

**High-Frequency Audiometry:
A Tool to Indicate Probable Inner Ear Damage
in Middle Ear Diseases**

Register No.M9908

**This Independent Project submitted as part fulfilment
for the First Year M.Sc, (Speech and Hearing),
submitted to the University of Mysore, Mysore.**

**ALL INDIA INSTITUTE OF SPEECH AND HEARING
MYSORE 570006**

MAY 2000


Dedicated

*In the loving memory of
Nanaji and Ma*

CERTIFICATE

This is to certify that this Independent Project entitled: High-Frequency Audiometry : A Tool to Indicate Probable Inner Ear Damage in Middle Ear Diseases is the bonafide work in part fulfilment for the degree of Master of science (Speech and Hearing) of the student with Register No.M9908.

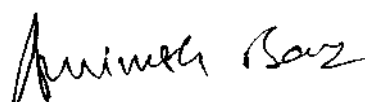
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CERTIFICATE

This is to certify that this Independent Project entitled: *High-Frequency Audiometry : A Tool to Indicate Probable Inner Ear Damage in Middle Ear Diseases* has been prepared under my supervision and guidance.

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DECLARATION

This Independent Project entitled : *High-Frequency Audiometry: A Tool to Indicate Probable Inner Ear Damage in Middle Ear Diseases* is the result of my own study under the guidance of Mr.Animesh Barman, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other diploma or degree.

Mysore
May, 2000

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INTRODUCTION

Human being is bestowed with five special senses, hearing being one of them, which enables adequate speech and language development. It also helps in communication, and development of communication skills. This special sense of hearing, aids us in gaining more knowledge and reacting accordingly by enhancing the perception of different situations and therefore enabling us to adjust with the environment

Any damage to the sense of hearing can deprive a person to gain knowledge about the environment and affect his communication ability. This loss of hearing can be estimated using conventional audiometry. Depending on the gap between the air conduction and bone conduction thresholds, hearing loss can be classified as sensorineural, conductive or mixed hearing loss.

Sensorineural hearing loss has a more adverse effect on speech communication as compared to conductive hearing loss which has lesser effects.

Middle ear pathologies like infections of the middle ear cleft or fixation of the ossicles especially stapes etc. usually lead to conductive hearing loss. Until the sixth decade of 20th century it was believed that ears with middle ear pathology behave like a "plug in the ear". Thus, resulting hearing loss only attenuates the energy reaching the cochlea, does not affect the physiology of higher auditory system and therefore signal processing remains intact.

Recently many scientists do not accept the above concept (Dobie and Berlin, 1979; Webster and Webster, 1979; Gunnarson and Finitzo, 1991). They have reported that effect of conductive hearing loss is not just limited to the attenuation of overall energy but it may have effects on higher central auditory nervous system (CANS), at least when the pathology is a long lasting one. Downs (1981) suggests that even minimal transient hearing loss should not be ignored as it can be educationally handicapping. Children with OME can have language or auditory processing problems (Downs. 1985).

Conductive hearing loss also is known to have affect on the CANS development. Gunnarson and Finitzo (1991) reported long term effect of conductive hearing loss during infancy on later auditory brainstem electrophysiology, i.e. more of a central than a peripheral effect

Long term middle ear infection is reported to have pathologic involvement of the round window membrane such as diapedesis of leukocytes, abnormal permeability of albumin associated with localized inflammatory changes in the adjacent basal turn of the cochlea. The damage to the cochlea starts from the basal end and proceeds to the apical end.

The possible routes between the inner ear and middle ear are through the round or oval window, through the facial canal or through microfissures hematogenically.

Saijo and Kimura (1984) suggest that inflammatory products from the middle ear can stimulate the endolymphatic sac directly even if they are harmless to sensory cells.

Hence, monitoring cochlear damage due to middle ear pathology has become important. Conventional audiometry is the most economical and widely used method to diagnose conductive hearing loss. But, conventional audiometry has a limitation that it can only tell about the sensorineural component, when it has affected the speech frequencies and thus is not very helpful, as damage to the cochlea due to middle ear pathology starts from the basal end.

Here, high-frequency audiometry (HFA) plays an important role in prediction of the sensorineural component before it affects the speech frequencies. Thus, helping the clinician to take adequate steps for the rehabilitation of the patient.

High-frequency audiometry is threshold estimation at frequencies above 8 kHz (to 20 kHz) either through AC or BC. Specialized equipment and unique calibration procedures are required for high-frequency audiometry. High-frequency audiometry may have both diagnostic and rehabilitative ability (Fausti et al. 1979b; Berlin, 1982; Fausti and Rappaport, 1985). Threshold audiometry at frequencies above 8 kHz is instrumentally feasible, clinically reliable and appears to be valid indicator of auditory sensitivity. The increased knowledge gained by expanding the test frequency range in threshold audiometry may be significant and deserves further exploration.

High-frequency audiometry can be used to monitor or detect early damage to the cochlea due to noise exposure, ototoxicity, also due to middle ear pathology. It is also been used to predict the prognosis of the treatment or surgery in different conductive hearing losses (Laitila, et al. 1997; Margolis, et al. 1993; Margolis and Nelson, 1993; Lopponen, et al. 1992; Mair et al. 1989; Dieroff and Schuhamann, 1986; Hunter et al. 1996; Rosen and Siegel, 1969; Mair and Laukli, 1985; Domenech and Carulla, 1998; Mair and Hallmo, 1994).

High-frequency audiometry revealed that even though there is significant improvement in the conventional audiometric frequencies, high-frequency does not show much improvement and may show a decline after treatment (medication or surgery) in cases with conductive hearing loss.

Thus, conductive hearing loss and duration of conductive pathology can affect the inner ear in later stages which is irreversible. Hence high-frequency audiometry can be used to monitor cochlear changes due to middle ear diseases.

This study was taken up to monitor the involvement of inner ear in different conductive pathological conditions and with different durations.

The aim of the study was

- 1) To compare the HFA findings in conductive hearing loss with normal hearing population to predict the involvement of the inner ear in order to prevent spread of hearing loss to speech frequencies.
- 2) To compare the HFA thresholds in patients with conductive hearing loss pre and post treatment.
- 3) To monitor the duration of the conductive hearing loss and presence of sensorineural component.

REVIEW OF LITERATURE

Middle ear pathology and its complications can lead to sensorineural hearing loss due to cochlear damage, especially long lasting middle ear (ME) diseases. In a retrospective study on adults suffering from long lasting recurrent middle ear disorders such as chronic otitis media, otitic perforation of tympanic membrane (TM) leading to cholesteatoma, Ballenger (1977), Morizono and Tono (1991) found that the bone conduction thresholds were also affected.

Otitis media has also got a deteriorating effect on cochlear dysfunction. English et al. (1973), Paparella et al. (1970), Tos (1988), Walby et al. (1984) reported cochlear losses associated with various type of chronic otitis media. Similar findings are reported in other types of otitis media of purulent otitis media (Munker, 1981), and otitis media with effusion (Arnold et al. 1977; Aviel and Ostfeld, 1982; Munker, 1977).

Beales (1987) on the basis of previous research summarized the possible causes of cochlear degeneration seen in otosclerosis as bony invasion of the scala tympani of the cochlea or circulatory changes in the cochlea as a result of abnormal bony foci or it may be due to damage to the cochlea by toxic metabolites from abnormal bone or an upset of equilibrium between enzymes in the microfoci of otosclerosis.

The cochlear damage caused by ME diseases effects the basal end first and then the apical end i.e. the high-frequencies will be affected initially. To assess the early cochlear damage due to ME diseases, HFA can be utilized.

Over the last 10-15 years as the high-frequency audiometry has become commercially available, it has become increasingly popular for both diagnosis as well as rehabilitation. Though HFA has been available for such a long time the validity and reliability is still questionable.

Lopponen and Sorri (1991) reported from their study, that an additive frequency dependent correction is needed to obtain similar loudness sensation increases with these audiometers. Reproducibility with the electric bone conduction audiometer was better than with the AC audiometer, especially in the high-frequency range.

Stelmachowicz et al. (1988) evaluated both intra- and inter-tester reliability of auditory thresholds in the 8- to 20 kHz range. Twenty normal hearing listeners were tested four times, twice by each of two examiners. In higher frequencies, accurate calibration functions could not be obtained for many subjects; in these cases, values extrapolated from lower frequencies were used estimate SPL. Findings revealed that the standard error of measurement for both intra and inter-tester measures increases as a function of frequency. Inter-tester variability was only slightly higher than intra-tester variability. In most cases, variability of threshold estimates in dB SPL was higher than that observed for the unconnected attenuator settings. Exclusion of extrapolated values improved reliability substantially.

In another study Henry and Fast (1984) used a quasi free-field technique to assess detection thresholds of puretones at frequencies ranging from 2-24 kHz in young adults. In the first experiment, 78

university subjects from Northern California aged between 18-24 years were exposed to tones in 2 kHz increments. All but 12 of these persons could reliably detect the highest frequency, with females being slightly more sensitive. But from 10-20 kHz, there were no differences between the sexes. In a second group of 20 students, tested in 1 kHz increments from 8 to 16 kHz, the previously observed 10 to 14 kHz threshold plateau was revealed as a pronounced 13 kHz low threshold region. The threshold at this frequency was approximately 14 dB lower than the 1 kHz threshold.

Stelmachowicz et al. (1989) obtained auditory thresholds in the 8 to 20 kHz range for 240 subjects ranging in age from 10-60 years. The largest changes in sensitivity with age occurred between 40 to 59 years. Below approximately 15 kHz, the inter-subject variability of threshold estimates increased as a function of both age and frequency. Further analysis revealed that the age-related changes in variability were related to absolute thresholds rather than to age per se. When data are converted to dB HL (relative to the youngest group tested), the region of maximum hearing loss shifts to lower frequencies with increasing age, and threshold shifts with age are greatest in the 13 to 17 kHz range.

Green et al. (1987) measured the hearing thresholds of 37 young adults (18-26 years) at 13 frequencies (8, 9, 10 ...20 kHz). The average SPL at threshold was 23 dB at 8 kHz, 30 dB at 12 kHz, and 87 dB at 18 kHz. Despite the homogeneous nature of the sample, the younger subjects in the sample had reliably better thresholds than the older subjects. Repeated measurements of threshold over an interval as long as one

month showed a standard deviation of 2.5 dB at the lower frequencies (8-14 kHz) and 4.5 dB at the higher frequencies.

In another article Schechter, et al. (1986) presented high-frequency (8 to 20 kHz) auditory threshold measurements for 157 subjects, with normal conventional hearing, ranging in age from 6-30 years expectation of a gradual diminution of high-frequency sensitivity through the adolescent and early adult years.

Better and Talley (1976) examined human auditory sensitivity for the frequencies 8, 10, 12, 16, 18 and 20 kHz in a group of 41 female university students ranging from 14-22 years. Statistical analysis revealed no significant threshold differences between right ears and left ears for all subjects at any frequency. These results are in general overall agreement

Matthews, et al. (1997) measured the extended high-frequency thresholds for 162 older listeners within the age range of 60-79 years. Results showed that extended-high-frequency(EHF) threshold of older listeners with normal hearing at conventional audiometric frequencies were substantially higher than the thresholds reported for younger listeners, with normal hearing. EHF thresholds of older listeners with hearing at conventional audiometric frequencies were further elevated as compared to older listeners with normal hearing. Differences in EHF thresholds between females and males were either not present or were reduced when gender differences in conventional audiometric thresholds were taken into account. No significant differences were seen in

thresholds at 8 kHz and higher between the 60 to 69 and 70 and 79 year old age groups. Results indicated that thresholds above 8 kHz can be measured in older listeners within a clinically acceptable +/- 10 dB test-retest range.

Hallmo, et al. (1994) determined air conduction and bone conduction thresholds in both the conventional audiometric frequency range and the extended high frequencies, for otologically healthy subjects in different age spans. Subjects younger than 30 years had conventional frequency air conduction thresholds less than or equal to 10 dB HL whereas the corresponding thresholds of older subjects. Thresholds increase with both age and frequency in the range 8-16 kHz, and there is a largely non-significant tendency for thresholds to be higher in males. Threshold deterioration at the highest frequencies is already present at age 18-24 compared with the youngest (8-14 years) age group.

In another study Lopponen et al. (1991) examined 208 subjects representing both sexes and five age groups (15-70 years) to obtain age related threshold values for high-frequency electric bone conduction (EBC) audiometry. The measurements also included conventional puretone audiometry and air conduction (AC) HF (8-18 kHz) audiometry. The measured EBC thresholds were comparable to the values obtained with AC audiometers, and were equal to ISO standards at the frequencies of 0.5-6 kHz. The 15 to 20 year old groups' EBC thresholds at 8 kHz were equivalent to thresholds of 15 year old people from a cross-sectional material in Northern Finland. Thresholds deteriorated as a function of age, particularly in the HF range. The males had poorer thresholds than females, especially in the age groups of 40 and 60 years.

Sakamoto et al. (1998) measured the sound pressure level thresholds in the extended high-frequency range (8 to 20 kHz) in 25 non-hearing impaired young adults from 20 to 29 years of age. The results are not unlike those obtained by previous investigators. The thresholds increased gradually as a function of frequency. However, two notable points were found: one that the threshold reached a plateau above 18 kHz, and the other that it decreased slightly at 12 kHz. As the subjects might respond to the low-frequency noise of the stimulus wave, the threshold became a plateau above 18 kHz. An acoustic resonance in the ear canal caused the threshold to decrease at 12 kHz.

In another study done by the same authors in 1998, they measured sound pressure thresholds at the extended high-frequencies of 8 to 20 kHz for 65 normal subjects aged between 10 and 69 years. The thresholds increased gradually as a function of frequency, except around 12 kHz and above 19 kHz, and also as a function of age.

There is an increase in trend to use HFA with the increase in reliability of the HFA with the clinical population.

High-frequency audiometry purportedly can reveal sensorineural hearing losses related to ototoxicity (Dreschler et al. 1985; Goldstein, et al. 1987; Rappaport et al. 1986; Vander et al. 1988), before the hearing loss is evident in frequencies at or below 8000 Hz.

Of all the diagnostic tools, EHF was found to be most sensitive to ototoxicity (Park, 1996). It can be used to monitor the ototoxic effects of various drugs (deEspaña et al. 1992; Tange et al. 1997) and estimation

of safe dosage of an ototoxic drug (Martin et al. 1994; Domenech et al. 1993). Collet et al. (1992) and Tange et al. (1995) employed HFA to compare the effects of ototoxicity of two or more drugs.

Studies have shown the selective or early involvements of high-frequency hearing in certain auditory and extra auditory pathologies, Rosen and Olin (1965) in peripheral vascular disease, Cunningham and Goetzinger (1974) in patients with hyperlipemia.

Differentiation between noise induced hearing impairment and other high-frequency sensori-neural hearing impairment such as presbycusis (Laukli and Mair, 1985) can be aided by HFA.

Sataloff et al. (1967), Robertson and Williams (1975), Osterhammel (1979), Dieroff (1982), Luts (1982) and Filipo and Deseta (1983) have shown relationship between high-frequency thresholds and industrial noise.

Zislis and Fletcher (1966) imply that if intense acoustic stimulation first affects the high-frequency hearing, perhaps the detection of a hearing loss at high frequencies can prevent further hearing loss at more important (lower) frequencies by indicating the need to change the individual's sound environment.

Fausti et al. (1981) concluded that threshold shift were prominent for the steady state noise subjects as compared to impulsive noise in the EHF region.

Morton et al. (1991) revealed that there is a significant difference on noise exposure to control groups and group with history of noise exposure.

Cunningham et al. (1983) studied 25 young adult nonsmokers and 18 smokers with respect to differences in their EHF auditory sensitivity. Although no statistically significant differences were found between their EHF thresholds, there was some evidence that smokers have poorer EHF hearing. Therefore, EHF audiometry can be used as a predictor of arteromatous damage to blood vessel walls.

High-frequency hearing loss has also been correlated with otosclerosis, hereditary sensorineural hearing loss and Meneires disease (Osterhammel, 1980).

Hallmo (1997) performed AC and BC audiometry in the frequency ranges 0.125-18 kHz and 0.25 - 16 kHz respectively in 38 patients with unilateral traumatic tympanic membrane perforation. SN threshold elevation was found in 16 ears. In nine of these this was permanent and in 4 restricted to the frequency range > 8 kHz. However both SN threshold elevation and tinnitus diminished with time.

Ahonen and McDermott (1984) compared the hearing sensitivity for frequencies 250 through 20000 Hz between children with repaired cleft palate on a history of otitis media, and children without cleft palate or middle ear disease. Although children in cleft palate group had consistently poor hearing throughout the auditory range, statistical analysis demonstrated significant differences in hearing levels above

9000 Hz only. No statistical difference was observed for the standard clinical audiometric frequencies.

Numerous attacks of acute otitis media (AOM) may have a harmful effect on high-frequency hearing in the long term. Laitila et al. (1997) evaluated EHF hearing of 573 children with a mean age of 13.8 years. All their ear related morbidity was recorded since birth and they had been examined at the ages of 7 months, 2 years and some of them at 5 years. The results were related to the number of attacks they had experienced and revealed that thresholds increased with increase in the number of attacks of AOM.

Margolis et al. (1993) studied EHF hearing in children with and without histories of chronic or recurrent otitis media, (OM). The EHF thresholds were found to have good test-retest repeatability. Children with OM histories had poorer EHF hearing than children without OM histories. The EHF hearing in OM children appeared to be related to OM severity. Children with residual tympanometric abnormalities had poorer EHF hearing than OM children with normal ME function. The results suggest evidences of both ME and IE components in the EHF hearing losses in children with OM.

Dieroff and Schuhmann (1986) studied 78 children who had been treated for secretory otitis media at the age of 4.4, on the average. At the age of 18.6, their hearing was checked by means of conventional and HF audiometry. In conventional range, the average hearing level was nearly normal and no air-bone gap could be found. In HF range, the

average hearing level decreased clearly but the HF hearing level was significantly affected only in which an air-bone gap exceeding 40 dB and more had existed during the active secretory otitis.

Hunter et al. (1996) studied the sequelae after tympanostomy tube insertion (intubation) on children with OM. Hearing thresholds in conventional and HF regions were compared with those of an age-matched control group of children who had less than 2 attacks of OM since birth.

High-frequency hearing loss was associated with OM after ME disease resolved and after middle ear (ME) dysfunction was excluded. Relatively poorer HF hearing thresholds found for older children with OM histories appeared to be attributable to time spent with ear disease. Children at greatest risk for HF hearing loss were those who required multiple intubations. Older children tended to have poorer hearing in both conventional and HF regions, suggesting that the effects of OM on hearing thresholds may be progressive.

Lopponen et al. (1992) invited 31 children with a mean age of 13 years who had been tympanostomy treated for SOM for follow up examination. 29 normal age peers were enrolled as controls. The test battery included pneumotoscopic examination and conventional puretone audiometry as well air conduction and electric bone conduction HF audiometry. High-frequency hearing losses (6 to 18 kHz) were found as sequelae of secretory otitis media (SOM).

Mair et al. (1989) made a comparison of AC threshold changes upto 1 year after myringotomy, aspiration of ME fluid, and insertion of ventilation tubes in 10 patients with bilateral and 12 with unilateral secretory otitis media (SOM). In the LF and HF ranges, significant improvement came during the first 24 hours after intubation, while in the EHF range, threshold lowering occurred generally over the following 2 months.

In a study by Domenech and Carulla (1998) 25 patients were examined with conventional and HFA before and after successful stapedectomy. Conventional audiometry showed a good postoperative improvement in the low and middle frequencies. HFA showed a lowering of the auditory thresholds above 8 kHz in 20 patients (83.4%) which was not evident with conventional audiometry because it occurred above its upper frequency limit. HFA is a very valuable means of assessing the results of ear surgery, and can be used to compare different surgical techniques.

Mair and Lauldi (1986) made a comparison of air conduction thresholds after myringoplasty and stapes surgery for otosclerosis in both the conventional (0.25 to 8 kHz) and high-frequency (8 to 20 kHz) ranges. Significant threshold losses occurred in the high frequencies following both procedures. Threshold improvement was significantly greater at the lower frequencies following stapes surgery, while high-frequency threshold deterioration was significantly less in the myringoplasty group. High-frequency audiometry may prove to be a sensitive monitor of ME surgical techniques.

Mair and Hallmo (1994) made a comparison of the pre and post operative AC and BC thresholds in 22 subjects in whom successful myringoplasty was performed in the conventional and EHF ranges. AC thresholds improved through 4 kHz, but were elevated post operatively for the frequencies 6 through 18 kHz. Post operative BC thresholds were elevated at 0.25 and 0.5 kHz. were lower by 2-8 dB for 1 through 3 kHz and not significantly altered in the EHF range of 8 through 16 kHz. The EHF AC threshold loss following myringoplasty in this study is, therefore due to changes in the ME transmission and is not indicative of iatrogenic cochlear damage.

Tange and Dreschler (1992) carried out a study to evaluate the value of HFA in stapes surgery in cases of otosclerosis. The hearing function was measured pre and post operatively by means of conventional and HFA. The operative findings of the gradation of otosclerosis were compared with the pre and post operative audiometrical measurements. The results pointed out a clear relation exists between the preoperative high tone audiogram and the gradation of otosclerosis. Therefore, HFA can predict the state of stapes fixation in otosclerosis and that can be important in stapes surgery.

In 1969 Rosen and Siegel selected 335 cases and did a preoperative high-frequency response at 12000 CPS and a post operative high-frequency test. Only two operations were performed (1) Rosen mobilization and (2) stapedectomy. Results indicated that the mean change for Rosen mobilization is significantly better at 4000 CPS and

12000 CPS at the end of one year as compared to the mean change of the stapedectomy. This same significance is also seen after 4 years in the 12000 CPS frequency. At the lower speech frequencies a significant difference was not found between any of the operations. But, the analysis was only done in 85% of cases with significant improvement in SRT.

Since the advent of HFA to the field of audiology it has been extensively used to monitor cochlear damage due to ototoxicity, noise induced hearing loss (NIHL) or presbycusis. Now it is clear that HFA can also be extensively used to monitor how ME pathologies can affect inner ear (IE) structures and lead to HF hearing loss. It can also be used to classify the different stages of different ME pathologies and also to see the effectiveness of different techniques used for ME surgery. Hence this study was taken up.

METHODOLOGY

The aim of the study was to monitor whether middle ear pathology can cause cochlear changes and also if the severity of damage can change with the duration.

Subjects

Subjects were selected under two groups. Group-A was the control group and consisted of 46 normal hearing ears. Group-B was the experimental group and consisted of 46 ears with conductive hearing loss.

Criteria for selection of subjects with normal hearing:

- Age range was between 18-25 years
- Their general health was good and they had a psychologically well balanced personality with an average IQ.
- There was no history of any otological problems like noise exposure, ototoxicity, otorrhoea or any operation.
- Audiological findings.

Threshold was within 25 dB HL in the conventional frequency ranges in 250 Hz to 8 kHz in octave. Involvements of middle ear pathology was ruled out using GSI-33 Immittance audiometer. All the subjects under group 'A' had 'A' type of tympanogram with reflexes at normal level.

Criteria for selection of subjects with conductive hearing loss:

- Age range was 10-50 years.
- Audiological findings: There was an AB gap which was more than 10 dB in the conventional audiometric frequency range, in 250 Hz to 4000 Hz, with the BC thresholds within the normal limit. Immittance showed an abnormal tympanogram with absent reflexes.

Equipment

Grason and Stadler (GSI-61) clinical audiometer was used which was calibrated according to the ANSI 1969 for high-frequency as well as for conventional frequencies audiometry.

High-frequency Sennheiser HDA 200 were used which gives a flat frequency response upto 20 kHz. For conventional frequencies TDH 50 P headphones were used which give a flat frequency response upto 8 kHz.

Grason and Stadler (GSI-33) version-2 immittance audiometer was used which was calibrated according to ANSI 1969. to find out middle ear condition.

Test Environment

Testing was done in a sound treated room where the ambient noise level was within the specified limits according to ANSI 1977. Patient was made to sit comfortably before the testing was started.

Procedure

Standard instructions were given to the patient in a language familiar to the subjects. Stimulus used was puretones and testing was done using the Carhart-Jerger Modified Hughson-Westlake method (1959).

Puretone audiometry in the conventional audiometric frequencies was done along with immittance audiometry. Based on the findings of these two tests the subjects were divided into group A (control group) and group B (experimental group).

The high-frequency audiometry was done for both the groups at 10kHz, 12 kHz, 14kHz, 16 kHz, 18 kHz and 20 kHz and the thresholds were recorded. After the 1st assessment, the data obtained from Group B subjects on the first occasion was also categorized according to the onset and duration of middle ear pathology i.e. from 0.02 years (7 days) to 17 years.

The Group B subjects were then sent to ENT Department for treatment which could be either medicinal/surgical, or combination of both. Out of 46 ears, 16 ears in Group B were evaluated again after the completion of medication (i.e. 1-2 weeks later).

On the second occasion both puretone audiometry in the conventional frequency range for both AC and BC was done. Immittance audiometry was carried out to monitor the improvement in conductive

component. Subsequent to the conventional puretone audiometry, high-frequency audiometry was done and thresholds were recorded.

Statistical analysis:

The data was then tabulated and subjected to analysis. During statistical analysis if there was a 'no response' at the maximum intensity level, the maximum level was taken as the threshold. Paired T- test was used to find out whether there is any significant difference between ears with conductive hearing loss and ears with normal hearing. Similarly pre-and post treatment comparison was made using paired T-test. Correlation between the duration of the ME disease and degree of HF hearing loss was found out by Karl Pearson's correlation coefficient.

RESULTS

In the present study the main aim was to investigate the cochlear damage caused by the middle ear diseases using extended high-frequency audiometry (EHFA). The data was analyzed by subjecting it to descriptive statistical procedures.

Results obtained are discussed as follows:

Comparison of puretone threshold (PTT): conductive pathological ears and normal hearing ears

PTT obtained from 46 normal hearing ears were compared with 46 ears with middle ear pathology from 0.25 to 20 kHz in octaves and in 2 kHz increments from 10 kHz and above. The mean, SD and range for both the groups is tabulated. T-test was administered on this data to find out whether there is a significant difference between the PTT obtained from the ears with normal hearing and ears with conductive hearing loss. The t-value obtained was highly significant at 0.01 level ($P < 0.01$) for all the frequencies.

Table-1: Shows the mean, SD and range for the PTT obtained from the ears with normal hearing and conductive hearing loss ears along with the t-value.

Frequency (kHz)	Mean		SD		Range		t-value
	Ears with normal hearing	Ears with conductive HL	Normal ears	Conductive HL (ears)	Normal ears	Conductive HL (ears)	
0.25	14.02	45.54	4.29	15.08	5-20	15-75	12.98
	15	45.43	4.47	14.85	5-25	20-70	12.94
1	14.56	43.91	5.65	15.01	5-25	15-70	13.92
2	15.1	39.23	4.65	15.09	5-25	5-70	10.23
4	16.52	42.5	6.13	14.86	5-25	5-75	10.75
8	15.1	46.7	4.9	14.6	5-55	10-75	13.91
10	1.63	38.0	8.37	15.1	-15-15	10-65	15.4
15	1.73	46.1	12.91	21.08	-20-25	10-95	11.33
14	6.41	56.7	12.93	10.17	-75-50	15-55	14.15
16	0.54	36.4	18.14	11.4	-20-4-5	0-45	10.25
18	11.73	34.5	14.4	9.5	-20-40	5-40	7.5
20	4.45	15.8	19.9	6.4	-20-20	-20-20	6.71

It is clear that the average PTT obtained in the ears with normal hearing falls within normal limits at all the frequencies; whereas, average PTT obtained from ears with conductive hearing loss shows elevated thresholds at all the frequencies, though EHF thresholds was expected to be normal. It is also seen that there is a dip at 14 kHz and 18 kHz with a better sensitivity at 12 kHz and 16 kHz in normal hearing ears. However, conductive hearing loss ears show raising pattern from 12 kHz to 20 kHz having best threshold at 20 kHz.

Comparison of pre and post treatment thresholds

The mean thresholds, SD and range for the 16 ears pre and post treatment is tabulated for all the frequencies from 0.25 - 20 kHz. To find out whether there is a significant difference between pre and post treatment thresholds, t-test was used. The difference was significant only at 4 kHz and 8 kHz at 0.05 level ($P < 0.05$) as shown in the table.

Table-2 : Show the mean, SD and range at all frequencies for pre and post treatment ears along with the t-value.

Frequency (Hz)	Mean		SD		Range		t-value
	Pre Rx	Post Rx	Pre- Rx	PostRx	Pre Rx	Post Rx	
0,25	38.75	35.62	15.65	14.24	15-75	15-70	1.14
0.5	40.62	36.56	13.27	15.78	20-70	15-80	1.67
1	37.5	35	12.7	17.12	15-60	15-70	3.855
2	30.6	26.87	13.6	16.41	5-50	5-60	1.23
4	37.18**	31.56	11.8	15.99	15-55	0-55	2.05
8	39.68	30.93	14.4	16.80	10-65	5-60	2.36
10	36.8	35.62	13.2	21.59	15-60	10-85	0.289
12	42.81	45.62	17.88	21.59	10-65	15-75	0.6
14	55.93	59.37	19.76	21.59	25-85	15-85	1.09
16	33.4	33.12	12.20	15.37	10-45	5-45	1.141
18	32.8	35	10.16	10	5-40	5-40	0.89
20	15.62	16.8	4.03	4.03	10-20	5-20	0.77

It is seen that there is slight improvement in AC thresholds at the frequencies 4 kHz and 8 kHz. Similar affect was also expected at EHF, but deterioration in AC threshold is seen at these frequencies.

Correlation between the duration of middle ear disease and thresholds at EHF:

Karl Pearson's correlation coefficient was found for the duration of the middle ear disease and the thresholds at EHF's which has been tabulated below.

Table-3: Shows the Karl Pearson's correlation coefficient at all EHF for the duration of ME pathology and degree of EHFHL.

Frequency (kHz)	Pearson's correlation coefficient
10	0.047
12	0.094
14	0.124
16	0.182
18	0.2
20	0.127

A low positive correlation is obtained at all the frequencies, which indicated that with increase in duration of ME problem, there is chances of increase in damage to the cochlea.

Extended High-frequency hearing loss

DISCUSSION

EHF audiometry indicates that the technique provides a reliable, accurate method for evaluating high- frequency hearing. Past concerns regarding reliability and stability appear to be manageable, provided that proper calibration methods are employed.

Results in this study indicated that conductive hearing loss can also cause HFHL. There was a significant difference between the PT threshold from 0.25 to 20 kHz in normals and conductive hearing loss ears. The mean threshold for normal ears showed a dip at 14 kHz and 18 kHz. The adding effect resulted from standing waves produced within the ear canal at 14 kHz (Sakamoto et al. 1998). As the subjects might respond to the low-frequency noise of the stimulus wave, the threshold became a plateau above 18 kHz. This finding was supported by Sakamoto et al. 1998). However, PTT obtained at 12 kHz and 16 kHz is relatively better than other frequencies in normal hearing ears which could be due to the resonance properties of the external auditory canal, as reported by Sakamoto et al. (1998).

HFHL in conductive pathologies can be caused due to inner ear or middle ear damage. Inner ear damage is permanent damage which is irreversible. Persistent middle ear infection is reported to cause damage to the round window membrane with localized inflammatory changes in the adjacent basal turn. The damage to cochlea starts from the basal end and proceeds to the apical end. The spread of infection from the middle ear to the inner ear can be through the round or oval window. Mair et al.

* High-frequency hearing loss

(1989) reported that cochlear damage can be caused as a result of leakage of toxins from the middle ear through the round window membrane, through the facial canal or through the microfissures hematogenically. These findings are supported by Downs (1985); English et al. (1973); Paparella et al. (1970); Tos (1988); Walby et al. (1984); Munker (1981); Arnold et al. (1977); Aviel and Ostfeld (1982); Munker (1977).

As cited by Mair et al. (1989) permanent damage to the basal cochlear duct has been also postulated to occur as a result of a local hypoxia, since perilymphatic oxygen tension in the basal coil is partly dependent on gas diffusion from a ventilated tympanic cavity. Thus, EHFA is sensitive to detect the permanent damage caused by middle ear diseases.

However, as electric bone conduction audiometry was not done, it is difficult to say whether the HFHL is due to IE or ME involvement. It is seen that even ME involvement shows a HFHL. This is in agreement with Mair and Hallmo (1994). who say that the feline tympanic membrane (TM) vibrates in phase upto 1 kHz and out of phase upto 20 kHz, the TM resonances at these frequencies being, however, largely averaged out in the response to malleus handle (Decraemer. et al. 1989). In addition it has been shown that the mode of vibration of the malleus at high frequencies is not around the classical incudo-malleal rotation axis but can be translational, rotational with different axis which may even be inferior to umbo, or a mixture of both modes (Decraemer et al. 1989, 1991). Middle ear transmission is, therefore, very complex in the EHF range. The shape, thickness and anisotropy of the TM have been shown to be important factors for sound transmission in the ME (Funnell and

Laszlo, 1982; Williams and Lesser, 1990). It is, therefore perhaps not surprising that replacement of the structurally highly organized lamina propria of the pars tensa of the TM (Lim, 1970) by the histologically different temporalis fascia used in myringoplasty will result in poorer transmission in the EHF range where mechanical coupling is more complex. The defective transmission in the EHF range found in this study could also be ascribed to minor changes in the ossicular chain secondary to previous otitis media or ear trauma, or to a combination of these factors. Also mass load on TM due to ME fluid can alter vibration pattern of the TM (Mair et al. 1989).

However, in this study, there is more possibility of a IE damage causing HFHL. As the ME transmission system is described as stiffness controlled at low frequencies and ME pathologies considered in this study (negative ME pressure, ME effusion, otosclerosis) have the greatest effect on the transmission of low frequency signals. Accordingly, a low frequency conductive hearing loss is called a stiffness tilt, and thus having lesser effect on HF. Therefore the HFHL obtained in this study shows more possibility of IE damage as the cause, which is due to the ME pathology. Hence, this leaves us with less option for the justification of the present findings.

Moreover, IE damage is known to start from the basal end of the cochlea and could be the reason for the significant threshold shift at the HF regions.

It was noticed that a few cases with unilateral conductive losses also showed an increase in threshold in the EHF region in the normal

ear. So, it may be indicated that ME pathology not only can affect the inner ear structures, it can also affect the higher centers having affect on the contralateral ear.

The post treatment findings in the EHF range imply that there is not a significant improvement, which can be attributed to the permanent cochlear damage. However, there is a significant improvement at midfrequencies (4 and 8 kHz). This may be possible, as the medication would have partially restored the ME mechanical properties. The literature is supportive of this finding as reported by Hallmo (1997), Mair and Hallomo (1994), Lopponen et al. (1992) and Mair et al. (1989).

Residual epitympanic or circumossicular disease could result in a permanent increase in mass of the system and thus persistently high EHF thresholds. However, at the lower frequencies (250 Hz to 2 kHz) there was not a significant difference between pre and post treatment thresholds; one reason for this can be that at the lower frequencies ME is stiffness dominant and as the post treatment was taken after 1-2 weeks itself, may be the ME disease had not fully resolved.

There was a low positive correlation between the duration of the ME disease and elevation of HF threshold, i.e. an increase in duration shows an increase in HF threshold as well. This is in agreement with Laitila et al. (1997). So, as duration of the hearing loss increases the IE involvement also increases. However, there may not be a one to one correlation, i.e. all patients need not show an elevation of HF thresholds with an increase in the duration. But still there are more chances of

effecting the BE. Therefore early detection and prevention of the IE damage is important

Clinical Implication

As the availability of extended high frequencies is increasing commercially, HFA has become more popular and can be used as a clinical tool to detect even mild cochlear damage which may be caused by ME pathology, ototoxic drugs or noise exposure. In this way cochlear damage can be predicted before, effecting the speech frequencies and thus adequate rehabilitative measures can be taken up to prevent the spread of permanent cochlear damage to speech frequencies. Indication of the prognosis of a treatment in the ME disorders can also be done using HFA.

Thus HFA helps us in detection and prediction of cochlear damage as well as tell us about the prognosis of treatment.

SUMMARY AND CONCLUSION

EHFA appears to be a reliable clinical tool for the indication and prediction of cochlear damage. Cochlear damage caused by middle ear diseases or noise exposure or ototoxicity can be detected before it affects the speech frequencies. Various studies imply that conductive hearing loss might resolve at speech frequencies post treatment but not so at EHF, thus causing a permanent damage. This is true even for minimal transient conductive pathologies.

Therefore, the present study was taken up with the aim to investigate whether the ME pathology can lead to permanent IE damage which is known to start from basal end which in turn can have a significant threshold shifts at EHF.

Fourty six normal hearing ears and fourty six ears with conductive pathology were taken up and audiometry done in both speech as well as EH frequencies. Then the pathological cases were arranged in order of duration of the disease. Sixteen ears were again tested on all me frequencies after medication using the GSI-61 audiometer.

The results obtained are :

1. There was a significant difference between the thresholds of normals and ME pathological ears at frequencies from 0.25 to 20 kHz HF involvement could be due to IE damage (English et al. 1973; Paparella et al. 1970; Tos, 1988; Walby et al. 1984; Munker, 1981; Arnold et al. 1977; Aviel and Ostfeld, 1982; Munker 1977) or ME damage (Mair

et al. 1989; Mair and Hallomo (1994) or a combination of both. There is a greater possibility of the threshold elevation being an IE damage because ME pathology exhibits more effects on the lower (speech) frequency region, whereas IE damage initiates from basal end which could result in the hearing loss at HF regions.

2. There was no significant difference between pre and post treatment at EHF which could be due to permanent damage to the cochlea, but there is a significant improvement at 4 kHz and 8 kHz which may be attributed to the resolution of mechanical properties as supported by Hallmo (1977); Mair and Hallmo (1994); Lopponen et al. (1992); Mair et al. (1989).
3. There was a low positive correlation found between the duration of the ME disease and the degree of HFHL which implies that with increase in duration there will be an increase in the HF threshold as supported by Laitila et al. (1997). But this may not be true in all cases.

Thus, the above results suggest that HFA can be used as clinical tool for detection of hearing loss at HF which can be used to predict further damage to the speech frequencies later on.

HFA also provides us with knowledge about the choice of surgical techniques or medication depending upon the amount of improvement post treatment. Although the reliability of HFA was questioned, many times. But with appropriate calibration procedures HFA can be used to assist in clinical situation. Earlier detection of HL can help inadequate rehabilitative procedures,

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APPENDIX

Calibration of High-frequency Audiometer,

The calibration procedure and instruments required to calibrate the high-frequency audiometer is similar to that of the conventional puretone audiometer, but the coupler and the reference level used is different for the Sennheiser HDA-200 earphones.

AN IEC 318 flat plane coupler must be used in the calibration of the high-frequency tones for the desired output.

The reference levels for puretones with sennheiser HDA-200 earphones for 0 dB HL setting are given below :

Frequency (Hz)	10k	12.5k	14k	16k	18k	20k
Sennheiser HDA-200	21.5	27.5	37.5	58.0	83	105