the frontal lobes that may represent switch of attention (McCarthy et al., 1989, cited in Tonnquist-Uhlen, 2000). The absence of N2 observed in the present study may indicates a possible disorder of attention and discrimination in these children. The prolonged latency of LLR components may be an effect of delayed or disturbed maturation due to conductive loss. The fluctuating conductive loss may disturb the maturational process by causing auditory deprivation, and strech it into the adolescence period too. In such an instance, we tend to find immature LLR response even in later periods.

Though it is difficult to account for the prolonged LLR latency solely as a result of conductive loss or maturational delay or an interaction of both, the prolongation of latency of LLR in almost all the subjects, supports the maturational delay as a major contributing factor to these LLR abnormalities. Also the absence of N2 component provides additional support for this hypothesis. A comparison between the subjects of same age in the present study, showed that the frequency of otitis media attacks and age of onset was directly related to LLR abnormalities. Eg. subject 5 has longer LLR peaks when compared with subject 7 even though both were of same age. This could be a result of longer and more severe history of OME in subject 5 compared to subject 7.

Investigations studying the effect of otitis media with effusion during childhood on MMN could not be traced in the literature. In the present study, out

of eight subjects, only subject 2 and subject 3 had prolonged latency of MMN for both the ears stimulated separately. Both of these subjects had long term history of OME. Subject 2 (aged 10 years) had initial onset of OME at 1 year of age while subject 3 (aged 10 years) had OME since the age of 5-6 although only in right ear. Hence we can infer a relation between the prolonged MMN latency and duration of OME from these subjects.

The amplitude of MMN was found to be normal in all the subjects. Among the three measures of MMN, duration was found to be abnormal in most of the subjects. Duration of MMN was greater than normals in either 1 or both the ears. Although a definite relation between the duration of OME and the duration of MMN could not be obtained, subjects with OME for longer period of time did show relatively longer duration of MMN. Unilateral / bilateral hearing loss could not be related to MMN abnormality on duration parameter. The MMN duration was found to be more even when the subjects had unilateral hearing loss since its onset eg: subject 6. The MMN was absent in Pz recording of the right ear in subject 2 and 8 while it was present in Cz recording. Such findings showing presence of MMN in recording from one electrode but absent waveform from other electrode indicates, deviance in the scalp distribution of the response. Kraus et al., (1993) have shown deficits correlated to an abnormal MMN in patients with CAPD. Radhika (1998) found absent MMN for frequency derivation in three of the LD subjects. Increased latency has also been reported

by other investigators (Koripilahti and Lang, 1994, cited in Shafer et al., 2000; Radhika, 1998). But in the present study, prolonged latency of MMN was not seen consistently in all subjects instead it was duration which was maximally abnormal across the subjects. The duration of MMN has been a strong indicator of abnormality in underlying process. Increased MMN duration probably reflect delay in memory trace in time (Naatanen, 1992, cited in Lang et al., 1995) indicating a prolonged processing duration.

The overall, results of MMN in these patients are grossly variable, this can be due to following factors. First, OME in most cases causes a fluctuating conductive hearing loss which indicates that there is an intermittent, partial auditory deprivation to the higher cortical centers. Binaural stimulation via bone conduction (eg. own voice) can have ameliorating effects on auditory deprivation due to conductive hearing loss (Stollman et al., 1996). This means that some auditory input of extraneous sounds is possible. Hence the patient in never totally deprived of the auditory stimulation as in case of profound hearing loss or ablation of cochlea. Second, the cortical change expected to occur due to auditory deprivation may have been compensated by neural plasticity that allows widespread and rapid changes in brain organisation in early life and less widely spread and slower change in later life (Knudsen, Knudsen and Esterly, 1984, cited in Hogan, Meyer, & Moore, 1996).

In conclusion, though there was a lot of variability in the results, it can be observed that brainstem dysfunction can occur if the onset of otitis media with effusion was very early (eg. subject 2 and 6) whereas abnormalities on LLR and MMN were seen even when the onset was late. This late onset conductive loss might have interfered with the normal process of maturation by causing auditory deprivation during the developmental period. Such changes at lower as well as at the higher cortical centers are thought to be major cause of CAPD.

SUMMARY AND CONCLUSION

Auditory deprivation in early life has been shown to give rise to central auditory processing disorders. Otitis media with effusion is a condition which results in auditory deprivation due to fluctuating conductive hearing loss. Speculations have been expressed of the effect of early onset otitis media on central auditory system. Various behavioural as well as eletrophysiologic tools have been employed to investigates the different auditory processes in children with central auditory processing disorders. The electrophysiologic tests are not only supplementary to the behavioural test result but are also more objective and free of linguistic boundaries.

Though it has been established that children with recurrent attacks of otitis media with effusion may develop central auditory processing disorder due to auditory deprivation. Literature regarding electrophysiologic investigations on children with history of otitis media with effusion is scares.

Hence, the present study was taken-up to investigate central auditory processing in children with history of otitis media with effusion using ABR, LLR and MMN for intensity deviations. ABR was recorded at repetition rate of 11.1/sec, 60.1/sec and 90.1/sec. The LLR and MMN was were recorded at the repetition rate of 3.1/sec. Stimulus was presented at 50dBSL. These potentials were recorded using Bio-logic evoked potential system (navigator). Eight

children in the age range of 6-13 years with history of otitis media with effusion were tested. The various parameters measured from ABR waveform were absolute latency of wave I, III, and V, interpeak latency of I-III, III-V and I-V and V/I amplitude ratio. In LLR waveform absolute latency and amplitude of PI, N1, P2 and N2 were measured from, the frequent waveform of MMN in Cz recording. The MMN analysis included peak latency of MMN, amplitude and the duration of MMN. The diagnosis of conductive hearing loss was established based on pure-tone audiometric results and the immittance evaluation.

The analysis of the results revealed that majority of the children had normal ABR results as reflected by normal interpeak latency. Abnormality in ABR indicating brainstem dysfunction was noticed in only two subjects. The onset of otitis media was earlier in these two subjects when compared to others.

Both the exogenous (P_1, N_1 & P_2) and the endogenous potentials (N_2 and MMN) were found to be abnormal on one or the other parameter for all the 8 subjects. The LLR waves were prolonged for all the subjects even though ABR was normal in some of the subjects. This indicates a abnormal process underlying the LLR generation mechanism. For MMN, duration was the most commonly affected parameter followed by peak latency. The amplitude measure was normal for all the subjects.

To conclude, even though the results of all the three potentials showed wide variability in this population of children, there is a trend towards the underlying deficit in central auditory processing. Based on the results, these deficits seem to be more at the higher cortical levels as reflected by abnormalities on LLR and MMN with normal ABR results. The duration since the onset and age of onset of otitis media with effusion also showed a weak agreement with the abnormalities evident on these tests.

Future Directions

1. Present study considered a very small group of children which was a heteropgeneous group. Future studies can select a large sample to form a homogenous group for the study.

2. It was difficult to control the variables like onset of otitis media duration of attacks, number of attacks and degree of hearing loss etc, in the present study and therefore further studies can be taken up to investigate the effect of each of these variables on central auditory processing.

3. Normal hearing subjects with history of otitis media need to be evaluated in the further studies.

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