COCHLEAR AND NEURAL FUNCTIONS IN INDUSTRIAL WORKERS EXPOSED TO OCCUPATIONAL NOISE

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This Dissertation is submitted as part fulfillment for the Degree of

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CERTIFICATE

This is to certify that this dissertation entitled **"COCHLEAR AND NEURAL FUNCTIONS IN INDUSTRIAL WORKERS EXPOSED TO OCCUPATIONAL NOISE"** is a bonafide work in part fulfillment for the degree of Master of Science (Audiology) of the student (Registration No. 13AUD034). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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DECLARATION

This is to certify that this dissertation entitled **"COCHLEAR AND NEURAL FUNCTIONS IN INDUSTRIAL WORKERS EXPOSED TO OCCUPATIONAL NOISE"** is the result of my own study under the guidance of Mr. Sreeraj K., Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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Abstract

As the industries are growing faster, the auditory and non-auditory effects in industrial workers exposed to hazardous noise level will add to global burden. Hence, there is a need to study the auditory effects of noise as it might lead to permanent sensori neural hearing loss. Therefore, the present study was conducted in order to detect, monitor and identify early cochlear and neural changes in industrial workers exposed to occupational noise of >85 dB (A). Thresholds from conventional pure tone audiometry (250 Hz to 8 kHz), extended high frequency audiometry (9 to 16 kHz), amplitude and SNR of DPOAE (8 point per octave from 1001 to 7996 Hz), absolute latency, inter peak latency of click evoked ABR and absolute latency of wave V in CE-chirp ABR were assessed in 20 industrial workers (40 ears) exposed to occupational noise and were compared with 20 individuals (40 ears) not exposed to occupational noise. Individuals exposed to occupational noise were again compared within for the same parameters to see the effect of noise exposure duration. There was significant threshold elevation observed in extended high frequency audiometry and significant monotonic relation as the duration of noise exposure increased. Although, similar findings were found in conventional pure tone audiometry, the shift in threshold was really small. Most of the ears had absent or significantly decreased amplitude and SNR and these changes were according to duration of exposure. In click evoked ABR, mean absolute latencyand inter peak latency were within normal limits but found significant prolongation in wave III along with inter peak latency (I-III,III-V). Also, there was significant prolongation of wave I and V observed when duration of noise exposure increased. CE chirp ABR was found to be sensitive as the response was either absent or prolonged. Hence, it can be concluded that using comprehensive test battery approach, early cochlear and neural pathological changes due to noise can be detected, identified and monitored.

Chapter 1

Introduction

Sound is an integral component of the environment, and plays a very important role in our normal development and survival by providing a valuable channel of sensory-environmental contact. Often, however, sound may interfere with our activities; disturb sleep, lead to auditory or other physiologic harm, or cause irritation and annoyance. When this occurs, sound is generally unwanted or undesirable, and is often referred as noise. Sound becomes noise only when it is physiologically or psychologically defined as unwanted (Kryter, 1970). This subjective definition of noise involves intricate (complex) physiologic, emotional, and psychological responses to sound (Burns, 1973; Kryter, 1970).Noise would be defined as audible acoustic energy (or sound) that is unwanted because it has adverse auditory and non-auditory physiological effects on humans (Kryter, 1994).

Noise is present in our day to day situation in the form of traffic noise, household noise or even when more than two people talking etc. But, usually these sounds are within safe levels of hearing which do not damage our ear sensitive structures. When these noise levels exceed safe level of hearing i.e. >85 dB (A), it causes damage to the ear structures leading to noise induced hearing loss (NIHL). NIHL can occur immediately or after a probation period which might take days or even years (Miller, Watson, & Covell, 1963; National Institute of Health and other communication disorders, 2014).

Noise is one of the major preventable causes of hearing loss. Noise induced hearing loss (NIHL) is a permanent impairment of hearing resulting from abrupt exposure to loud sound or protracted exposure to high levels of noise. Once, an

individual is exposed to damaging sound at his work place or at home, inner ear sensitive structures might get damaged, leading to noise-induced hearing loss (Miller, Watson, & Covell, 1963). After presbycusis, NIHL is the second most common form of sensory neural hearing loss (Nandi &Dhatrak, 2008) and has its prevalence of 16 % worldwide (Nelson, Nelson, Barrientos, &Fingerhut, 2005). National Institute of Occupational Safety and Health (1998) suggests individuals exposed to noise >85dB (A) are at risk of developing NIHL.

NIHL is one of the major health issue (Department of Health and Human Services, 2009), as there have been increased opportunities to noise exposure in recent days which might lead to damage of hearing. Noise exposure can cause two kinds of health effect namely non-auditory effects and auditory effects. Non auditory effects include disturbance with sleep, stress and anxiety reaction etc. (Cohen, 1977). Auditory effect includes temporary threshold shift (TTS) and permanent threshold shift (PTS). Threshold shift recovers in TTS, but remains at an elated value in PTS (Miller, Watson, & Covell, 1963). Permanent NIHL is because of the destruction of cochlear hair cells or damage to their mechano-sensory hair bundles (Liberman & Dodds, 1984). Usually hair cells transduces acoustically evoked mechanical motion into receptor potential which leads to transmitter release at their glutamatergic synapses with cochlear afferent fibers. After over exposure to noise, hair cell damage may be seen within minutes or hair cell death might continue for days (Wang, Hirose, & Liberman, 2002). However, noise induced loss of spiral ganglion cells, cochlear afferent neurons contacting hair cells can get delayed by months or can continue till years (Kujawa & Liberman, 2006).

As these early cochlear changes might really take longer time (days to years) to express on a standard pure tone audiometry as thresholds might be within normal

limits. The thresholds get affected might initially get affected in the higher frequency region greater than 8 kHz. As the duration of exposure to noise increases, audiometric thresholds gets affected in the region of 3 to 6 kHz then extending to lower frequencies comparatively (Riga, Korres, Balatsouras & Korres, 2010). Hence, we might not be able to monitor early cochlear changes which might not be seen in regular pure tone audiometry which estimates hearing thresholds from 250 Hz to 8 kHz and might show hearing thresholds within normal hearing sensitivity.

However, normal hearing thresholds might not necessarily indicate normal cochlear function. Kujawa and Liberman (2009) revisited the issue of neural degeneration in ears with noise-induced threshold shifts in mice subjected to mild acoustic trauma. A temporary shift in hearing thresholds was noted, but also 50-60% permanent deafferentation of the auditory nerve fibers in the high frequency region of the cochlea was seen. Results of this study suggests that normal hearing thresholds can be accompanied by impaired function of efferent fibers that project from the brainstem to the cochlea (Kim & Frisina, 2002; Jacobson, Kim, Romney, Zhu, & Frisina, 2003; Zettel, Zhu, O'Neill, & Frisina, 2007; Zhu, et al., 2007). It can also be hypothesized that deafferentation of auditory nerve fibers, as observed in mice following "temporary" hearing loss might lead to develop a neural correlate in central auditory structures. Dean, Chen, Nahm, Mattiace and Kim (2013) conducted a study in mice subjected to 110-120 dB noise for 6 hours and found that they had absent auditory brainstem response even when otoacoustic emissions were present which shows there is damage after cochlear level. Electrophysiological research has shown that after exposure to noise, spontaneous neuronal activity and compound action potentials in the auditory nerve are decreased (Dallos, Harris, Ozdamar, & Ryan, 1978; Salvi, Ding, Wang, & Jiang, 2000). Similar findings have been reported within

the first day post- exposure of intense noise in the central structure of the dorsal cochlear nucleus of cats (Liberman & Kiang, 1978). Further, clinical sequel of noise exposure, like tinnitus and hyperacusis are usually present after the hearing loss has recovered which cannot be explained with cochlear pathology satisfactorily. These discrepancies might possibly be related to additional, central mechanisms involved in the generation of NIHL (House &Brackmann, 1981).

Recovery of threshold sensitivity after noise exposure has been related to indicate reversal of damage to delicate mechano-sensory and inner ear neural structures and no constant or postponed effect for auditory function. Results from study done by Kujawa and Liberman (2009) on mice to revisit the issue of neural degeneration in ears with noise-induced threshold shifts suggest that noise-induced damage to the ear is progressive in nature that are considerably more widely diffused than the findings in routine pure-tone audiometry. This primary neuro-degeneration will contribute to difficulties hearing in noisy situations, and could lead to tinnitus, hyperacusis and other perceptual problems usually associated with inner ear damage. Hence, tests other than standard pure tone audiometry which are sensitive to early cochlear changes to be included while testing an individual exposed to hazardous occupational noise.

Wang,Yang, Li, Hou and Han (2000) reported that extended high frequency audiometry is sensitive in early diagnosis of NIHL. The study consisted of 120 individuals exposed to occupational noise between 95-115 dB (A). Conventional audiometry from 500 to 6 kHz and extended high frequency audiometry from 9 to 20 kHz was done. Results revealed that there was significant difference in threshold shift in the region 10 kHz and 16 kHz compared to 500 Hz to 6 kHz. These results can be supported by studies done bySomma, Pietroiusti, Magrini, Coppeta, Gardi et al.

(2008); Turkkahraman, Gok, Karlidaq, Keles and Ozturk (2003); Riga, Korres, Balatsouras and Korres, (2010).

Vinck, Cauwenberge, Leroyi and Corthals (1999) studied the sensitivity of DPOAEs in monitoring the effects of TTS on outer hair cells. They exposed broad band noise of 90 dBSPL to normal hearing individuals for 1 hour. DP-gram was recorded soon after the exposure and after 6 hours of exposure. Also, pure tone threshold was calculated from 250 Hz to 8 kHz. The amplitude in DP-gram was significantly reduced soon after exposure especially at frequencies 4 kHz and above even when audiometric thresholds were within normal limits. After six hours of exposure, amplitude in DP-gram reverted back to normal. They concluded that changes due to exposure to noise are first seen in DPOAE's than in conventional audiometry. Hence, DPOAE's are more sensitive to noise induced changes compared to conventional audiometry. Studies done by Korres, Balatsouras, Tzagaroulakis, Kandiloros, Ferekidou and Korres(2009); Seixas, Kujawa, Norton, Sheppard, Neitzel and Slee(2004), and Balatsouras (2004) reported that DPOAE's might serve as a sensitive and objective tool in diagnosing early changes in outer hair cells in individuals exposed to hazardous level of noise.

It may be seen from the literature that detailed audiological assessment is very important in evaluating, tracking and managing NIHL, since it can lead to manifestation of different auditory symptoms apart from hearing loss. A classic NIHL is of a sensory neural type which involves damage to the inner ear. It is usually bilaterally symmetrical high frequency hearing loss between 3 and 6 kHz especially a notch at 4 kHz called boiler's notch and then advancing to the lower frequencies 0.5 kHz, 1 kHz or 2 kHz (Nandi &Dhatrak, 2008).Several studies have suggested that Oto Acoustic Emissions may provide early indication of noise induced hair cell damage

up to 30%, before it is seen in standard audiometry (Seixas, Goldman & Sheppard, 1995; Desai, Reed, Cheyne, Richards &Prasher, 1999). The auditory brain stem response (ABR) has become widely recognized as a sensitive and cost-effective screening modality in neuro-otologic diagnosis (Telian,Kemink, Graham & Kileny, 1989).A study done by Attias and Pratt (1985) showed that there are latency changes in click ABR with individuals exposed to noise. There are limited studies regarding ABR especially monitoring the early cochlear or neural changes in individuals exposed to occupational noise. ABR can be recorded using various kinds of stimuli like click, tone burst etc. Recently, chirp stimulus compensating for basilar membrane traveling wave delay has come into existence which yields better amplitude and early latency of peaks compared to click evoked ABR. Hence, it might help in monitoring the early cochlear changes in industrial workers.

This area has not much probed which might help us identifying the early neural changes or 'hidden hearing loss' of NIHL. Routine Audiological testing along with ABR might play a very important role in early identification and monitoring occupational noise induced hearing loss.

Need for the Study

Noise is more prevalent in day today life and can cause both auditory and non-auditory health effects. NIHL remains highly common in occupational settings. Worldwide, 16 percent disabling hearing loss of adults is ascribed to occupational noise and around 7 to 21% in other sub regions (Nelson, Nelson, Barrientos, &Fingerhut, 2005). Also 39% of industrial workers who were exposed to noise level >87.3 dB (A), for 8-12 hour/day suffered from NIHL (Ranga, Yadav, Yadav, &Ranga, 2014). As industries are growing in a faster rate in the present scenario, NIHL will progressively add to the global burden of disability.So, extensive

studies are to be done in this area for populations exposed to noise. There are studies reported in the literature regarding the effects of occupational noise on peripheral hearing structures; but there is dearth of literature regarding the neural component in subjects exposed to noise (Groschel, Ryll, Gotze, Ernst, &Basta, 2014) across varying duration of noise exposure. As it is proven that long duration exposure to occupational noise cause permanent threshold shift (Ferrite & Santana, 2005), finding the effect of duration of occupational noise on auditory system exposed to varying durations is important, which might help in monitoring and preventing auditory effects.

Aim of the study

To examine the cochlear and auditory neural functions in industrial workers exposed to occupational noise.

Objectives of the study

1. To compare the cochlear and neural findings between individuals exposed to occupational noise and those who are not exposed to occupational noise according to National Institute for Occupational Safety and Health (1998) guidelines on the following audiological tests

- Pure tone Audiometry along with extended high frequency audiometry.
- Auditory Brainstem Response

  Click evoked ABR

  CE-chirp evoked ABR
- Distortion Product Oto Acoustic Emissions

2. To find the effect of noise exposure duration on cochlear and neural components based on the above tests.

Chapter 2

Review of Literature

Noise is unwanted, but an unavoidable aspect prevalent in most of the work places. Exposure to noise can cause both auditory and non-auditory effects. Non-auditory effects include disturbance with sleep, stress and anxiety reaction etc. (Cohen, 1977).Auditory effect includes Noise induced hearing loss (NIHL) which is of two forms namely temporary threshold shift and permanent threshold shift.NIHLcan be defined as permanent or temporary impairment of hearing resulting from abrupt exposure to loud sound or protracted exposure to high levels of noise. Once, an individual is exposed to damaging sound at his work place or at home, inner ear sensitive structures might get damaged, leading to NIHL (Miller, Watson, & Covell, 1963).

After overexposure, NIHL recovers with aggressive course of time (Miller, Watson, & Covell, 1963) for 2 to 3 weeks, depending on severity initially.Thresholds might recover completely (temporary threshold shift) or maintained at an elated value (permanent threshold shift). Permanent NIHL is because of the destruction of cochlear hair cells or damage to their mechano-sensory hair bundles (Liberman & Dodds, 1984). After over exposure to noise, hair cell damage may be seen within minutes or hair cell death might continue for days (Wang, Hirose, & Liberman, 2002). In comparison, noise-induced loss of spiral ganglion cells, cochlear afferent neuron's cell body contacting these hair cells, can be delayed by months and can continue for years (Kujawa & Liberman, 2006).National Institute of Occupational Safety and Health (1998) suggests individuals exposed to noise >85dB (A) for equal to or greater than 8 hours per day are at risk of developing NIHL. With growing industries in the

present scenario, there is high risk for individuals exposed to occupational noise to develop NIHL.

Prevalence of NIHL

Noise is one of the most common causes of hearing loss which is preventable. Worldwide, 16 percent disabling hearing loss of adults is ascribed to occupational noise (Nelson, Nelson, Barrientos, &Fingerhut, 2005). Statistics computed by National Institute on Deafness and other Communication Disorder (2014) revealed that 15% i.e. around 26 million American individuals in the age range 20 to 69 developed high frequency hearing loss due to occupational noise.

Fuente and Hickson (2011) reviewed prevalence of NIHL in Asia based on several studies in sub regions of Asia. A study done in Taiwan by Wu et al.(1998), revealed that 34% among 10,000 industrial workers exposed to >85 dB (A) of noise showed hearing loss greater than 40 dBHL above 4000 Hz. A study done in China by Zhi, Sheng and Levine (2000) showed 34% of individuals working in rural industry exhibited NIHL.A study done in India by Pawardhan,Kolate and More(1991), reported that 75% of bus drivers had sensori neural hearing loss who were exposed to noise level between 86 to 106 dB(A). Further, study done in India by Kumar, Mathur, Varghese, Mohan, Singh and Punnet(2005),reported that 48 % of agricultural workers (tractor drivers) who were exposed to 110 dB (A) of noise exhibited significant high frequency hearing loss compared to agricultural workers who were non tractor drivers. They concluded that a total prevalence NIHL is of 17% in Asia.

Singh, Bharadwajand Kumar (2012) studied prevalence of permanent threshold shift among iron and steel small and medium enterprises (SME).Study was conducted on 572 individuals randomly selected from 3 SME casting and 3 SME

forging units in northern India. Pure tone audiometry was done in three frequency region; low frequency (250 to 1000 Hz), mid frequency (1500 to 3000 Hz) and high frequency (4000 to 8000 Hz) and threshold obtained were compared with control group who were not exposed to occupational noise. Results revealed that 90% of them exhibited significant hearing loss at medium and high frequencies (1 to 8 kHz). Hence, it can be concluded that a greater number of individuals are at risk of developing NIHL at high frequencies.

A study done by Ranga, Yadav, Yadav and Ranga (2014) reported the effect of noise pollution and effect of different noise levels at two industries (textile and hard strip rolling) at Bhiwani (Haryana), India. The study included 100 male workers in the age range of 19 to 55 years exposed to occupational noise from 50 dB (A) to 120 dB (A) for 8 to 12 hours a day. Hearing threshold were obtained using standard pure tone audiometry from 250 Hz to 8000 Hz where 25 dB was considered as cut off criteria for the presence of hearing loss. Results revealed that 39% of individuals exposed to noise level >87.3 dB (A), in textile and hard strip rolling industries for 8-12 hour/day suffered from NIHL. They also reported that chronic exposure to noise affect cochlea leading to sensori neural hearing loss especially a notch at 4 kHz initially.

Based on the above studies, we can note that there is increased prevalence of NIHL. Hence, it is important to study the effects of NIHL and pathophysiology behind, to prevent or monitor auditory and non-auditory effects of noise exposure. Prevention programs for individuals working in industries should include awareness and education plan about auditory and non-auditory affects due to exposure of hazardous noise, hearing conservation programs and frequent assessment of hearing to monitor the changes (Henderson, Bielefeld, & Harris, 2006).

Pathophysiology of NIHL

Individuals Exposed to noise >85dB (A) for eight hours per day are at risk of developing NIHL (NIOSH, 1998). Exposure to noise results in injury to inner ear sensitive structures leading to hearing loss. Hearing loss may be temporary in nature which is reversible or permanent in nature.

Temporary threshold shift (TTS) is a temporary increase in hearing sensitivity when an individual is exposed to high intensity sounds i.e. > 85dB (A) (NIOSH, 1998). TTS may last up to 16 hours or more, and then the thresholds improve gradually (Gelfand, 2001). Exposure to more high intensity sounds results in greater threshold shifts. The threshold shift reaches a saturation level, further which there is no increase in thresholds. When this maximum threshold has reached, it is called asymptomatic threshold shift. The thresholds are usually measured after the cessation of noise for two minutes as the thresholds are unstable before, known as TTS2. The course of recovery is measured from TTS2. As the duration and intensity of noise exposure increases, the threshold shift increases until it reaches asymptomatic threshold shift (Gelfand, 2001).

Temporary threshold shift seems to be an indication of temporary changes in outer hair cells resulting from exposure to hazardous sound or noise. As the exposure to high intensity sound increases the damage to hair cells and its associated structures becomes permanent leading to permanent threshold shifts. When there is no recovery from temporary threshold shift, it manifests as permanent threshold shift which is irreversible (Gelfand, 2001).

As seen from several animal studies, the outer hair cells are more sensitive in the inner ear. Hence, damages are seen initially in these areas when they are exposed

to continuoushigh intensity noise. Outer hair cells in the mid frequency region 3000 to 4000 Hz gets damaged first followed by high frequency region 6000 to 8000 Hz followed by low frequency region. Also, there can be decreased blood flow to inner ear which might cause damage of OHC's, IHC's, also death and or loss of auditory nerve fibers(Narasimhan, Jayashankaran, Rajagopalan, &Sreesailapathy, 2014).

Gao, Ding, Zheng, Ruan and Liu (1992) compared the type of stereocilial damage from temporary threshold shift and permanent threshold shift in guinea pigs. The study was conducted on57 guinea pigs in two experiments. In the first experiment, they subjected 10 guinea pigs to 110 dB broadband white noise for 30 minutes and other 10 guinea pigs were subjected to 120 dB noise for 150 minutes. The thresholds were monitored from 250 Hz to 8000 Hz up to 80 days. The first group exhibited more threshold shift at 2 and 4 kHz and the threshold gradually improved and returned to normal by 96 hours. The latter group subjected to 120 dB noise exhibited threshold shift from 1 kHz to 8 kHz. The thresholds especially at 4 and 8 kHz did not revert back to normal even after 30 days. In the second experiment, similar method was followed. One group of 16 guinea pigs were subjected to 110 dB broadband white noise for 30 minutes and other group of 16 guinea pigs were subjected to 120 dB noise for 150 minutes. They conducted scanning and transmission electron microscopy on both groups. The acute temporary threshold shift group exhibited damage which was only restricted to tips of stereocilia of 3rd row of outer hair cells. The permanent threshold shift group, entire length of stereocilia was affected in all three rows of OHC's and or 1st row of IHC's. They concluded that status of stereocilia plays an important role in deciding the reversibility of hearing loss. From the above study we can draw a conclusion that prolonged exposure to high

intensity sound leads to permanent damage to hair cells resulting in permanent noise induced hearing loss.

A model developed by Henderson, Bielefeld and Harris (2006) indicates that noise might lead to increase reactive oxygen species (ROS) causing death of hair cells.

ROS can be caused by:

- increased activity of mitochondria at hair cells in cochlea,
- excitotoxicity at the junction of inner hair cells and afferent nerve fibers present in auditory nerve,
- limited supply of oxygen through blood flow to cochlea (Narasimhan et al., 2014).

Henderson, Bielefeld and Harris (2006) also supports role of Reactive oxidative stress causing hair cell death due to exposure of noise. Le Prelle, Yamashitha, Minami, Yamasoba and Miller (2006) also reported ROS formation leads to damage of hair cells mechanisms.

These pathophysiological changes findings in cochlea can be seen symptomatically within days or it might take years. When these pathological findings become evident, the threshold shift can be measured using standard pure tone audiometry. Shah, Baig and Vaidya(2013); Kirchner, Evenson, Dobie, Rabinowitz, Crawford, Kopke and Hudson(2012);McBride and Williams(2001) studied the effect of noise exposure on pure tone audiometry in the region of 250 Hz to 8 kHz. They observed elevated thresholds in these regions in individuals exposed to hazardous noise especially in the region 3 kHz to 6 kHz. Also, this threshold shift becomes permanent when the duration of exposure increases.

Pure Tone Audiometry in individuals exposed to occupational noise

Pure tone audiometry is a quantitative measurement of air conduction thresholds in the octave and mid octave frequency region 250 Hz to 8000 Hz and bone conduction threshold are obtained from 250 Hz to 4000 Hz. Air conduction thresholds are measured using head phones and bone conduction threshold are measured using bone vibrator connected to audiometer. Hearing thresholds can be classified based pure tone thresholds. Based on pure tone average, Goodman (1967) classified hearing thresholds in dB HL into six categories ranging from normal hearing sensitivity (within 15 dBHL) to profound hearing loss (above 90 dB HL). This helps in identification of threshold shift due to various causes.

Kirchner et al. (2012)listed typical characteristics of noise induced hearing loss as seen in pure tone audiometry. NIHL is typically sensorineural in origin resulting from damage to hair cells in cochlea. It is typically bilateral as the exposure to noise is similar to both ears. On Audiogram it shows a "notch" also known as "boiler's notch" in the frequency region 3000Hz to 6000 Hz reverting at 8000Hz.

Mantysalo andVuori (1984) studied the effect of impulse noise and continuous noise on hearing in 3 Groups. Group 1 consisted of 30 individuals exposed to impulse noise for various duration of exposure and Group 2 consisted of 12 individuals exposed to continuous noise for various duration of exposure. Group 3 consisted of 10 individuals who were not exposed to occupational noise and they served as controls. The audiometric thresholds were calculated for frequencies from 1 to 8 kHz. Results revealed that threshold shift is similar between individuals exposed to shorter duration (3 to 4 years) of impulse noise and individuals exposed to longer duration of continuous (5 to 6 years). Also, they suggested that exposure of impulse noise for duration of 5 to 6 years lead to permanent threshold shift at 4000 Hz and 6000 Hz and

exposure to a duration of greater than 10 years leads to permanent threshold shift in the audiometric range from 1000 to 8000 Hz. We can conclude from the study that exposure to impulse noise has detrimental effects on threshold shift compared to continuous noise.

McBride and Williams(2001) studied the relationship between different kinds of noise exposure (continuous and impulse) and classic notch at high frequencies. 682 individuals were identified with risk of NIHL among 5678 individuals who were exposed to occupational noise of hazardous level. Pure tone thresholds were calculated using Bekesymethod in an audiometric booth. Individuals were not exposed to noise to at least 16 hours before the testing. The audiograms were given to three clinicians to identify the notch and found notch was present between 3000 to 6000 Hz. They concluded that 4 kHz notch is sensitive compared to 6 kHz notch in identifying NIHL. Hence, we can infer that there is a strong relationship between 4 kHz notch and NIHL.

Shah, Baig and Vaidya(2013) studied threshold shift in 256 textile industrial workers. They were divided into two groups consisting of 128 each based on the intensity of exposure to noise. Group 1individuals were exposed to noise below 80 decibels and Group 2 individuals were exposed to noise greater than 80 dB. Pure tone thresholds were calculated for air conduction between 125 Hz to 8000 Hz and bone conduction between 250 Hz to 4000 Hz. They considered individuals with normal hearing thresholds and mild hearing loss as ‘not affected’. Individuals with moderate to profound hearing loss were considered as ‘affected’. Results revealed that a total number of 120 ears were ‘affected’ in Group 1 and a total number of 160 ears were affected in Group 2. We can conclude that individuals exposed to both the noise

levels are at risk of developing NIHL, but more chances when exposed to noise level greater than 80 dB.

As reported from several authors (Fausti, Erickson, Frey, Rappaport, & Schechter, 1981; Wang, Yang, Li, Hou, & Han 2000;Riga, Korres, Balatsouras & Korres, 2010), High frequency audiometry is comparatively more sensitive compared to standard pure tone audiometry in detecting early cochlear changes in individuals exposed occupational noise.

Extended high frequencyaudiometry in individuals exposed to occupational noise

Extended high frequency audiometry is an extension of conventional audiometry which assesses hearing threshold in the frequency region greater than 8000 Hz. The audiometric frequency region extends from 9 kHz up to 20 kHz.

Fausti, Erickson, Frey, Rappaport and Schechter (1981) studied the threshold shift in individuals exposed occupational noise. Total of 36 subjects were considered in the study and were divided into two groups based on type of noise exposure. Group 1 individuals were exposed to impulse noise and Group 2 individuals were exposed to steady state noise. Pure tone thresholds were estimated for octave frequencies from 250 Hz to 8000 Hz. Extended high frequency thresholds from 8000 Hz to 20000 Hz at 1000 Hz interval also were calculated for both groups and was compared with controls. Group 1 exhibited prominent threshold shift greater than 45 dBHL compared to individuals with normal hearing in 9000 Hz to 20000 Hz as well as from 2000 Hz to 8000 Hz. Group 2 exhibited prominent threshold shift around 45dBHL than individuals with normal hearing in 13000 Hz to 20000 Hz region and around threshold shift of 5 to 20dBHL in the region between 250 Hz to 12000 Hz. Hence, it

can be concluded that high frequency audiometry is more sensitive compared to conventional audiometry in identifying NIHL.

Wang, Yang, Li, Hou and Han (2000) also reported that extended high frequency audiometry is sensitive in early diagnosis of NIHL. The study consisted of 120 individuals exposed to occupational noise between 95 to 115 dB (A). Conventional audiometry from 500 Hz to 6 kHz and extended high frequency audiometry from 9 to 20 kHz was done. Results revealed that there was significant difference in threshold shift in the region 10 kHz 16 kHz compared to 500 Hz to 6 kHz. This study also reveals the sensitivity of extended high frequency audiometry in identifying early cochlear changes compared to standard pure tone audiometry.

Somma, Pietroiusti, Magrini, Coppeta, Gardi et al. (2008) the threshold shift in extended high frequency audiometry in 184 industrial cement workers exposed to noise level greater than 80 dB(A). Conventional audiometry from 250 to 8 kHz and extended high frequency audiometry from 9 to 18 kHz was done.The thresholds were then compared 98 workers who were not exposed to occupational noise. Results revealed that there was significant difference in threshold shift in the region above 13 kHz between cement workers exposed to noise and workers not exposed to noise. This study also suggests extended high frequency audiometry is more sensitive than conventional audiometry to early noise related changes.

Riga, Korres, Balatsouras and Korres (2010)studied the effect of duration of exposure to noise and its implication on conventional and extended high frequency audiometry. The study consisted of 151 subjects working for 8 hours per day and exposed to 90-110 dB (A) of occupational noise. The subjects were divided into three groups based on duration of exposure. Group 1 had 0 to 10 years of exposure, Group

2 had 10 to 20 years of exposure and Group 3 had 21 to 33 years of exposure. Conventional audiometry from 250 Hz to 8000 Hz as well as high frequency audiometry from 9000 Hz to 20000 Hz was carried out. Results from conventional audiometry revealed normal threshold in Group 1, significant elevated threshold at 2000 Hz and 4000 Hz in Group 2 and significant elevated threshold at 250 Hz, 500 Hz and 1000 Hz in Group 3. Results from extended high frequency audiometry revealed significant elevated thresholds at 12500 Hz, 14000 Hz and 16000 Hz in Group 1. Further, exposure to noise did not reveal any significant changes. Hence, from the study we can conclude that extended high frequency audiometry is more sensitive to noise induced changes in the earlier years compared to conventional audiometry.

Turkkahraman, Gok, Karlidag, Keles and Ozturk(2003) compared standard and extended high frequency threshold in 64 individuals exposed to occupational noise for longer durations. Pure tone thresholds at frequencies from 250 Hz to 16 kHz were assessed. There were significantly higher thresholds found from 4 kHz to 16 kHz especially at frequencies 4 kHz, 6 kHz, 14 kHz and 16 kHz. Hence, he concluded that extended high frequency audiometry along with standard audiometry as it will be more sensitive in early diagnosis of NIHL. Hence, extended high frequency audiometry along with conventional audiometry plays a very important role in diagnosing early noise induced hearing loss.

Mehrparvar, Mirmohammadi, Ghoreyshi, Mollasadeghi and Loukzadeh(2011) designed a study to compare conventional audiometry and extended high frequency audiometry in early diagnosis of NIHL. The study consisted of two groups of textile workers exposed and not exposed to noise greater than 85 dB (A).They found that

hearing thresholds were greater for 4 kHz, 6 kHz and 16 kHz. But, 16 kHz had significantly greater thresholds compared to 4 kHz.

Based on the above studies, we can conclude that extended high frequency audiometry is more sensitive to noise induced hearing loss and hence it has to be used along with conventional audiometry which helps audiologists to diagnose NIHL at an early stage.

Also, along with extended high frequency audiometry, Oto acoustic emissions has proved to be a sensitive test in identifying early cochlear changes in various cochlear pathologies (Mauermann, Uppenkamp, Kollmeier & Hengel, 2015). Studies done by Balatsouras (2004);Vinck, Cauwenberge, Leroyi and Corthals(1999); Seixas, Kujawa, Norton, Sheppard, Neitzel and Slee (2004) reports that Distortion product reflects early cochlear changes compared to conventional audiometry. They recommendDPOAE as an important toolin identifying and monitoring the effects of noise on hair cells.

Distortion Product Oto Acoustic Emission in individuals exposed to occupational noise

Otoacoustic emissions (OAEs) are sounds of cochlear origin, which can be recorded by a microphone fitted into the ear canal. They are caused by the motion of the cochlea's sensory hair cells as they energetically respond to auditory stimulation. OAEs provide a simple, efficient and non-invasive objective indicator of healthy cochlear function.

Distortion product oto acoustic emission is a type of OAE which is simultaneously measured from presentation of two pure tone f1 and f2, at the level L1 and L2 respectively where f2 < f1. DPOAEs are recorded in the frequency region where these primary tones interact to determine the functioning of OHC's.

Vinck, Cauwenberge, Leroyi and Corthals(1999) studied the sensitivity of DPOAEs in monitoring the effects of TTS on outer hair cells. They exposed broad band noise of 90 dBSPL to normal hearing individuals for one hour. DP-gram was recorded soon after the exposure and after 6 hours of exposure. Also pure tone threshold was calculated from 250 Hz to 8 kHz. The amplitude in DP-gram was significantly reduced soon after exposure especially at frequencies 4 kHz and above, even when audiometric thresholds were within normal limits. After 6 hours of cessation of exposure, amplitude in DP-gram reverted back to normal. They concluded that changes due to exposure to noise are first seen in DPOAE's than in conventional audiometry. Hence, DPOAE's are more sensitive to noise induced changes compared to conventional audiometry.

Balatsouras (2004) studied conventional audiometry and DPOAE's in 34 subjects (64 ears) in the age range 31to 51 years who were exposed occupational noise from 8 to 31 years. Pure tone thresholds were calculated for frequencies from 250 Hz to 8 kHz. DPOAE's were obtained using two primary tones f1 and f2 (f2<f1) at a constant ratio of 1.22 and equal level (L1=L2). DPOAE's were computed as DP-grams in the frequency region 1.001 to 6.348 kHz recorded in 1/3rd octave steps. Results were compared with 30 normal hearing individuals (60 ears) who served as controls. Audiometric thresholds in all the individual exposed to noise were within normal limits (<15 dBHL) in low and mid frequency region but there was a notch seen in 3 kHz to 6 kHz region and thresholds reverted back to normal at 8 k Hz. DP-gram of control group had clear response to f1 and f2 product, but there was significant decrease in amplitudes or even absent response at all or some of the frequencies. The significant difference was mainly at high frequencies (1587-6348 Hz). Hence, they concluded that DPOAE's might serve as a sensitive and objective

tool in diagnosing NIHL. We can conclude that DPOAE's as a sensitive tool in identifying early cochlear changes.

Seixas, Kuwaja, Norton, Sheppard, Neitzel and Slee (2004) evaluated noise induced hearing loss with DPOAE's along with conventional audiometry. Conventional audiometry between 250 Hz to 8 kHz and DPOAE's were recorded for 456 participants (393 constructional workers and 63 controls). DPOAE's were obtained using two primary tones f1 and f2 (f2<f1) at a constant ratio of 1.22 and equal level (L2=L1-10). DPOAE's were computed as DP-grams in the frequency region between 1031 Hz and 10028 Hz. Both hearing threshold level and DPOAE deteriorated at frequency 4, 6 and 8 kHz. Hence, DPOAE serves as a sensitive tool to assess damage to inner ear structures due to exposure of noise.

Korres, Balatsouras, Tzagaroulakis, Kandiloros, Ferekidou and Korres (2009) evaluated noise induced hearing loss using DPOAE's along with conventional audiometry. The study included 105 individuals exposed to noise level 92 - 93 dB (A) for 8 hours per day. Pure tone thresholds were calculated for frequencies from 250 Hz to 8 kHz. DPOAE's were obtained using two primary tones f1 and f2 (f2<f1) at a constant ratio of 1.22 and equal level (L1=60 dB and L2=45dB). Audiometric thresholds were significantly elevated at 4 kHz region and reverting back to normal hearing at 8 kHz. DPOAE amplitude was significantly reduced compared to control group in the frequency region of 3000 Hz to 6000 Hz especially at 4000 and 6000.

Based on the above studies, we can conclude that DPOAE's serve as a reliable test in monitoring the cochlear changes in individuals exposed to occupational noise. However, All the above mentioned tests, assesses cochlear and early cochlear changes but they do not account for early neural changes which might be associated with

individuals exposed to occupational noise. Several times, normal hearing thresholds might not necessarily indicate normal cochlear function. Even when audiometric thresholds are within normal limits as the pathological changes might take days or years to express its symptoms, there might be physiological changes seen at cochlear or neural level which can be monitored through other tests assessing at brainstem or cortical level.

Kujawa and Liberman (2009) revisited the issue of neural degeneration in ears with noise-induced threshold shifts in mice subjected to mild acoustic trauma. A temporary shift in hearing thresholds was noted, but also50-60% permanent deafferentation of the auditory nerve fibers in the high frequency region of the cochlea was seen. Results of this study suggests that normal hearing thresholds can be accompanied by impaired function of efferent fibers that project from the brainstem to the cochlea (Kim & Frisina, 2002; Jacobson, Kim, Romney, Zhu, & Frisina, 2003; Zettel, Zhu, O'Neill, & Frisina, 2007; Zhu, et al., 2007). It can also be hypothesized that deafferentation of auditory nerve fibers, as observed in mice following "temporary" hearing loss might lead to develop a neural correlate in central auditory structures.

Dean, Chen, Nahm, Mattiace and Kim (2013) conducted a study in mice subjected to 110-120 dB noise for six hours and found that they had absent auditory brainstem response even when otoacoustic emissions were present which shows there is damage after cochlear level. Hence, it can be concluded that there are damage beyond cochlear level which cannot be identified using OAE's or pure tone audiometry.

Electrophysiological research has shown that after exposure to noise, spontaneous neuronal activity and compound action potentials in the auditory nerve are decreased (Dallos, Harris, Ozdamar, & Ryan, 1978; Salvi, Ding, Wang, & Jiang, 2000). Similar findings have been reported within the first day post-exposure of intense noise in the central structure of the dorsal cochlear nucleus of cats (Liberman & Kiang, 1978). Further, clinical sequel of noise exposure, like tinnitus and hyperacusis are usually present after the hearing loss has recovered which cannot be explained with cochlear pathology satisfactorily (House &Brackmann, 1981). These discrepancies might possibly be related to additional, central mechanisms involved in the generation of NIHL.

Electrophysiological tests like Auditory Brainstem Response might prove its role in identifying early neural correlate in individuals exposed to occupational noise. Hence, a single audiological test will not be sufficient to notice the early cochlear and neural changes resulting from exposure to noise. A test battery approach comprising of different test assessing both cochlear and neural changes to be included when assessing an individual exposed to hazardous occupational noise. A test battery approach might include test like pure tone audiometry, extended high frequency audiometry, distortion product oto acoustic emission to assess cochlear changes and auditory brainstem response which might help in finding early neural changes.

Hence, auditory brainstem response along with conventional audiological tests might give an insight to early neural correlate along with cochlear changes which would give holistic view of pathological changes in individuals exposed to occupational noise.

Auditory Brainstem Response in individuals exposed to occupational noise

ABR is a representation of synchronous discharge of onset sensitive single unit of 1st through 6th order neurons of the peripheral and central auditory neurons system to an external stimulus. Stimulus can vary from broad band click stimuli to frequency specific tone burst or chirp. ABR serves as a sensitive tool in identifying and differentiating normal brainstem versus pathologies related to brainstem. Also it might help in identifying and monitoring early neural changes which might not be seen in conventional audiometry, extended high frequency audiometry or distortion product OAE's.

Click ABR

Attias and Pratt (1985) studied the changes in ABR in individuals exposed to occupational noise >90dB (A). They recorded ABR using click stimuli of alternating polarity at two repetition rate of 10/sec and 55/sec at 70 dBHL in 16 new industrial workers with normal hearing soon after an exposure to pink noise of 95 dBHL through TDH 39 headphones for 15 minutes accounting for temporary threshold shift. ABR were again recorded for the same individuals when they developed permanent threshold shift. They assessed waveform morphology, absolute latencies for I III and V peak and Inter peak latencies for I-III, III-V and I-V. Results revealed prolongation of wave I, III and V was found and also IPL values increased as the repetition rate increased from 10/sec to 90/sec. They conclude that ABR with faster repetition rate are sensitive to noise induced changes than lower repetition rate. But, they should have recorded ABR before exposure to pink noise which would have served as baseline for the measurement.

Attias, Urbach, Gold andShemes (1993) studied involvement of central auditory neural activities in chronic tinnitus patients with noise induced hearing loss

using ABR and event related potential. The study consisted of 12 individuals with chronic tinnitus for 5 years who were exposed to impulse noise. All of them had typical NIHL (loss at frequencies >2 kHz and bilaterally symmetric). They recorded bilateral ABR for a click stimuli of alternating polarity at 120 dBSPL using 10/ sec repetition rate. They assessed absolute latencies for I, III and V peak and Inter peak latencies for I-III, III-V and I-V. Results of ABR revealed that the values fell within normal limits when compared with control group who were not exposed to occupational noise. But, they had reduced amplitude in event related potentials compared to control group. Their results suggested involvement of central auditory problems in individuals with chronic tinnitus and NIHL.

Almadori et al. (1998) studied any possibility of retro cochlear pathology in individuals with noise induced hearing loss. The study consisted of 54(108 ears) individuals exposed to occupational noise at least for years and having bilateral and symmetric sensori neural hearing loss at 1000 Hz to 4000 Hz region. They recorded ABR for two stimulation rates 21/sec and 51/sec at 70 dBnHL for clicks of alternating polarity. They assessed waveform morphology, absolute latencies for I, III and V peak and Inter peak latencies for I-III, III-V and I-V. The results revealed absolute latencies and Inter peak latencies were within normal limits. They observed poor waveform resolution was found especially in the I peak in 12 ears. The results also revealed absence of ABR in 5 ears which was not according to the hearing loss. From results of this study, we can conclude that hearing loss due to exposure of noise might also have some neural correlate but has to be probed more to know more details about the changes in central auditory pathway.

Xu, Vinck, Vel and Cauenberge(1998)studied the localization of pathological findings using ABR and EOAE in individuals exposed to occupational noise and

compared with normal hearing individuals not exposed to occupational noise. The study consisted of 22 individuals having bilateral symmetric sensori neural hearing loss at one frequency (4 kHz) or more than frequency. ABR were recorded using click stimuli at 80 dBnHL and 90 dBnHL at repetition rate of 21.3/sec. They assessed absolute latencies for V peak, Interpeak latencies for I-III, III-V and I-V and amplitude ratio of I/V. Results revealed that there was no significant difference between these parameters and values fell within normal limits. But amplitude ratio ofI/V decreased as the affected frequencies increased. Results of broad band TEOAE revealed decreased amplitude compared to control group. This suggests that pathological findings first appear in OHC's as OAE's were affected before ABR.

Dean, Chen, Nahm, Mattiace, and Kim (2013) conducted a study in mice subjected to 110-120 dB noise for 6 hours and found that they had absent auditory brainstem response even when otoacoustic emissions were present which shows there is damage after cochlear level.

There has been only limited number of studies regarding auditory brainstem response in individuals exposed to occupational noise with normal hearing sensitivity. Hence, this area to be probed in future research.

CE-chirp ABR.

Chirp stimuli are designed to overcome travelling time delay in basilar membrane to increase temporal synchrony which is abrupt in clicks. The Clauss Elberling (CE) chirp was designed using a delay model based on derived band ABRs to overcome cochlear travelling wave delay and to increase synchronicity (Elberling & Don, 2010). Elberling and Don (2008) used chirp stimuli which had frequency range from 200 Hz to 10000 Hz. But, now new broadband CE-chirp has been

included in Interacoustics EP-25 which has a flat spectrum in five octave bands from 350 to 11300 Hz.

Elberling and Don (2010) study consisted of 25 individuals with normal hearing having pure tone thresholds within 15 dBHL in the frequency region 500 Hz to 8000 Hz. They recorded ABR using CE-chirp with alternating polarity at repletion rate of 27/sec at different presentation level from 10 dBnHL to 80 dBnHL in 10 dB steps. They analyzed wave V latency at these levels. The mean latency of wave V was found to be 2.85 msec at 80 dBnHL. The latencies got prolonged as the intensity decreased in 10 dB steps. The CE-chirp also resulted in larger ABR. We can conclude that CE-chirp gives better amplitude and early latency compared to click ABR because of more synchronous activity resulting from travelling wave delay compensation.

Cebulla, Lurz, and Sheheta - Dieler (2014) compared click ABR and Chirp ABR thresholds in newborns. Latency and amplitude of Wave V was compared between click and chirp ABR. The result revealed that chirp ABR had shorter latency and larger amplitude which was easier to detect wave V at near threshold when compared to click ABR. Hence, chirp ABR can be used as a better tool to estimate hearing thresholds in infants as it is easier to identify the peak near threshold as it has larger amplitude.

Xu, Cheng and Yao (2014) studied the efficacy of Level Specific (LS) chirp stimuli evoked ABR in young children with mild to moderate hearing loss. LS chirp is a level specific broad band chirp which has frequency range similar to click stimuli where the edge frequencies were 0.1 and 10 kHz. They evaluated LS chirp thresholds evoked at 21.1/sec repetition rate with maximum level of 100 dB HL in 68 infants in

the age range of 6 to 12 months. They also obtained behavioral thresholds from visual reinforcement audiometry. They compared both behavioral and evoked thresholds and found LS chirp evoked threshold was much closed to behavioral threshold, the difference between two was within 5 dB. However, they also found that the thresholds were correlated better with severe hearing loss compared to mild hearing loss. They conclude that chirp ABR is an effective tool in identifying hearing loss. Hence, we can conclude that LS chirp ABR is a valuable tool in measuring hearing threshold.

Maloff and Hood(2014) compared thresholds click evoked ABR and Chirp ABR in individuals with normal hearing sensitivity and individuals with sensori neural hearing loss. The study consisted of 25 adult individuals with normal hearing and 25 adult individuals with mild to moderately severe SNHL. They recorded Click ABR and CE-chirp ABR with condensation polarity at a repetition rate at 27/sec. The initial presentation level of the stimuli were 100 dB peak SPL and was decreased in 10 or 20 dB steps until there was no response present. They analyzed absolute latency of wave V and peak to peak amplitude of wave V. The thresholds for both Click ABR and CE-chirp ABR did not differ significantly but thresholds of CE-chirp ABR were relatively closer to behavioral threshold. Also, Wave V peak to peak amplitude was larger in CE-chirp ABR compared to click ABR. Hence CE-chirp gives better identification of wave V near threshold as it gives relatively larger amplitude which leads to a better identification and diagnosis of the condition.

There is a dearth of information on CE-chirp ABR especially in different pathological conditions of hearing impairment. Hence, in the present study, we are evaluating CE-chirp ABR in individuals exposed to Occupational noise.

Based on the literature, we can conclude that a single audiological test is not sufficient identify cochlear and neural pathology. Hence, test battery approach including conventional pure tone audiometry, extended high frequency audiometry, distortion product oto acoustic emission to assess and monitor cochlear and early cochlear changes and auditory brainstem response or any other audiological test assessing neural changes would give a holistic view of early pathological changes seen in individuals exposed to occupational noise which helps in early identification, prevention and monitoring issues related to auditory effects of occupational noise.

Chapter 3

Method

The present study aims at comparing pure tone thresholds, amplitude and SNR of fine structure DPOAE's and latencies of ABR in individuals exposed to occupational noise (Group 2) with individuals not exposed to occupational noise (Group 1).

3.1. Participants

A total number 40 individuals participated in the study. They were divided into two groups:

Group 1: consisted of 20 individuals with normal hearing sensitivity in the age range of 18 to 45 years with the mean age 26.33 years.

Group 2: consisted of 20 individual who were working in industry in the age range of 20 to 45 years with the mean age 31.8 years.

3.1.1. *Participant selection criteria*

Group 1: Individuals with normal hearing sensitivity who were not exposed to occupational noise > 85 dB (A).

- Detailed case history was taken in order to rule out any past history or complaint of otological problems, neurological problems, hereditary hearing loss or any other major illness.
- Auditory thresholds were within 15 dB HL over the frequency range of 250 Hz to 8000 Hz for air conduction stimuli and 250 Hz to 4000 Hz for bone conduction stimuli.
- All the participants had 'A' type tympanogram along with normal acoustic reflexes at 500Hz, 1000Hz and 2000Hz indicating normal middle ear function.

- None of the participants had any history or complaints of retro cochlear pathology.
- All the participants had speech identification score greater than 90%.
- All the participants were not exposed to occupational noise >85dB (A).
- Consent for willingness to participate in the study from each individual was taken in prior.

Group 2: Individuals exposed to occupational noise.

- Detailed case history was taken in order to rule out any past history or complaint of otological problems, neurological problems, hereditary hearing loss or any other major illness.
- Auditory thresholds were within 15 dB HL from 250 Hz to 2 kHz and within 25 dB HL > 2 kHz region.
- All the participants had 'A' type of tympanogram along with presence of acoustic reflexes at 500 Hz, 1000 Hz and 2000 Hz indicating normal middle ear function.
- All the participants had speech identification score greater than 80 %.
- None of the participants had any history or complaints of retro cochlear pathology or any otological problems.
- All the participants were exposed to noise >85 dB (A) for greater than or equal to 8 hours per day (National Institute of Occupational Safety and Health, 1998) at their place of work.

  All the participants selected for the study met the selection criteria.
- This group was further divided into 3 Groups depending on the duration of exposure to occupational noise namely T1, T2 and T3.
    - T1- 0 to 5 years of noise exposure

- T2- 5 to 10 years of noise exposure
- T3- > 10 years of noise exposure

Table 3.1: Distribution of participants in Group2 based on their duration of exposure

Noise exposure duration	(T1) 0 - 5 years	(T2) 5 -10 years	(T3) >15years
Total number of ears	16	12	12

3.2. Instrumentation

Following instruments were used for the study:

- Piano-Inventis, a two channel diagnostic audiometer calibrated according to ANSI S3.6 (1991) standards coupled with the following transducers:
    a) TDH 39 headphone to estimate air conduction threshold from 250 Hz to 8 kHz.
    b) Sennheiser HDA-200 to estimate air conduction threshold from 9 kHz to 16 kHz.
    c) Bone vibrator (Radio ear B-71) was used to estimate bone conduction hearing threshold from 250 Hz to 4000 Hz.
- A calibrated Immittance meter - GSI Tympstar with visual display was used for tympanometry and to obtain acoustic reflexes.
- A calibrated diagnostic OAE instrument ILO-V6 was used to record distortion product oto acoustic emissions (DPOAEs) with 8 points/octave.
- Inter- acoustics Eclipse EP-25 was used for recording Click evoked ABR and CE-Chirp ABR.

3.3. Testing Environment

All the behavioral and electrophysiological tests were carried out in a sound treated room, with ambient noise levels well within permissible limits as per American National Standard Institution ANSI S3.1 (1991) specifications.

3.4. Test Stimuli

The Clauss Elberling chirp (CE Chirp) stimuli designed by Elberling and Don (2008) was used in the study for recording ABR along with click stimuli. The CE chirp is designed using a delay model based on derived band ABRs to overcome cochlear travelling wave delay and to increase synchronicity (Elberling & Don, 2010). Broadband CE-chirp has been included in EP25 which has a flat spectrum in five octave bands from 350 to 11300 Hz (Elberling & Don, 2010).

3.5.Procedure

The following tests were administered for both Group 1 and Group 2.

3.5.1. *Pure tone audiometry*

Pure tone thresholds were obtained at octaves and mid-octaves between 250 Hz to 8 kHz for air conduction and between 250 Hz to 4 kHz for bone conduction using modified Hughson Westlake procedure (Carhart & Jerger, 1959).

3.5.2. *Immittance*

- Tympanometry was done to rule out middle ear pathology using 226 Hz probe tone and pressure was swept from +200 to -400 dapa at 85 dB SPL.
- Acoustic reflexes were obtained at 500Hz, 1 kHz and 2 kHz.

3.5.3. *High frequency audiometry*

- High frequency air conduction thresholds were obtained using Sennheizer HDA 200 headphones for frequency between 9 kHz to 16 kHz using modified Hughson Westlake procedure (Carhart & Jerger, 1959).

3.5.4. *Speech Audiometry*

- Speech recognition threshold (SRT) was calculated for each individual using spondee word list. Speech identification scores were obtained for each individual at the level 40 dB above SRT using phonetically balanced words (Yathiraj & Vijayalakshmi, 2005). SIS scores were greater than 90 % for Group 1 and greater than 80 % for Group 2.

3.5.5. *Fine Structure Distortion Product Oto Acoustic Emission*

- DPOAEs were recorded at 8 points per octave in the frequency region of 2f1-f2 at the level L1= 65 dB and L2 = 55 dB at the ratio of 1.22. DPOAE's were measured from 800 Hz to 8000 Hz with 8 point per octave resolution. Total of 27 frequencies were measured.

3.5.6. *Auditory brainstem Response*

- ABR recording for both Click and CE-Chirp stimuli was done monaurally. The subject were seated in a reclining chair and the skin surface was cleaned using skin abrasive at the mastoid (M1 and M2), and forehead (Fz and Fpz) impedance obtained was less than 5KΩ for all the electrodes. Electrodes were placed in the respective places using skin conduction gel and were secured with the surgical plaster.
- The participants were instructed to relax and avoid extraneous body movements to keep the artifacts minimum.

- Click and CE-Chirp ABR was recorded using following stimulus and acquisition parameters which is given in the table 3.2

Table 3.2. Stimulus and Acquisition parameters for Click ABR and CE-chirp ABR

	Click ABR	***CE-Chirp ABR***
Stimulus parameters		
Transducer	Insert(ER 3A)	Insert(ER 3A)
Duration	0.1 msec	0.1 msec
Polarity	Rarefaction	Alternating
Intensity	90dBnHL	80 dBnHL
Repetition rate	11.1	11.1
Acquisition parameters		
Mode	Ipsilateral	Ipsilateral
Analysis time	12msec	12msec
Filter setting	100-3000Hz	100-3000Hz
Electrode Montage	Inverting- M1,M2 Ground- Fz and Fpz	Inverting- M1,M2 Ground- Fz and Fpz
No of sweeps	1500	1500
Inter electrode impedance	<5KΩ	< 5KΩ
No of channels	One	One
No of replication	Two	Two
	Hall (2006)	(Elberling & Don, 2010)

3.6. Waveform Analysis

Quantitative and qualitative analysis was done for waveforms of each individual obtained from Click ABR and CE-chirp ABR. Qualitative analysis was done by visual inspection of absolute latency of wave I, III and V in click ABR and absolute latency of wave V in CE-chirp ABR. Quantitative analysis was carried out by marking the absolute latency of wave I, III and V in click ABR and absolute latency of wave V in CE-chirp ABR. Latencies was measured. Two qualified Audiologists were given the recorded waveforms for both qualitative and quantitative analysis. The peaks were then marked based on the suggestion given and was considered for analysis.

Chapter 4

Results

The aim of the study was to assess cochlear and neural changes in individuals exposed to occupational noise. The tests included pure tone audiometry, extended high frequency audiometry, DPOAE (8 points per octave), click ABR and CE-chirp ABR. Data from 80 ears (40 normal hearing and 40 ears exposed to occupational noise) were analyzed using the statistical package for social sciences (SPSS) software version 20. Shapiro-wilk's test, a test of normality was done to check for normality of data. As the data did not fall under the normal distribution, non-parametric tests were selected to check for the significant differences. The variability is accounted to heterogeneity in the participants of the study.

Following analysis were carried out between the two groups for the entire tests:

- Descriptive statistics (mean and standard deviation) was done for all the parameters.
- Mann Whitney U test was administered to compare overall difference between two groups across all the selected tests.

Within group analysis was done using following statistical measures:

- Descriptive statistics (mean and standard deviation) was done for all the parameters.
- Kruskal Wallis test was done within groups across all the parameters so as to see the significant difference.
- Mann Whitney U test was administered wherever there was significant difference noticed, to compare overall difference within 3 Groups (T1, T2 and T3) across all the test parameters.

Results of the analysis are discussed under following sections:

4.1. Pure tone audiometry

4.2. Extended high frequency audiometry

4.3. Fine structure distortion product oto acoustic emissions

4.4. Auditory brainstem responses

4.1. Pure tone audiometry

The mean pure tone thresholds obtained were compared between Group 1 and Group 2 to check whether the latter had significant threshold shift. Further, the same comparison was done within the subgroups of Group 2 (T1, T2 and T3) to find out the effect of duration of exposure on threshold shift.

4.1.1. *Comparison of pure tone thresholds between Group 1 and Group 2.*

Descriptive statistics (mean and standard deviation) was done between Group 1 and Group 2. The mean threshold and SD obtained for pure tones from 250 Hz to 8 k Hz are depicted in the Figure 4.1. Mann Whitney U test was done to check for the significant difference. Z values and p values obtained are as mentioned: 250 Hz ($|Z| = 1.002$, $p > 0.05$), 500 Hz ($|Z| = 1.758$, $p > 0.05$), 750 Hz ($|Z| = 3.768$, $p < 0.05$), 1000 Hz ($|Z| = 3.995$, $p < 0.05$), 1500 Hz ($|Z| = 4.961$, $p < 0.05$), 2000 Hz ($|Z| = 3.767$, $p < 0.05$), 3000 Hz ($|Z| = 4.303$, $p < 0.05$), 4000 Hz ($|Z| = 3.113$, $p < 0.05$), 6000 Hz ($|Z| = 4.563$, $p < 0.05$) and 8000 Hz ($|Z| = 5.192$, $p < 0.05$).

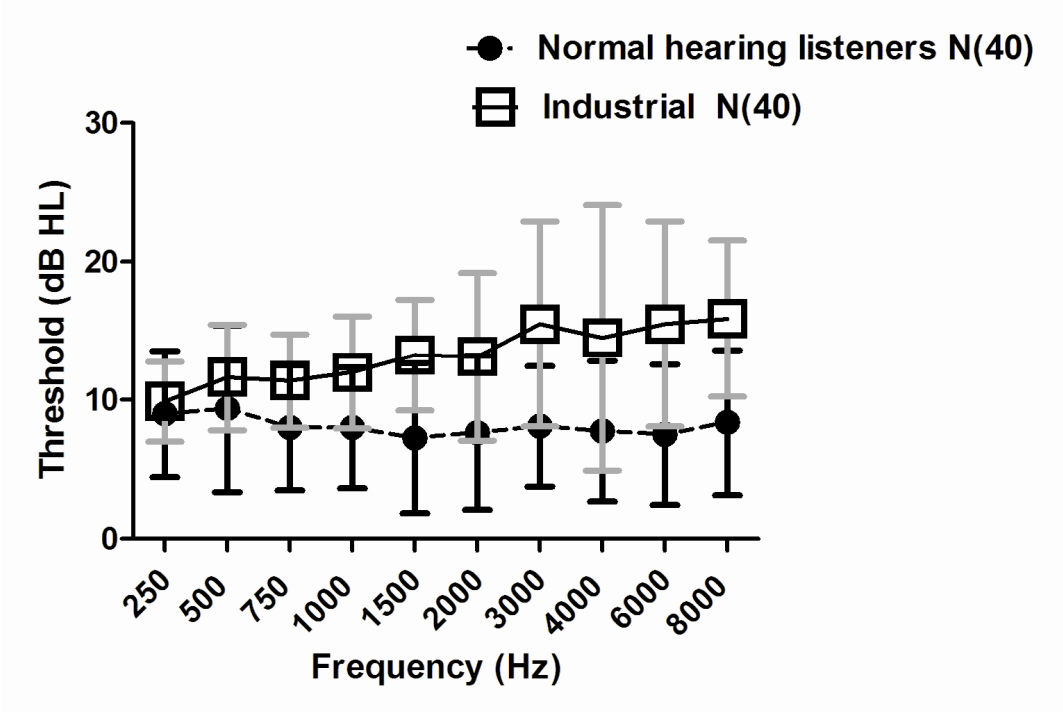

Figure 4.1. Mean and SD of pure tone thresholds of Individuals with normalhearing and individuals exposed to occupational noise

4.1.2.*Comparison of pure tone thresholds within Group 2*

Descriptive statistics (mean and standard deviation) was carried out. The mean threshold and SD obtained for pure tones from 250 Hz to 8 k Hz are depicted in the Figure 4.2. Kruskal Wallis test was administered and results revealed that there was a statistically significant difference across 3 Groups across all frequencies except for 250 Hz and 500 Hz. The values of χ^2 (Chi square) are as mentioned: 250 Hz (χ^2 = 2.368, $p>0.05$), 500 Hz (χ^2 = 5.577, $p>0.05$), 750 Hz (χ^2 = 19.807, $p<0.05$), 1000 Hz (χ^2 = 19.552, $p<0.05$), 1500 Hz (χ^2 = 26.351, $p<0.05$), 2000 Hz (χ^2 = 30.301, $p<0.05$), 3000 Hz (χ^2 = 39.277, $p<0.05$), 4000 Hz (χ^2 = 31.601, $p<0.05$), 6000 Hz(χ^2 = 35.098, $p<0.05$) and 8000 Hz (χ^2 = 40.577, $p<0.05$). Mann Whitney U test was carried out from 750 Hz to 8000 Hz to check for the significant difference across Groups. Z values and p values for the same are represented in Table 4.1.

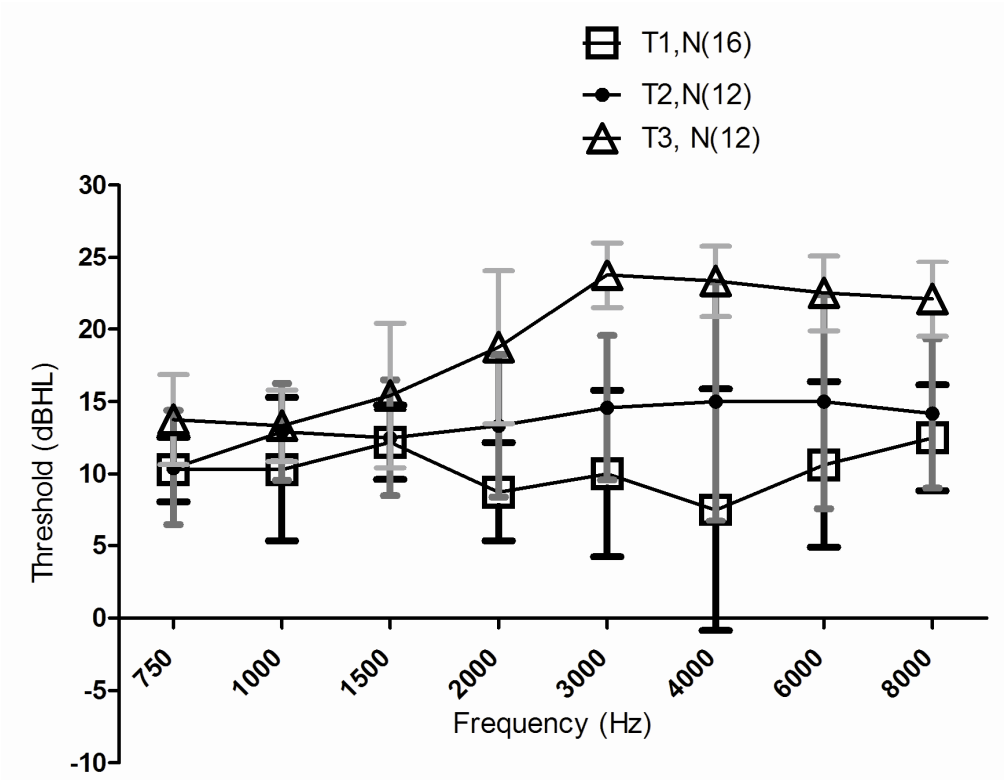

Figure 4.2. Mean and SD of pure tone thresholds of individuals exposed to occupational noise for different durations

Table 4.1. | Z | values for pure tones from 750 Hz to 8 k Hz within Group 2

Frequency	T1 v/s T2	T1 v/s T3	T2 v/s T3
750	0.552	2.960*	2.157*
1000	1.430	1.640	0.140
1500	0.681	1.824	1.246
2000	2.477*	4.072*	2.269*
3000	2.071*	4.463*	3.978*
4000	2.323*	3.833*	2.480*
6000	1.559	4.295*	2.529*
8000	1.007	4.322*	3.550*

Note:*p*< 0.05, T1: 0 to 5 years of exposure, T2: 5 to 10 years of exposure, T3: >10 years of exposure.

4.2. Extended high frequency audiometry

The mean extended high frequency threshold obtained for frequencies from 9000 Hz to 16000 Hz were compared between Group 1 and Group 2 to check whether the latter had elevated threshold shift. Further, the same comparison was done within the subgroups of Group 2 (T1, T2 and T3) to find out the effect of duration of exposure on threshold shift.

4.2.1. *Comparison of extended high frequency thresholds between Group 1 and Group 2*

Mean and standard deviation was calculated using descriptive statistics between Group 1 and Group 2. The mean threshold and SD obtained for extended high frequency pure tones from 9000 Hz to 16000 Hz are represented in the Figure 4.3. Mann Whitney U test was done to check for the significant difference. Z values and p values obtained are as mentioned: 9000 Hz ($|Z| = 6.011$, $p<0.05$), 10000 Hz ($|Z| = 4.995$, $p<0.05$), 11200 Hz ($|Z| = 6.055$, $p<0.05$), 12000 Hz ($|Z| = 6.121$, $p<0.05$), 14000 Hz ($|Z| = 6.820$, $p<0.05$), 16000 Hz ($|Z| = 6.717$, $p< 0.05$). There was statistically significant difference at all frequencies between two groups.

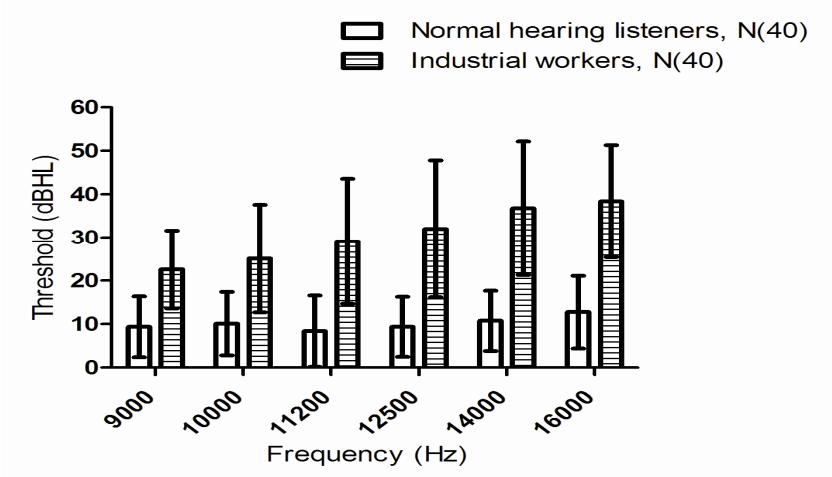

Figure 4.3. Mean and SD of extended high frequency pure tone thresholds of normal hearing listeners and individuals exposed to occupational noise

4.1.2. *Comparison of extended high frequency thresholds within Group 2*

The mean threshold and SD obtained for pure tones from 9000 Hz to 16000 Hz using descriptive statistics are depicted in the Figure 4.4. Kruskal Wallis test was administered to look for significant difference across 3 sub-groups (T1, T2 and T3) and results revealed that there was a statistical difference across 3 groups for all frequencies from 9000 Hz to 16000 Hz. The values of χ^2 are as mentioned: 9000 Hz (χ^2 = 42.683, $p<0.05$), 10000 Hz (χ^2 = 39.314, $p<0.05$), 11200 Hz (χ^2 = 46.856, $p<0.05$), 12500 Hz (χ^2 =45.746, $p<0.05$), 14000 Hz (χ^2 = 53.770, $p<0.05$), 16000 Hz (χ^2 = 52.939, $p<0.05$). Mann Whitney U test was carried out for all the frequency to check for the significant difference across groups. Z values and p values for the same are represented in Table 4.2.

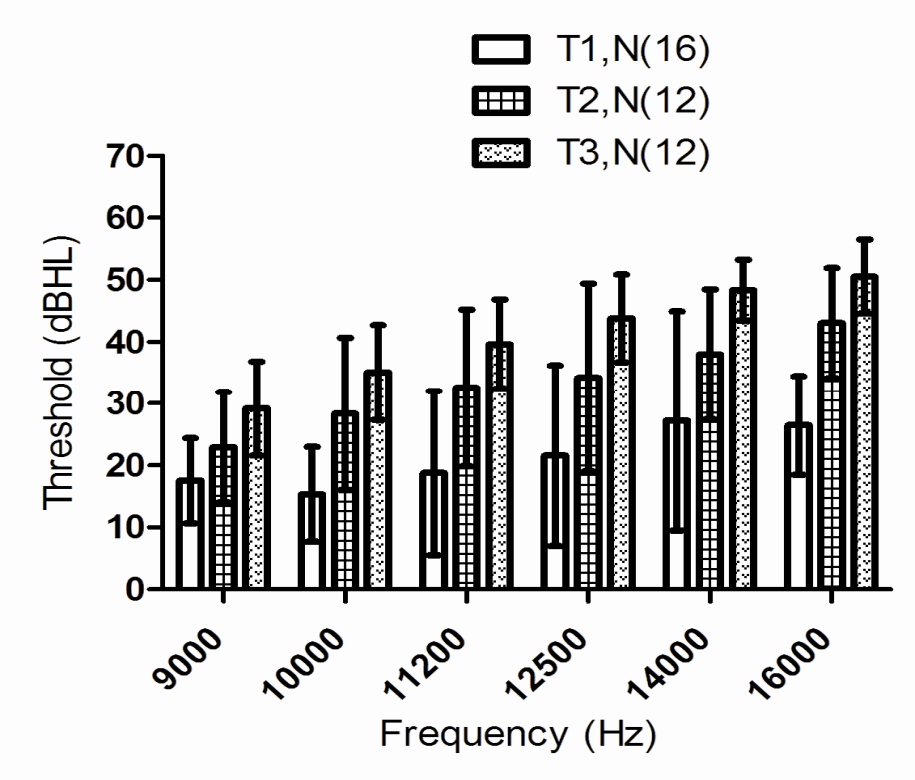

Figure 4.4. Mean and SD of extended high frequency pure tone thresholds within Group 2

Table 4.2. │Z│ values for pure tones from 9000 Hz to 16000 Hz within Group 2

Frequency(Hz)	T1 v/s T2	T1 v/s T3	T2 v/s T3
9000	1.704	3.398*	1.672
10000	2.691*	4.058*	1.223
11200	2.813*	3.493*	1.664
12000	2.276*	3.620*	1.779
14000	2.512*	3.401*	2.715**
16000	3.600*	4.112*	1.891*

Note:*p*< 0.05, ** *p=0.5* (approaching significance), T1: 0 to 5 years of exposure, T2: 5 to 10 years of exposure, T3: >10 years of exposure.

4.3. Fine structure distortion product oto acoustic emissions

Amplitude and SNR of fine structure DPOAE across frequencies 1001 Hz and 7996 Hz were measured in Group 1 and Group 2 and were compared to see significant change between the groups. The amplitude and SNR were also compared within subgroups (T1, T2 and T3) of Group 2 to check for the effect of noise exposure duration on amplitude and SNR of fine structure DPOAE. Some of the data's were excluded when OAE's were absent and SNR <3 dB at some of the frequencies which is mentioned in Table 4.5 and Table 4.8 respectively.

4.3.1. *Comparison of OAE amplitude between Group 1 and Group 2*

Mean and standard deviation was obtained for amplitude of DPOAE in Group 1 and Group 2 from frequency 1001 Hz to 7996 Hz using descriptive statistics. The mean amplitude across frequenciesare depicted in the Figure 4.5 and SD's are mentioned in Table 4.3.Mann Whitney U test was done to assess the significant difference between two groups. Z values and p values obtained for DPOAE's 8 point per octave from 1001 Hz to 7996 Hz are shown in Table 4.4. Ears which had presence

of OAE were considered and others were excluded from the data. The excluded number of ears across frequency is given in Table 4.5.

Table 4.3.SD for amplitude and SNR of OAE of Group 1 and Group 2 across frequencies

Frequency (Hz)	SD (Group 1)		SD (Group 2)	
	Amplitude	SNR	Amplitude	SNR
1001	3.81	3.03	3.58	4.69
1086	5.12	2.87	3.97	4.47
1184	4.22	2.97	4.20	5.23
1294	3.99	3.17	4.73	6.08
1416	4.49	3.18	5.22	7.80
1538	4.97	3.16	4.17	5.79
1685	6.26	5.42	4.99	6.45
1831	5.36	5.23	4.79	6.3
2002	4.24	5.02	4.05	6.2
2185	4.82	5.53	3.37	5.44
2380	3.49	3.03	3.85	6.2
2600	3.66	4.62	3.26	5.1
2832	3.02	3.43	2.70	4.23
3088	2.48	2.36	2.07	4.02
3369	3.48	4.27	2.84	14
3662	3.10	4.26	3.15	5.5
4002	4.09	3.82	3.06	4.59
4358	2.85	3.10	4.86	6.31
4761	3.48	3.43	4.66	14.64
5188	4.99	4.90	6.85	6.98
5652	4.17	4.20	7.17	8.33
6165	1.51	3.99	4.97	17.01
6726	2.72	4.54	0.17	2.59
7336	2.31	5.59	0.45	1.43
7996	4.83	7.92	**	2.37

Note:**p*< 0.05, ** only one data available

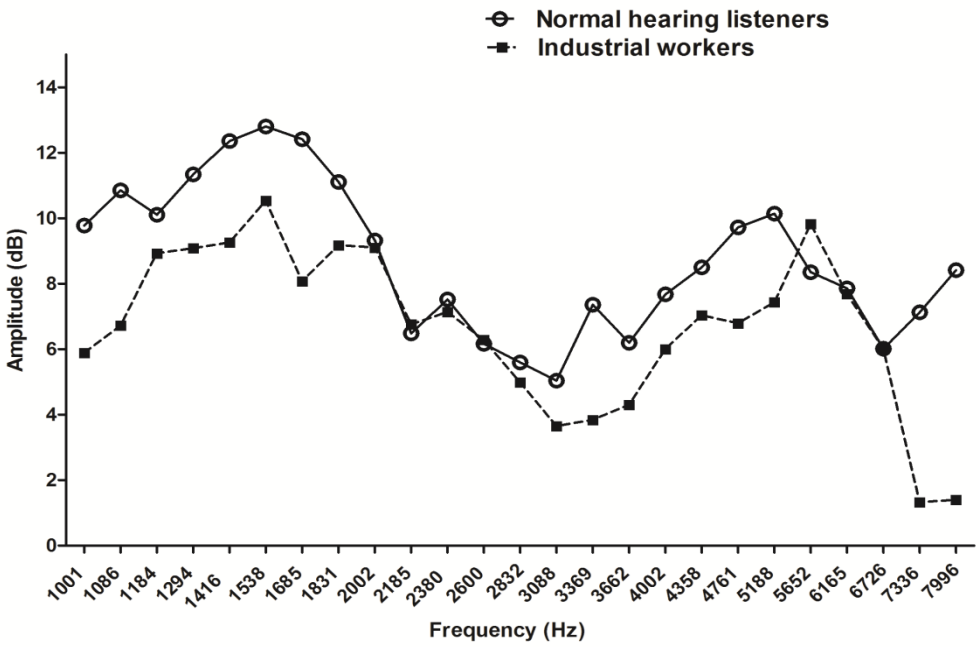

Figure 4.5. Mean of OAE amplitude of normal hearing listeners and individuals exposed to occupational noise

Table 4.4. | Z | values for amplitude and SNR of DPOAE from 1001 Hz to 799 Hz between Group 1 and Group 2

Frequency (Hz)	Z value (amplitude)	Z value (SNR)
1001	3.823*	1.730
1086	3.514*	1.993*
1184	1.412	1.701
1294	2.241*	1.971*
1416	2.361*	0.039
1538	2.322*	1.746
1685	3.283*	0.135
1831	1.183	0.631
2002	0.111	1.524
2185	0.482	2.406*
2380	0.649	0.512
2600	0.171	0.161
2832	0.408	1.367
3088	1.912	0.390
3369	2.735*	1.131
3662	1.213	0.691
4002	1.741	2.770*
4358	0.878	0.265
4761	2.334*	1.333

5188	2.291*	1.665
5652	0.083	0.040
6165	1.664	0.166
6726	0.935	0.386
7336	2.646*	1.505
7996	1.697*	1.033

Note: *$p<0.05$

Table 4.5. Excluded ears across frequency and across groups for OAE amplitude

Frequency (Hz)	Normal	Industrial workers		
		0-5 years of exposure	5-10 year of exposure	>10 years of exposure
1001	0	6	2	3
1086	0	3	1	2
1184	0	5	3	3
1294	0	4	3	4
1416	0	4	5	2
1538	0	6	5	4
1685	0	3	5	4
1831	0	5	5	4
2002	0	7	5	4
2185	1	6	5	5
2380	0	8	4	6
2600	4	8	3	6
2832	6	9	3	5
3088	0	10	6	9
3369	7	9	7	10
3662	0	9	6	10
4002	0	9	7	11
4358	0	7	6	8
4761	0	10	5	11
5188	0	8	3	10
5652	8	11	5	11
6165	6	12	8	10
6726	6	15	10	12
7336	0	15	10	11
7996	0	16	12	11

Note: T1: 0 to 5 years of exposure, T2: 5 to 10 years of exposure, T3: > 10 years of exposure.

4.3.2. *Comparison of OAE amplitude within Group 2*

The mean amplitude obtained using descriptive statistics for DPOAE's of 8 point per octave from 1001 Hz to 7996 Hz are depicted in the Figure 4.6. Using Kruskal Wallis test, statistical analysis was done to check for significant difference between groups and results revealed that there was a statistical difference across 3

groups only in 1001 Hz (χ^2=15.425, *p<0.05*), 1086 Hz (χ^2=13.372,*p<0.05*), 1416 Hz (χ^2=11.256, *p<0.05*), 1685 Hz (χ^2=11.631, *p<0.05*), 3369 Hz (χ^2=9.022, *p<0.05*). Mann Whitney U test was carried out across these frequencies, Z and *p*values are given in Table 4.7. Chi square and p values across all frequencies are given in Table 4.6.

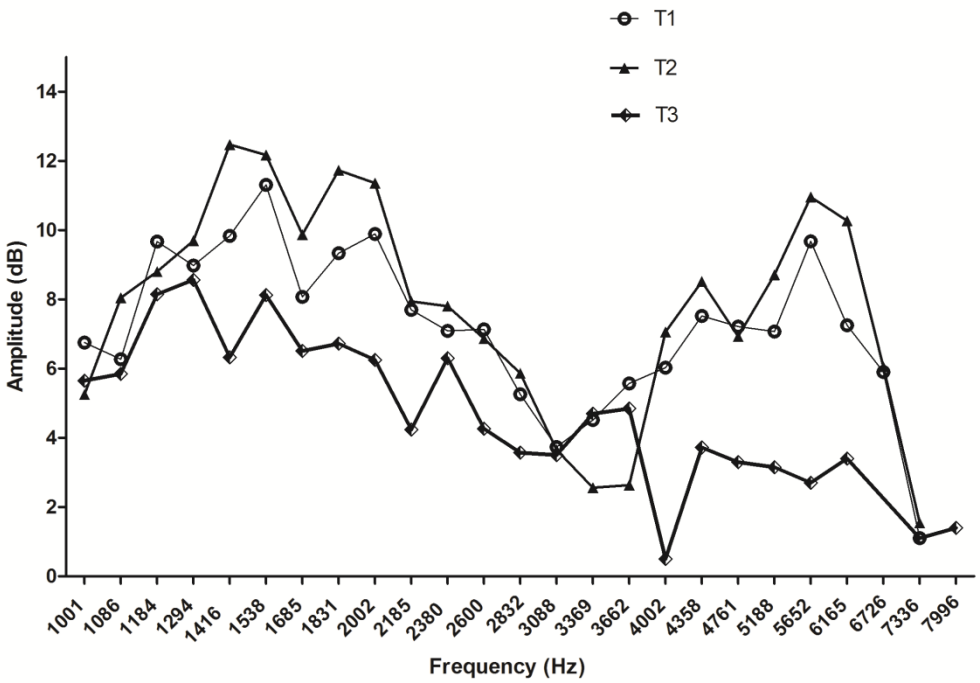

Figure 4.6. Mean of OAE amplitude within Group 2

Table 4.6. χ^2values for OAE amplitude and SNR from 1001 Hz and 7996 Hz across group 2

Frequency (Hz)	χ^2 value (amplitude)	χ^2 value (SNR)
1001	15.425*	5.098
1086	13.372*	4.012
1184	3.162	4.178
1294	6.212	6.446
1416	11.256*	1.825
1538	7.198	5.609
1685	11.631*	5.272
1831	4.676	3.278
2002	5.732	6.010
2185	4.756	7.611

2380	1.256	0.316
2600	2.014	1.506
2832	3.323	4.045
3088	3.665	2.631
3369	9.022*	6.464
3662	4.220	1.346
4002	5.129	12.805*
4358	4.720	3.282
4761	6.002	1.907
5188	7.176	4.752
5652	1.930	6.963
6165	7.200	3.625
6726	0.879	0.392
7336	7.019	2.299
7996	2.878	1.592

Table 4.7. $|Z|$ values for OAE amplitude from 1001 Hz and 7996 Hz within Group 2

Frequency	T1 v/s T2	T1 v/s T3	T2 v/s T3
1001	1.063	0.858	0.082
1086	0.871	0.434	1.445
1416	1.062	1.518	2.296*
1685	0.716	0.725	1.565
3369	0.828	0.738	1.954

Note:*p* <0.05; T1: 0 to 5 years of exposure, T2: 5 to 10 years of exposure, T3: >10 years of exposure

4.3.3. *Comparison of SNR of OAE between Group 1 and Group 2*

Descriptive statistics (mean and standard deviation) was done for the measurement of SNR between Group 1 and Group 2. Data were extracted only when SNR was >3dB and the others below were excluded from the analysis which is represented in table 4.8. The mean value of SNR in dB was obtained for DPOAE of 8 point per octave from 1001 Hz to 7996 Hz are depicted in the Figure 4.7 and SD's are mentioned in Table 4.3. Mann Whitney U test was done to evaluate the significant difference between Group 1 and 2. Z values and p values obtained for DPOAE's 8

point per octave from 1001 Hz to 7996 Hz are represented in Table 4.4. Ears which had presence of OAE were considered and others were excluded from the data. The excluded number of ears across frequency is given in Table 4.8.

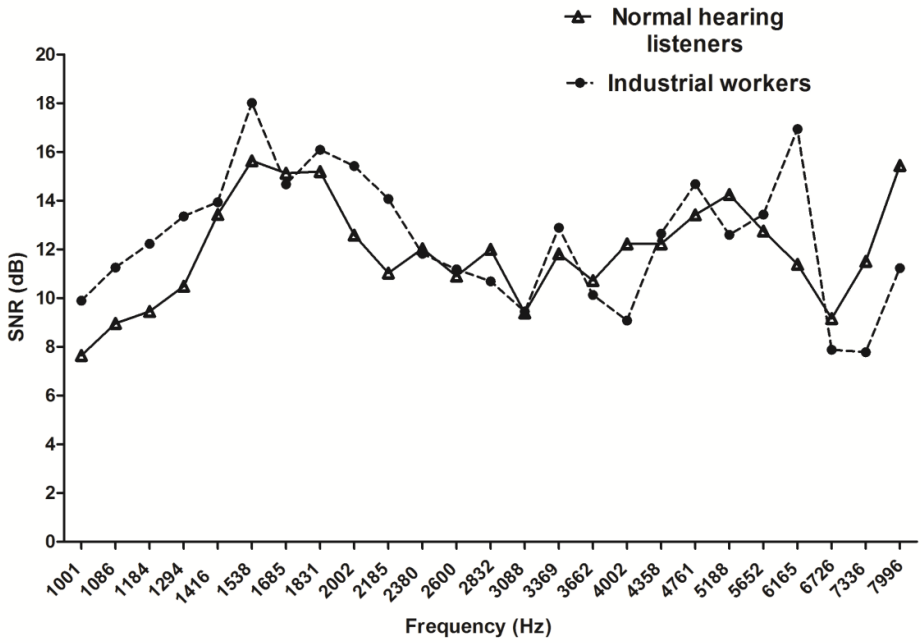

Figure 4.7. Mean value of DPOAE SNR in Group 1 and Group 2

4.3.4. *Comparison of SNR of OAE within Group 2*

Descriptive statistics (mean and standard deviation) was carried out. The mean value of SNR obtained for DPOAE's of 8 point per octave from 1001 Hz to 7996 Hz within subgroups of Group 2 are depicted in the Figure 4.8. Kruskal Wallis test was administered to assess the significant difference across groups. There was no significant difference across groups except for frequency 4004 Hz (χ^2=12.805, $p<0.05$). Mann Whitney test for the particular frequency revealed: $|Z| = 0.640$ ($p>0.05$) between T1 and T2, $|Z| = 0.542$ ($p>0.05$) between T1 and T3 and $|Z| = 0.95$ ($p<0.05$). There was statistical difference between T1 and T3 Group for that particular frequency. Chi square and *p*values for SNR across all frequencies are given in Table 4.6.

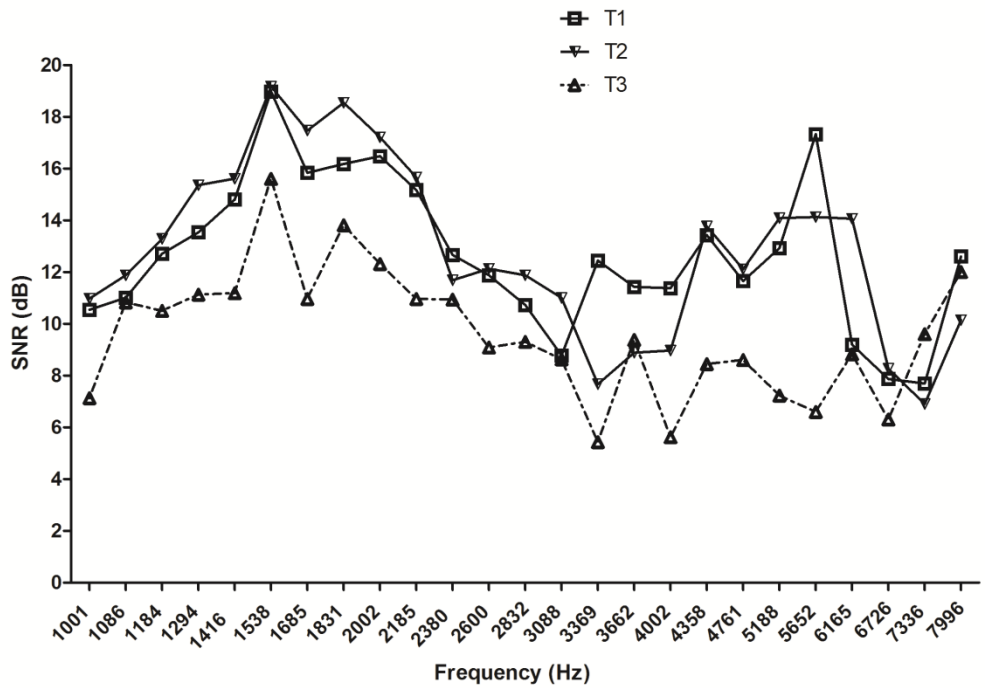

Figure 4.8. Mean of SNR values from 1001 Hz and 7996 Hz within Group 2.

Table 4.8. Excluded ears for SNR across frequency and across groups in Group 2

Excluded ears				
Frequency (Hz)	Normal	Industrial workers		
		0-5 years of exposure	5-10 year of exposure	>10 years of exposure
1001	8	9	6	8
1086	6	6	4	6
1184	6	5	4	4
1294	2	6	4	4
1416	0	6	4	4
1538	0	7	5	5
1685	0	5	6	4
1831	0	5	5	4
2002	0	6	4	4
2185	4	5	4	4
2380	0	6	2	4
2600	8	6	4	5
2832	6	7	3	4
3088	0	7	6	8
3369	4	9	3	8
3662	0	7	5	8
4002	0	8	3	7
4358	0	6	4	8

4761	0	9	4	9
5188	0	8	3	9
5652	0	12	5	9
6165	0	11	7	9
6726	6	13	8	11
7336	6	15	10	11
7996	2	15	10	11

4.4. Auditory brainstem response

The mean absolute latency of 1, III, V, inter peak latency I-III, III-V, I-V of click evoked ABR and wave V of CE-chirp ABR were compared between Group 1 and 2 to look for the significant in two groups. Also, the same parameters were compared within Group 2 to see the effect of varying duration of noise exposure on latency parameters.

4.4.1. *Comparison of absolute latency and inter peak latency of click evoked ABR between Group 1 and Group 2*

Descriptive statistics (mean and standard deviation) was done between Group 1 and Group 2. The mean Latency, SD obtained for peak I, III, V in click evoked ABR are depicted in the Figure 4.9 and for inter peak latencies of I-III, III-V, I-V in click evoked ABR are represented in Figure 4.10. Mann Whitney U test was done to check for the significant difference between both the groups. Z values and p values obtained are as follows: I peak ($|Z| = 0.149$, $p>0.05$), III peak ($|Z| = 4.442$, $p<0.05$), V peak ($|Z| = 1.419$, $p>0.05$), I-III inter peak ($|Z| = 4.367$, $p<0.05$), III-V inter peak ($|Z| = 3.326$, $p<0.05$), I-V inter peak ($|Z| = 1.523$, $p>0.05$). Results showed significant difference between two groups in absolute latency of wave III and inter peak latency of I-III, III-V.

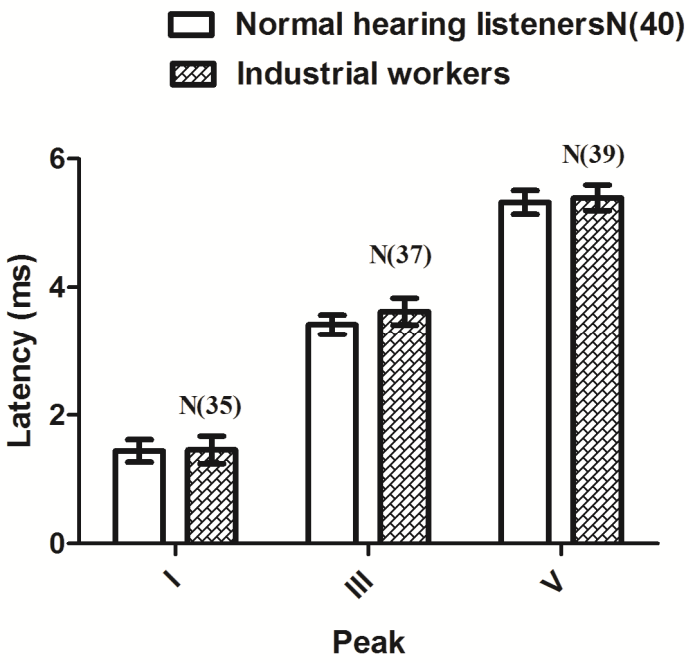

Figure 4.9. Mean of latency of I, III and V in click evoked ABR of group 1 and group 2

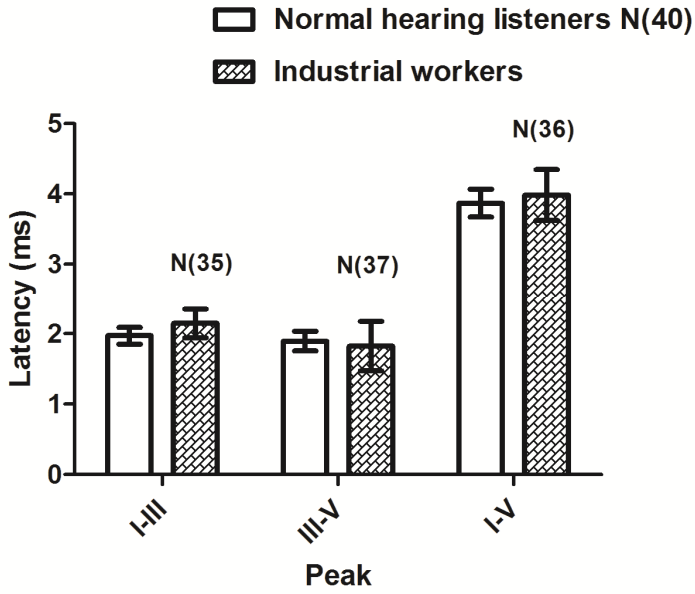

Figure 4.10. Inter peak latency of I-III, III-V, I-V in click evoked ABR of Group 1 and Group 2

ABR within Group 2

Descriptive statistics (mean and standard deviation) was carried out. The mean Latency and SD obtained for peak I, III, V in click evoked ABR are depicted in the

figure 4.11 and for inter peak latencies of I-III, III-V, I-V in click evoked ABR are represented in Figure 4.12. Kruskal Wallis test was initially administered to check for significant difference across groups. Results revealed that there was a statistical difference across 3 Groups for peak III ($\chi 2 = 22.509$, $p<0.05$), peak V ($\chi 2 = 10.938$, $p<0.05$) and for inter peak I- III ($\chi^2 = 26.377$, $p<0.05$), III-V ($\chi^2 = 24.515$, $p<0.05$) and there was no significant difference found for Peak I and inter peak I-V. Mann Whitney U test was carried out for: peak III and peak V, inter peak I-III and III-V to check for the significant difference across Groups. Z values and p values for the same are represented in Table 4.9.

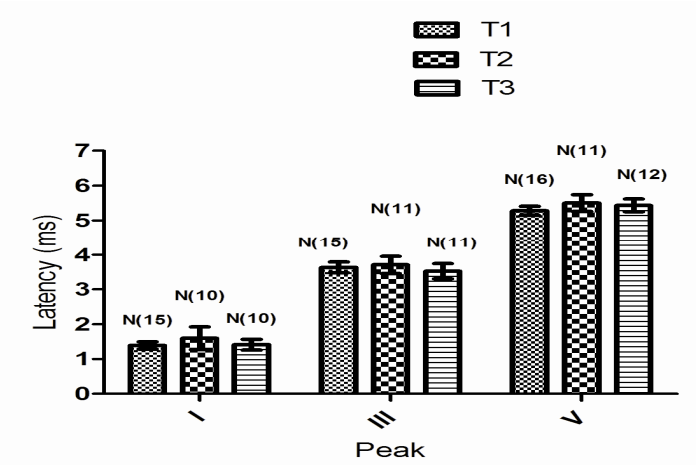

Figure 4.11. Mean and SD of absolute latency of I, III and V in click evoked ABR within group 2.

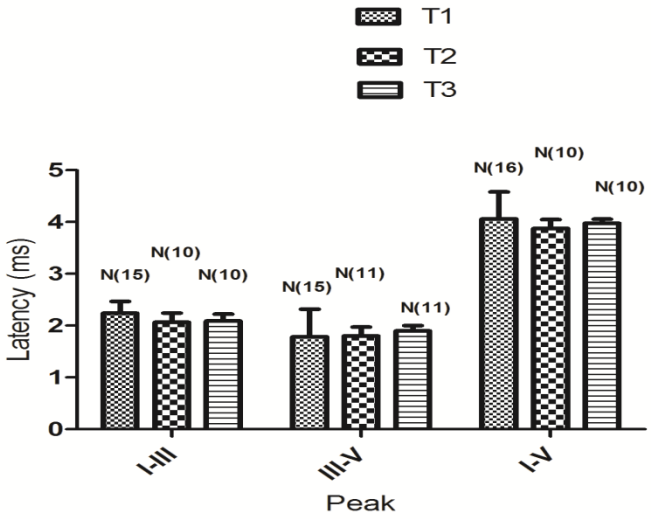

Figure 4.12. Mean and SD of Inter peak latency of I-III, III-V, I-V in click evoked ABR within group 2.

Table 4.9. | Z | values for peak III, V and inter peak latency within Group 2

Peak	T1 v/s T2	T1 v/s T3	T2 v/s T3
III	0.313	0.704	1.089
V	2.773*	2.263*	0.558
I-III	2.129*	2.394*	0.114
III-V	2.248*	3.592*	1.992*

Note:*p*<0.05; T1: 0 to 5 years of exposure, T2: 5 to 10 years of exposure, T3: >10 years of exposure.

4.4.3. *Comparison of absolute latency of wave V in CE-chirp ABR between Group 1 and 2*

Descriptive statistics (mean and standard deviation) was done between Group 1 and Group 2. Total number of 11 ears had absent CE-chirp ABR which were excluded from the data. The mean latency and SD obtained for peak V in CE-chirp evoked ABR is shown in the figure 4.13. Mann Whitney U test was administered to evaluate for the significant difference between both the groups. Z values and p value obtained for peak V (| Z | = 3.754, p<0.05) showed statistically significant difference between groups.

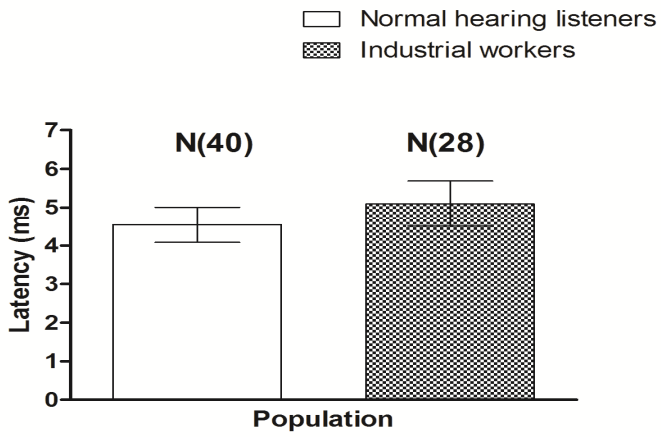

Figure 4.13. Mean and SD of Peak V in CE-chirp ABR between group 1 and group 2

4.4.3. *Comparison of absolute latency of wave V in CE-chirp ABR within Group 2*

Descriptive statistics (mean and standard deviation) was carried out. The mean Latency and SD obtained for peak V across groups are depicted in the figure 4.14. Kruskal Wallis test was initially administered to check for significant difference across groups. Results revealed that there was a statistical difference across 3 Groups for peak V ($\chi 2 = 22.509$, $p<0.05$). Mann Whitney U test was administered. Z values and p values for the same are as mentioned: $|Z| = 0.528$ ($p>0.05$) between T1 and T2, $|Z| = 2.756$ ($p<0.05$) between T1 and T3 and $|Z| = 0.326$ ($p>0.05$). Significant difference was found between T1 and T3.

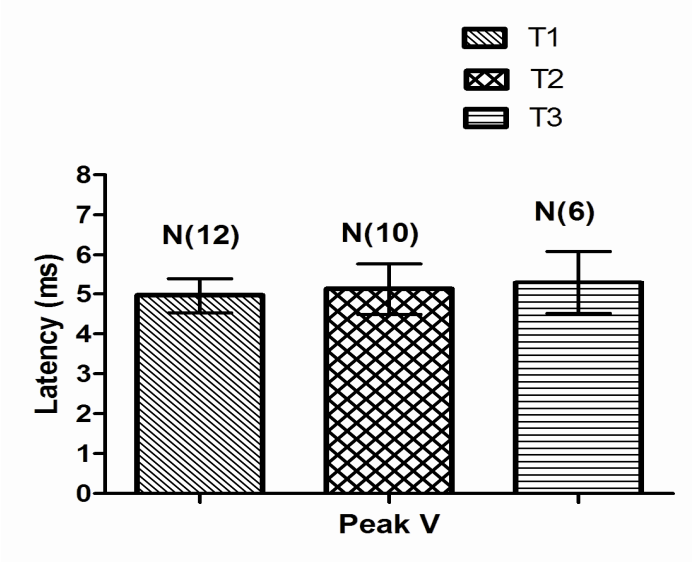

Figure 4.14. Mean and SD of Peak V in CE-chirp ABR within Group 2

Chapter 5

Discussion

The study was conducted with the objective to compare the cochlear and neural findings of individuals exposed to occupational noise with those who are not exposed to the same based on the following audiological tests.

- Pure tone Audiometry along with extended high frequency audiometry.
- Auditory Brainstem Response
    - Click evoked ABR
    - CE-chirp evoked ABR
- Distortion Product Oto Acoustic Emissions

The results show that there is a significant effect of duration of exposure on the test results.

The findings of the study will be discussed under following sections:

5.1. Pure tone audiometry

5.2. Extended high frequency audiometry

5.3. Fine structure distortion product oto acoustic emissions

5.4. Auditory brainstem response

5.1. Pure tone audiometry

Pure tone threshold from 250 Hz to 8 kHz of Group 1 and Group 2 are as shown in the Figure 4.1. Based on the results obtained, there issmall, but significant threshold shift between two Groups from 750 Hz to 8 kHz. But, this elevation of thresholds in conventional pure tone audiometry is very subtle and cannot be identified as the thresholds sometimes might fall within normal limits. The results are

in agreement with studies Kirchner et al. (2012)**;** Mantysalo and Vuori (1984); Shah, Baig and Vaidya (2013); McBride and Williams(2001) which shows significant difference at higher frequency (>2 kHz). From the Figure 4.2, it can be seen that there is a monotonic relation between pure tone thresholds and duration of exposure. There was a significant increase in pure tone thresholds across groups as the duration of noise exposure increased at frequency above 2 kHz. The findings are in consensus with the studies done by Nixon and Glorig (1961); Seixas, Goldman, Sheppard, Neitzal, Norton and Kujawa (2005). The conventional pure tone thresholds do not give an actual insight to the damage that is happening earlier at the cochlear level or at neural level. These changes might take months or years together to express as significant threshold shift. Hence, pure tone audiometry itself cannot be used as a susceptibility test for noise induced hearing loss. However, the magnitude of elevation of pure tone threshold in the present study might be smaller, but shows imminent long standing effect of noise on hearing.

5.2. Extended high frequency audiometry

In the present study it can be seen that the magnitude of hearing loss is greater than conventional audiometry in individuals exposed to occupational noise. The mean thresholds are significantly higher in Group 2 compared to Group 1 as shown in fig 4.3. The findings of the study suggest that regions at higher frequencies >9000 Hz especially 14 kHz and 16 kHz get affected initially before the changes are seen in standard audiometric frequency range. We can assume that these changes might be because of tonotopical arrangement of cochlea where high frequencies are arranged in the basal region and low frequencies are arranged in apical region of cochlea. So, the direction of travelling wave for a signal or noise is always from basal to apex (Bekesy & wever, 1960) and hence making basal region more prone to damage when the ear is

exposed to hazardous noise levels. Also the nerve fibres in the basal region are longer (Starr, 2009) making high frequency more prone to damage when compared to low frequency nerve fibres. In addition, according to equal loudness contour (ELC), high frequencies require minimum SPL to get stimulated when compared to lower frequencies (Fletcher and Munson, 1933), but ELC are given only till 8 kHz. We can presume that frequencies greater than 8 kHz would require still lesser SPL. Therefore hazardous level of noise would damage these regions. Hence, over all it can be concluded that preliminary pathological changes might be seen in the most basal region of cochlea and then extending to lower frequency or speech frequency region. The findings of the study are in agreement with the results obtained by Faustiet al. (1981); Wang et al. (2000); and Soma et al. (2011). Also, there is significant monotonic relationship between extended high frequency audiometric thresholds and duration of exposure as seen in Figure 4.4. Similar findings have been reported by Riga et al. (2010). Hence, Extended High Frequency audiometry can be used as a sensitive tool or even as a screening tool to identify and monitor the early cochlear changes due to exposure of noise as the changes are seen initially in the most basal regions.

5.3. Fine structure distortion product oto acoustic emissions

Distortion product oto acoustic emissions were absent in most of the ears exposed to occupational noise across different frequencies as seen in Table 4.4.It can be seen that there is variability in DPOAE's results among subjects and also across frequencies. The absences of OAE's are because that the outer hair cells are more sensitive to noise induced changes which are responsible for generation of DPOAE's. Hence, damage to OHC's are reflected in DPOAE's showing absence of response. Even when the oae's were present there was a significant decrease in amplitude and

SNR at some of the frequencies (1001 Hz to 1600 Hz and 4000 Hz to 8000 Hz) as shown in Table 4.3 and 4.7 respectively. These observations are in agreement with studies done by Balatsouras(2004); Seixas et al. (2004); and Korres et al. (2009). As the duration of exposure to noise increased, the OAE's became more affected at frequencies above 2 k Hz.

There was decrease in amplitude as well as SNR when the duration of noise exposure increased and most significant difference was seen in T3 Group (Fig 4.6) at frequencies 4000 Hz to 8000 Hz for amplitude and at 4002 Hz for SNR. But, the difference was not statistically evident for amplitude may be because the sample considered was less as most of the ears exposed to occupational noise had absence OAE's. There was significant decrease in SNR for 4004 Hz in T3 Group compared to T1 Group. As the duration of exposure to occupational noise increased the SNR at 4004 Hz significantly decreased as the hair cells at this frequency would have undergone pathological changes which were not seen in the initial exposure duration. But, 7 out of 12 ears had SNR <3 dB at this frequency showing the sensitivity of OAE's in identifying hair cell damage before showing in conventional audiometry. This observation from the present study is in accordance to findings reported from Seixas et al. (2004). As DPOAE's reflect normal functioning of outer hair cells, even subtle changes in the functioning of outer hair cells can be detected and monitored. These findings suggest that DPOAE's are more sensitive compared to conventional pure tone audiometry in detecting early cochlear changes. Moreover, it is an objective method which does not require the active participation of an individual and less time consuming. Hence, it can be used as an efficient tool to identify and monitor early cochlear changes.

5.4. Auditory brainstem response

Click ABR

The study compared absolute latencies of peak I, III, V and inters peak latencies of I-III, III-V, I-V between Group 1 and Group 2 to see the susceptibility to noise induced changes in brainstem. The mean absolute latencies and inter peak latencies shown in Figure 4.9 and Figure 4.10 respectively did not differ from Group 1 and Group 2 as the values were within normal limits which shows no significant difference in functioning of auditory nerve and brainstem showing the noise induced changes do not essentially involve abnormality of AEP. This findings is in consensus with studies reported by Attias, Urbach and Shemes (1993); Almadori et al. (1998) and Xu, Vinck, Vel and Cauenberge(1998). However, there was small but statistically significant difference found in peak III and inters peak latencies involving the III peak between Group 1 and Group 2. As the III peak originate at the level of cochlear nucleus, we can presume that there could be subtle damage to the cells in the structure which might indicate cochlear nucleus in central auditory pathway could be slightly sensitive to noise induced changes. But it is very difficult to monitor these changes as the latency parameter might fall within the normative.

It can also be seen that there is small, but significant prolongation of absolute latency of I and V peak, and inter peak latencies of I-III and III-V as the duration of exposure duration is increased from T1 to T3. Thus, as the duration of noise exposure increases, there might be subtle damage to the brain stem structures which might have decreased the conduction minimally but significantly. Therefore we can assume that there is small but significant positive relation between exposure duration and latency parameters.

CE-chirp ABR

CE-chirp ABR results in enhanced synchronization of cochlea compared to standard click stimuli due to overlapping of responses at different frequency ranges. As a result the amplitude of the response is larger and the latencies will be earlier when compared to standard click ABR.

In the current study, Group 1 individuals had mean latency of 4.542 ms for wave V in CE-chirp ABR which is earlier compared to wave V latency of click evoked ABR as a result of enhanced synchrony due to compensation of basilar membrane delay. However, in Group 2 individuals, 11 out of ears had absent CE-chirp ABR. But when present the mean latency was 5.0982 ms showing small latency prolongation which is statistically significant. This can be attributed to damage in auditory nerve fibers at some particular frequency region especially at basal region affecting the overall firing rate of auditory nerve fibers leading to prolonged conduction time. This finding can take support by a study done by Kujawa and Liberman (2009) where they found 50-60% permanent deafferentation of the auditory nerve fibers in the high frequency region of the cochlea in mice subjected to acoustic trauma even when acoustic thresholds returned to normal. Frequency specific NB chirp ABR can be carried out to see frequency specific damage of nerve fibers. Also, synchronous activity could be adversely affected in some of the individuals exposed to occupational noise which might be the reason of absence of CE-chirp ABR.

There was small, but significant latency prolongation of wave V from 4.9692 ms to 5.3017 msec as the duration of exposure to noise increased from T1 to T3. This indicates the damage to auditory nerve fibers increases as the exposure duration to noise increases. Hence, we can assume that CE-chirp ABR can be used as an effective tool to identify the early neural changes in individuals exposed to occupational noise.

Over all it can be concluded that the early cochlear and changes at neural level are difficult to monitor through conventional audiometry as thresholds might fall within normal limits. Hence, extended high frequency audiometry to be used to monitor the early cochlear changes at more basal region which is more susceptible to noise induced damage. Also, as DPOAE's monitor the status of outer hair cell and its damage due to noise exposure, this test to be used as it is more sensitive to any subtle changes in OHC's. Thus, extended high frequency audiometry and DPOAE's together serve as an excellent combination to monitor early changes at cochlear level. As evidenced by the present study, CE chirp ABR can be used as an effective tool to identify and monitor early neural changes at the level of brainstem in individuals exposed to occupational noise. Hence, comprehensive tests to be administered including all the above mentioned tests to prevent, identify and monitor the noise induced changes in auditory system.

Chapter 6

Summary and Conclusion

Noise is present in our day to day situation in the form of traffic noise, household noise or even when more than two people talking etc. But, usually these sounds are within safe levels of hearing which do not damage our ear sensitive structures. When these noise levels exceed safe level of hearing i.e. >85 dB (A), it causes damage to the ear structures leading to noise induced hearing loss (NIHL). NIHL can occur immediately or after a probation period which might take days or even years (Miller, Watson, & Covell, 1963; National Institute of Health and other communication disorders, 2014)

The present study was conducted with the aim to examine the cochlear and auditory neural functions in industrial workers exposed to occupational noise. The objective of the current study was to compare the cochlear and neural findings between individuals exposed to occupational noise and those who are not exposed to occupational noise on the following audiological tests: pure tone audiometry along with extended high frequency audiometry, distortion product otoacoustic emissions, click evoked ABR and CE-chirp evoked ABR. Also, to find the effect of noise exposure duration on cochlear and neural components based on the above tests.

To attain the goal, 20 participants comprising 40 ears in the age range 20-45 years with the mean age of 31.8 years participated in the study with a history of exposure to occupational noise (Group 2). Also 20 participants (40 ears), who were not exposed to occupational noise in the age range of 20-45 years with the mean age of 26.33 years participated in the study served as control group (Group 1). Group 2 was further divided into 3 subgroups (T1, T2 and T3) based on the duration of

exposure. The Pure tone thresholds were obtained at octaves and mid-octaves between 250 Hz to 16 kHz using modified Hughson Westlake procedure (Carhart & Jerger, 1959).DPOAEs were recorded at 8 points per octave in the frequency region of 2f1-f2 at the level L1=65 dB and L2=55 dB at the ratio of 1.22. DPOAE's were measured from 1000 Hz to 8000 Hz with 8 point per octave resolution. Total of 25 frequencies were measured. ABR was recorded using click stimuli at 90 dBnHL at 11.1/sec repetition rate and CE-chirp stimuli at 11.1/sec repetition rate at 80 dBnHL. Statistical analysis was done using statistical package for social sciences (SPSS) software version 20.

Conclusions

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

There is small, but significant elevation of pure tone threshold in individuals exposed to occupational noise when compared to control group. Also, there is a monotonic relation between pure tone thresholds and duration of exposure but the changes are very subtle.

The magnitude of hearing loss is greater in extended high frequency audiometry than conventional audiometry in individuals exposed to occupational noise. EHF mean thresholds are significantly higher in individuals exposed to occupational noise when compared to controls and also there is significant linear relationship between extended high frequency audiometric thresholds and duration of exposure.

DPOAE's were absent in most of the ears especially at frequencies between 4000 Hz to 8000 Hz as shown in and even when present there was decrease in amplitude and SNR at or above 4000 Hz. Also, there is decrease in amplitude and

SNR when the duration of exposure exceeded above 10 years. Hence, it serves as an effective tool in identifying early cochlear changes.

In Click evoked ABR, statistically significant difference was found in peak III and inter peak latencies involving the III peak between Group 1 and Group 2 indicating that cochlear nucleus in the central auditory pathway is more susceptive to noise induced changes at brainstem level. There is small, but significant prolongation of wave I and V, also significant latency prolongation can be seen in inter peak latency of I-III and III-V as the duration of exposure to noise increased.

In CE-chirp ABR, There was statistically significant prolongation of wave V latency in individuals exposed to noise compared to controls which could be attributed to damage of auditory nerve fibres at some particular frequency region especially at basal region leading affecting the overall firing rate of auditory nerve fibres leading to prolonged conduction time. Further, there was absence of response in 11 ears which might be because of affected synchronicity of auditory nerve fibres. Hence, CE-chirp ABR can be used as an effective tool to identify the early neural changes in individuals exposed to occupational noise.

Hence, we can conclude that the early cochlear changes are seen mainly at the basal region of cochlea and then extending to lower frequency regions. Also, early neural damages might be due to destruction of afferent connections in auditory nerve fibres. Hence, to detect these early cochlear and neural changes in individuals exposed occupational noise, a test battery approach comprising of conventional pure tone audiometry along with extended high frequency audiometry, distortion product oto acoustic emissions, click evoked ABR and CE-chirp evoked ABR to be used to prevent, identify and monitor noise induced pathological changes to auditory system.

Implications of the study

1. Extended high frequency audiometry and fine structure DPOAE's to be used clinically while testing an individual exposed to occupational noise as these tests are sensitive tool to identify and monitor the effect of duration on early cochlear changes.
2. CE-chirp ABR is promised to be an effective tool in identifying early damage to auditory nerve fibers, hence this test also to be included in a test battery approach to individuals exposed to occupational noise.
3. As there are no studies about CE-chirp ABR in individuals exposed to occupational noise, the findings will add information to the literature.
4. Finding of the study provides information on importance of diagnostic significance of test battery to be used in individuals exposed to occupational noise.
5. Findings of the study can be utilized in creating awareness and prevention programs on auditory effects of occupational noise.

Future research direction

1. Click evoked ABR to be carried out in individuals exposed to occupational noise using different repetition rates.
2. More studies to be done in the area of CE-chirp ABR in individuals exposed to occupational noise, so as to validate the efficacy of the test in identifying early damage to auditory nerve fibres.
3. Studies to be done in Frequency specific NB chirp ABR in individuals exposed to occupational noise so as to find frequency specific damage to auditory nerve fibres.

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