# EVALUATION OF SOME ASPECTS OF AUDITORY TEMPORAL PROCESSING DEFICITS IN SUBJECTS WITH CONDUCTIVE HEARING LOSS

Reg No. A0390010

A DISSERTATION SUBMITTED IN PART FULFILMENT OF THE MASTER'S DEGREE (AUDIOLOGY), UNIVERSITY OF MYSORE, MYSORE

> ALL INDIA INSTITUTE OF SPEECH AND HEARING MANASAGANGOTHRI MYSORE-570006

> > **MAY-2005**

# Dedicated to..... My dearest Mom and dad, & My dear god, "Sri Sadguru Saibaba"

# CERTIFICATE

This is to certify that this dissertation entitled "**Evaluation of some aspects of auditory temporal processing deficits in subjects with conductive hearing loss**" is a bonafide work in part fulfillment for the degree of Master of Science (Audiology) of the student (Register No. A0390010).

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# CERTIFICATE

This is to certify that this dissertation entitled "**Evaluation of some aspects of auditory temporal processing deficits in subjects with conductive hearing loss**" 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 "**Evaluation of some aspects of auditory temporal processing deficits in subjects with conductive hearing loss**" is the result of my own study under the guidance of Mr. Animesh Barman, Lecturer in Audiology; All India Institute of Speech and Hearing, Mysore and has not been submitted earlier in any other university for the award of any Diploma or Degree.

Mysore

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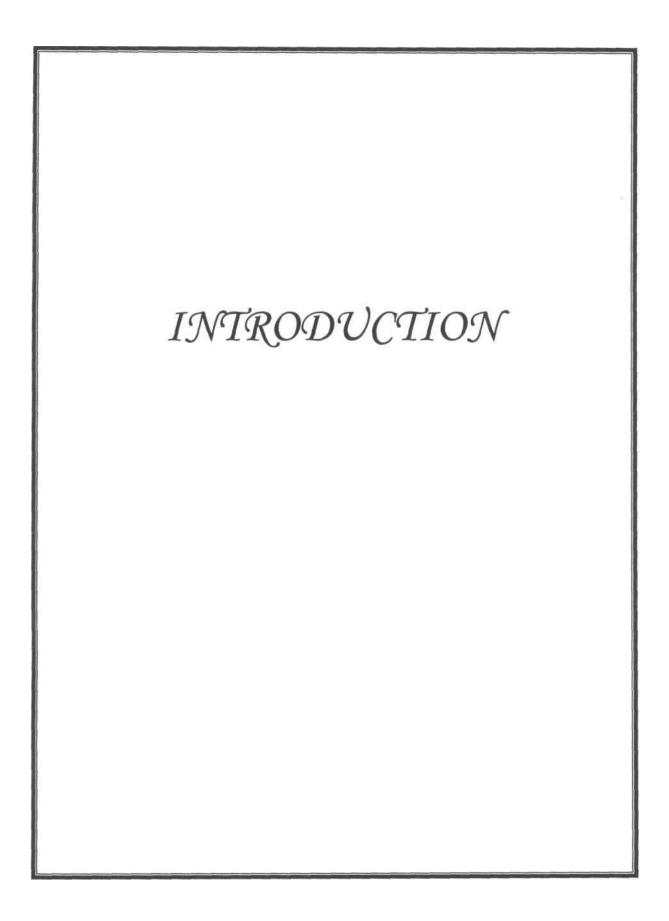
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### INTRODUCTION

Otitis media is a common and fairly ubiquitous condition of early childhood with the prevalence of otitis media greatest in the first three years of life (Teele, Klein, and Rosner 1980). Nearly all children have at least one episode of acute otitis media (AOM) or otitis media with effusion (OME), and at least 80% of children have three or more episodes before 3 years of age. OME typically causes hearing loss that lasts as long as the fluid persists and has been hypothesized to disrupt children's ability to process language at a rapid rate, affecting both comprehension and production in phonology, vocabulary, syntax, and discourse

Infants show higher prevalence rates than children in elementary school (Howie, 1975; Daly et al, 1998). Episodes of OME can resolve spontaneously, although many children experience persistent OME for prolonged periods.

The median number of days before resolution of OME was 72 days as reported by Roland and colleagues (1989). Most pre-school-and school-aged children who develop OME will experience the disease for more than two months before spontaneous resolution (Casselbraudt et al, 1985). In any case, when OME is present, one associated factor is some degree of conductive hearing loss. Severity can vary from mild to moderate degree (kokko, 1974, fria et al, 1985; hunter, 1993).

Until 1960's it was believed that ears with middle ear pathology behaved like "plug in the ear". Thus resulting hearing loss only attenuates the energy reaching to the cochlea, does not affect the physiology of higher auditory system and therefore signal processing remains intact. The effect of conductive loss is not just limited to attenuation of overall energy but it may have effects on higher central auditory nervous system at least when the pathology is long lasting one was reported by Dobie and Berlin, 1979, Webster and Webster 1979, Gunnarson and Finitzo, 1991.

Apart from its deleterious effects on language development, children with histories of recurrent otitis media with effusion (OME) may be at risk for abnormal auditory development. Impact of OME on central auditory processing have effects on functions believed to originate in the lower brainstem and require equal (binaural) hearing by the two ears.

Although the specific relation between OME in children and auditory/linguistic development remains unclear, there is some association between OME and deficits in language, cognitive skills, or attention (Brandes and Ehinger, 1981; Roberts et al, 1986; Sak and Ruben, 1982; Teele, Klein, and Rosner, 1980; Zinkus and Gottlieb, 1980).

Some studies involving central auditory function (higher order tasks) have reported an association between early OME history and complex auditory tasks, such as understanding speech in fluctuating background noises. Also, histories of hearing loss due to OME after age 3 years were associated with poorer high-frequency hearing as reported at the University of Minnesota by Lisa Hunter and colleagues (1993).

Conductive loss extended over a period has a potential impact on speech perception, Dobie and Berlin, 1979. Also, a mild conductive hearing loss significantly impacted speech perception in normal-hearing adult subjects, both in quiet and background noise as reported by Crandell and Flannagan, 1998. Thus, one can expect larger impact in young children who are developing speech and language skills. The general consensus among hearing scientists thus is that long-standing conductive pathology and resulting hearing loss could have deleterious effects on structure and function of higher central auditory system. Inconsistent and decreased auditory stimulation during the sensitive period of auditory pathway maturation may lead to permanent disturbance, damage or changes of these pathways, which might result in deficits of various types, such as, temporal processing deficit, integration deficit, so on and so forth. These deficits in the auditory processing can be studied using several tests.

Such changes or deficiencies can be evaluated using psychoacoustic tests such as masking level difference (MLD), temporal processing tests, and also higher potentials such as MLR, LLR, MMN and P300 (Jerger, 1992).

MMN, which is one of the most efficient electrophysiological tests to find out the fine grade discrimination ability of the individual, can be elicited by any discriminable change of a repetitive sound differing in terms of frequency, intensity, duration, or even complex phonemic changes. Currently MMN is being used as a powerful tool to evaluate automatic processing of such features in the CNS. Tyagi, 2002 has observed prolonged peak latency and abnormal MMN duration in children with otitis media.

Although such many tools are available to assess the processing deficits, very few are satisfactory for effective diagnosis.

### **NEED FOR THE STUDY:**

It is evident now that auditory deprivation in early developmental period due to OME may lead to APD. They are shown to have abnormalities at the brainstem level as reflected by ABR and are also suspected to have higher-level dysfunction. Cortical processing of temporal aspects, such as duration, is reported to be an important parameter for speech perception, which could be disturbed in these children. The children who are identified early can be provided with early intervention and thus would help in significantly better performance. Hence, processing deficits should be identified at an early age and thus would help to give appropriate training.

Several electrophysiological studies have taken neurological conditions affecting brainstem and higher level dysfunction. But very fewer studies have been undertaken for the evaluation of functioning of auditory system in individuals with a history of OME. Also, there is a dearth of literature about the psychophysical aspects of the duration discrimination in subjects with conductive hearing loss. Thus, this indicates a need to study processing of temporal aspects in conductive hearing loss using both psychophysical and electrophysiological tests.

#### AIM OF THE STUDY:

Thus, the present study is designed to investigate:

- 1. Whether some aspects of temporal processing are affected in individuals with conductive hearing loss?
- 2. If it is affected, how is it different in conductive hearing loss population from normals?
- 3. Is there any difference exist in subjective and objective measurement of temporal processing in individuals with conductive hearing loss and normal hearing?

4. Does duration and severity of conductive hearing loss has any correlation with temporal processing deficits?



#### **REVIEW OF LITERATURE**

Hearing loss of postnatal onset is reported by Shimizu (1976), to be the primary cause in 50% of the hearing impaired children under 19years of age. In this group, the most common disorder was otitis media with an incidence of 33.4%, and it is reported to be the most commonly occurring childhood disorder (Teele, D. W., Klein, J. O., and Rosner, B. A. 1980; Klein, J., Chase, C., Teele, D., Menyuk, P., and Rosner, B.1988; Roland et al 1989). The presence of fluid in the middle ear due to otitis media (SOM) generally results in a hearing loss.

The hearing loss associated with OME is temporary, but some times persistent or recurrent, and variable (fluctuating) in degree (Bess, 1986; Fria, Cantekin, and Eichler, 1985) and symmetry (Gravel and Ellis, 1992; Hall and Grose, 1993a). In theory, such a reduced and fluctuating input signal could make learning the auditory linguistic code challenging for some children.

The wide spread existence of recurrent OM and associated conductive hearing loss in children who are at a critical age for acquiring speech and language has recently received considerable attention because of its possible relation to linguistic and cognition development.

In addition to uniformly dampening the input, the conductive nature of the hearing loss may distort the speech signals, resulting in significant periods of abnormal incoming information during the perception of speech.

Studies on the effects of otitis media on hearing have shown the effects of early auditory deprivation on central auditory processing, development of auditory neurons, and discrimination of sounds (Finitzo et al., 1988; Paden, 1994). Finitzo et al. (1988) studied the long term effects of middle ear effusion in children and found that the hearing loss due to middle ear effusion causes deficits in processing speech and non-speech aspects of the auditory signal.

In a study by Eimas and Clarkson, 1986, showed that 5-year old language delayed children with significant histories of OM had considerably more difficulty than a matched group of control children in using phonetic information for processing the voicing distinction in syllable initial stop consonants.

Paradise et al., 2000, reported that persistent early-life otitis media actually causes later small, circumscribed impairments of receptive language and verbal aspects of cognition in certain groups of children or that unidentified, confounding factors predispose children both to early-life otitis media and to certain types of developmental impairment.

In a study by Bax, 1981, suggested that children as young as 2 and 3 years of age showed significant language delays if they suffered OM with effusion during the past six months. Teele et al, 1980 found that OM involved children from higher socioeconomic status (SES) scored less well on language assessment instruments when compared with children of comparable SES who had little or no middle ear involvement.

Gdowski et al, 1986, in a comprehensive study, reported that, the children with mild to moderate conductive hearing losses have problems in the classroom such as misunderstanding short words in connected speech or the reflexive endings of words. Welsh et al, 1983, stated that there is a central disturbance of auditory perception in some children who have had recurrent middle ear disease in early childhood. Experimental work both in animals and humans has shown anatomical abnormalities and electrophysiologic deficits in auditory processing as sequelae of OME in early life (Ruben, 1996). Gravel and Ellis (1992) put forth a hypothesis that COM 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.

Throughout early childhood, auditory structures in the brain depend upon innervations in the cochlea and ascending synaptic activity for normal development of neurons (Rubel and Fritzsch, 2002). Children need exposure to a variety of simple and complex auditory stimuli from their environment to develop normal auditory pathways and abilities. The acoustic stimuli must adequately and appropriately excite ascending neural pathways in order for the brain to develop normally. Any disruption in acoustic input from the environment or from the neural pathways, can lead to behavioral deficits that are commonly characterized as auditory processing disorders.

Zinkus and Gottlieb (1980) compared two groups of children with identified auditory processing deficits, one of which had a history of recurrent OM. The OM children were found to be slower in developing word combinations, to have decreased verbal intelligence scores, to manifest pervasive auditory processing deficits, and to be significantly, poorer readers.

Finitzo et al., 1988, in study found that children with central auditory processing disorders have difficulty with discriminating foreground and background noise, problems with auditory attention, and a reduced ability to sequence auditory information. With respect to problems with foreground and background discrimination, one researcher points out that similarities between foreground and background noise cause greater difficulties for infants than adults under normal circumstances and would be a particular problem if the infant were experiencing mild hearing loss (Paden, 1994).

In this context, it is always better to discuss a few related to conductive pathologies. They are:

1. Possible routes of infection spreading between middle ear and inner ear, which could be through the oval and round windows, through the facial canal, through the microfissures or hematogenic route (Morizono and Tono, 1991).

Morizono et al., 1991, studied the function of the cochlea using a model of otitis media induced by an antigenic protein of human serum albumin. They reported that, a significant, dose related central auditory processing threshold increase at higher frequencies, the degree of which is minimized following the steroid treatment.

Harris and Ryan, 1985, reported that the concentration of immunoglobin in the perilymph can be increased in the presence of middle ear effusion. Mogi et al. (1988) reported that various vasoactive mediators may cross round window membrane and the concentration of these mediators in the perilymph rises in the presence of middle ear effusion.

 Target structures in the inner ear – inflammatory products from the middle ear can stimulate the endolymphatic sac directly and even if they are harmless to the sensory cells can cause cochlear dysfunction as the endolymphatic sac is assumed to play a central role in the immunological reactions (Saijo and Kimura, 1984; Rask – Anderson and Stahle, (1980). It may stimulate and spread the inflammatory process releasing mediators (Golddek and Harris, 1989). Mogi et al. (1988) reported that various vasoactive mediators may cross round window membrane and thus may affect the cochlear function.

3. These mediators (e.g. Histamine, prostaglandins, leukotrienes etc) first reach the scale tympani of the cochlear basal end, where the spiral modiolar vein runs interiorly and drains into the vein of the cochlear aqueduct and inflammatory cells migrate into the inner ear from systemic circulation (Harris et al. 1990).

Lenhardt et al., 1985 reported that, when there is middle ear pathology, in this case, otitis media, the auditory sensitivity is reduced, since the transformer action is comprised. Otitis media can theoretically influence normal basilar membrane function. The membrane which acts 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 the 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. These changes in the stimulus character may influence the morphology, producing a delay in the peak latencies of MMN.

Webster and Webster, 1977; Dobie and Berlin, 1979; Trune, 1988 reported that due to early conductive hearing loss, there is a probability of reduced CANS development due to auditory deprivation.

Experimentally induced conductive hearing loss and the resulting auditory deprivation in animals are known to cause alterations in anatomy and effect physiological responses during specific critical period. In these cases, CANS anatomy and physiology are influenced by peripheral system dysfunction only during a species – specific critical

developmental period (Webster and Webster, 1979; Trune, 1988; Blachley et al. 1983; Clopton and Silverman, 1977; Evans et al. 1983).

Clopton and Silverman (1977) reported that monaural ear canal occlusion in rats caused altered response patterns in binaurally sensitive cells of inferior colliculus.

In a review, Downs, 1985, has identified 30 separate studies implicating otitis media with language learning or auditory processing problems. Downs reported that mild conductive hearing loss during the critical period, produces the same results on the language test that have been reported as the classic "central processing disorder".

According to the author, the otitis prone children found to present more auditory processing problems due to hearing loss are those who have suffered more than 3 attacks before the age of 3 years. This may be possibly due to persistence of the disorder for a longer period (Casselbraudt et al., 1984; Tos et al., 1984).

To understand the effects of mild hearing loss on the auditory processing different models have been proposed such as, operational definitional model, organic model, developmental / psychological model (Downs, 1985). All the models consider one common aspect of auditory processing, i.e., language is learned auditorily. Therefore, any reduction in the quality or quantity of the language input during critical periods of development results in auditory deprivation and thus causes language difficulties or delay in development.

The 1996 report on central auditory processing: current status of research and implications for clinical practice (ASHA, 1996) published definitions of major issues relative to central auditory processing. In that consensus statement "central auditory processes" included the auditory system mechanisms and processes responsible for the following behavioral phenomena:

- ✓ Sound localization and lateralization
- ✓ Auditory discrimination
- ✓ Auditory pattern recognition
- ✓ Temporal aspects of audition
  - Temporal resolution (i.e. detection of changes in duration of auditory stimuli and intervals between auditory stimuli over time
  - Temporal ordering (i.e. detection of response of sounds over time) Temporal resolution (i.e. detection of changes in duration of auditory stimuli and intervals between auditory stimuli over time.
  - Temporal ordering (i.e. detection of response of sounds over time)
  - Temporal integration (i.e. summation of power over durations less than 200 sec).
  - Temporal masking (i.e. obscuring of probe by pre or post stimulatory presentation or masker).
- ✓ Auditory performance decrements with competing acoustic signal
- ✓ Auditory performance with degraded signals

### **TESTS TO ASSESS APD:**

Evaluation of central auditory processing requires administration of multiple tests spanning the processing categories. CAPD is assessed through the use of special tests designed to assess the various auditory functions of the brain.

The CAPD test battery is very different from those used during traditional audiometric testing. Most tests of central auditory function involve speech stimuli that have been modified in some fashion to make their understanding more challenging. The goal in all evaluations is to identify a lack of ability in processing of auditory information which would count for the individual's communication problems in his / her everyday listening environment.

Tests to assess auditory processing disorders can be grossly categorized into the following types:

- Behavioral tests
- Electrophysiological tests

Behavioral tests are most commonly used to assess the auditory system. The behavioral tests are often broken down into four subcategories:

- Monaural low-redundancy speech tests
- Dichotic speech tests
- Temporal patterning tests
- Binaural interaction tests

The above mentioned are the tests used to study the discrimination abilities of the auditory system behaviorally. Among the above mentioned tests, temporal processing test such as gap detection test, temporal sequence test, auditory fusion test and pitch patterning test are used to study the temporal processing abilities of the auditory system.

### APD TEST IN SUBJECTS WITH HISTORY OF OTITIS MEDIA

Masking level difference (MLD) is the most commonly used behavioral test in OME population. Many investigators have reported reduced MLD in children with previous history of otitis media (Pillsbury, Grose, Joseph and Hall, 1991; Moore, Hutchings and Meyer, 1991, Hall and Grose 1993).

Welsh, Welsh and Healy (1983) reported test results from a central auditory battery in children with history of OME. Results showed that more than 75% of the children with history of OME failed one or more of the test components.

Pillsbury, Grose, and Hall (1991) demonstrated reduced MLDs in children just prior to surgical intervention for prolonged histories of otitis media with effusion in comparison to MLDs obtained from children of same age. While reduced MLDs could have been the result of the pre-surgical conductive hearing loss, many children continued to demonstrated lowerthan-normal MLDs after surgical restoration of their hearing. Similarly, More, Hutchings and Meyer, 1991, demonstrated smaller MLDs for children with reported histories of persistent early otitis media versus children without such backgrounds.

Morrongiello (1989) studied the immediate effect of unilateral otitis media on localization abilities of infants. The infants' horizontal plane localization abilities were adversely affected by the presence of unilateral otitis media.

It has been noted by a number of investigators (Keith, Rudy, Donahue and Katbamna, 1989; Willeford and Burleigh, 1985) that there is much variability in the behavioral tests commonly used for the assessment of central auditory function. Much of the inconsistency in behavioral tests probably occurs because of the extreme heterogenecity found within the population (Willeford and Burleigh, 1985). Lastly, there are practical difficulties in administrating behavioral tests to children, where in the interpretation of results become highly subjective. Thus there is a need for more objective test like event related potentials, for the accurate assessment of central auditory processing in young population.

### **ELECTROPHYSIOLOGIC TESTS:**

Electrophysiological tests are measures of the brains' response to sounds. Some electrophysiologic tests are used to evaluate processing lower in the brain (ABR), others assess functioning higher in the brain (MLR, LLR, P300 responses).

### **MISMATCH NEGATIVITY:**

The mismatch negativity is an automatic cortical evoked potential that is elicited by infrequent sounds ("deviant") sounds occurring in a sequence of repetitive ("standard") sounds. MMN is an event-related brain potential (ERP) which can be elicited even in the absence of attention to these sounds (Naatanen and Alho 1992). MMN signifies the brain's detection of acoustic change. In other words the MMN reflects the neurophysiologic processes that underlie auditory discrimination and it indicates attention independent, perceptual change detection. Thus by using MMN, one can study what is processed as the same as, and what as different from, the previous stimuli by the auditory system. Importantly, the MMN is elicited even by small stimulus changes that approximate perceptual discrimination thresholds (the just detectable difference; Kraus et al., 1993; Sams, Paavilainen, Alho, and Naatanen, 1985b; Tiitinen, May, Reinikainen and Naatenen, 1994).

The fact that the MMN is a change detector for sequentially presented sounds implicates accurate neural representations of the preceeding stimuli. Therefore, the MMN may serve as an index of these neural stimulus representations. Naatanen and Alho, (1992) proposed that the MMN provides the best available physiological measure of automatic central processing in audition. Even though the classic paradigm for recording MMN involve presenting a regular train of auditory 'standard ' stimuli in which occasional 'deviant ' stimuli differ from the others in terms of some physical attribute, there are a host of other factors that have been reported to affect MMN. Some of these factors are mentioned here.

1. Age of the subject

2. Gender

- 3. Type of stimulus deviance
- 4. Magnitude of deviance
- 5. Rate of stimulus presentation

### **Clinical applications of MMN:**

MMN is an automatic cortical evoked potential that signifies the brains detection of acoustic change. It provides an objective tool for evaluating central auditory perceptual mechanisms involved in speech perception. Discrimination of small acoustic differences is an important aspect in speech perception (Kraus et al, 1995). There are several features of MMN, which makes it a specially suitable tool for auditory research and clinical practice. Naatanen (1992) summarizes the clinical applications of MMN as follows.

- 1. The MMN is elicited by any discriminable change of a repetitive sound and can be elicited by stimulus differences that approximate the behavior discrimination threshold. Therefore it provides an individuals discrimination ability for simple and complex (such as phonemic) sound features.
- 2. As it can be elicited without attention, the MMN is free from attentional variants that contaminate the behavioral measures and attention dependent physiological measures of

auditory function. In addition, auditory function can be studied even in individuals who are unable to cooperate.

- 3. MMN provides a unique window to view the neurophysiological processes underlying normal hearing.
- 4. MMN also provides a means for studying auditory short-term memory, which is of crucial importance for correct speech processing and understanding. Consequently, MMN opens a view to the temporal dimension of auditory function which in contrast to vision, is to a great extent, sequential in nature.

The term temporal processing refers to time related aspects of the acoustic signal. Temporal processing is critical to a wide variety of everyday listening tasks, including speech perception and perception of music (Hirsh, 1959).

Several authors have discussed temporal processing as it relates to the perception of speech and specifically speech discrimination. A brief summary of their findings is that auditory analysis of the temporal aspects is important to the understanding of speech and language. Disorders in the discrimination of temporal (timing) or spectral cues of speech can lead to break down in phonemic discrimination, and consequent disorders in receptive and expressive language and reading.

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

Sreevidya, 2002 did a study and found that there is a reduction in amplitude and duration of MMN in children with learning disability.

# ELECTROPHYSIOLOGIC TESTS IN SUBJECTS WITH HISTORY OF OTITIS MEDIA:

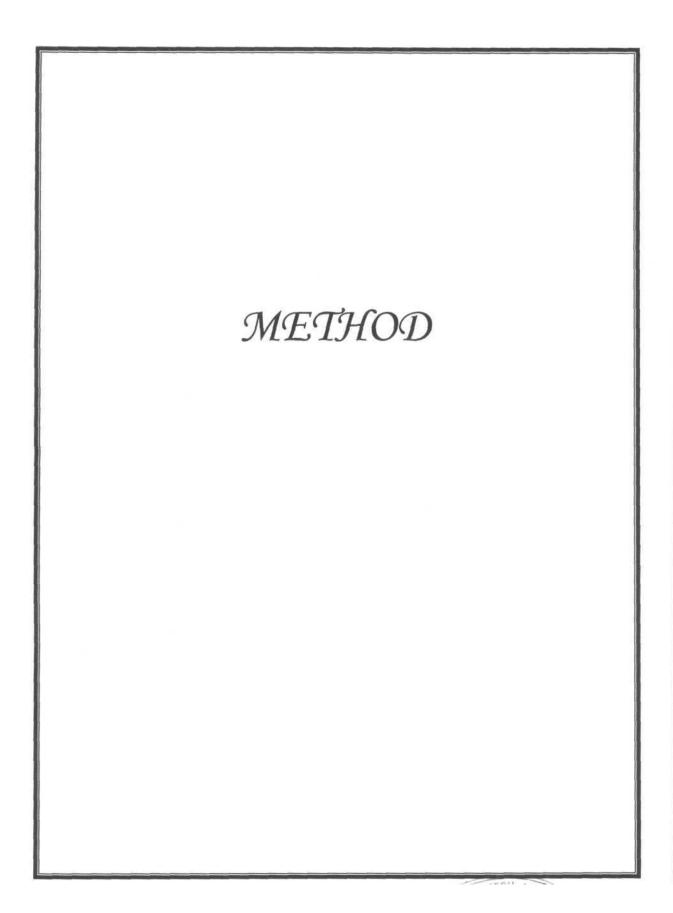
Most of the available information in this area indicates that the vast majority of children with otitis media have abnormal ABRs (Folsom, Weber and Thompson, 1983; Hall and Grose, 1993). Gunnerson and Finitzo (1991) recorded ABR in children with a history of OME, and found delays in both waves III and V.

Hall and Grose (1993a) demonstrated a small but significant relationship between MLD and interaural ABR wave asymmetries in children with histories of otitis media. Their work demonstrated abnormal brainstem processing, utilizing both psychoacoustic and electrophysiological measures in the same children with early otitis media histories.

Tyagi 2002 did a study on conductive hearing loss children using intensity deviance. In which, he observed decreased MMN duration with no change in amplitude. Latency seems to increase in few cases but was not consistent.

Several electrophysiological studies have taken neurological conditions affecting brainstem and higher level dysfunction. But very fewer studies have been undertaken for the evaluation of brainstem functioning in individuals with a history of OME.

Thus from the review of literature, it is evident that there can be brain dysfunction in subjects with a history of OME. These deficits can be evaluated using higher auditory evoked potentials and psychophysical tests.



### **METHOD**

The present study aimed at investigating auditory temporal processing deficits in subjects with otitis media. To achieve the above mentioned goal the following method was used for the study.

### I. SUBJECTS:

The study consisted of two groups of subjects.

### **Group I - Control group:**

Eighteen subjects in the age range of 18 to 35 years were selected for the study.

### Selection criteria:

- All the subjects had hearing sensitivity within 15 dBHL at frequencies from 250 Hz 8000 Hz and 'A' type tympanogram with normal reflexes in both ears.
- 2. They did not have any history or presence of any otological problems (like ear discharge, ear ache etc) and neurological symptoms.
- 3. The subjects in this group did not report of having any difficulty in understanding speech in the presence of noise.

### **Group II - Experimental group:**

Twelve subjects with age range of 21 to 52 years were taken for the study.

### Selection criteria:

- 1. All the subjects in this group had a history of otitis media, which resulted in conductive hearing loss ranging from mild to moderately severe degree.
- 2. Immitance findings revealed abnormal tympanograms with absence of acoustic reflexes in all the subjects.

- 3. All the subjects were diagnosed as having conductive hearing loss by an audiologist, based on the results obtained in PTA and immitance findings.
- 4. Otologist findings also revealed presence of middle ear effusion.
- 5. Subjects did not report of having any history or presence of any neurological symptoms.

### **II. INSTRUMENTATION:**

The following instruments were used for the study:

- 1. Pure tone audiometer a diagnostic audiometer, GSI-61/ OB-922 was used to estimate the hearing sensitivity in both the ears for both the groups.
- 2. Immitance meter GSI tympstar was used to evaluate the middle ear status in both control and experimental groups.
- Auditory evoked potential system Intelligent Hearing Systems, SmartEP software was used to generate stimuli and to find out physiological just noticeable difference (JND) and psychophysical just noticeable difference (JND).

### **III. PROCEDURE:**

Hearing sensitivity was estimated in both the ears in all the subjects for both the groups, frequencies between 250 Hz - 8000 Hz in octaves followed by immitance measurements in a sound treated room. Based on these findings and selection criteria, subjects were categorized into control and experimental groups. Subjects were then taken for psychophysical JND and later prepared to find out physiological JND using MMN recording. **Stimulus generation:** 

The Stimuli required during the psychophysical and physiological testing were generated using the auditory stimulus generator software in intelligent hearing systems software DSP version 4. The stimuli were generated using the following protocol.

Туре	pure tones
Frequency	1000 Hz
Duration	50, 51, 53, 55, up to 120 msec stimuli in
	5 msec steps
Envelope	Blackman window

The stimuli were stored in the hard disk of the system to avail during the testing. These stimuli were presented using ER 3A insert earphones. Once the stimuli were generated, the same stimuli were used for all the subjects for both the testings.

### **PSYCHOPHYSICAL JND:**

### 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. Insert earphones were placed in the ear being tested. Each ear was tested separately to obtain psychophysical JND.

### **Procedure:**

SmartEP software was used to present the stimuli to the subjects. Subjects were presented with two different signals, differing in duration, and they were instructed to indicate whether both are the same or different in duration or length. The initial pair was taken with a difference of 50 msec to make the subject understand the instructions clearly. If they perceive the difference, the difference was decreased to 25 msec, and then in 10 msec steps, till they fail to perceive the difference. When they failed to detect, the difference was increased by 5 msec till they perceived the difference. The same procedure was repeated

thrice with different initiation difference between the stimuli. Psychophysical JND is defined as the minimum duration difference that the subject can identify in two out of three trials.

Todd et al. 2000, in a study, used a stimulus pair, with an initial deviance of 50 msec, and evaluated the behavioral just noticeable difference. They report that as the initial difference is more, the better the subject can understand the instructions. Hence, the initial pair in this study was taken with an initial deviance of 50 msec.

### **PHYSIOLOGICAL JND:**

Physiological JND was obtained using MMN. The following steps were followed to obtain physiological JND.

### 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:**

Four electrodes were used for recording MMN. The electrode sites for the two channel recording were selected as follows:

- Vertex (Cz) non inverting
- upper forehead (Fz) non inverting
- Lower fore head (FPz) ground / common
- Nose tip inverting

The electrode sites in the present study were selected on the basis of the evidence from different psychophysiological MMN studies, in which vertex (Cz) and upper forehead (Fz) electrodes are used in order to obtain a better morphology and amplitude of MMN. Naatanen (1992) recommends the use of nose tip as reference instead of ear or mastoids because the phase shift in the parasagittal and temporal derivations makes it easier to identify MMN topographically and to distinguish it from N2 waveform.

Silver chloride disc electrodes were fixed at the above mentioned sites after thorough skin surfaces cleaning with surgical spirit and an abrasive gel. Standard ten-20 EEG paste was used to increase the conductivity. Electrodes were secured tightly in the above mentioned sites using surgical adhesive tape.

The impedance of the electrodes was measured with reference to the common electrode for both the channels. MMN was recorded only when the impedance values were less than 5K for each electrode and the inter-electrode impedance difference was less than 3K.

### **Procedure for recording:**

The insert earphones were placed in the ear being tested. Each ear was stimulated separately to obtain physiological JND. Subjects were instructed not to pay attention to the stimuli. The following protocol was used to record MMN.

Stimulus parameters used to record MMN are as follows:

Туре	pure tones
Polarity	alternating
Frequency	1000 Hz
Intensity	40 dBSL
Rate	1.10/sec
Number	500 (standard + deviant stimuli)
Ratio	1:4 (deviant: standard)
Duration	

	Standard stimuli	50 msec		
	Deviant stimuli	60 / 55 / 53 / 51msec		
Acquisition parameters:				
	Analysis time	500 msec		

r marysis time	Job misee
Gain	50 K
Filter setting	0.1- 30 Hz

The MMN was obtained in all the subjects at 40dBSL using stimuli differing in duration (frequent and infrequent differing in msec). The initial pair of frequent and infrequent stimuli had a difference of 10 msec (frequent-50 msec, infrequent- 60 msec). The difference was reduced to 5 msec, 3 msec and finally to 1 msec, if the MMN was present in previous higher deviance pair. The minimum deviance at which the MMN could be obtained was noted as the physiological JND.

Korpilhati and Lang (1994) reported that the more the difference between frequent and the infrequent stimuli, better is the MMN morphology. Thus the initial pair was taken with 10 msec deviance to obtain a clear morphology of MMN. However, MMN can also be elicited by finer differences in standard and deviant stimuli. Sreevidya, 2003, reported that the minimum duration deviance needed to elicit MMN was found to be 1 to 3 msec. Thus the later pairs had a difference of 5, 3 and 1 msec differences, to obtain MMN.

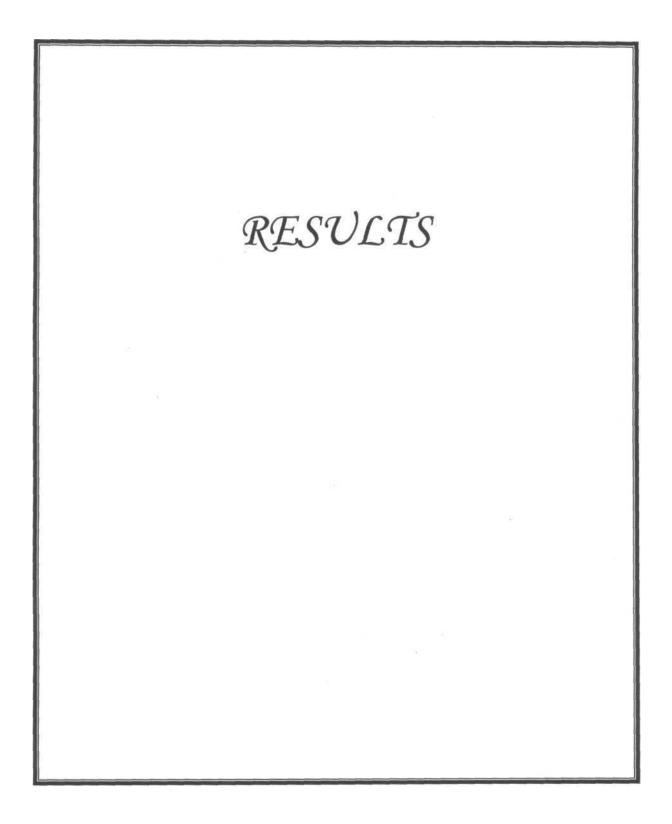
MMN was obtained by subtraction of standard waveform from infrequent waveform. The following parameters were analyzed from the recorded waveform.

- a) Peak latency of MMN
- b) Amplitude of MMN
- c) Duration of MMN

These parameters were noted in both control and experimental groups for comparison between the groups.

## **III. ANALYSIS:**

- a) The just noticeable difference for duration (psychophysical and physiological) in normals and conductive hearing loss were obtained and t - test for independent samples was administered to see if there is any difference in JND values between the groups and paired t- test was used to see significant difference in subjective and objective JND within the groups.
- b) The MMN parameters duration, amplitude and peak latency were obtained in both the groups and t test for independent samples was administered to see if there is any significant differences exist between the control and the experimental groups.
- c) Two tailed correlation test was administered to see if the duration of the conductive pathology and severity of the hearing loss has any effect on psychophysical and physiological JND.



## RESULTS

The present study is designed to investigate:

- 1. Whether some aspects of temporal processing are affected in individuals with conductive hearing loss, if so, how is it different from normals?
- 2. Is there any difference exist in subjective and objective measurement of temporal processing in individuals with conductive hearing loss and normals?
- 3. Does duration and severity of conductive hearing loss has any correlation with temporal processing deficits?

To investigate the above aims, two groups of subjects were selected. The first group, the control group, consisted of subjects with normal hearing, while the second group, the experimental group had subject with conductive hearing loss.

Subjects in both the groups have undergone psychophysical and electrophysiological testings to obtain psychophysical JND followed by physiological JND testings. Both the psychophysical and electrophysiological testing were carried at 40 dB SL. Physiological JND was found using MMN. Psychophysical JND and physiological JND values were obtained from both the groups which can be seen in table 1.

S. No	VARIABLE		Mean	Range	Standard	t - value
			(in msec)		deviation	
1.	Psychophysical Normal		17.14	15 - 25	2.74	10.38*
	JND	Conductive loss	36.45	20 - 55	10.68	
2.	Physiological	Normal	1.16	1 – 3	0.56	7.91*
	JND	Conductive loss	5.04	1 - 10	2.866	

Significant at 0.05 level

Table 1: The mean, SD and t - values for psychophysical and physiological JND obtained in both the groups

It can be seen that the mean psychophysical JND obtained in normal population is much lesser than that obtained in conductive hearing loss group, which is 17.14 msec and 36.45 msec respectively. The mean difference is also high and it differs significantly at 0.01 level. Similarly, physiological JND obtained in normal group is lower than that obtained in conductive hearing loss group which is 1.16 msec and 5.04 msec respectively. This difference is also statistically significant at 0.01 level.

It is also evident that psychophysical JND obtained in both the groups are much higher than the physiological JND. The variation in JND noticed in control group is much lesser for physiological and psychophysical JND. Where as, in the conductive hearing loss group, the variation in both psychophysical and physiological JND is high compared to control group.

The variation in psychophysical and physiological JND is also evident from the range given in the table 1 for both the groups. The Physiological JND value obtained in control group 1 - 3 msec, where as in conductive hearing loss group it was 1-10 msec. Similarly the

psychophysical JND value obtained in normals was 15 - 25 msec, where as in the conductive hearing loss, it was 15 - 55 msec.

Thus, it is evident from the above data that, the conductive hearing loss group has deficits in some aspects of temporal processing which resulted in the higher mean values in both the subjective and objective measurements, and a higher degree of variability within the group.

To observe the temporal processing deficits in conductive hearing loss population, several parameters of MMN also were analyzed.

MMN in both the groups was obtained by subtraction of standard waveform from infrequent waveform. The following parameters were analyzed from the recorded waveform.

- 1. Peak latency of MMN
- 2. Amplitude of MMN
- 3. Duration of MMN

These parameters were noted in both control and experimental groups for comparison between the groups.

## PEAK LATENCY

	GROUPS	No. of	Mean	Std. Deviation	t-values
		ears			
60 C	Normal	36	147.08	5.66	6.855*
	Conductive hearing loss	23	168.70	17.74	
60 D	Normal	36	146.21	4.51	7.248*
	Conductive hearing loss	24	167.540	16.82	
55 C	Normal	36	151.27	6.04	8.299*
	Conductive hearing loss	20	176.48	16.71	
55 D	Normal	36	151.00	6.79	8.923*
	Conductive hearing loss	20	179.30	17.18	
53 C	Normal	36	155.71	5.65	11.268*
	Conductive hearing loss	16	186.95	15.174	
53 D	Normal	36	154.74	6.16	12.519*
	Conductive hearing loss	16	185.21	12.53	
51 C	Normal	32	159.08	5.17	18.948*
	Conductive hearing loss	2	181.20	2.948	
51 D	Normal	34	154.73	24.86	5.038*
	Conductive hearing loss	2	180.50	2.97	

\* Significant at 0.05 level (C: recorded from upper forehead; D: recorded from vertex)
Table 2: Depicts the mean peak latency values for MMN and t - values in normals and conductive hearing loss groups.

The mean peak latency of MMN in normals at 10, 5, 3 and 1 msec deviance levels were found to be, 146.21, 151, 154.71 and 154.73 respectively (The higher value among C & D recordings, has been considered for both the groups). In the subjects with conductive hearing loss, the mean peak latency levels, at 10, 5, 3 and 1 msec deviance levels were found to be, 168.70, 176.48, 185.21 and 181.20, respectively. The mean peak latency values in

conductive hearing loss population are prolonged compared to that of normals. This difference was found to be statistically significant at 0.05 level.

The standard deviation values in subjects with conductive hearing loss are high compared to normals at all levels of deviance as seen from table 2. This indicates a high degree of variance in the mean peak latencies of the subjects with conductive hearing loss.

In conclusion the peak latencies in subjects with conductive hearing loss are prolonged and are highly variable compared to peak latencies observed in normals hearing group.

	GROUPS	No. of ears	Mean	Std. Deviation	T- values
60 C	Normal	36	-2.90	.78	4.474*
	Conductive hearing loss	23	-1.99	.72	
60 D	Normal	36	-3.17	.93	4.208*
	Conductive hearing loss	24	-2.21	.74	
55 C	Normal	36	-2.27	.81	5.405*
	Conductive hearing loss	20	-1.97	.66	
55 D	Normal	36	-2.92	.73	4.486*
	Conductive hearing loss	20	-1.93	.63	
53 C	Normal	36	-2.08	.72	3.399*
	Conductive hearing loss	16	-1.44	.69	
53 D	Normal	36	-2.39	.69	5.353*
	Conductive hearing loss	16	-1.36	.78	
51 C	Normal	32	-1.61	.64	2.572
	Conductive hearing loss	2	-1.27	.63	
51 D	Normal	34	-2.11	.66	6.060*
	Conductive hearing loss	2	-1.27	.15	

## PEAK AMPLITUDE

Significant at 0.05 level. (C: recorded from upper forehead; D: recorded from vertex)

Table 3: Depicts the mean, standard deviation and t - values for the peak amplitude of MMN for both normal and conductive hearing loss groups.

The mean peak amplitude of MMN obtained in normals at 10, 5, 3 and 1 msec deviance levels were found to be, -3.17, -2.92, -2.39, and -2.11uv respectively (The higher value among C & D recordings, has been considered). In the subjects with conductive hearing loss, the mean peak amplitude levels, at 10, 5, 3 and 1 msec deviance levels were found to be, -1.99, -1.97, -1.44, and -1.27uv respectively.

The standard deviation values in subjects with conductive hearing loss are high compared to normals at all levels of deviance. This indicates a high degree of variance in the mean amplitude values of the subjects with conductive hearing loss. The mean peak amplitude values in conductive hearing loss population are lesser compared to that of normals at a same deviance level. This difference was found to be statistically significant at 0.05 level

## PEAK DURATION

	GROUPS	No. of	Mean	Std. Deviation	t- values
		ears			
60 C	Normal	36	60.48	14.36	7.204*
	Conductive hearing loss	23	31.77	16.21	
60 D	Normal	36	61.66	14.92	7.123*
	Conductive hearing loss	24	32.74	16.12	
55 C	Normal	36	54.32	9.92	6.942*
	Conductive hearing loss	20	33.21	13.69	
55 D	Normal	36	54.88	12.96	8.066*
	Conductive hearing loss	20	29.18	10.62	
53 C	Normal	36	48.29	10.72	7.511*
	Conductive hearing loss	16	25.84	12.22	
53 D	Normal	36	47.03	10.17	6.6973*
	Conductive hearing loss	16	24.76	14.59	
51 C	Normal	32	33.17	9.98	4.475*
	Conductive hearing loss	2	24.00	0.94	
51 D	Normal	34	37.18	11.67	10.912*
	Conductive hearing loss	2	10.90	1.79	

\* Significant at 0.05 level (C: recorded from upper forehead; D: recorded from vertex)
Table 4: Depicts the mean, standard deviation t - values for the peak duration of MMN for both normal and conductive hearing loss groups.

The mean, standard deviation and t - values for duration were obtained for both the control and experimental groups. The mean peak duration of MMN in normals at 10, 5, 3 and 1 msec deviance levels were found to be 61.6, 54.8, 48.2 and 37.1 msec respectively (The higher value among C & D recordings, has been considered for both the groups). In the subjects with conductive hearing loss, the mean peak duration values, at 10, 5, 3 and 1 msec were found to be, 32.74, 33.21, 25.84 and 24 msec respectively.

The standard deviation values in subjects with conductive hearing loss are high compared to normals at 10, 5 and 3 msec deviance levels of deviance. However, at 1 msec deviance, the standard deviation of peak duration is high in normals compared to subjects with conductive hearing loss. This indicates a high degree of variance in the mean duration values of the subjects with conductive hearing loss.

Also the peak duration in this group was found to be reduced more with the decrease in the level of deviance when compared to normal hearing subjects. Thus the peak duration values in the subjects with conductive hearing loss are less compared to subjects with normal hearing and this difference is also statistically significant at 0.05 level.

In conclusion, all the three parameters of MMN differ significantly between the groups, peak latencies being longer, amplitude and duration of MMN being lesser in conductive hearing loss group. With the decrease in the deviance level, it was observed that the latencies were prolonged, amplitude reduced and the duration decreased. This trend was seen in both control and the experimental groups.

In addition, the no. of ears in which MMN was obtained in the experimental group was less compared to the no. of ears in control group. The no. of ears in which MMN was present in subjects with conductive hearing loss at 10, 5, 3, and 1 msec deviance levels are 24, 20, 16 and 2 respectively out of 24 ears. It can be noticed from the above data that, as the deviance level was decreasing the no. of ears in the experimental group in which MMN could be obtained also decreased. Where as in the control group, at all levels of deviance, MMN was present in almost all the ears except at 1 msec deviance in which MMN could be recorded from 34 ears.

Thus, the above results also indicate that conductive hearing loss population have deficit in some aspect of temporal processing.

Group	JND	Mean	S.D	t- test	Correlation
	Psychophysical JND	17.14	2.74		
Normals				33.83*	0.759
inormais	Physiological JND	1.16	0.56		
	Psychophysical JND	36.45	10.68		
Conductive				14.46*	0.493
hearing loss	Physiological JND	5.04	2.86		

#### **CORRELATION BETWEEN SUBJECTIVE AND OBJECTIVE JND**

\* Significant at 0.05 level

Table 5: Shows the mean, S.D, t - values and the correlation values for both the groups separately.

t - test for paired samples was administered to find out the presence of significant difference between both the subjective and objective JND measurements in both the groups. Results indicate a statistically significant difference at 0.05 level exists between both the psychophysical and physiological JND in both the groups.

Pearson's correlation test has been administered to find out any correlation exists between the psychophysical and physiological JND in both the groups. The results indicate that there is no one to one strong correlation found between the psychophysical and the physiological JND in both the groups. This indicates that, there was no increase in psychophysical JND with increase or decrease in the physiological JND either in control or experimental groups. Thus, it suggests that psychophysical JND is more compared to physiological JND in both groups which is statistically significant. However there is no relation exists between the two in both the groups.

# RELATIONSHIP BETWEEN DURATION OF OTITIS MEDIA, PURE TONE AVERAGE AND BOTH PSYCHOPHYSICAL AND PHYSIOLOGICAL JNDs:

The mean value for the duration of otitis media in the subjects with conductive hearing loss was found to be 15 years and the mean PTA value is 44.44 dBHL. On the other hand, the mean PTA value in the subjects with normal hearing is 6.66 dBHL. The standard deviation in the PTA values for the control and the experimental groups are 1.72 and 10.32 respectively. These values indicate a high variability in the degree of loss in subjects with otitis media.

Variables	Groups	Mean	Standard deviation
History	Normals		
	Conductive hearing loss	15.31	11.56
PTA	Normals	6.66	1.72
	Conductive hearing loss	44.44	10.32

Table 7: Shows the mean and the standard deviation values for the duration and PTA values in both the groups.

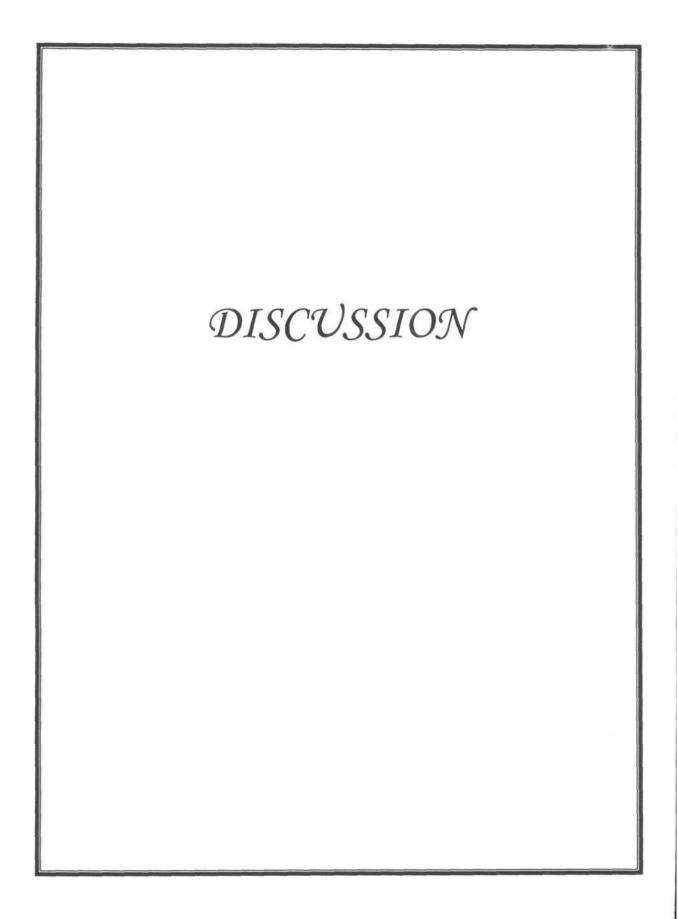
		Physiological	Duration of otitis
Variables	PTA	JND	media
РТА		0.73	0.650**
Physiological JND	0.73		0.115
Psychophysical JND	0.679**	0.147	0.603**

## PEARSONS CORRELATION - CONDUCTIVE HEARING LOSS

\*\* Correlation is significant at 0.01 level

Table 7: Shows the physiological and psychophysical JND, PTA and duration of otitis media in subjects with conductive hearing loss

Two – tailed Pearson's correlation test was administered to find out the relation between the duration of otitis media, PTA and psychophysical and physiological JND. It can be observed in table 7 that there is good correlation between pure tone average and duration of otitis media. Duration of otitis media also had a good correlation with psychophysical JND and psychophysical JND has good correlation with PTA. This suggests that, as the duration of otitis media increases, the psychophysical JND is also likely to increase. Similarly, with the duration one can expect increase in PTA too. Similarly, correlation was not seen for physiological JND with other factors. Thus one must consider the psychophysical JND to be better indicator to observe long term effect of any pathological condition to study temporal processing rather than physiological JND.



#### DISCUSSION

#### **TEMPORAL PROCESSSING DEFICITS**

It is evident from the results that, some aspects of temporal processing are affected in subjects with conductive hearing loss which resulted in the higher mean values in both the subjective and objective measurements. In the present study, the mean value of physiological JND obtained in normals is between 1 - 3 msec. This result was found to be in synchronization with the study done by Sreevidya 2002, in which the physiological JND in normals was reported to be around 1 msec to 3 msec.

Results in the current study, indicate that the MMN latencies were more prolonged in subjects with otitis media than normal hearing individuals. Reduced amplitude and the duration were noticed in the subjects with otitis media compared to normals. As the deviance level decreased from 10 msec to 1 msec, there is a steady increase in latency of MMN, decrease in amplitude and duration of MMN in both the groups.

The mean values of psychophysical JND obtained in the subjects having conductive hearing loss was found to be more compared to the normal hearing group. This finding indicates that the normals can perceive the minimum duration deviance at a lower deviance level, while the subjects with a history of otitis media and a considerable degree of hearing loss require more duration deviance to identify the difference between the stimuli.

This finding suggest the inability to process the time differences in stimulus which occur due to the effect of long term otitis media, and thus lead to temporal processing deficit. Increased JND both (psychophysical and physiological) could be attributed to the effect of otitis media in different levels of auditory system. The long term effect of otitis media can alter the physiology of the inner ear, (Morizono and Tono 1991). The possible reason for such effect is due to the entry of middle ear fluid into inner ear. The possible routes between the inner ear and the middle ear are through the round or oval window, the facial canal, the micro fissures, or hematogenically. According to Morizono and Tono (1991), a hematogenous route to the inner ear in otitis media may be of less significant. Harris and Ryan (1985) reported that the concentration of immunoglobin in the perilymph can be increased in the presence of middle ear effusion.

The inflammatory products from the middle ear can stimulate the endolymphatic sac directly even if they are harmless to the sensory cells (Saijo and kimura 1984). As the endolymphatic sac is assumed to play a central role in the immunologic reactions (Rask-Anderson and Stahle, 1980; Saijo and kimura, 1984) it may stimulate and spread the inflammatory process releasing mediators such as interleukins (Gloddek and Harris, 1989). Various vasoactive mediators may cross round window membrane and the concentration of these mediators in the perilymph rises in the presence of middle ear effusion (Mogi et al. 1988). These mediators (e.g. Histamine, prostaglandins, leukotrienes etc) first reach the scale tympani of the cochlear basal end, where the spiral modiolar vein runs interiorly and drains into the vein of the cochlear aqueduct and inflammatory cells migrate into the inner ear from systemic circulation (Harris et al. 1990). Thus, resulting in altered cochlear physiology.

Subjects with cochlear impairment often perform more poorly than normals in the tasks such as gap detection (Buus and Florentine, 1985). Temporal modulation transfer function is worse for impaired listeners at high modulation rates (Bacon and Viemeister, 19885). Fitzgibbons and Wightman (1982) also reported that some measures of temporal resolution are slightly worse in hearing impaired subjects than normals at equal sensation

levels. The above studies indicate that the cochlear abnormality reduces the ability to process temporal cues of the sounds. Thus, in the current study effect on cochlea would have lead to poor discrimination abilities of temporal aspect, which might have manifested in overall psychophysical perception or MMN findings.

Another reason could be attributed to the auditory deprivation due to otitis media in the central auditory nervous system. Auditory deprivation in animals due to otitis media causes alterations in anatomy and effect physiological responses (Trune 1982; Blachley et al, 1983; Evans et al. 1983). In these cases, CANS anatomy and physiology are influenced by peripheral system dysfunction only during a species – specific critical developmental period.

Dobie and Berlin (1979) and Trune (1982) reported that due to early conductive hearing loss, there is a probability of reduced CANS development due to auditory deprivation. Clopton and Silverman (1977) observed that monaural ear canal occlusion in rats caused altered response patterns in binaurally sensitive cells of the inferior colliculus. Webster and Webster (1979) also observed CANS anatomical and physiological change, due to peripheral system dysfunction.

All this suggest that if the peripheral structure do not transmit adequate sounds, or in this case is otitis media, the physiological responses of central auditory nervous system are likely to be altered. This would have resulted in poor JNDs obtained in conductive hearing loss in the current study.

Prolonged MMN latency observed in otitis media group could be due to its effect on the basilar membrane. When there is middle ear pathology, in this case, otitis media, the auditory sensitivity is reduced, since the transformer action is comprised. Otitis media can theoretically influence normal basilar membrane function. The membrane which acts 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, 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 would have influenced the morphology and produced a delay in the peak latencies of MMN.

The amplitude and duration of MMN has been a strong indicator of abnormalities underlying the processing. Decrease in MMN amplitude and duration probably reflects the delay in memory trace in time (Naatanen, 1992; cited in Lang et al. 1995), indicates a prolonged processing duration.

This can be attributed to the fluctuating nature of conductive hearing loss which led to an intermittent or partial deprivation to the higher cortical centers. The cortical changes can be expected due to auditory deprivation. At the same time it should be kept in mind that the air bone gaps in these individuals itself will significantly alter the stimulus characteristics leading to abnormalities in the higher potentials.

There are a few studies to suggest or contradict the present study findings where MMN or psychophysical JND were obtained in conductive population. Hall and Grose (1993a) demonstrated a small but significant relationship between MLD and interaural ABR wave asymmetries in children with histories of otitis media. Their work demonstrated abnormal brainstem processing, utilizing both psychoacoustic and electrophysiological measures in the same children with early otitis media histories.

Tyagi 2002 did a similar study on conductive hearing loss children using intensity deviance. In which, he observed decreased MMN duration with no change in amplitude. Latency seems to increase in few cases but was not consistent. This difference could be either

due to number of subjects taken for the study or the age range, or the parameters considered were not sensitive enough to detect such variation. Thus, this suggests that the temporal aspects which are most important for speech perception could be the sensitive acoustic parameter to observe such effect in the auditory system.

There is no literature seen which has considered both psychophysical and physiological JND to study the effect of otitis media on auditory system. However, Petinou, Schwartz, Gravel and Raphael, in 2001, studied the speech perception in children with otitis media and the results suggested that the fluctuating hearing loss associated with OME has a negative impact on speech perception, due to which the phonological and morphophonological perception is impaired.

Thus the results of the above study indicate that the conductive hearing loss population has deficits in some aspects of temporal processing which resulted in elevated JND.

#### DIFFERENCE BETWEEN PSYCHOPHYSICAL AND PHYSIOLOGICAL JND:

As can be seen from the results, a statistically significant difference between physiological JND and psychophysical JND could be obtained. The physiological JND being much lower compared to the psychophysical JND in both the groups. Though there is no correlation in both, an increase in both psychophysical and physiological JND has been seen in subjects with conductive hearing loss.

The reason for this finding can be attributed to the fact that, the MMN provides the best available physiological measure of automatic central processing in audition, as proposed by Naatanen and Alho, (1992). Also, MMN is an event-related brain potential (ERP) which can be elicited even in the absence of attention to these sounds (Naatanen, 1990; Naatanen & Alho 1992, 1995). MMN signifies the brain's detection of acoustic change. In other words the MMN reflects the neurophysiologic processes that underlie auditory discrimination and it indicates attention independent, perceptual change detection. Thus by using MMN, one can study what is processed as different from, the previous stimuli by the auditory system. Importantly, the MMN is elicited even by small stimulus changes that approximate perceptual discrimination thresholds (the just detectable difference); (Kraus et al., 1993; Sams, Paavilainen, Alho, & Naatanen, 1985b; Tiitinen, May, Reinikainen & Naatenen, 1994). Thus the smallest differences in the stimuli which were not readily discriminated through behavioral tests could be discriminated by the higher center which can be recorded in electrophysiological tests as it is evident in this study.

## RELATIONSHIP BETWEEN THE DURATION OF OTITIS MEDIA, PURE TONE AVERAGES, PSYCHOPHYSICAL AND PHYSIOLOGICAL JNDs:

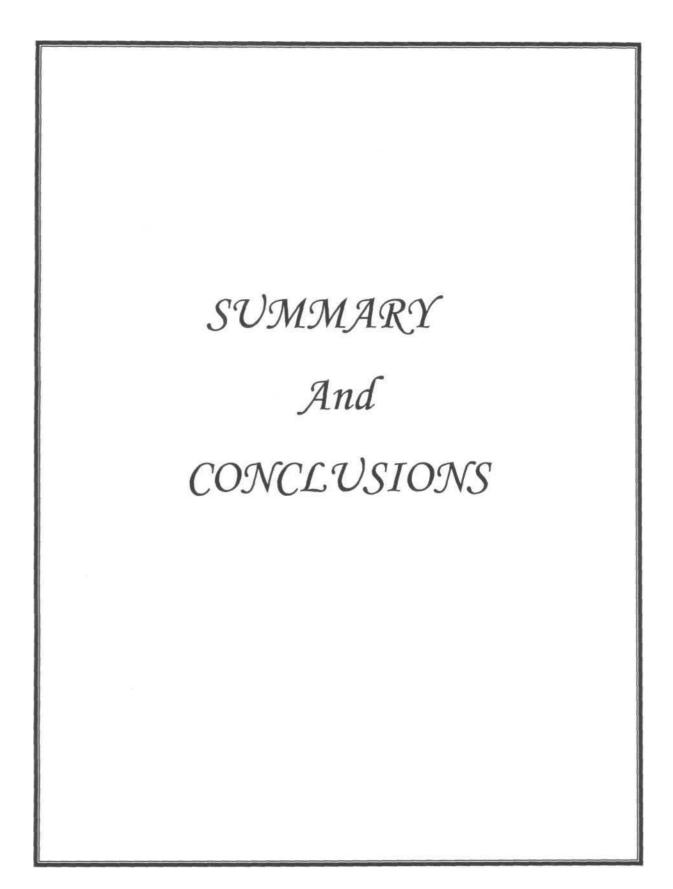
The severity of the hearing loss is an important factor in determining the auditory deprivation caused due to the pathology. The results of this study indicate a high correlation between the degrees of loss, the duration of pathology, and the subjective JND. As the duration of pathology increases the severity of the loss likely to increase and this would have resulted in the elevated subjective JND values in the subjects with otitis media. Thus, suggesting an auditory processing deficit in cases with otitis media. Though the physiological JND was abnormal in subjects with conductive hearing loss, there was no significant correlation with any of the other variables is observed.

This finding could be attributed, to the degree of hearing loss present at the time of testing and, secondly, to the long standing conductive pathology which resulted in deficits in auditory temporal processing. According to Welsh et al (1983), a long standing middle ear disease should be cured to prevent a defect of auditory perceptual disorder. As stated in the above study, a long standing middle ear disorder, which was not treated in this group of subjects, resulted in an elevated JND values compared to the normal hearing population.

The more the severity of the hearing loss, the more is the elevation in both subjective and objective JNDs. This could be due to greater impact on auditory system due to higher degree of hearing loss and, if it persists, leads to increase in impairment of perception of certain temporal aspects of the acoustic stimulus.

Investigations studying long term effects of otitis media on MMN in literature are scarce. In a study done by Tyagi, 2002, found that the MMN latencies were prolonged in subjects who had a long term history of otitis media.

It can be noticed in the current study that the physiological JNDs are lower compared to the psychophysical JND. However no correlation was found between the physiological JND and the other variables, such as, PTA, duration, and psychophysical JND. Thus, it can be concluded that the psychophysical JND, which has a good correlation with the other variables, is a better indicator to observe the effect of any long term pathological condition on auditory system to investigate the temporal processing deficits.



#### SUMMARY AND CONCLUSIONS

Otitis media with effusion is one of the most common childhood disorders which results in a conductive hearing loss of a considerable degree. This hearing loss due to otitis media results in auditory deprivation if left unnoticed. Auditory deprivation in early life has been shown to give rise to auditory processing disorders. Various behavioral and electrophysiological tests have been employed in these subjects to investigate the presence of auditory processing deficits.

It has been already established that auditory deprivation in early developmental period due to OME may lead to APD. These children are shown to have abnormalities at the brainstem level as reflected by ABR and are also suspected to have higher-level dysfunction. Cortical processing of temporal aspects, such as duration, is reported to be an important parameter for speech perception, which could be disturbed in these children. However, there is a dearth of literature in this area; thus, indicating a need to study processing of temporal aspects in conductive hearing loss.

Hence, the present study was aimed at investigating the effects of otitis media on temporal processing in subjects with conductive hearing loss and if it has any affect, how it is different from normals. To achieve the above aim, the subjects were selected and categorized into two groups, one with history of otitis media (experimental group) and the other with no known ear disease (control group). Control group had 18 subjects within age range of 18 - 35 years; where as experimental group had 12 subjects with age range between 21 - 52 years.

Psychophysical and electrophysiological testings were carried out on all the subjects to obtain the psychophysical and the electrophysiological JNDs. Both the testings were carried out at 40 dB SL. MMN was recorded in all the subjects at a repetition rate of 1.10/sec. Recording was done from two different locations by placing non – inverting electrodes at upper forehead and vertex. These potentials were recorded in response to duration deviant stimuli having deviances of 10, 5, 3 and 1 msec for 1 KHz. The psychophysical testing was also done using the same protocol. The stimulus level for this testing was varied in 5 msec steps to determine the psychophysical JND.

The MMN parameters noted for the analysis were, peak latency, peak amplitude and peak duration in both the groups. The analysis of MMN waveforms show a prolonged latencies, reduced amplitude and duration in subjects with conductive hearing loss compared to the subjects in the control group. Also, the psychophysical JND was found to be elevated in subjects with conductive hearing loss. Thus the psychophysical and physiological JNDs were significantly different in both the groups. This could be due to altered physiology at the inner ear and or auditory pathway due to auditory deprivation which might have taken lace due to the long term effect of otitis media.

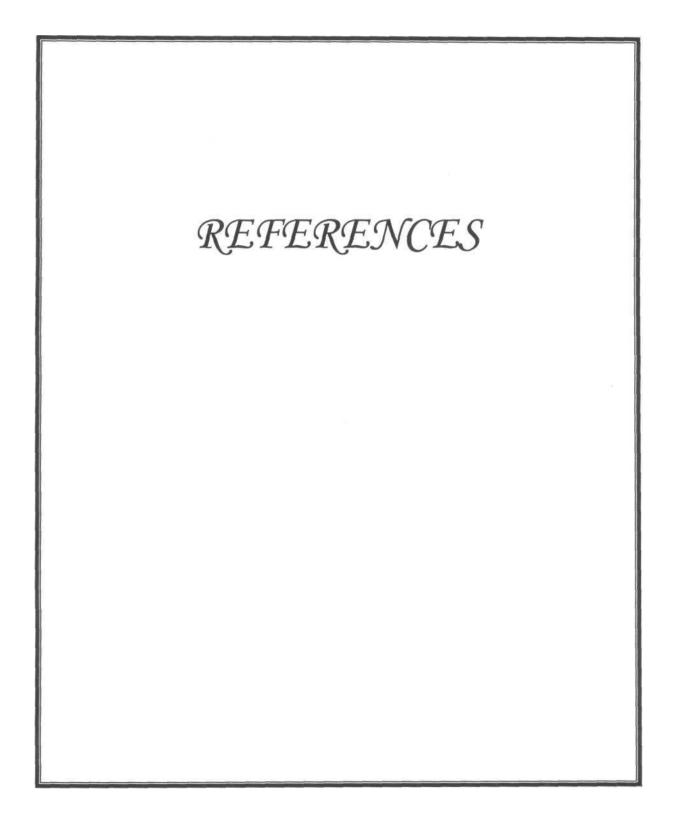
Another finding in this study is that the psychophysical JND has a better correlation with the duration of otitis media and also the pure tone threshold of the conductive hearing loss. Where as such good correlation is not obtained for physiological JND though the conductive hearing loss group showed elevated physiological JND in comparison to normal population.

Thus, it can be concluded from the present study that a long standing conductive pathology results in impairment of some aspects of temporal processing like duration discrimination. This could be due to the conductive hearing loss, which resulted in auditory deprivation.

These affects are more likely to increase with duration as well as the severity of the hearing loss. To identify such deficits in conductive hearing loss population, psychophysical JND could be a better tool than electrophysiological test as the former had a better correlation with both, duration of otitis media and severity of hearing loss.

## **IMPLICATIONS OF THE STUDY**

- ✓ This study highlights the importance of acoustic parameters to be considered to investigate the effects of long term pathology on auditory system.
- ✓ It highlights the importance of early identification of individuals with conductive hearing loss who are at risk of developing temporal processing deficits.
- $\checkmark$  This also highlights the importance of early intervention of otitis media



#### REFERENCES

- Alho, K., Woods, D.L., Algaki, A., Knight, R.T., & Naatanen R (1994). Lesions of frontal cortex diminish the auditory mismatch negativity. *Electroencephalography and Clinical Neurophysiology*, 11; 91(5), 353-62.
- American Speech-Language -Hearing Association (1996). Central auditory processing: current status of research and implications for clinical practice. Task force on central auditory processing consensus development. *American Journal of Audiology*, 5, 41-54.
- Anderson, K. L., (1985). A case study of central processing following long standing unilateral conductive hearing loss. *Journal of Auditory Research*, 25, 201.
- Bacon, S. P. & Viemeister, N. F. (1985). Temporal modulation transfer functions in normal hearing and hearing impaired subjects. *Audiology*, 24, 117 – 134.
- Bax, M. (1981). The intimate relationship of health, development and behavior in the young child. In Brown CC: *Infants at risk: pediatric round table 5*. New Brunswick, NJ: Johnson & Johnson, 1981, pp 106 – 113.
- Bess, F. H. (1986). The unilaterally hearing-impaired child: a final comment. *Ear and Hearing*, 7(1), 52-4.
- Blatchley, B., Williams, J., & Coleman, J. (1983). Age dependent effects of acoustic deprivation and spherical cells of the rat antero ventral cochlear nucleus. *Experimental Neurology*, 80, 81.
- Brandes & Ehinger. (1981). The effects of early middle ear pathology on auditory perception and academic achievement. *Journal of Speech Hearing Disorders*. 1981, 46(3), 3017.

- Buus, S., & Florentine, M. (1985) Gap detection in normal and hearing impaired listeners: the effect of level and frequency. In: Michelson A., Ed., *Time resolution in auditory systems*. New York; Springer Verlag; 159 179.
- Cacace, A. T., & McFarland, D. J. (1988). Central auditory processing disorders in school aged children: A critical review. *Journal of Speech, Language and Hearing Research*, 41, 355 - 373.
- Casselbraudt, M., Okeowo, P. A., & Flaherty, M. R., et al (1985). Prevalence and incidence of otitis media in a group of preschool children in the united states. In *recent advances in otitis media*, Philadelphia B. C., Decker Inc.
- Chermak, G. D., & Musiek, F. E. (1997). *Central auditory processing disorders: new perspectives*. San Diego: Singular Publishing Group.
- Clopton, B. M., & Silverman, M. S., (1978) Changes in latency and duration of neural responding following developmental auditory deprivation. *Experimental Brain Research*, 32, 39 – 47.
- Clopton, B., & Silverman, M. (1977). Plasticity of binaural interaction II critical period and changes in midline response. *Journal of Neurophysiology*, 40, 1275
- Crandell, C., & Flanagan, R. (1998). Effects of otitis media with effusion on speech perception. Paper presented at the American Academy of Audiology annual meeting, Los Angles, CA.
- Daly, Giebenk, G.S. & Batalden, P. B., (1988). Resolution of with effusion using a stepped treatment regimen of trimethoprim - sulfamethoxazole and predisone, *Pediatr Infections and disorders Journal*, 7:471, 1988.

- Dobie, R., & Berlin, C. (1979). Influence of otitis media on hearing and development. *Annals of Otology, Rhinology & Laryngology*, 88 (Suppl 60), 48.
- Downs, M. (1985). Effects of mild hearing loss on auditory processing, peripheral effects on central nervous system. *Otolaryngological clinics of north America*, 18 (2), 337.
- Eimas, P. D., & Clarkson, R. L. (1986). "Speech perception in children Are there effects of otitis media?", in *Otitis media and child development*, edited by J. F. Kavanaugh (York, Parkton, MD).
- Evans, W., Webster, D., & Cullen, J. (1983). Auditory brainstem responses in neonatally sound deprived CBA/ J mice. *Hearing Research*, 10, 269.
- Finitzo, T., Roland, P., & Friel Patti, S. (1988). Incidence prevalence and duration of Otitis media in infants. In D. Lim, C. Bluestone, J. Klein, J. Nelson (eds). *Recent advances in otitis media*. (P - 16), Philadelphia B. C. Decker.
- Fitzgibbons, P. J., & Wightman, F. L. (1982). Gap detection in normal and hearing impaired listeners. *Journal of the Acoustical Society America*; 72: 761 – 765.
- Folsom, R. C., Weber, B. A., & Thompson, G. (1983). Auditory brainstem responses in children with early recurrent middle ear disease. *Annals of Otology, Rhinology and Laryngology*, 92, 249 – 253.
- Fria, T. J., Cantekin, E. I., & Eichler, J. A. (1985). Hearing acuity of children with otitis media with effusion. Archives of Otolaryngology, 111(1), 10-6.
- Friel-Patti, S., & Finitzo, T. (1990). Language learning in a prospective study of otitis media with effusion in the first two years of life. *Journal of Speech and Hearing Research*, 3 ;33(1), 188-94.

- Gdowski, R., Sanger, D., & Decker, T.N. (1986). Otitis media: Effect on a child's learning. *Academic Therapy*, 21 (3), 283.
- Gloddek, B., & Harris, J. P. (1989). Role of lymphokines in the immune response of the inner ear. *Acta Otolaryngologica*, 7 8; 108(1-2), 68-75.
- Gottlieb, G. (1978). Development of species specific perception caused by auditory deprivation. *Journal of Composite Physiology and Psychology*, 92, 375 387.
- Gottlieb, M. I., Zinkus, P. W., & Thompson, A. (1979). Chronic middle ear disease and auditory perceptual deficits. *Clinical Pediatrics*, 18, 725 732.
- Gravel, J. S., & Ellis. (1992). Listening and language at 4 years of age: Effects of early otitis media, *Journal of Speech and Hearing Research* Gunnarson, A. D., & Finitzo, T. (1991). Conductive hearing loss during infancy: effects on later auditory brainstem physiology. *Journal of Speech and Hearing Research*, 34 (5), 1207.

, 34, 1207 – 1215.

- Hall, J. W., & Grose, J. H. (1993a). The effect of otitis media on masking level difference and the auditory brainstem response. *Journal of Speech and Hearing Research*, 36, 210 217.
- Harris & Ryan. (1985). Effect of a middle ear immune response on inner ear antibody levels. Annals of Otology, Rhinology, and Laryngology, 3 - 4; 94, 202-6.
- Harris, J. P., Fukuda, S., & Keithley, E. M. (1990). The spiral modular vein inner inflammation. *Associative Research Otolaryngology*, 13, 413.
- Hirsh, I. (1959). Auditory perception of temporal order. *Journal of the Acoustical Society* of America, 31, 759-767.

- Howie, V. M., Ploussard, J. H., & Sloyer, J. (1975). The "otitis prone" condition. American Journal of Disabled Child, 129, 679, 1975.
- Hunter, L.L. (1993). Long term sequelae of chronic otitis media with effusion. Doctoral dissertation, University of Minnesota.
- Ikeda, K., & Morizono, T. (1990). Round window permeability during experimental purulent otitis media: altered cortisporin ototoxicity. Annals of Otology, Rhinology, and Laryngology. (suppl. 148), 99, 46
- Jerger, J. (1992). Reply to Kileny and Shepard, Face to Face, *American Journal of Audiology*, 1, 11 – 12.
- Keith, R.W., Rudy, J., Donahue, P.A., & Katbamna, B. (1989). Comparison of SCAN results with other auditory and language measures in a clinical population. *Ear and Hearing*, 1989 Dec; 10(6):382-6.
- Klein, J., Chase, C., Teele, D., Menyuk, P., & Rosner, B. et al. (1988). Otitis media and development of speech, language, and cognitive abilities at seven years of age. In Lim D et al (Eds) *Recent advances in Otitis Media*. Toronto: BC Decker, PP.396 397.
- Kokko, E. (1974). Chronic secretory otitis media in children. A clinical study. Acta Otolaryngologica Suppl, 327, 1-44.
- Korpilahti, P., & Lang, H. A. (1994). Auditory ERP components and mismatch negativity in dysphasic children. *Electroencephalography and Clinical Neurophysiology*, 91(4), 256-64.
- Kraus, N., McGee, T., Ferre, J., Hoeppner, J.A., Carrell, T., Sharma, A., & Nicol, T. (1993). Mismatch negativity in the neurophysiologic/behavioral evaluation of auditory processing deficits: a case study. *Ear and Hearing*, 14(4), 223-34.

- Lang, A. H., Eerola, O., Korpilahti, P., Holopainen, I., Salo, S., & Aaltonen, O. (1995).
  Practical issues in the clinical application of mismatch negativity. *Ear & Hearing*, 16, (1), 118-30.
- Lenhardt, M., Shaia, F.T., & Abedi, E. (1985). Brainstem evoked response waveform variation associated with recurrent otitis media. *Archives of Otolaryngologica*, 111, 315 316.
- Mogi, et al, (1988). Staphylococcus epidermidis and Staphylococcus aureus in otitis media with effusion. Archives of otolaryngology Head and Neck Surgery, 11; 114(11), 1262-5.
- Moore, D.R., Hutchings, M.E., & Meyer, S. E. (1991). Binaural masking level differences in children with a history of otitis media. *Audiology*, 30:91 101.
- Morizono, & Tono, (1991). Middle ear inflammatory mediators and cochlear function. Otolaryngology Clinics of North America. 8;24(4), 835-43.
- Morrongiello, B. A. (1989). Infants, monaural localization of sounds: effects of unilateral ear infection. *Journal of the Acoustical Society of America*, 86:597 602
- Naatanen R, Teder W, Alho K, Lavikainen J. (1992). Auditory attention and selective input modulation: a topographical ERP study. *Neuroreport*, 3(6), 493-6.
- Paden, E. P. (1994). Predictors of phonologic inadequacy in young children prone to otitis media. *Journal of Speech and Hearing Disorders*, 60, 232 – 242.
- Paradise, J., & Hoberman A. (2000). Acute otitis media: diagnosis and management in the year 2000. *Pediatric Annals*, 29(10), 609-20.
- Paradise, J. (1981). Otitis media during early life: how hazardous to development? A critical review of the evidence. *Pediatrics*, 68, 869 873.

- Petinou, K.C., Schwartz, R.G., Gravel, J.S., & Raphael, L.J. (2001). A preliminary account of phonological and morphophonological perception in young children with and without otitis media. *International Journal of Language and Communication Disorders*, 1 – 3 ; 36(1), 21-42.
- Pillsbury, H. C., Grose, J.H., & Hall, J.W. (1991). Otitis media with effusion in children: binaural hearing before and after corrective surgery. *Archives of Otolaryngology*, 117, 718 – 723.
- Rask Anderson, H., & Stahle, J. (1980). Immunodefense of the inner ear: Lymphocyte macrophage interaction in the endolymphatic sac. Acta Otolaryngologica, 89, 283.
- Roberts, J. E., Burchinal, M. R, Collier, A. M., Ramey, C. T., Koch, M. A, & Henderson, F. W. (1986). Otitis media in early childhood and cognitive, academic, and classroom performance of the school-aged child. *Pediatrics*, 83, 477-485.
- Roland, P. S., Finitzo, T., Friel Patti, S., Brown, K. C., Stephens, K. T., Brown, & Coleman,
   J.M. (1989) Otitis media: incidence, duration, and hearing status, Archives of
   Otolaryngology, Head and Neck Surgery, 115 1049 1053.
- Rubel, E.W., & Fritzsch, B. (2002) Auditory system development: primary auditory neurons and their targets. *Annu Rev Neurosci.* 25, 51-101.
- Saijo & Kimura (1984). Distribution of HRP in the inner ear after injection into the middle ear cavity. *Acta Otolaryngologica*, 5 6 ; 97(5-6), 593-610.
- Sak, R. J., & Ruben, R. J. (1982). Recurrent middle ear effusion in childhood: implications of temporary auditory deprivation for language and learning. *Annals of Otology, Rhinology, and Laryngology*, 90, 546 – 551.

- Sams, M., Paavilainen, P., Alho, K., Naatanen, R. (1985b) Auditory frequency discrimination and event-related potentials. *Electroencephalography and Clinical Neurophysiology*. 11; 62(6), 437-48.
- Shimizu, H. (1976). Identification of hearing impairment in the neonatal intensive care unit population: Outcome of a five-year project at the Johns Hopkins Hospital. *Seminars in Hearing*, 11(2), 150-160.
- Silverman, M. S., & Clopton, B. M. (1977). Plasticity of binaural interaction. I. Effect of early auditory deprivation. *Journal of Neurophysiology*, 40, 1266 1274.
- Sreevidya, B. A. (2002). Mismatch Negativity and duration deviants in dyslexic children, Unpublished Dissertation, *University of Mysore*, Mysore.
- Teele, D. W., Klein, J. O., & Rosner, B. A. (1980). Epidemiology of otitis media in children. Annals of Otology, Rhinology, and Laryngology, 89, 5-6.
- Tiitinen, H., May, P., Reinikainen, K., & Näätänen, R. (1994). Attentive novelty detection in humans is governed by pre-attentive sensory memory. *Nature*, 370, 90-92.
- Tos, M., Strangerup, S., & Anderson, U. K. (1984). Natural history of secretory otitis media in D Lim, C. Bluestone, J. Klein, J. Nelson (eds)., *Recent advances in Otitis media with effusion.*, Philadelphia B. C. Decker.
- Trune, D. R., & Morgan, C. R. (1988). Influence in developmental auditory deprivation on neuronal ultra structure in the mouse anteroventral cochlear nucleus. *Brain Research* 470: 304 – 308.
- Tyagi, M. (2002), Auditory evoked potentials in children with history of otitis media with effusion, *Unpublished Dissertation*, University of Mysore, Mysore.

- Ventry, I. (1980). Effects of conductive hearing loss: fact or Fiction? Journal of Speech and Hearing Disorders, 45:143 -156.
- Wallace, I. F., Gravel, J. S., Mc Carton, C. M., & Ruben, R. J. (1988a). Otitis media and language development at one – year of age. *Journal of Speech and Hearing Disorders*, 53:245 – 251.
- Webster, D. B., & Webster, M. (1979). Neonatal sound deprivation affects brain stem auditory nuclei. *Archives of Otolaryngology*, 103:392 396.
- Welsh, L. W., Welsh, J., & Healy, M.P. (1983). Effects of sound deprivation on central hearing. *Laryngoscope*, 93, 1569 – 1575.
- Willeford, J., & Burleigh, J. (1985). *Handbook of central auditory processing disorders in children*. New York: Grune & Stratton.
- Zinkus, P. W., & Gottlieb, M. I. (1980). Patterns of perceptual and academic deficits related to early chronic otitis media. *Pediatrics*, 66:246 253.