EFFICACY OF EXTENDED HIGH FREQUENCY AUDIOMETRY AND DISTORTION PRODUCT OTOACOUSTIC EMISSIONS AS EARLY IDENTIFIERS OF NOISE INDUCED HEARING LOSS

Register No. M0120

An Independent Project submitted in part fulfillment for the first year M.Sc, (Speech and Hearing)
University of Mysore, Mysore

All India Institute of Speech and Hearing
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MAY-2002

Dedicated

To

My Parents who have been my second Teachers
and
My teachers who have been my second Parents...

CERTIFICATE

This is to certify that the independent project entitled "EFFICACY OF EXTENDED HIGH FREQUENCY AUDIOMETRY AND DISTORTION PRODUCT OTOACOUSTIC EMISSIONS AS EARLY IDENTIFIERS OF NOISE INDUCED HEARING LOSS" is the bonafide work in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student with Register No. MO 120.

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Director
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Mysore
May 2002

CERTIFICATE

This is to certify that the independent project entitled "EFFICACY OF EXTENDED HIGH FREQUENCY AUDIOMETRY AND DISTORTION PRODUCT OTOACOUSTIC EMISSIONS AS EARLY IDENTIFIERS OF NOISE INDUCED HEARING LOSS" has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any Diploma or Degree.

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DECLARATION

I hereby declare that this independent project entitled "EFFICACY OF EXTENDED HIGH FREQUENCY AUDIOMETRY AND DISTORTION PRODUCT OTOACOUSTIC EMISSIONS AS EARLY IDENTIFIERS OF NOISE INDUCED HEARING LOSS" is the result of my own study under the guidance of Ms. **Manjula, P.** Lecturer, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any other University for the award of any Diploma or Degree.

Mysore
May 2002

Reg. NoMO**120**

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INTRODUCTION

"Ear is the gate to the sour - Wedenberg (1981). Hearing is one of the most important senses of human beings. It is one of the channels through which we communicate and interact with the society. Unfortunately, there are a multitude of factors that can affect the hearing of an individual. Of the various factors, one factor which can have an adverse effect on our hearing is 'Noise' which has subjectively been defined as any unwanted sound. Based on the physical properties, it can be defined as a sound, generally random in nature, the spectrum of which does not exhibit clearly defined frequency components (Behar, Chasin, and Cheesman, 2000).

The American College of Occupational Medicine (ACOM) noise and hearing conservation committee (1987) (Cited in Dobie, A.R., 1995) defined occupational noise induced hearing loss as a slowly developing hearing loss over a long period (several years) as the result of exposure to continuous or intermittent loud noise. The effect of noise upon the auditory system has become a major problem in today's highly technological society. Hearing loss due to occupational noise exposure is our most prevalent industrial malady and has been recognized since the Industrial revolution. Continuous exposure to loud noise, especially as in industries, can lead to noise induced hearing loss, depending on variables, such as the individual's susceptibility, amount and duration of noise exposure. The noise to which people are exposed at their work places leads, at first, to a high tone hearing defect and later to a reduction of hearing in the speech frequency region (Schwetz, Doppler, Schewczik, and Welleschik, 1980).

Early identification of noise induced hearing loss is very essential. Noise induced hearing loss is almost always preventable at relatively little costs. If the loss is identified at a very early stage (before the speech frequencies are affected), then measures can be taken to prevent further spread of hearing loss to the other

frequencies. This can be done by measures such as enforcing the usage of ear protective devices or a change in the work environment, etc. This will also help the management by reducing the claims for compensation. The clinical audiological measures must therefore, identify auditory changes with special attention to early detection, base line function and monitoring the changes in hearing sensitivity in industrial workers (Fausti, Erickson,Frey, Rappaport, and Schechter, 1981).

Conventional frequency audiometry (CFA), from 250 Hz to 8 kHz is used routinely in monitoring procedures for NIHL. This has indicated a tendency for noise induced threshold shifts from 3000 Hz to 6000 Hz, more often in 4000 Hz. Fowler (1929) (Cited in Fausti, S.A. et al., 1981) has termed this configuration a 4000 Hz dip. Histological research on animals and a limited number of human subjects has indicated the vulnerability of the base of the cochlea to NIHL (Fausti et al., 1981; Pye, Knight, and Arnett, 1984). So, by monitoring high frequency hearing, changes above 8 kHz can be seen well before the loss shows up in 3-6 kHz region. This measurement of hearing between 9 kHz to 20 kHz is called Extended High frequency audiometry (EHFA).

Since the demonstration of significantly lower AC thresholds above 8 kHz in the African Mabaans compared with age matched Western controls (Rosen, Plester, El-Mofty, Rosen, 1964, cited in Hallmo P., Borchgrevink, H.M., and Mair, I.W.S., 1995) several authors have hypothesized that NIHL may be detectable at an early stage by EHFA (Osterhamrnel, 1979, cited in Hallmo, P. et al., 1995; Dieroff, 1982). Beiter and Talley (1976) reported that HFA may be valuable as an early indicator of the traumatic effects of high intensity noise. Subjects with excellent high frequency hearing possess a good resistance against noise damage (Osterhammel, 1980).

Several studies of high frequency auditory function in noise exposed humans have been reported. Flottorp (1973) examined the high frequency hearing

in students who had military noise exposure. He suggested that threshold deviations from 250 Hz to 12,000 Hz generally were associated with decreases in the upper frequency limit of hearing. Reports of cochlear histologic data and high frequency sensitivity in noise exposed human subjects suggest that high frequency audiometry above 8 kHz may provide greater clinical definition and differentiation of NIHL (Fausti et al., 1981).

Another clinical measure that has gained importance in the recent years is Otoacoustic emissions (OAEs). OAEs were first identified by Kemp (1978). OAEs are acoustic signals that can be detected in the external auditory meatus. They originate in physiologically vital and vulnerable activity inside the cochlea, i.e., they are believed to originate from the electromechanical process in the outer hair cells (OHC) of the organ of Corti (Kemp, 1997). OAEs are an indication of the active mechanisms in the inner ear.

There are two basic OAE phenomena (Norton and Stover, 1994).

i) Spontaneous OAE (SOAE)
ii) Evoked OAE (EOAE)

Spontaneous otoacoustic emissions occur in the absence of external stimulation. They occur in 60% of normal ears and are of limited clinical value. Evoked OAEs are those which occur during or after external stimulation. Evoked OAEs further include the following :

i) Stimulus frequency OAE (SFOAE).
ii) Transient Evoked OAE (TEOAE).
in) Distortion product OAE (DPOAE).

Stimulus frequency OAEs are recorded using a continuous puretone and do not have any significant clinical value. Transient evoked OAEs are recorded using

click or tone burst stimuli and they have a good clinical value (Hall, 2000). Distortion product OAEs are recorded using two puretones and they too have a good clinical value. Hall (2000) has reported that both TEOAEs and DPOAEs are present in 99+% of normal ears. DPOAEs are more sensitive than TEOAEs in the region of 4000 Hz to 6000 Hz (Gorga, Neely, Bergman, Beauchaine, Kaminski, Peters, Schulte and Jesteadt, 1993).

Noise primarily damages the mechano - electrical transduction process located in the hair bundle of outer hair cells (Gao, Ding, Zheng, Raun, and Liu, 1992). OAEs are known as OHC function reference and to be affected preferentially during the initial stages of noise damage (Clark and Bohne, 1978; Davis, Ahroon, and Hamernik, 1989; Hamernik, Patterson, Turrentine, and Ahroon, 1989). DPOAEs have assumed an important role as an electrophysiologic index of the cochlear status in experiments involving exposure to noise (Hall, 2000).

Need **for the study**

Early identification of Noise-Induced hearing loss is very important. As the noxious agent is known, NIHL is preventable. But once acquired, it is not treatable, i.e. the hearing of the individual cannot be reverted back to normalcy after occurrence of NIHL. So, it is very essential that NIHL be identified before the speech frequencies are affected.

From the psychological viewpoint of the industrial worker, measures for early identification of NIHL has got a lot of advantages. When the worker is assured that sufficient care is taken by the industry inorder to protect his welfare, then his contributions in the work environment will be better. He will work without the fear of incurring any hearing loss. This will indirectly improve the productivity. The industry is also benefited a lot by adopting measures for early identification of

NIHL. These measures prevents the industry in paying out huge sums in the form of compensation by preventing the spread of loss to the speech frequencies.

Thus, for the above mentioned reasons, it is crucial that NIHL gets identified at an early stage. For early identification of NIHL, it is essential that the most appropriate clinical measure is used. From the review of literature, it is evident that various measures such as Extended High Frequency Audiometry and Distortion Product Otoacoustic Emissions can be used as early identifiers of NIHL. It is of utmost importance to know which of these tests is most efficient with respect to time, cost and early identification. Hence this study was carried out with the aim of finding out,

i) Extended High Frequency Audiometry results among industrial workers with normal hearing in Conventional Frequency Audiometry.
ii) Distortion Product Otoacoustic Emissions results among industrial workers with normal hearing in Conventional Frequency Audiometry.
iii) Comparison between EHFA results and DPOAE results in terms of the efficacy in early identification of NIHL.

REVIEW OF LITERATURE

Noise induced occupational hearing loss remains a widespread and serious problem in modern industry. A good program for the conservation of hearing requires both direct and indirect measures such as measuring and reducing noise levels, protecting hearing, and monitoring hearing thresholds at regular intervals. Regular monitoring of hearing thresholds is very important so that early intervention measures can be taken and thus, prevent the industry from paying large sums for compensatory claims.

Conventional audiometry is the test routinely used in many industrial hearing conservation programs for monitoring NIHL. Threshold shifts produced by noise exposure have been intensively studied with the aid of behavioural techniques in several animal species such as chinchilla, cat, monkey and also in human beings. Prolonged exposure to high - intensity noise results in sensorineural hearing loss that is greatest at 4000 Hz or between 3000 Hz to 6000 Hz. Fowler (1929) (cited in Fausti, S.A. et al., 1981) termed this configuration 4000 Hz dip. A bilateral and symmetrical 4000 Hz dip is the prominent feature seen in early stages of NIHL (Sataloff and Sataloff, 1987).

Rosier (1994) has compiled eleven investigations from 1950s to 1970s and summarized the results. He concluded stating that hearing deterioration due to noise exposure begins in the frequency range of 4 to 6 kHz. During the first 5 to 10 years of noise exposure, factors such as frequency, level and temporal pattern of noise affect the hearing loss. An average hearing loss of 5 to 9 dB at 1 kHz, 20 dB at 2 kHz and 35 to 50 dB at 4 kHz was noted during the first ten years of noise exposure. After long lasting noise exposure for 30 to 40 years, the total median hearing loss increased to 60 to 70dB in the frequency region of 3 to 8 kHz.

Table **2.1** : Summary of a few studies on **N1IIL.**

Authors and years	***Description of noise***	***Year, at which 4kHz dip was evident***
Taylor, Pearson, Mair and Burns (1965) (cited in Rosier, G., 1994)	Continous broad band noise in 500-4 kHz at 99-102 dB	5-9 years
Nixon and Glorig (1961) (cited in Rosier, G., 1994)	300 to 4 kHz at 100.5 dBSPL	3.2 years
Szanto and Ionescu, (1983)	Continuous broad band noise in 500 - 4kHz, at 98 dBL_{Aeq}	1-5 years
Ivarsson, (1987) (cited in Rosier, G., 1994)	90.4 to 91.3 dB (A)	11-20 years
Salmivalli (1967) (cited in Rosier, G., 1994)	168 dBSPL-188 dBSPL (impulse noise)	0-5 years
Counter and Klareskov (1990) (cited in Rosier, G., 1994)	Impact noise of rifles and shot gun (Noise measurement not done)	0-5 years

From Table 2.1 it is clear that the first sign of NIHL, i.e., a dip at 4000 Hz is evidenced only after certain period of noise exposure has elapsed. The variability obtained can be attributed partly to the difference in the type, level and duration of noise they have been exposed to. It can be evidenced that, the effect of noise is noticed first in conventional audiometry only after a few years of exposure during which the hearing loss has already occurred in the speech frequencies. The onset of NIHL is generally insidious, the hearing loss is cumulative and not currently treatable (Consensus Conference, 1990, cited in Hallmo, P. et al., 1995). Early recognition of incipient NIHL is therefore, desirable. It is important that sensitive methods which identify hearing loss even before the speech frequencies are affected be employed so that early preventive measures and precautions can be adopted by the industrialists to prevent NIHL. Tests such as Extended High Frequency

Audiometry (EHFA) and Otoacoustic Emissions (OAEs) can be employed for early identification of those with a greater susceptibility for developing NIHL.

EHFA AND NOISE INDUCED HEARING LOSS :

Measurement of hearing in the frequency range of 8 kHz to 20 kHz is called EHFA (Osterhammel, 1980). Clinically, EHFA is of value because of its extreme sensitivity in the early detection of cochlear pathology, because the pathological process tends to start in the basal, high - frequency region, as a result of ototoxic drugs and NIHL (Osterhammel, 1979, cited in Hallmo, P. et al., 1995; Fausti et al., 1981, Dieroff, 1982; Halimo, Borchgrevink and Mair, 1995). Several hydrodynamic effects have been proposed as possible contributors to basal noise induced damage. These factors include (i) greater traveling wave amplitude at the base, (ii) greater acoustic load at the base, and (iii) a possible basal locus for shock from impulse energy abnormally conducted to the cochlea (Jordan, Pinheiro, Chiba, and Jimenez, 1973). Several histologic patterns of basal damage from noise or combined noise and other degenerative factors have been reported (McGill and Schuknecht, 1976). These general patterns of primary damage are (i) extreme basal degeneration for 1 to 3 mm from the oval window, (ii) first turn and extreme basal degeneration, (iii) complete basal degeneration for 1 to 8 mm from the oval window, and (iv) complete basal degeneration for 1 to 15 mm from the oval window.

Several studies of high - frequency auditory function in noise - exposed humans have been reported. Corliss, Doster, Simonton and Downs (1970) (cited in Fausti, S.A. et al., 1981) have reported high - frequency thresholds from 250 Hz to 18,000 Hz for high school students who were rifle team members, rock band musicians and non - noise - exposed subjects. The region above 12,000 Hz seemed to be particularly vulnerable to noise damage. High - frequency sensitivity changes above 8000 Hz were not always accompanied by abnormal sensitivity below 8000 Hz. Dieroff (1982) has also reported that the frequency range between 11 and

12 kHz is relatively unaffected for a long time. He concluded saying that with high sound levels, there occurs distinct shifts in the high frequency limits of detectability as well as marked changes in threshold. Apart from temporary threshold shift, there is also a reduction in the frequency range due to overloads in the high frequency region. Flottorp (1973) examined the high frequency limit in students with noise exposure and suggested that threshold deviations from 250 Hz to 12,000 Hz were associated with decreases in the upper frequency limit of hearing.

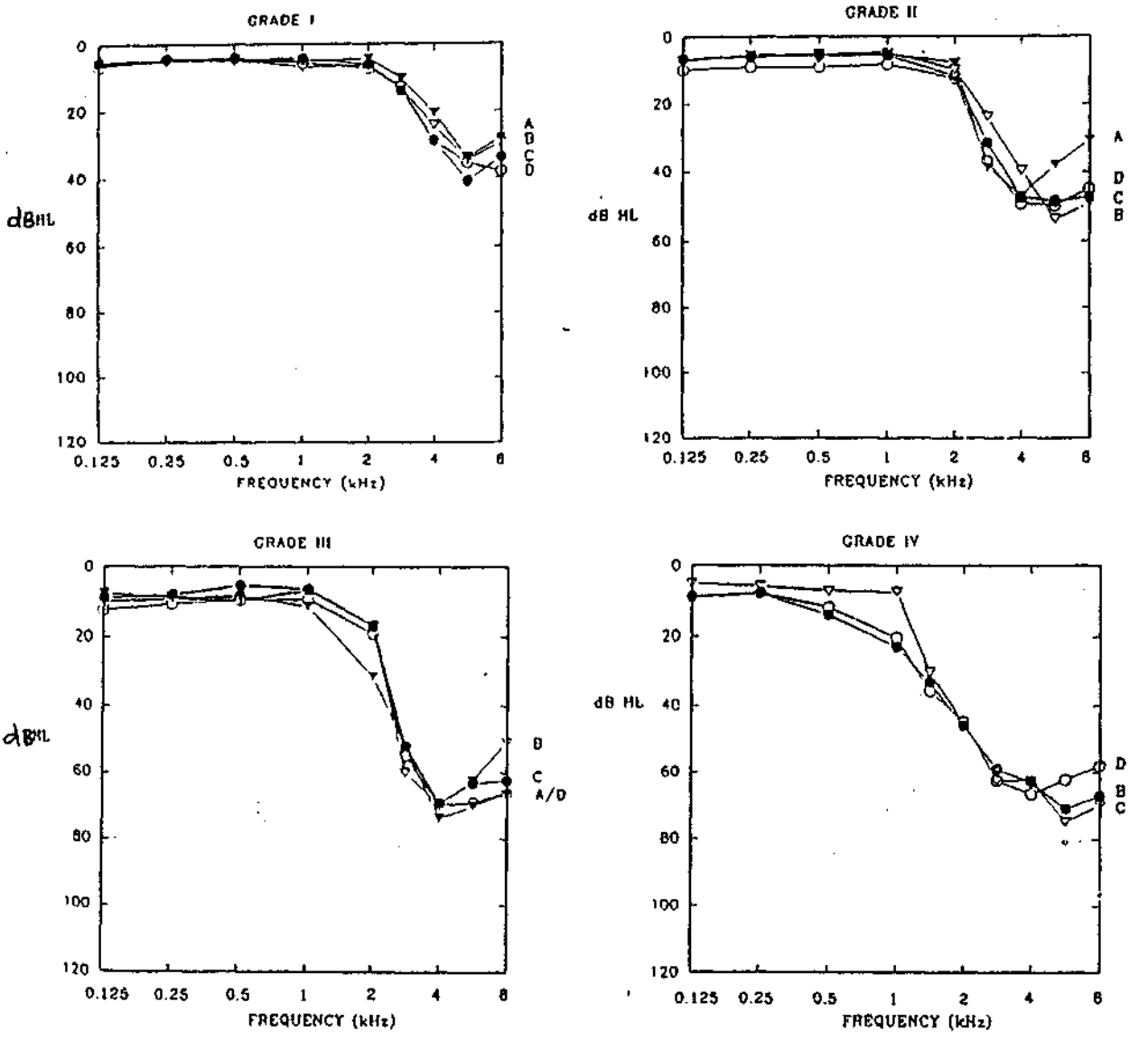
Fausti et al. (1981) compared the high frequency hearing sensitivity of thirty - six, 20 to 29 years old noise exposed (both impulsive and steady state) individuals with the non - noise exposed individuals. The normal hearing estimate revealed mean thresholds from 500 through 12,000 Hz that were 20 dB SPL, rising slightly to 35 dB SPL at 17,000 Hz then rising steeply to 82 dB SPL at 20,000 Hz. The impulsive noise sample exhibited prominent threshold shifts from 9000 to 20,000 Hz and from 2000 to 8000 Hz as well. The steady state noise sample revealed mean thresholds from 250 Hz to 12,000 Hz that were only 8 to 20 dB poorer than the normal hearing estimate. The region from 13,000 to 20,000 Hz showed the greatest changes which were as much as 45 dB poorer than normal at 17,000 Hz. From this study it is clear that despite variability in the noise exposure, there is an effect seen in the high frequency regions. The study done by Fausti et al. (1981) on the effects of impulsive noise upon high frequency hearing also support the above results.

There are a few factors which hinder the usage of EHFA routinely in industries. In EHFA, the sound source (transducer), the placement of the sound source relative to the ear canal, and the size and shape of the external ear are critical variables that have limited the clinical application of this method. These problems are largely due to the extreme directionality of high frequency tones. Procedures with acceptable reliability were not established until some 30 years ago. Since then, different laboratories have largely employed different techniques,

making it difficult to establish normative thresholds and recommended procedures. An ISO standard is still lacking for EHFA. Also, test retest reliability in the same subject corresponds to that in the conventional frequency range, whereas the inter subject variability is much higher (Northern, Downs, Rudmose, Glorig, Fletcher, 1972). Age related deterioration of hearing starts in the EHF range from the first decade of life, requiring different normative data for each decade (Osterhammel, 1980; Buren, Solem and Laukli, 1992). The above mentioned limitations restrict the clinical utility of EHFA. Inspite of these limitations, EHFA can be used to the maximum in industries if adequate norms are developed.

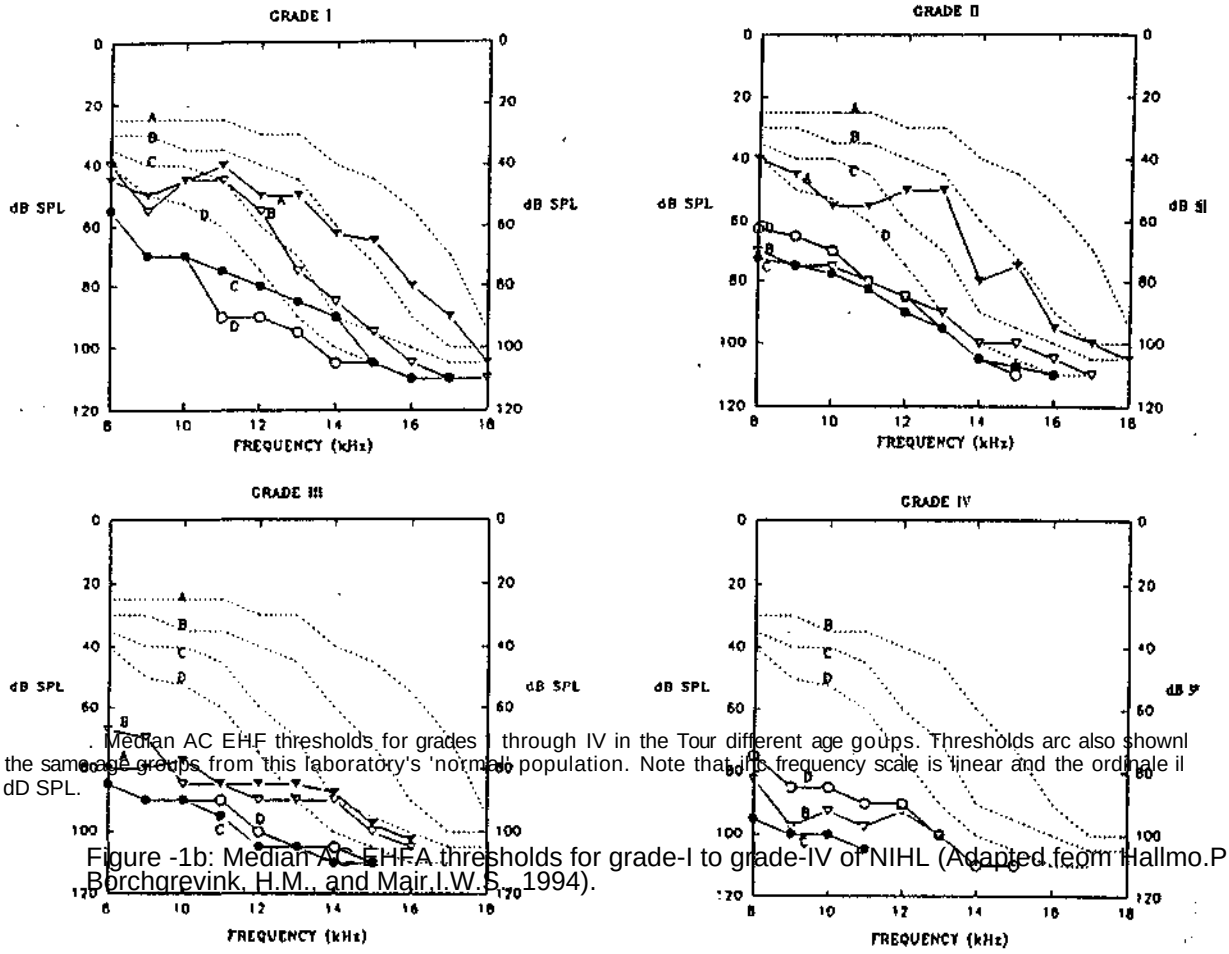
Hallmo et al. (1995) compared the EHFA thresholds and conventional audiometric thresholds across individuals with varying grades of NIHL (refer Figure 1). They reported that elevation of AC thresholds in NIHL occured both at 3-6 kHz and throughout the EHF range of 9-18 kHz. For increasing grade of conventional frequency NIHL, the EHF hearing loss progresses towards, more wide-spread dips which ultimately merge and lead to extensive EHF deterioration until a ceiling effect is reached. Moderate level exposures lead to asymptotic EHF threshold shift. Variability is considerable across individuals, and susceptibility seemed to increase with age.

It is thus evidenced that, high - frequency audiometry from 8,000 Hz to 20,000 Hz provides n more complete mnp of auditory sensitivity in the basal region of the cochlea, and strengthens the clinical test battery. The measurement of hearing sensitivity above 8,000 Hz holds promise for better early detection and description ofNIHL.



Median AC CF thresholds for grades I through IV in the four different age groups. In these and subsequent figures the following symbols are used: 18–24 years—A/filled triangle; 30–39 years—B/open triangle; 40–49 years—C/filled circle; 50–59 years—D/open circle.

Figure-1a: Median AC Conventional Frequency threshold for grace -I to grade-IV of NIHL (Adapted from Hallmo, P., Borchfrevink, H.M., and Mair, I.W.S., 1994).



. Median AC EHF thresholds for grades I through IV in the Tour different age goups. Thresholds arc also shownl the sameage groups from this laboratory's 'normal' population. Note that the frequency scale is linear and the ordinale il dD SPL.

Figure -1b: Median AC EHFA thresholds for grade-I to grade-IV of NIHL (Adapted feom Hallmo.P. Borchgrevink, H.M., and Mair.I.W.S., 1994).

Distortion Product Otaacoustic Emissions and Noise Induced Hearing Loss:

Another clinical measure that can be used lor early identification of N1HL is OAE. OAEs, discovered by Kemp (1978), has gained a lot of importance during the past decade. Otoacoustic Emissions (OAEs) are acoustic signals that can be detected in the ear canaL Recent studies have demonstrated that within the organ of Corti, an active mechanical process makes use of metabolic energy to create additional microvibrations that enhance the sound induced motion of the cochlear structures ami increase the sensitivity and frequency selectivity of the ear (Davis, 1983; Johnstone, Patuzzi and Yates, 1986; Sellick, Patuzzi, and Johnstone, 1982). Thus, the cochlea actively produces energy as a part of the normal hearing process. Some of this added energy propagates towards the base of the cochlea, to the stapes footplate, through the ossicles and to the external ear canal, where it can be detected by the sensitive microphone (Kemp, 1978; Wilson, 1980). The sound produced in this manner is called OAE.

OAEs are generated only when the organ of Corti is in near normal condition, and they can emerge or be detected only when the middle ear system is operating normally (Kemp, 1997). Various studies in the past have indicated that the effects of noise lead to damage in the organ of Corti. Clark and Bohne (1978) and Davis, Hamernik and Ahroon (1993) and many other investigators have shown that noise can damage the hair cells and that the outer hair cells are particularly vulnerable. Davis (1983) has pointed out that the outer hair cells are the morphological correlate of an active cochlear process that has been postulated by Gold (1948) (cited in Delb, W., Hoppe, U., Liebel, J., and Iro, H., 1999). An epiphenomenon of this active amplification process is the generation of otoacoustic emissions. Therefore, it is a reasonable hypothesis that the measurement of otoacoustic emissions could be an ideal tool for examining damage that affects the outer hair cells, such as acute trauma caused by noise. The clinical utility of OAEs, especially evoked OAEs as objective tests of auditory function, is greatly enhanced by their ability to test discrete, frequency - specific regions of the cochlea so that frequency areas of impaired hearing can be adequately distinguished from regions of normal functioa (Lonsbury - Martin, Whitehead, and Martin, 1991).

OAEs can be classified into different types. The various types of OAEs can be summarized as follows. (Hall, 2000; Robinette and Glattke, 2000).

Table 2.2 : **Description of types of OAEs**

Type of Emission	***Stimulus***	***Recording / Analysis techniques***	***Prevalence in ears***	***Clinical value***
Spontaneous OAE (SOAE)	None	Signal from microphone is submitted to high-resolution spectral analysis inorder to reduce noise artifacts.	Approximately, 60%	Limited
Synchronized spontaneous OAE (SSOAE)	Click at 70 dB p SPL	Signal from microphone is submitted to time averaging over an 82 msec period. This is followed by a spectral analysis of the waveform. A synchronized response detected in the time period between 60 and 80 msec after stimulus presentation is considered to be an SSOAE.	Not established	
Transient Evoked OAE (TEOAE)	Click at 80 dB p SPL or tone burst	Signal from microphone is submitted to time averaging. Average response waveform in buffer A is compared with average response waveform in buffer B. Correlation between waveforms is expressed as response reproducibility. Response amplitude is based on comparison of the gross power spectrum of the A and B buffer contents with a waveform computed as the difference between AandB.	99+ %	Yes
Stimulus frequency OAE (SFOAE)	Swept sinusoid (low SPL)	SPL of signal in the ear canal is monitored as stimulus at constant SPL is swept through the frequency region of interest. Changes in SPL are reflections of combinations of incident energy and emission produced by the cochlea	Unknown	No
Distortion product OAE (DPOAE)	Paired sinusoids (No standard SPL)	Signal from microphone is subjected to time averaging. Spectral analysis is obtained for average waveform. Energy at the appropriate frequency is considered to be the DPOAE.	99+%	Yes

Of the various types of OAEs, DPOAEs and TEOAEs have maximum clinical value. Robinette and Glattke (2000) have stated that the choice between TEOAE and DPOAE instrumentation may be influenced by the frequency range of interest. In terms of separating patients with normal hearing sensitivity (20 dB or better) from those with hearing loss, TEOAEs are more sensitive at 1000 Hz, TEOAEs and DPOAEs are essentially equivalent for 2000 and 3000 Hz, and DPOAEs are more sensitive from 4000 Hz through 6000 Hz (Gorga et al., 1993). Probst and Harris (1993) have stated that TEOAEs may be preferable for screening purposes, whereas, DPOAEs may be more valuable for monitoring cochlear changes clinically. Kim, Paparello, Jung, Smurzynski, and Sun (1996) have stated that the sensitivity, specificity, and predictive efficiency of DPOAE is 85-89% at 6 kHz and 4 kHz, 82-83% at 2 kHz and 78-79% at 1 kHz. So there is no doubt that DPOAEs can form a useful frequency - specific objective test of cochlear function.

The largest human DPOAEs are recorded at the frequency $2f_1$-f_2, where the ratio between f_1 and f_2 is of the order of 1.2. The $2f_1$ - f_2 DPOAE are believed to be generated at the cochlear partition corresponding to the site of f_2 frequency (Kemp and Brown, 1983, cited in Attias, J., Bresloff, I., Reshef, I., Horowitz, G., and Furman, V., 1998) or the geometric mean of the primary frequencies (Martin, Lonsbury - Martin, Probst and Coats, 1987; Harris, Probst, Xu, 1992).

Liebel, Delb, Andes, and Koch (1996) (cited in Delb, W. et al, 1999) have reported that the detection of acute noise effects on the cochlea using DPOAE with stimulation levels of $L_1 = L_2 = 70$ was inadequate and resulted in low sensitivity. A classic study done by Skellet, Crist, Falloon, and Babboon (1996) (cited in Delb, W. et al., 1999), exposed gebrils to a small band noise of 65dB (A) for eleven days. They determined the input - output functions at $L_1=L_2$ and measured significant reductions of the amplitude of the $2f_1$- f_2 distortion product even at this low noise exposure. Significant changes were noticed only when the stimulation level was between 40 and 55 dB. No significant changes was noticed at higher

stimulation level. They concluded that detection of noise effects is only possible at stimulation levels at which the cochlear amplification process contributes considerably to the amplitude of the displacement of the basilar membrane.

Many investigators including Brown and Gaskill (1990), and Hauser and Probst (1991), have opined that lowering of L_2 compared to L_1 can increase the amplitude of DPOAE. Whitehead, McCoy, Lonsbury - Martin, and Martin (1995) systematically studied the different variations of L_1 and L_2, varying L_2 and keeping L_1 constant and vice versa. They noted a maximum amplitude at high stimulation levels when $L_1=L_2$. At lower stimulation levels, DPOAE amplitudes were the highest when L_1 was larger than L_2. Better results can be obtained when the stimulation level is lowered. (Delb, Hoppe, Liebel and Iro, 1999 ; Skellet et al., 1996, cited in Delb, W. et al., 1999). Smurzynski, Leonard, Kim, Lafreniere, and Jung (1990) reported that, when the stimulus frequencies fell in an impaired region, detection of DPOAEs required higher stimulus levels than were required for a normal ear. When the hearing impairment was severe, DPOAEs could not be elicited at the highest stimulus levels tested (80 dB SPL).

Another possible variation in the stimulus combination when measuring DPOAEs is the variation of f_2 / f_1 ratio. Harris, Lonsbury - Martin, Stagner, Coats and Martin (1989) systematically investigated the dependency of the DPOAE amplitude on the variation of the f_2 / f_1 ratio. They observed that at low stimulation amplitudes (65 dB SPL) and high stimulation frequency (4000 Hz), DPOAE amplitudes were maximum when the f_2 / f_1 ratio was between 1.16 and 1.2. When the stimulation frequency was 1000 Hz, maximum amplitudes were measured at a f_2 / f_1 ratio between 1.12 and 1.24.

The results of the study done by Delb et al. (1999) reveal that the best separation between noise - exposed and non - exposed subjects was obtained at the stimulus paradigm L_1=60dB, L2=35dB and f_2 / f_1 = 1.18. Stover, Gorga, and

Neely (1996) performed receiver operating characteristic curves (ROC curves) to distinguish between normal and impaired ears. When plotting sensitivity against specificity for different DPOAE amplitude thresholds, one receives the ROC curves. They concluded that the area under ROC curves was best at L_2 levels of 45-65dB. This paradigm gives better results for detection of NIHL.

A classical study by Attias, Bresloff, Reshef, Horowitz and Furman (1998) assessed the efficacy of screening for NIHL with DPOAE (refer Figure 2). They used the paradigm $L_1=L_2=70$ dB SPL and $f_2 / f_1 = 1.22$. They observed that the DPOAE levels of the exposed ears were significantly reduced in amplitude as compared with the non - exposed ears at the test frequencies 1 kHz, 2 kHz, 3 kHz, and 4 kHz. Atleast 25% of the noise exposed ears had an absence of emissions at 1 and 6 kHz. They also concluded that as the hearing loss severity increased, the amplitude and frequency range of DPOAE decreased significantly. The correlation between the audiometric hearing thresholds and DPOAE levels was found to be moderate. The inter-and intra-subject variability values were great. The sensitivity of DPOAE ranged between 0.51 and 0.9 while the associated specificity ranged between 0.63 and 0.25.

Gorga, Neely, Bergman, Beauchaine, Kaminski, and Liu (1994) have determined an absence of low frequency DPOAE among normal hearing subjects. Also, Moulin, Bera, and Collet (1994) have reported DPOAE in the presence of 65 dB HL hearing losses. So, there is a general inability to predict audiometric thresholds with a high degree of certainty (Bonfils, and Avan, 1992). The DPOAEs in subjects with NIHL were also investigated by Martin, Ohlms, Franklin, Harris, and Lonsbury - Martin (1990). They reported frequency - specific reduction of DPOAE for stimulus frequencies corresponding to hearing impairment. Kim, Leonard, Smurzynski, and Jung (1992) concluded that in ears afflicted with NIHL, DPOAEs are reduced or eliminated when two - tone stimulus frequencies fall within a hearing - impairment region, thus providing sensitive and frequency - specific information about cochlear dysfunction.

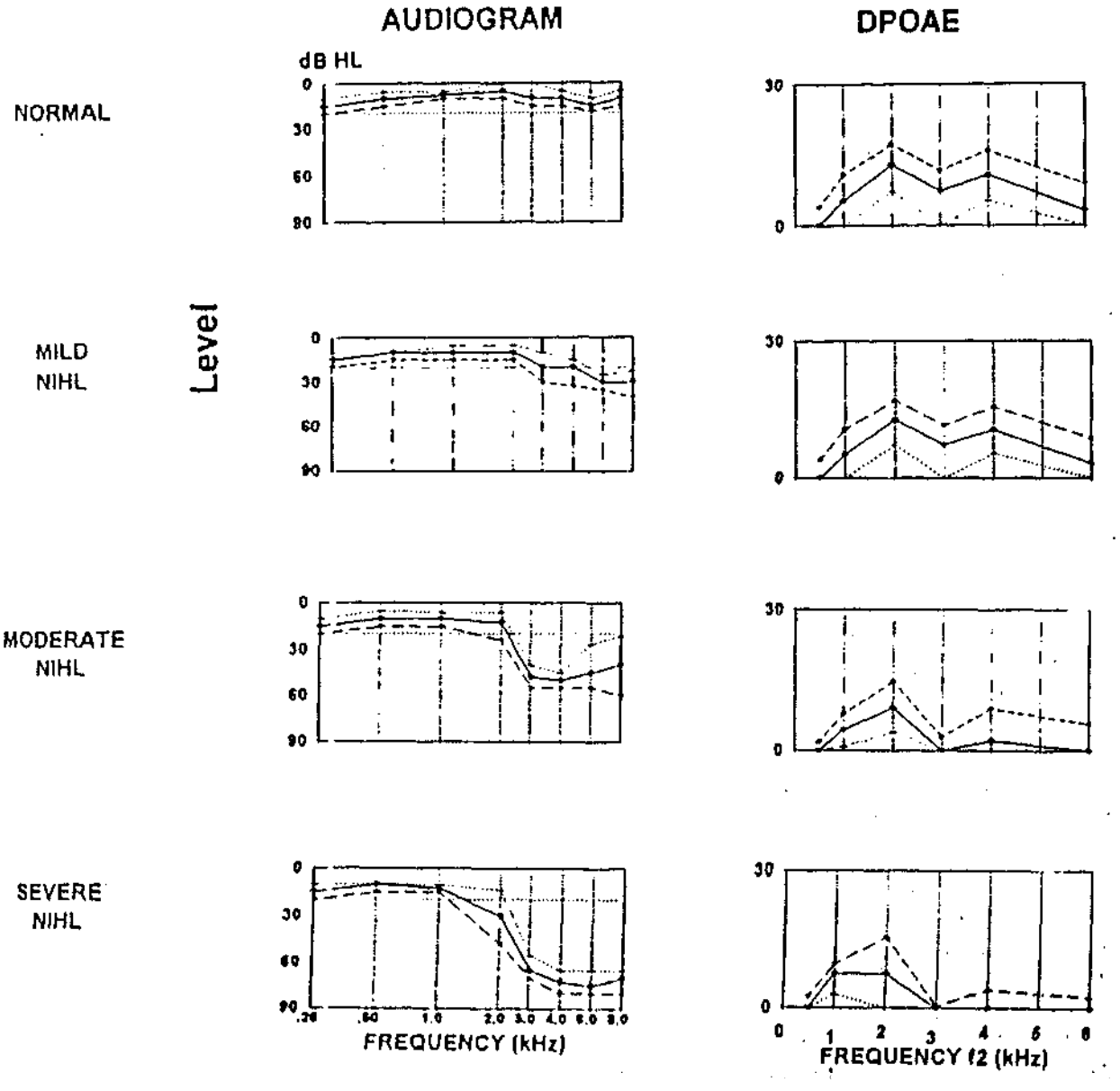


Figure -. *Median audiograms and DPOA E levels of the normal and NIHL subjects. The dotted lines represent the high and low quartites.*
Figure-2: Adapted from Attias,J., Bresloff, I., Reshef,I., Horowitz, G., and Furman, V./, 1998.

METHOD

The present study aimed at finding out the efficacy of extended high frequency audiometry and distortion product otoacoustic emissions as early identifiers of industrial noise induced hearing loss. In order to investigate the same, the following method was used :

Subjects :

Two groups of subjects were taken

Group I : Subjects with history of industrial noise exposure (Experimental group).

Group II : Subjects with no significant history of hazardous noise exposure (Control group).

The following Table 3.1 provides the details regarding the 80 ears that were studied.

Table 31: Number of ears, mean age and age range of subjects in experimental and control group.

		Experimental Group	***Control Group***
Number of right ears		19	20
Number of left ears		21	20
Age (in years)	Mean	27.7	23.8
	Range	20-35	20-35

Subject Selection Criteria:

The experimental group comprised of subjects who met the following criteria:

a) History of atleast 2 years of industrial noise exposure at the workplace. The minimum noise level to which the subjects were exposed was 74 dB for 7 hours / day. The data regarding the noise exposure was obtained from records of the industry.

b) The subjects were selected irrespective of the usage of ear protective devices (EPDs).

There were 26 subjects using EPDs and 14 of them not using EPDs.

c) Hearing thresholds less than or equal to 20 dB HL in the frequencies 250 Hz, 500 Hz, 1 kHz, 3 kHz, 4 kHz, 6 kHz and 8 kHz.

d) Normal middle ear functioning - confirmed through immittance evaluation.

e) 16 hours of rest from industrial noise exposure prior to evaluation to prevent the temporary effects of noise.

f) No significant history of other conditions such as ototoxicity, neurological disorders, etc.,

The control group comprised of subjects who met the following criteria :

a) No significant history of exposure to hazardous noise.

b) Hearing thresholds less than or equal to 20 dBHL in the audiometric frequencies 250 Hz, 500 Hz, 1 kHz, 2 kHz, 3 kHz, 4 kHz, 6 kHz and 8 kHz.

c) Normal middle ear functioning - confirmed through immittance evaluation.

d) No significant history of other conditions such as ototoxicity, neurological disorders, etc.,

Instruments used :

a) Otoscope was used for inspecting the earcanal and to rule out any contra indication for audiological evaluation.

b) A calibrated clinical audiometer, Grason Stadler Incorporation, Model - 61 (GSI - 61), Revision 2.4 connected to TDH 50P head set and B-71 bone vibrator was used for conventional frequency audiometry.

c) The same audiometer, connected to HDA 200 Sennheiser headphones was used for extended high frequency audiometry.

d) A calibrated Grason stadler Incorporation, Model - 33 GSI-33, (Version 2), middle ear analyzer was used to rule out middle ear pathology.

e) A calibrated ILO 292 Otodynamics, DP Echoport plus, version 5, was used to record DP gram.

Test environment:

The tests were carried out in an air conditioned, sound treated room with the ambient noise levels within permissible limits (American National Standards Institute, 1991, cited in Wilber, L.A., 1994). Conventional and high frequency audiometry were carried out in a double room situation, whereas, DPOAE and immittance evaluations were done in a single room situation.

Test procedure:

The testing was done in the following steps:

a) Case history

b) Otoscopic examination

c) Immittance evaluation

d) Conventional audiometry

e) Extended High frequency audiometry

f) Recording of DPOAEs.

Casehistory :

A detailed case history was taken to collect information about demographic data, and to rule out any significant history of hazardous noise exposure in the control group. In the experimental group, demographic data and information about the type and duration of noise exposure, use of ear protective devices, etc., were collected. (Refer to Appendix A and B for questions).

Otoscopic Examination :

Otoscopic examination was done in order to inspect the condition of the earcanal and tympanic membrane. Only subjects with normal otoscopic findings were taken up for the study.

Immittance evaluation :

Tympanometry, acoustic reflexes and reflex decay test were administered to rule out middle ear pathology, retrocochlear pathology and neural adaptation.

Conventional audiometry :

Each subject was seated comfortably in the patient room. The following instructions were given to each subject. "Raise your forefinger whenever you hear the sound. Pay attention and raise it even for the slightest sound you hear". Then, using the modified Hughson - Westlake procedure (Carhart and Jerger, 1959, cited in Silman, S., and Silverman, C.A., 1991), the hearing thresholds of the subjects across the audiometric frequencies 250 Hz to 8 kHz were found out. At frequencies beyond 2 kHz, the thresholds for mid - octaves were also obtained. The bone conduction thresholds were found out for the audiometric frequencies from 250 Hz to 4 kHz.

The above steps were carried out in order to ensure that the subjects met the specified selection criteria.

Extended High frequency audiometry:

The hearing thresholds of the subjects at the frequencies 9 kHz, 10 kHz, 11.2 kHz, 12.5 kHz, 14 kHz, 16 kHz, 18 kHz and 20 kHz were found out using the same procedure that was used for conventional audiometry.

Recording of DPOAE:

Each subject was seated comfortably and was instructed to relax and minimize any extraneous movements during the test. The probe was inserted gently into the ear canal with an appropriate probe tip. The DP - gram option was selected from the test menu and the test resolution was chosen. Then, the click stimulus checkfit routine was carried out to ensure that the best fit was achieved. This was followed by the instrument automatically adjusting the DP tones to the pre-set level. After all these preliminaries, the actual test was carried out. The following protocol was used for recording of DP gram

Table 3.2 : Protocol used for DPOAE

Parameters	*Values*
Primary stimuli	$F_1 < F_2 ; F_1 : F_2 = 1.2$
Stimulation levels	L_1 = 65 dB SPL ; L_2 = 55 dB SPL
Emissions recorded at	2f1-f2
Number of points per octave	3 points / octave
Frequency swept	250 Hz to 6 kHz
Minimum number of sweeps	112. If emissions were not seen, then the number of sweeps was increased to 208 to confirm the results.

After the recording of DPOAE, the difference between the level of emissions and the level of noise floor (S/N value) was noted at 86% replicability.

RESULTS AND DISCUSSION

The aim of the present study was to check the efficacy of EHFA and DPOAE as early identifiers of NIHL. EHFA and DPOAE were administered to the experimental and control group and the obtained data was subjected to statistical analysis. The statistical analysis carried out and the results obtained are as follows.

The mean and standard deviation (S.D.) of the data was found out across each frequency. The independent sample T-test was carried out using Statistical Program Software System Inc., version 10. to find out whether the difference between mean of the experimental group and control group was statistically significant or not. Table-4.1 summarises the results of the T-test for EHFA. It was evidenced that a statistically significant difference existed between the control and experimental group across all frequencies at $p<0.01$, except at 9 kHz, 12.5 kHz, where $p<0.05$. The obtained results were in par with the reports of Corliss et al. (1970), which stated that in subjects exposed to noise, changes occur above 8 kHz before they are noticed below 8 kHz.

Figure 3 gives the DP gram of a subject of the control group and Figure 4 gives the DP gram of a subject of the experimental group. Table - 4.2 summarises the results of the T-test for DPOAE in experimental group and control group. It is clear that the decrease in the S/N (Emission - Noise) value in the experimental group is statistically significant ($p<0.01$). The results of this study is supported by the study conducted by Attias et al. (1998).

Table 4.1 : Mean, S.D. and t-values across frequencies of extended high frequency audiometry of control (C) and experimental (E) groups.

Frequency	*Group*	TV	*Mean*	*S.D.*	*'t' value*
9 kHz	C	40	4.75	8.24	-2.273*
	E	40	9.38	9.88	
10 kHz	C	40	-2.25	9.67	-2.781**
	E	40	4.25	11.18	
11.2 kHz	C	40	11.5	8.02	-3.763**
	E	40	20	11.82	
12.5 kHz	C	40	3.75	12.39	-2.397*
	E	40	12	17.93	
14 kHz	C	40	10.25	10.31	-3.661**
	E	40	21.8	17.23	
16 kHz	C	40	1	15.7	-3.475**
	E	40	14.13	18.01	
18 kHz	C	40	10.88	12.7	-4.471**
	E	40	24.5	14.49	
20 kHz	C	40	2.25	6.79	-2.877**
	E	40	6.88	7.57	

= p<0.05;**=p<0.01

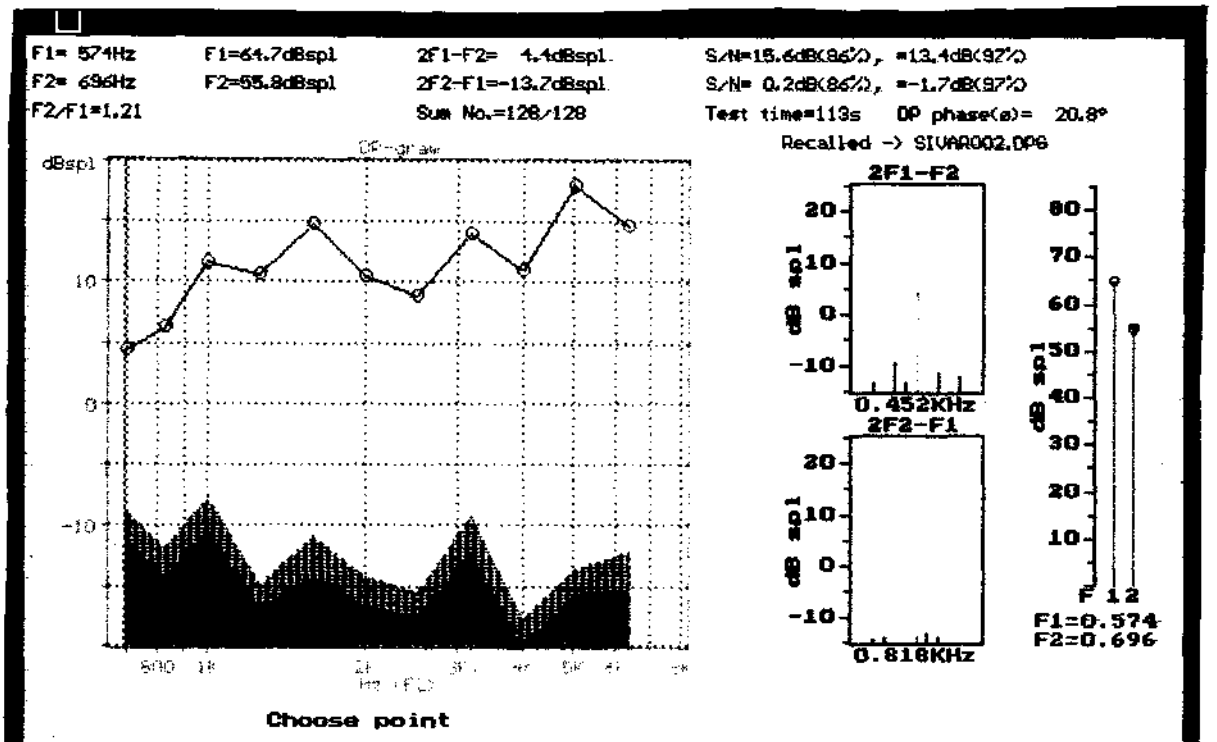


Figure - 3 : DPgram of a subject of control group.

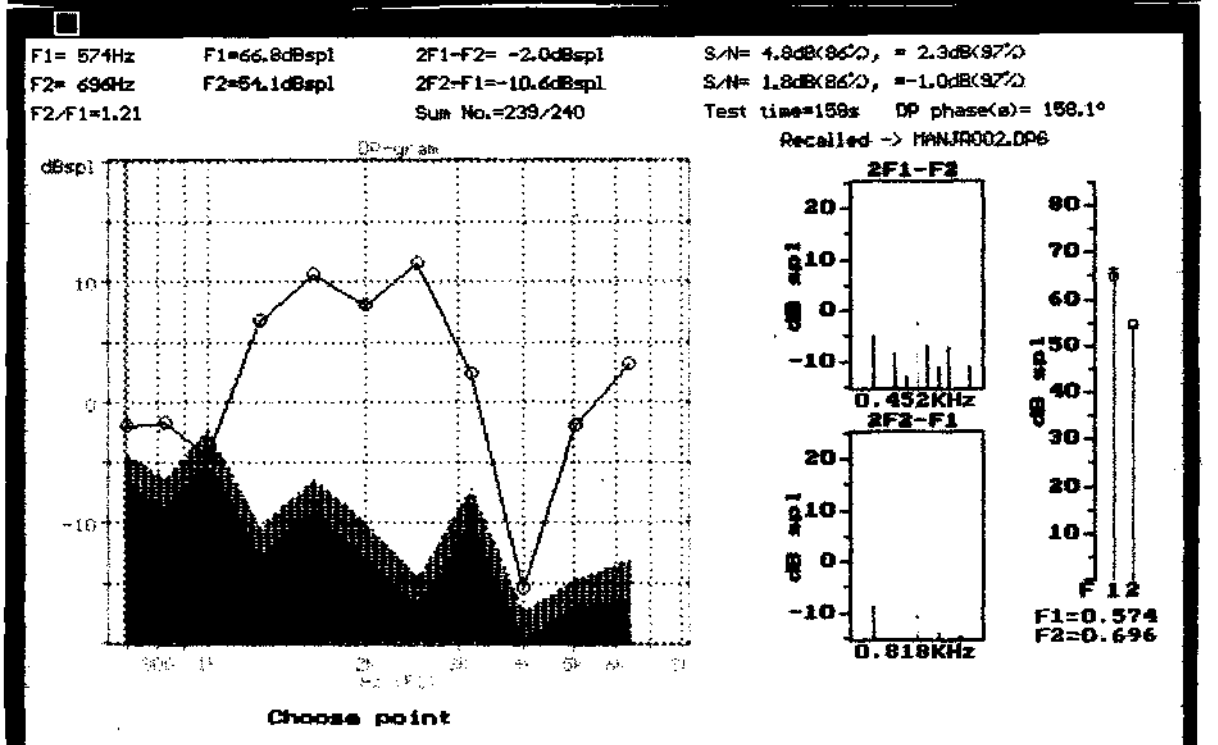


Figure - 4 : DPgram of a subject of experimental group.

Table 4.2 : Mean, S.D. and t-values of DPOAE of control (C) and experimental (E) group.

Frequency	*Group*	***N***	*Mean*	***S.D.***	*'t' value*
0.452 kHz	C	40	15.25	3.5	5.818**
	E	40	8.3	6.68	
0.537 kHz	C	40	18.83	4.24	6.179**
	E	40	11.66	5.99	
0.635 kHz	C	40	21.4	4.85	5.13**
	E	40	14.22	7.4	
0.818 kHz	C	40	24.22	4.88	4.118**
	E	40	17.88	8.44	
1.025 kHz	C	40	26.37	5.28	4.735**
	E	40	19.87	6.9	
1.270 kHz	C	40	24.92	5.68	3.696**
	E	40	19.9	6.45	
1.611kHz	C	40	23.92	7.31	3.398**
	E	40	18.36	7.33	
2.026 kHz	C	40	24.26	5.45	4.087**
	E	40	18.63	6.82	
2.563 kHz	C	40	24.08	6.83	3.945**
	E	40	17.95	7.07	
3.210 kHz	C	40	26.26	6.61	2.877**
	E	40	21.95	6.79	
4.053 kHz	C	40	22.55	6.07	3.522**
	E	40	17.16	7.54	

** **= $p<0.01$

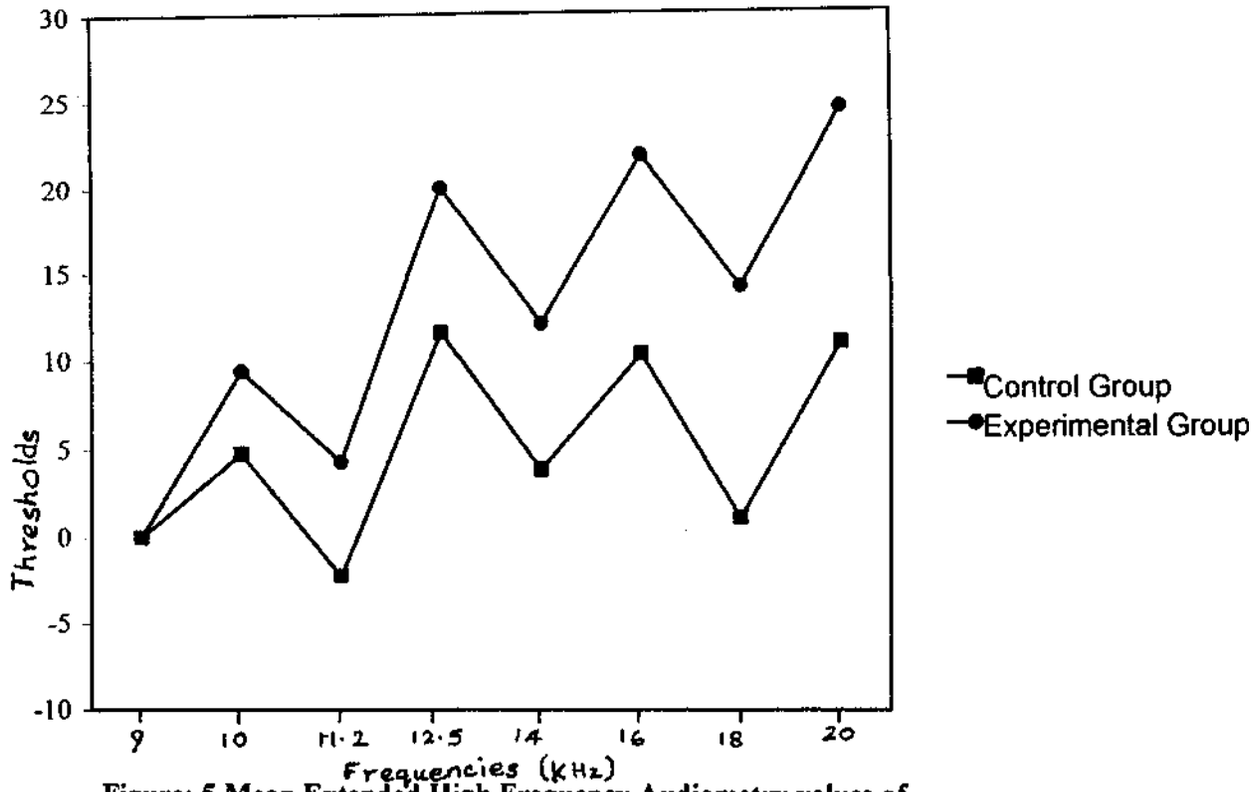


Figure: 5 Mean Extended High Frequency Audiometry values of Control and Experimental group

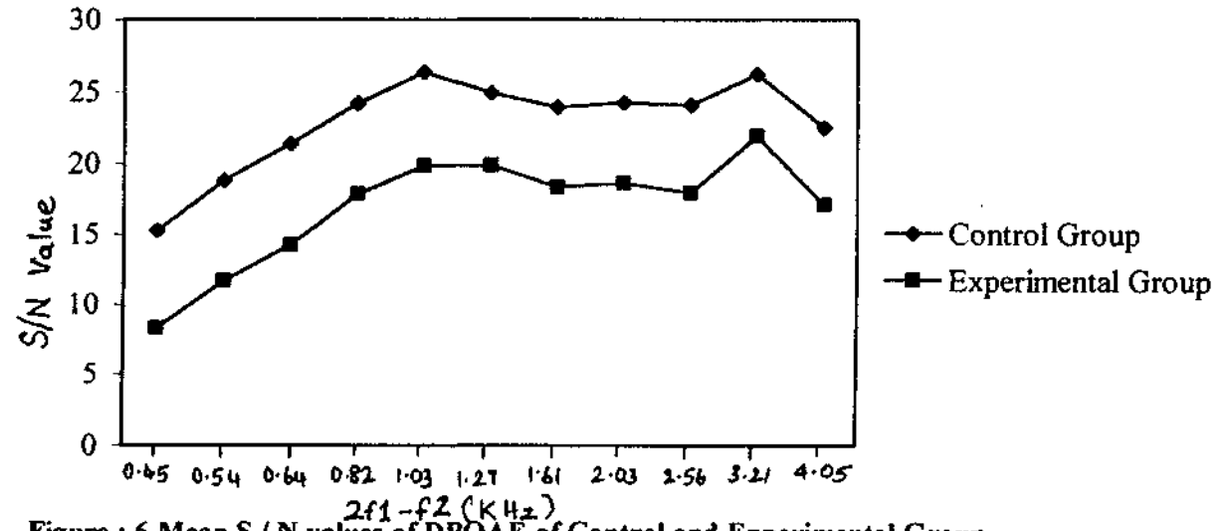


Figure : 6 Mean S / N values of DPOAE of Control and Experimental Group

Establishing cut - off point or Norm value:

For EHFA, the average threshold of all individuals was found out at each frequency. The mean of all the averaged frequency threshold was found out to obtain the average EHFA threshold. The S.D. was also found out for this value. "Mean + 2 S.D." was taken as the cut - off point or norm. The individual with thresholds greater than the cut off value at any one frequency was considered to have failed EHFA.

For DPOAE, the average S/N value was found out in a similar manner. Here, the cut - off point was "mean - 2. S.D." The individual with S/N values below the cut off at any one of the frequencies above 0.818lHz was considered to have failed DPOAE. Here, the frequencies below 0.818 kHz was not considered because of the contamination of the emissions with noise at the lower frequencies. Also, Gorga et al. (1994) have determined an absence of low frequency DPOAE even amongst normal hearing subjects. Table 4.3 gives the norm value of EHFA and DPOAE.

Table 4.3 : Norm value for EHFA and DPOAE.

	Mean	*S.D.*	*Mean ± 2.S.D*
EHFA (Average)	5.234	6.581	18.396
DPOAE (Average)	22.914	3.875	15.164

Table - 4.4 gives the percentage of individuals who have thresholds greater than the cut - off value (18.39) at each frequency. From Table - 4.4, it is evidenced that maximum number of individuals have been indicated as susceptible for NIHL at the frequency 18 kHz followed by 14 kHz, 16 kHz and 11.2 kHz. The results of Corliss et al. (1970) that the region above 12,000 Hz was particularly vulnerable to noise damage, supports this finding. Fausti et al. (1981) have also stated that the region between 13,000 Hz to 20,000 Hz showed the greatest changes when exposed to noise. So, when EHFA has to be used as a screening tool or when it has

to be used within a short span of time, it is advisable that the hearing thresholds of the individual be checked in the frequency range of 11.2 kHz to 18 kHz, starting from 18 kHz and then going on to 11.2 kHz.

Table 4.4 : Percentage of individuals failed in EHFA across frequencies.

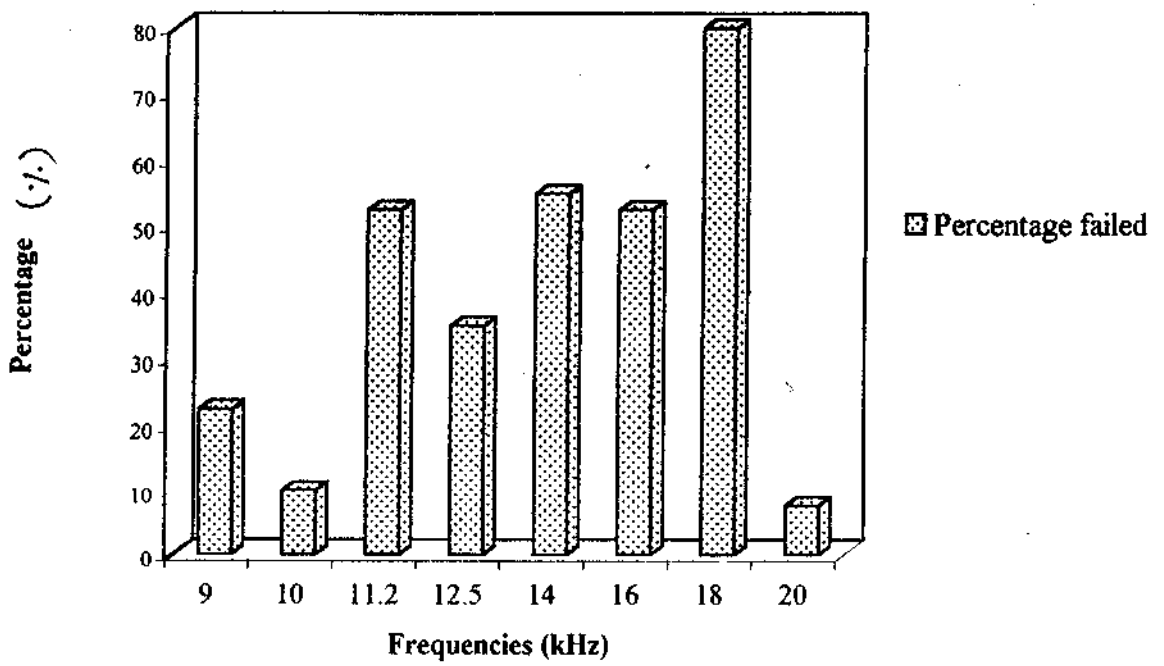
Frequencies	*No. of individuals failed (N=40)*	*Percentage*
9 kHz	9	22.5
10 kHz	4	10
11.2 kHz	21	52.5
12.5 kHz	14	35
14 kHz	22	55
16 kHz	21	52.5
18 kHz	32	30
20 kHz	3	7.5

Table - 4.5 gives the percentage of individuals who had DPOAE S/N values below the cut - off point (15.16). From Table - 4.5, it is evidenced that maximum individuals have failed the test at the frequency 4.053 kHz followed by 1.611 kHz, 1.025 kHz, 0.818 kHz, and then 2.026 kHz (excluding the low frequency DPOAE). Attias et al. (1998) have also reported significant reduction in DPOAE amplitude at the frequencies 1 kHz, 2 kHz, 3 kHz and 4 kHz in noise exposed individuals. So, if DPOAE has to be done in a short span of time to test for NIHL, it is advisable that it is done at f2 frequencies ranging between 1 kHz to 6 kHz.

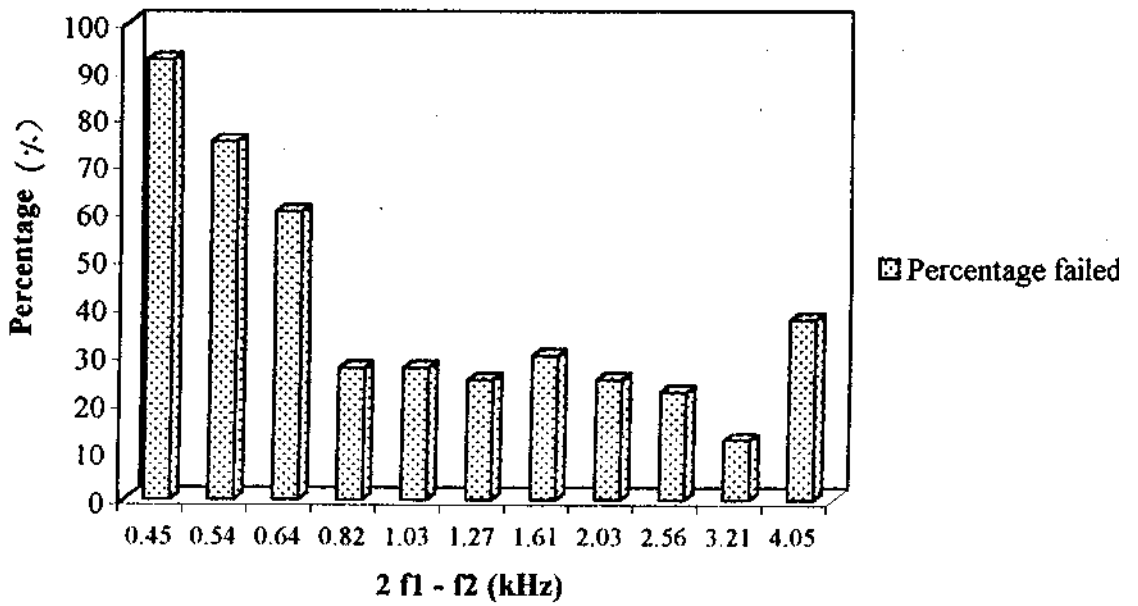
Table 4.5 : Percentage of individuals failed in DPOAE across frequencies.

Frequencies	*No. of individuals failed (N=40)*	*Percentage*
0.452 kHz	37	92.5
0.537 kHz	30	75
0.635 kHz	24	60
0.818 kHz	11	27.5
1.025 kHz	11	27.5
1.270 kHz	10	25
1.611kHz	12	30
2.026 kHz	10	25
2.563 kHz	9	22.5
3.210 kHz	5	12.5
4.053 kHz	15	37.5

On the whole, 92.5% have failed in EHFA and 70% of individuals have foiled in DPOAE. Chi-square analysis indicated a statistically significant difference ($p<0.01$) between these two. Thus, it can be inferred that EHFA identified NIHL better than DPOAE in the early stages.



Graph 3 : Percentage failed in EHFA across frequencies



Graph 4 : Percentage failed in DPOAE across frequencies

The experimental group was further divided into three groups depending on the level of noise the subjects were exposed to. The three groups are depicted in Table 4.6:

Table 4.6 : Grouping in experimental group based on noise levels

Group	*Noise level*	*No. of subjects*
X	< 90 dBA	15
Y	80-90 dBA	18
Z	< 80-90 dBA	7

The mean and S.D. of the EHFA and DPOAE was found out across frequencies in each of these groups separately. The small sample T-test was then administered between the three groups.

Table - 4.7 summarises the mean and S.D. values of EHFA across frequencies for all the three groups. Table - 4.8 summarises the results of T-test among the 3 groups. It indicated that there was no statistically significant differences between the means of the three groups. This implies that the EHFA findings remain almost the same inspite of the difference in the level of noise the subjects have been exposed to.

Table - 4.9 summarises the mean and S.D. of DPOAE across frequencies for the three groups. The small sample T-test results for this data is summarized in Table - 4.10. This indicated a statistically significant difference between the groups X and Y for the frequencies 1.611 kHz, 2.026 kHz ($p<0.05$). There was no statistically significant difference between the means of the groups X and Z. Between the means of the groups Y and Z, a statistically significant difference existed at the frequencies 1.025 kHz and 1.270 kHz ($p<0.05$). The reason for these differences can be partly attributed to the lack of homogeneity in the usage of ear protective devices. All the individuals in group X used ear protective devices. But, in group Y and Z, very few individuals used ear protective devices.

Table 4.7 : Mean and S.D. across frequencies of extended high frequency audiometry within the experimental group based on the level of noise exposure.

Frequency	*Mean*			***SD***		
	X(N=1S)	*Y(N=J8)*	*Z(N=7)*	*X(N=15)*	*Y(N=18)*	*Z(N=7)*
9 kHz	8.33	10	10	7.48	12.6	7.07
10 kHz	-0.67	6.39	9.29	9.79	12.34	7.32
11.2 kHz	15.67	21.39	25.71	9.97	11.09	15.39
12.5 kHz	8	13.33	17.14	10.66	19.55	25.79
14 kHz	21.67	21.94	22.14	13.45	17.33	25.63
16 kHz	21	9.17	12.14	13.78	20.09	17.99
18 kHz	29.33	19.72	26.43	6.22	18.03	14.92
20 kHz	8.67	5.56	6.43	6.94	8.38	6.9

Table 4.8 : t-values across frequencies within the experimental group based on the level to noise exposure.

Frequency	*(-value*		
	X VsY	*X VsZ*	*Y VsZ*
9 kHz	-0.45	-0.05	0
10 kHz	-1.79	-2.38	-0.58
11.2 kHz	-1.54	-1.85	-0.79
12.5 kHz	-0.94	-1.2	-0.4
14 kHz	-0.05	-0.06	-0.02
16 kHz	1.93	1.28	-0.34
18 kHz	1.96	0.65	0.1
20 kHz	1.15	0.71	0.24

Table 4.9 : Mean and S.D. of DPOAE within the experimental group based on the level of noise exposure.

Frequency	*Mean*			*SD*		
	X(N=15)	*Y(N=18)*	*Z(N=7)*	*X(N=15)*	*Y(N=18)*	*Z(N=7)*
0.452 kHz	6.11	10.88	6.34	8.86	4.55	3.49
0.537 kHz	10.29	13.59	9.61	7.34	4.99	3.96
0.635 kHz	14.29	15.31	11.29	8.86	6.84	5.16
0.818 kHz	18.37	20.19	10.91	7.25	7.41	10.55
1.025 kHz	20.94	21.77	12.67	6.84	4.8	7.81
1.270 kHz	17.93	22.97	16.2	5.2	5.62	7.96
1.611 kHz	14.83	21.78	17.1	7	7.15	14.68
2.026 kHz	14.85	21.15	20.23	7.56	5.37	5.39
2.563 kHz	15.19	18.41	22.83	8.08	5.59	5.97
3.210 kHz	21.22	22.39	22.37	8.64	5.54	5.99
4.053 kHz	13.77	19.42	18.57	7.97	6.95	6.19

Table 4.10 : 't' values of DPOAE within the experimental group based on the level of noise exposure.

Frequency	*t-value*		
	XVsY	*X VsZ*	*Y VsZ*
0.452 kHz	-1.99	-0.06	2.37
0.537 kHz	-1.53	0.23	1.89
0.635 kHz	-0.41	0.83	1.4
0.818 kHz	-0.71	1.94	1.07
1.025 kHz	-0.41	1.99	2.64
1.270 kHz	-2.65*	0.61	2.41
1.61 Ik kHz	-2.8**	-0.65	1.09
2.026 kHz	-2.79**	-1.68	0.39
2.563 kHz	-1.35	-0.94	-1.75
3.210 kHz	-0.47	-0.35	0.01
4.053 kHz	-2.17*	-1.44	0.28

*= p< 0.05. **=PD <0.01

An individual who foiled in any one frequency in EHFA was considered to have failed EHFA (Refer Table 4.11). Likewise, an individual who failed in any one frequency beyond 0.818 kHz in DPOAE was considered to have failed DPOAE (Refer Table 4.12). In this manner, 100%, 83% and 100% failed in EHFA in the groups X,Y and Z respectively. 87%, 50% and 86% of individuals failed in DPOAE in the groups X, Y and Z respectively. Chi-square analysis indicated statistically significant differences ($p<0.01$) between EHFA and DPOAE in all the three groups. This implied that irrespective of the level of noise the subjects were being exposed to, EHFA is a better indicator of NIHL at an early stage or EHFA is a sensitive tool for early identification of NIHL.

Table 4.11 : Percentage of individuals failed in EHFA across frequencies in the three groups.

	9 kHz	10 kHz	11.2 kHz	12.5 kHz	14 kHz	16 kHz	18 kHz	20 kHz	Overall
X	13.3	0	40	20	60	60	93.3	13.3	100
Y	18.9	16.7	66.7	38.9	55.6	50	66.7	5.6	83
Z	0	14.3	42.9	57.1	42.9	42.9	85.7	0	100

Table 4.12 : Percentage of individuals failed in DPOAE across frequencies in the three groups.

	0.452 kHz	***0.537 kHz***	***0.635 kHz***	***0.818 kHz***	***1.025 kHz***	***1.270 kHz***	***1.611 kHz***	***2.026 kHz***	***2.563 kHz***	***3.210 kHz***	***4.053 kHz***	***Overall***
X	93.3	80	53.3	20	20	33.3	46.6	46.6	40	20	53.3	87
Y	88.9	61.1	55.6	16.7	11.1	5.6	11.1	11.1	16.7	5.6	27.8	50
Z	100	100	85.7	71.4	85.7	57.1	42.9	14.3	6	14.3	28.6	86

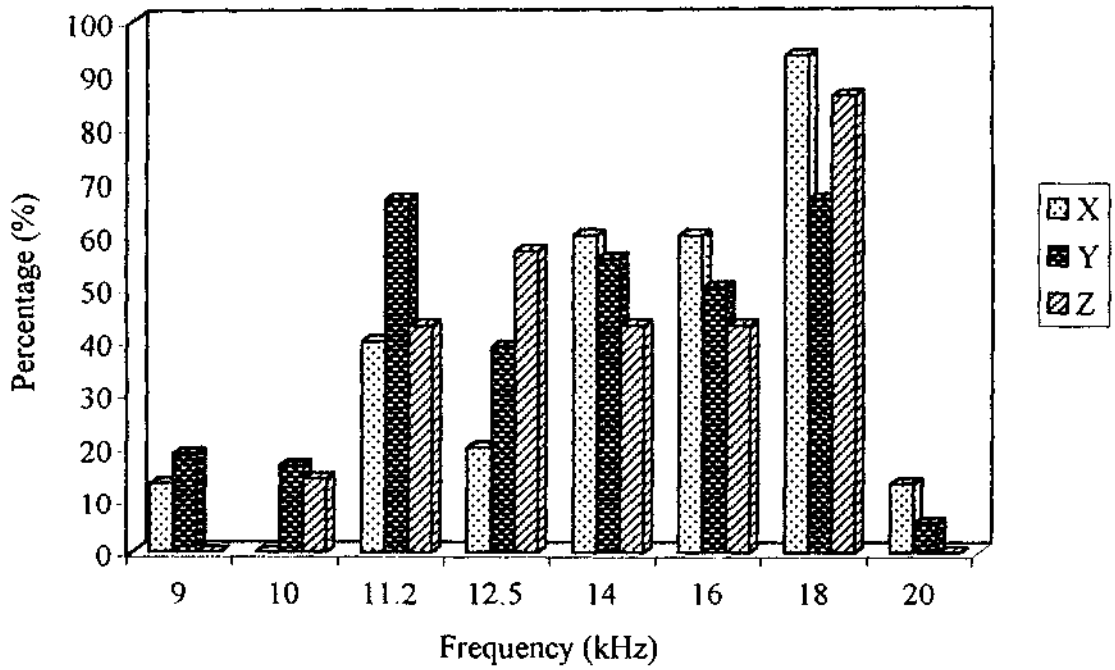


Figure 9 : Percentage failed in EHFA in the three groups within the experimental group

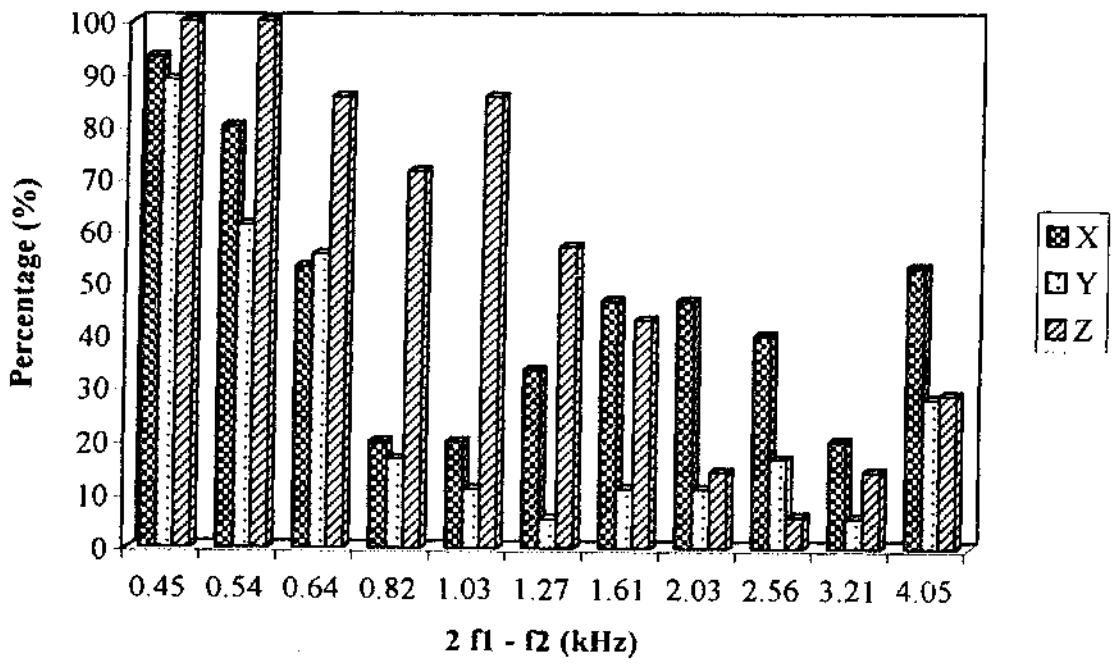


Figure 10 : Percentage failed in DPOAE in three groups within the experimental group

In an industrial set - up, apart from the sensitivity of the test, other factors such as efficiency in terms of time and cost are also important. If an industrial audiologist prefers to use EHFA routinely, then he or she has the option of getting a conventional audiometer with an additional facility of EHFA. If an industrial audiologist wants to use DPOAE routinely, then the option is to purchase an OAE system separately. Bearing in mind that a conventional audiometer is the basic requirement in an industrial audiological set up, it is understood that the former option would be better in terms of cost efficiency. So, EHFA would be a better option than DOPAE in terms of cost efficiency.

With respect to efficiency in terms of time, there is no major difference observed between the two tests. Depending upon the availability of time, the hearing threshold of the individual can be tested in the limited frequency range (11.2 kHz to 18 kHz) in EHFA. In DPOAE, the resolution can be varied depending upon the availability of time. But, if a very low resolution of testing is selected, then reliable information regarding the cochlear status cannot be obtained. Considering the above factors, it is preferable to use EHFA than DPOAE.

From the above discussions it can be implied that EHFA is a better tool in early identification of NIHL than DPOAE and is also efficient in terms of cost and time.

SUMMARY AND CONCLUSION

The effect of noise on the auditory system is a major problem in today's highly technological society. Exposure to loud noise for a long duration results in sensori neural hearing loss which is slow and progressive and initially affects high frequency. Clinical measures must identify auditory changes with special attention to early detection, baseline function and monitoring the changes in hearing sensitivity in industrial workers.

Early identification of NIHL is very important. Only if we identify it early (before the speech frequencies are affected), adequate measures can be taken to prevent further loss of hearing sensitivity. Literature states that EHFA and DPOAE are useful in identifying NIHL at an early stage. The present study was carried inorder to check for the efficacy of EHFA and DPOAE as early identifiers of NIHL. The following were investigated :

a) EHFA results among industrial workers with normal hearing in conventional frequency audiometry.
b) DPOAE results among industrial workers with normal hearing in conventional frequency audiometry.
c) Comparison between EHFA results and DPOAE results in terms of the efficacy in early identification of NIHL.

40 noise exposed ears (experimental group) with normal hearing in the conventional frequency audiometry and 40 normal non - noise exposed ears (control group) were taken. The subjects were in the age range of 20 to 30 years. The experimental and control group comprised of otologically normal ears with no history of ototoxicity or other middle ear problems. The testing was done in the experimental group after ensuring 16 hours of rest from noise exposure to prevent the temporary effects of noise.

Otoscopic examination, Immittance evaluation, conventional frequency audiometry, EHFA and DPOAE were administered to both the groups. The following results were obtained.

- There was a statistically significant difference between the control group and experimental group for both EHFA and DPOAE.
- In EHFA, 18 kHz was most sensitive to the effects of noise followed by 14 kHz, 16 kHz and then 11.2 kHz.
- In DPOAE, the frequency 4.053 kHz was most sensitive followed by. 1.611 kHz, 1.025 kHz, 0.818 kHz and then 2.026 kHz (excluding the low frequency DPOAE).
- EHFA was more sensitive to the effects of noise than DPOAE, irrespective of changes in the level of noise.
- In terms of time and cost efficacy, EHFA appears to be more effective than DPOAE.

Recommendations:

a) The study can be carried out by having a better control on variables such as level of noise exposure, duration of noise exposure, usage of EPDs, etc.

b) A study can be done with follow up of the subjects for a period of time so that the efficacy of these tests can be known at different stages of noise exposure.

c) Follow - up of these subjects will also facilitate measurements of sensitivity and specificity of the tests.

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APPENDIX - A

Questionnaire for the Experimental group :

Name:

Age/ Sex:

Work place :

1. Since when are you working in this industry?
2. How long do you work in a day?
3. In which unit / department of the industry do you work?
4. What type of product is manufactured in the department or industry?
5. Approximately, what is the level of noise in your unit / department?
6. Is the noise continuous or intermittent?
7. Do you use EPDs ? If yes, what type of EPDs do you use and for what duration?
8. Do you have difficulty in conversing with the person standing next to you (approximately, 1 m) in the work environment?
9. Do you have any problem in hearing soon after your work?
10. Do you hear ringing sound in your ears soon after your work ?
11. Did you have any problem in hearing before joining this industry?
12. Did you ever have drugs of the Mycin group, etc. for any illness for a long period ?
13. Were you working anywhere else before joining this industry? If yes, was that a noisy environment ?

APPENDIX-B

Questionnaire for the control group:

Name :

Age / Sex:

1. Did you ever have any infection or problem of the ear ?
2. Did you ever have any problem in hearing ?
3. Did you ever work in any noisy environment or were you exposed to loud noise ?
4. Did you ever have drugs of the Mycin group, etc. for any illness for a long period ?