ASSESSMENT OF HIDDEN HEARING LOSS IN INDIVIDUALS EXPOSED TO NOISE USING TONE BURST AUDITORY BRAINSTEM RESPONSE AND DISTORTION PRODUCT OTOACOUSTIC EMISSIONS

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CERTIFICATE

This is to certify that this dissertation entitled "**Assessment of Hidden Hearing Loss in Individuals Exposed to Noise using Tone Burst Auditory Brainstem Response and Distortion Product Otoacoustic Emissions**" is a bonafide work submitted as a part for the fulfilment for the degree of Master of Science (Audiology) of the student Registration Number 18AUD019. This has been carried out under the guidance of a faculty of this institute has not been submitted earlier to any other University for the award of any other diploma or Degree.

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CERTIFICATE

This is to certify that this dissertation entitled "**Assessment of Hidden Hearing Loss in Individuals Exposed to Noise using Tone Burst Auditory Brainstem Response and Distortion Product Otoacoustic Emissions**" has been prepared under my supervision and guidance. It is also being certified that this dissertation has not been submitted earlier to any other University for the award of any other Degree or Diploma.

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DECLARATION

This is to certify that this dissertation entitled "**Assessment of Hidden Hearing Loss in Individuals Exposed to Noise using Tone Burst Auditory Brainstem Response and Distortion Product Otoacoustic Emissions**" is the result of my own side under the guidance of a faculty at All India Institute of Speech and Hearing, Mysuru and has not been submitted to any other University for the award of any other Degree or Diploma.

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Abstract

As the industries 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 sensorineural hearing loss. Therefore, the present study was conducted in order to check the effect of noise exposure on cochlea and auditory nerve functioning using Tone Burst ABR and DPOAEs in noise induced hidden hearing loss individuals. Amplitude and SNR of DPOAE (from 1000 to 6000 Hz), absolute latency of Tone Burst ABR at different frequencies (500, 1000, 2000 & 4000 Hz) and different intensities (80 dB nHL to 30dB nHL) were assessed in 10 industrial workers exposed to occupational noise and compared with 11 individuals not exposed to occupational noise. All of the participants had average pure tone thresholds < 15 dB HL. The results showed that there is an overall reduction in the mean amplitude of DPOAE especially, at higher frequencies in clinical group when compared to control group. In Tone Burst ABR, there is a prolongation of different wave latencies, especially wave V at supra threshold level compared to control group. This difference was more pronounced for 4000 Hz than other Tone Burst frequencies. Hence from the current study, it can be concluded that noise exposure has a more significant effect on higher frequencies. Hence it can be concluded that using a comprehensive test battery approach including DPOAEs and Tone Burst ABR, early cochlear, and neural pathological changes due to noise can be detected, identified and monitored.

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Chapter 1

Introduction

Normal hearing sensitivity in general defined as acquisition of absolute threshold response within the normal auditory limits. However, normal hearing thresholds might not necessarily indicate normal cochlear function (Kujawa & Liberman, 2009). When hearing sensitivity is normal, the first assumption is that the auditory system is functioning well. Sensorineural hearing loss is the most frequent type of hearing loss in adults that can be triggered by damage to the cochlear hair cells or auditory nerve fibres that affect the normal functioning of the inner ear (Festen & Plomb, 1983).The presence of hearing loss not only results in threshold elevation rather it also impacts speech perception and other temporal-based processing abilities such as temporal discrimination, temporal integration, and temporal resolution (Kujala & Brattico, 2009; Plack et al., 2014; Liberman et al., 2016). Individuals may not perceptually recognize the reduced hearing sensitivity, though their auditory system may deteriorate in hearing acuity defined as hidden hearing loss (Schaette & McAlpine, 2011).

Hidden hearing loss (HHL) is lately described as an auditory disorder, which affects hearing acuity and neural processing, especially in noisy environments in individuals with audiometric thresholds within normal limits. Hidden hearing loss could be due to the loss of cochlear synapses associated with noise exposure or aging (Elberling & Parbo, 1987). As a result of neural degeneration, it creates an impact on auditory processing at the supra-threshold level because this neuronal loss found to be selective for auditory nerve fibres with a low spontaneous rate. (Furman et al., 2013;

Kujawa & Liberman, 2015). The identification of HHL is not possible based on a standard clinical audiogram, because thresholds remain unaffected due to high spontaneous rate fibres respond to the sound when nerve fibres with low spontaneous rate are missing (Furman et al., 2013). Even though the actual perceptual difficulties due to hidden hearing loss are still unclear, researchers suggest that it can affect speech understanding in the presence of noise, tinnitus, and hyperacusis (Schaette & McAlpine, 2011; Bharadwaj et al., 2015). Some of the known risk factors associated with HHL are ototoxic drugs, noise exposure, aging and peripheral neuropathies.

However, normal hearing thresholds might not essentially indicate normal cochlear function. Kujawa and Liberman (2009) revisited the issue of neutral degeneration in ears with noise induced thresholds shift in mice subjected to acoustic trauma. In hearing threshold, a temporary change was noted, but in the high frequency region of the cochlea there was a permanent differentiation of the auditory nerve fibres (50-60%). Results of this study indicated that even in normal hearing individuals the functioning of efferent fibres which project from the brainstem to the cochlea can be getting affected (Kim & Frisina, 2002). Hence test other than standard pure tone audiometry which is sensitive to early cochlear changes to be included while testing in individual exposed to hazardous occupational noise. Electrophysiological research has shown that after noise exposure, spontaneous neuronal activity and compound action potentials in the auditory nerve are decreased **(**Dallos et al., 1978).

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. Several studies have suggested that Otoacoustic emissions may provide early identification of noise induced hair cell

damage up to 30 %, before it is seen in standard audiometry (Seixas et al., 1995; Desai et al., 1999). Study done by Attias et al in year 2001 reported that there was a narrowing of emission range and decline in DPOAE amplitude as the severity of damage increased because of noise exposure.

Wang et al (2009) did a study on 120 individuals exposed to occupational noise. Results indicated that there was significant difference in amplitude of DPOAE. From the literature it can be concluded that DPOAE is sensitive in early diagnosis of NIHL. The ABR has become widely recognized as a sensitive and cost effective screening modality in neuro-diagnosis. A study done by Attias and Pratt (1985) showed that there are latency changes in click ABR with individuals exposed to noise. Other researchers also reported a significant delay in ABR Wave-V latency with increase in the levels background noise level (Sammeth et al., 1986; Burkard & Sims, 2002). There are studies reported a delay in ABRs peaks to supra-threshold click stimuli in individuals with high noise exposure in comparison to low-exposure group and this prolongation was evident for all the waves i.e. wave I, III and V but most significant for the Vth wave (Skoe et al., 2019; Ridley et al., 2018). However, there are limited studies which probed into identifying the early neural changes or hidden hearing loss in noise exposed individuals. Routine audiological testing along with ABR might play a very important role in early identification and monitoring occupational noise induced hearing loss.

1.1. Need for the Study

The outlook of sensorineural hearing loss in individuals with noise exposure has been altered by the reality that there is a hidden element which is not detected in a

conventional audiogram. Because of this reason, the loss of cochlear synapses associated with noise exposure or aging has been defined as hidden hearing loss.

Several researchers recorded ABR to identify hidden hearing loss in individuals with noise exposure (Kujawa & Liberman, 2009; Hickox et al., 2017; Stamper & Johnson, 2015). Hence, present study is designed to assess the frequency specific ABR recording in individuals with noise exposure and compared with healthy individuals who were not exposed to noise. Studies reported utility of the otoacoustic emissions i.e. either TEOAEs or DPOAEs in monitoring the deleterious effects in individuals with noise exposure (Attias et al., 2001; Hughes et al., 2009). Most of the studies reported early indication of reduction in emission in noise exposed individuals and compliments the finding noticed in ABR in individuals with hidden hearing loss due to the noise exposure. There is no strong evidence up to now, that synaptopathy causes (or does not produce) measurable hearing deficits. Therefore, there is a need to assess that even in individuals with normal audiometric threshold and presence of OAEs, there might be physiological changes seen at the neural level with or without cochlear damage in the noise exposed individuals.

The use of a short-term (two to four hours) noise exposure, to monitor any permanent changes in the amplitude of DPOAE and latency of ABR measured approximately 24 hours after the exposure is one of the paradigms that has become prevalent in recent years (Kujawa & Liberman, 2009). Using such paradigms, we can document the permanent auditory nerve pathology "hidden" behind normal hearing sensitivity and normal hair cell counts can be effectively documented. In animals, studies reported that there was a reduction in the amplitude of ABR wave, especially

Wave-I associated with noise induced synaptopathy. But there were only smaller number of studies in humans, because ABR Wave-I is difficult to detect and measure in human beings, this limits its clinical use. Bharadwaj and colleagues revealed evidence of delay in Wave-V latency with increase in the background noise level (Bharadwaj et al., 2015). These results indicated that ABR Wave-V latency can be used as an effective and sensitive tool to diagnose cochlear synaptopathy in humans as it efficiently represents the early neural changes due to noise exposure. From the above studies, it can conclude that in both animals and humans, there was a negative correlation between the latency shift due to masking noise and ABR wave I amplitude. The findings of these experiments suggested that the latency shift in masked ABRs could be a valuable measure of hidden hearing loss in humans. However, cochlear synaptopathy is suspected of affecting humans of normal hearing levels, a testing procedure that is an accurate predictor of such hidden loss has not yet been documented.

Based on the literature, we can conclude that a single audiological test is not sufficient in identifying cochlear and neural pathology. Hence, test battery approach including conventional pure tone audiometry along with distortion product otoacoustic emission to assess early cochlear changes, psycho-acoustical measures and auditory brainstem response will help in assessing neural changes. Further, above test battery approach would give a holistic view in identifying early pathological changes seen in individuals exposed to occupational noise, in turn will help in early identification, prevention and monitoring issues related to auditory effects of occupational noise. Hence present study aimed to detect hidden hearing loss in individuals exposed to occupational noise using DPOAEs and frequency specific ABR.

1.2. Aim of the study

The aim of the present study was to check the impact of noise exposure on cochlea and auditory nerve functioning using Tone Burst ABR and DPOAEs in noise induced hidden hearing loss individuals.

1.3. Objectives of the Study

1. To assess the cochlear functioning using DPOAEs in individuals with noise induced hidden hearing loss.
2. To assess the functioning of Auditory nerve using Tone Burst ABR at different frequencies (500 Hz, 1kHz, 2 kHz & 4 kHz) and different intensities (80 dB nHL to 30 dB nHL).
3. To study the latency-intensity function of TB-ABR among noise induced hidden hearing loss individuals and compare with healthy individuals.

Chapter 2

Review of Literature

Noise is unwanted, but an unavoidable aspect prevalent in most of the work places. Noise exposure induces both auditory and non-auditory effects. Non-auditory effects include disturbance with sleep, stress and anxiety reaction etc. (Kujawa, 2003). Auditory effects include Noise induced hearing loss (NIHL) which is of two forms namely temporary threshold shift and permanent threshold shift. Noise induced hearing loss can be defined as permanent or temporary impairment of hearing resulting from abrupt exposure to loud noise or exposure to high levels of noise. Once, an individual is exposed to sound at his work place or at home, inner ear sensitive structures might get damaged, leading to NIHL (Miller et al., 1970).

Noise-induced hearing loss (NIHL) is the second most predominant type of acquired hearing loss after aging-induced hearing loss (Nandi & Dhatrak, 2008). If it is possible to identify noise-induced hearing loss early and formulate appropriate preventive measures that would reduce the socio-economic burdens associated with this disorder. It mainly occurs in the sensory cells and related structures inside the organ of Corti. Most precisely, the stereocilia of the outer hair cells (OHCs). Exposure to lower noise levels leads to temporary threshold shift (TTS), and gradually recovers (Miller et al., 1970). Duration and level of noise exposure determine whether synaptopathy occurs due to noise exposure or not. Temporary threshold shifts result in reduced stiffness of the stereo cilia of outer hair cells (Dew et al., 1993). It causes damage to synaptic transmission and mechano-electrical transduction channels (Patuzzi, 1998). Irreversible damage to hair cells results in a permanent threshold shift (PTS). Acoustic trauma is

the sudden sensorineural hearing impairment caused due to the single exposure results from mechanical damage of the organ of Corti by extreme pressure waves.

Cochlear synaptopathy, a form of damage to the synapsis between the inner hair cells and the auditory nerve fibres (ANFs) is believed to be seen in individuals with noise exposure and aging. This has been termed as hidden hearing loss, because, it is still unclear whether even humans showcase with similar deficits and also the effects are not exposed in to any of the clinical tests, both in behavioural and physiological measures of absolute threshold (Oxenham, 2016). Animal studies have shown that the major two causes for the loss of a large percentile of auditory nerve fibres are noise exposure and aging, which does not bring significant changes in their behavioural thresholds when tested with pure tone audiometry (Bharadwaj et al., 2015). They suggested that conventional pure tone audiometry is not sensitive to inner hair cells loss, and only a less amount of these hair cells are needed to detect a sound in quiet. Due to these limitations with the conventional audiometry, it is very difficult to identify an individual with noise exposure or an aged individual who are at the risk of further damage to the auditory structures which lead to the use of OAEs and ABR for assessing the damage seen in them.

In order to check the functioning of auditory system among individuals exposed to occupational noise in humans, there are several psychoacoustics measures such as temporal processing tests and speech perception in noise (SPIN) test (Kujala & Brattico, 2009; Plack et al., 2014; Liberman et al., 2016) as well as objective measures such as distortion product otoacoustic emissions and auditory brainstem response. Both psychoacoustic and objective measures help in identifying the cochlear lesions, functioning of the auditory nerves including noise exposure caused by various

mediators (Marshall & Heller, 1998). The below mentioned sub-section will describe the studies done on individuals with and without exposure of noise using psychoacoustic measures as well as electrophysiological measures.

2.1. Electrophysiological measures and Hidden Hearing Loss

Electrophysiological measures include both otoacoustic emissions and auditory brainstem responses used for the individuals with and without exposure of the noise. These studies were explored to identify the probable cause of hidden hearing loss among noise exposed individuals though there is normal audiometric threshold.

2.1.1. Effect of noise exposure on DPOAE

In case of permanent damage to hair cells or damage to mechano-sensory function, the more widely used diagnostics test includes pure tone audiometry and OAEs, which would reveal an increment in the thresholds and a decrement in the amplitude or absence of OAEs in those damaged frequency regions. A new perspective approach to identify and monitor individuals for early signs of cochlear damage due to hazardous noise exposure is distortion product otoacoustic emission (DPOAE) level mapping. Otoacoustic emissions primarily reflecting the outer hair cell activity of the cochlea, which is the site that will getting more affected by noise exposure. Therefore, they are particularly appropriate for evaluating early cochlear damages due to noise exposure (Balatsouras, 2004; Korres et al., 2009). However, when compared to pure tone audiometry, OAEs are reported to have better sensitivity in identifying damage to the auditory system (Attias et al., 2015). Literature suggested that OAEs can be used as

an early indicator of damages in the cochlea because of exposure to noise (Attias et al., 2001; Heller et al., 2009).

Vinck and colleagues studied the sensitivity of DPOAE 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 of DPOAEs was recorded soon after noise exposure and 6 hours of post-exposure. The amplitude in DP-gram was significantly reduced (poorer) 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, study reported amplitude in DP-gram reverted back to normal. They concluded that changes due to exposure to noise are temporary as seen in DPOAEs than in conventional audiometry. Hence DPOAEs are more sensitive to noise induced changes compared to conventional audiometry (Vinck et al., 1999).

Study done by Sexias and colleagues evaluated noise induced hearing loss individuals using DPOAEs. Conventional pure tone audiometry between 250 Hz to 8 kHz and DPOAEs were recorded for 456 participants (393 constructional workers and 63 controls). DPOAEs were obtained using two primary tones f1 and f2(f2<f1) at a constant ratio of 1.22 and equal level (L2= L1-10). DPOAEs were computed as DP-grams in the frequency region between 1031 Hz and 10028 Hz. Results indicated that DPOAE found to be deteriorated (poorer) at higher frequencies especially at 4, 6 and 8 kHz. Hence, DPOAEs serve as a sensitive tool to evaluate damage to inner ear (hair cells) structures due to noise exposure (Sexias et al., 2004).

Balatsouras (2004) studied conventional audiometry and DPOAEs in 34 subjects in the age range of 31 to 51 years who were exposed to occupational noise from 8 to 31 years. PTA was calculated for frequencies starting from 250 Hz to 8kHz. DPOAEs were obtained using two primary tones f1 and f2 (f2<f1) at a constant ratio of 1.22 and equal level (L1 = L2). DPOAEs were computed as DP-grams in the frequency region of 1 to 6 kHz. Results were compared with 30 normal hearing individuals without noise exposure (60 ears) who served as controls. Audiometric thresholds in all the individuals exposed to noise were within normal limits (<15 dBHL). DP-gram of control group had clear response to f1 and f2 product, but there was a significant decrease (poorer) in amplitudes or even absent response at some of the frequencies was found for the group with noise exposure in comparison to the group without exposure. The significant difference was seen mainly at higher frequencies in noise exposed group. Hence they concluded that DPOAEs might serve as a sensitive and objective tool in diagnosing NIHL.

Korres et al (2009) evaluated noise induced hearing loss with DPOAEs along with Conventional audiometry. The study consists of 105 individuals exposed to noise level 92-93 dBA for 8 hours per day. DPOAEs were obtained using two primary tones f1 and f2 (f2<f1) at a constant ratio of 1.22 and equal level (L2 = 60 and L1 = 45 dB). Audiometric thresholds were elevated at 4 kHz and 6 kHz region and reverting back to normal after 6 hours of cessation of exposure. Results revealed that DPOAEs amplitude was significantly reduced compared to control group in the frequency region of 3000 Hz and 4000 Hz especially at 6000 Hz.

One of the study done by Kujawa and Liberman (2009) wherein they induced a temporary NIHL of up to 30 dB in guinea pigs and mice. Study reported recovery from temporary threshold shift (TTS), the auditory system was evaluated with DPOAEs. These results suggested that there was a permanent damage at the frequency region corresponding to maximum TTS in the afferent nerve ending between the IHCs and ANFs. Therefore, it is being said that the remaining afferent connections that are undamaged would take up the work and help in preserving the thresholds within normal range.

Based on the above studies, it can be concluded that DPOAEs serve as a reliable marker in monitoring the damages seen in cochlea in individual with noise exposure. These findings indicated that DPOAEs assess the early cochlear changes in the cochlea due to noise exposure and also to monitor the prolonged effect of the same. But they do not account for neural changes which might be associated in individuals with occupational noise exposure. Several times, normal hearing thresholds might not necessarily indicate normal cochlear function. Even if the audiometric thresholds are in the normal range as the pathological changes might take days or years to express it symptoms, there might be physiological changes seen at cochlear or neural level which can be monitors through test assessing at brainstem or cortical level.

In a similar line of physiological measures, electrophysiological research has shown that after exposure to noise, spontaneous neuronal activity and compound action potentials in the auditory nerve are decreased (Dallos et al., 1978; Salvi et al., 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, 1978). Hence,

electrophysiological tests like Auditory Brainstem Response are also one of the diagnostic tools to identify early neural changes in individuals exposed to occupational noise.

2.1.2. Effect of noise exposure on ABR

Recent work on animals shows that over exposure to acoustic stimulation results not only temporary threshold elevation, without any hair cell damage, but can also cause irreversible loss of synapsis between the inner hair cells and auditory nerve fibres (Kujawa & Liberman, 2009). Evidences also shows that the difficulty with hearing in day today life in understanding speech in the presence of noise with normal hearing could be due to the differences in the fidelity with which supra-threshold sound is coded in the auditory pathway (Furman et al., 2013; Mehrai et al., 2016**)**. Furman and colleagues in the year 2013 carried out an experiment on guinea pigs with noise of frequency ranges between 4000 Hz-8000 Hz octave bands at 106 dB SPL for 2 hours wherein they recorded potentials from single auditory nerve fibres. They found that 2 weeks after the exposure, ABR thresholds came back to normal, indicating that there was a recovery in the hair cell functioning. However, the supra-threshold ABR amplitude had reduced, and a loss of 30% of synapsis between ANFs and inner hair cells was confirmed by immunostaining pre and postsynaptic markers of sensory epithelium. They concluded that cochlear synaptopathy mainly affects the auditory nerve fibres with low spontaneous rates and higher thresholds **(**Furman et al., 2013). At supra-threshold level, possibly due to the cochlear synaptopathy which is specific for nerve fibres with higher thresholds. Cochlear synaptopathy due to noise exposure has been extensively studied in animals, wherein, there is a decrease in wave I amplitude at the supra-threshold level and not significant at threshold level (Hickox et al., 2007;

Kujawa & Liberman, 2009). However, there are very few studies to see whether the same results hold good for humans as well.

Attias and Pratt (1985) studied the changes in ABR in individuals (N=15) exposed to occupational noise of >90 dBA with normal hearing thresholds. They recorded ABR using click stimulus at 75 dB HL. They assessed waveform morphology, absolute and inter peak latencies of I, III and Vth wave. Results revealed that there was a prolongation in wave and inter peak latencies in noise exposed individuals. Across studies, only a smaller number of studies reported that there was a reduction in the amplitude for Waves I, III or V, but there were several reports states that the latencies of the I, III, and/or Vth wave were delayed in workers who were exposed to occupational noise in comparison to the control group. Moreover, several studies, failed to find out the connection between noise exposure and its effect on Wave I amplitude (Fulbright et al., 2017; Grinn et al., 2017; Spankovich et al., 2017). Burkard and Hecox (1987) reported a substantial shift in Wave-V latency of ABR at supra threshold level with increase in the levels of background noise. This loss indicates selective loss of low spontaneous rate fibres and this latency shift correlates with the perceptual measures of fine temporal encoding (Bharadwaj et al., 2015).

Attias et al., 1993 studied involvement of central auditory neural activities in subjects with noise induced hearing loss and who had tinnitus associated with it 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 mild sensorineural hearing loss (loss at frequencies > 2 kHz and bilateral symmetric). They recorded ABR for a click stimulus of alternating polarity at 120 dBSPL using 10/sec

repetition rate. They assessed absolute and inter peak latencies of waves I, III and V. Results of click evoked ABR showed that wave V and I-V interpeak latencies were delayed in individuals with noise exposure.

Burkard and Sims, 2001 studied the effect of ipsilateral broad band noise (BBN) on ABR wave latency and amplitude especially for the wave I and V across two groups, young adults (21- 27 years) versus older adults (62-78 years). All participants in the younger group had thresholds better than 20 dB HL and for older group it was 40 dB HL. ABR was done for high pass filtered clicks (1000 Hz-3000 Hz) at115 dB SPL. Noise was presented at different levels of 20, 30, 40, 50, 60, and 70 dB effective masking. Responses were analysed based on the latency, inter-peak latency and amplitude of wave I and V. Results of the study indicated that there were only minor changes in the latency of wave I for both groups, but there was a significant increase in wave V latency as the noise level increased. For both groups, there was a reduction in peak amplitude with increase in the noise level. Mean peak latencies were prolonged in the older group, compared to younger group across noise levels, but the I-V inter peak interval was similar between the two groups, across different noise levels.

In one of the study done by Bharadwaj and colleagues on 32 human subjects with normal hearing thresholds in the age range of 20 to 40 years. They assessed the changes if any due to acoustic exposure in different tests including inter-aural time difference (ITD), click evoked otoacoustic emission (CEOAEs) and ABR (analysis of 1st and Vth wave latency) in the presence of masker noise. In individuals without noise exposure, the shift in wave V latency was more compared to individuals without noise exposure. Also the performance in sound localization which requires discrimination of

ITDs in envelops of sound waves was better in group without noise exposure in comparison to the low noise exposure group (Bharadwaj et al., 2015).Other Studies also reported that there was a delay in ABRs to supra threshold click stimuli in individual's exposed to high noise exposure when compared to the low-exposure group and this prolongation was evident for all the waves I, III and V but noticed most significant for wave V (Ridley et al., 2018; Camera et al.,2019).

Prendergast et al (2017), did a study on young adults with normal hearing thresholds with occupational noise exposure. ABR was done for high pass filtered clicks (> 1500Hz) at 80 and 100 dB peak SPL. The bandwidth chosen was 3000 Hz - 6000 Hz for the ABR stimuli. They didn't find out any relationship between the noise exposure and amplitude of ABR waves, especially wave I. But there was a delay in the wave V latency as the noise exposure level increased.

2.2. Psychoacoustic measures and Hidden Hearing Loss

In general, a trend of reduced speech and temporal processing abilities in terms of temporal discrimination, temporal ordering, temporal integration, temporal masking (backward and forward masking), as well as localization and pitch perception were observed in individuals exposed to occupational noise (Moore, 2007). They also suggested that individuals with cochlear hearing loss perform poorly on tasks such as temporal integration, modulation detection and gap detection. These deficits were seen in spite of normal hearing sensitivity. Similar results were found by other researchers (Epstein et al., 2016; Kujala & Brattico, 2009; Guest et al., 2018). In a study done by Kumar and colleagues compared the speech perception and psycho-acoustical abilities in individual with occupational noise exposure in the age range of 30- 40 years, who

had PTA <20dB HL. Using gap detection and duration pattern tests they assessed temporal processing abilities. Speech recognition was tested in the presence of multi-talker babble at -5dB SNR. Results of the study indicated that speech recognition scores and temporal processing abilities were significantly reduced in noise-exposed group in the presence of background noise (Kumar et al., 2012).

The study done by Stone and Moore in the year 2014 aimed to assess the effect of noise exposure on amplitude modulation detection. There were two group of participants, young (18-24 years) and older (26-35 years) who were exposed to high level of noise (HN, i.e. >35 dBA) and low level noise (LN i.e. <38 dBA). All participants had PTA <15 dB HL. Amplitude modulation detection thresholds were made for 3,4 and 6 kHz at 10, 25 and 40 dB SL. Stimuli duration was 250 ms. Results of the study indicated that the HN group had poor amplitude detection thresholds at 10 dB SL in comparison to LN groups at 3 and 4 kHz.

Based on the above studies we can conclude that overall most of the studies do reflect the importance of auditory brainstem response and otoacoustic emissions estimation in NIHL individuals in early stage of noise exposure. So, DPOAEs can be used as an effective tool in monitoring early cochlear damages in individuals with noise exposure. But they do not account for early neural changes which might be also associated with these individuals. Therefore, these neural changes can be monitored through auditory brainstem responses. Hence, ABR along with conventional audiological tests might give an insight to early cochlear changes along with neural changes in individuals exposed to occupational noise.

Chapter 3

Methods

The present study aimed to check the effect of noise exposure on cochlea and auditory nerve functioning using Tone Burst ABR and DPOAEs in noise induced hearing loss individuals. To achieve the above aim, the below mentioned method was adopted.

3.1. Selection of participants

There were total of 21 individuals with normal hearing who were in the age range of 30 to 45 years (mean age- 36 years) considered for the study. Group I include 11 individuals with normal hearing who are not exposed to occupational noise served as the control group. Group II consisted of 10 individuals with normal hearing who were exposed to occupational noise for more than 5 years served as experimental group.

3.1.1. Participant Inclusion criteria

Participants who were having bilateral normal hearing up to 15 dBHL for all frequencies from 250 to 8 kHz was considered for both group I and II. Experimental group participants were having duration of exposure to noise for more than 5 years, 8 hours/day. Both group I and II participants were having normal middle ear functioning confirmed by 'A' type tympanogram and the presence of both ipsilateral and contralateral reflexes at 500 to 4000 Hz in both ears.

3.1.2. Participant exclusion criteria

For both control and experimental group, any participants who were reported to have the presence of tinnitus, middle ear disease, conductive pathology were excepted from the study based on the case history. The participants who had the habit of smoking or alcohol, exposed to ototoxic drugs, head trauma, under medication for Diabetes mellitus, and Hypertension were excluded from the study based on information collected through detailed case history.

3.2 Environment

All audiological testing was conducted in an acoustically treated room where the ambient noise levels were within the permissible limits (ANSI S3.1, 1999, R2013).

3.3 Instrumentation

The below mentioned audiological equipment's were used for the present study:

1. Calibrated dual channel Inventis Piano diagnostic audiometer with TDH 39 head phones was used for pure tone audiometry and speech audiometry.
2. Calibrated Immittance meter (GSI-Tympstar V 2.0) was used for evaluating middle ear status.
3. Calibrated Biologic Navigator Pro Evoked potential (version 7.2.0.) system was used to carry out Click evoked ABR and Tone Burst ABR.
4. Otodynamics ILO (Version 6) Echo port system was used for measuring DPOAEs.

3.4. Procedure

Preliminary evaluations: As a first step detailed structured case history was taken from all the participants to get information about their working environment, nature and duration of noise exposure and to rule out any middle ear pathologies. A questionnaire developed by Tharmar (1990) was also administered in the experimental group along with case history. First pure tone audiometry was done for octave frequencies starting from 250 Hz to 8000 Hz using calibrated double channel Inventis piano coupled to TDH-39 earphone to estimate air conduction threshold and Radio Ear B-71 for bone conduction threshold. The threshold was estimated by using modified Hughson and Westlake (Carhart & Jerger, 1959) procedure, air conduction well as bone conduction thresholds were estimated. The mean pure tone average for group I and group II were 12.5 dBHL and 13.25 dBHL respectively. Speech recognition threshold and speech identification scores were obtained by using Kannada paired words and phonetically balanced word list in Kannada respectively. Tympanometry was carried out by using calibrated GSI-Tympstar using 226 Hz probe tone, and reflexometry was done at 0.5 kHz, 1 kHz, 2 kHz and 4 kHz for both ipsilateral and contralateral reflexes. For all the participants both acoustic reflexes were present at 70 to 80 dBSL.

DPOAEs measurements. DPOAE fine structure was studied at 8 points per octave using calibrated Otodynamics ILO (Version 6) Echo port system to evaluate the function of outer hair cells. DPOAE recordings were made between DP frequencies of 1000 Hz to 6000 Hz using stimulus levels of 65 dB SPL (L1) and 55 dB SPL (L2) and a constant stimulus frequency ratio of (f2/f1) is 1.22. The DPOAEs were evaluated for amplitude parameter at various distortion product frequencies and the signal-to-noise ratio (SNR) was recorded. The response was considered to be present if the SNR > 6 dB for both groups at any three consecutive frequencies tested.

ABR recording was done in a sound treated room using Biologic Navigator Pro Evoked (version 7.2.0) system. The evoked potentials were recorded with electrodes placed at Fz, M1 and Fpz position. Electrode impedance considered was below 5kΩ for all the electrodes. ER-3A insert earphone was used to present the stimulus. Click evoked ABR was carried out at 11.1/s and 90.1/s repetition rate with rarefaction polarity to rule out retro-cochlear pathology. The stimuli used for assessment was tone burst and the intensity level was decreased in 20 dB steps from 80 dB nHL to 40 dB nHL and 10 dB steps from 40 to 30 dB nHL. A repetition rate of 11.1/s was used as it provides a better morphology at lower repetition rate. A band pass filter of 100 to 3000 Hz and recorded in a 12 ms time window. Thousand five hundred sweeps were averaged at each presentation levels and average was taken. The absolute amplitude and absolute latency of I, III and V were analysed for both groups (Control &experimental) at all the presentation levels (80 dB nHL to 30 dB nHL). The ABR measures considered for the analyses were absolute latency and absolute amplitude. The peaks considered were marked as wave I, III and V. The latencies of the different peaks were marked by considering the centre or midpoint when the waveform contained double peaks of equal amplitude and if the amplitude were unequal then it was marked at the centre of the larger peak. Thus the latency-intensity function of TB-ABR among group I and II were compared. The protocol used for click evoked ABR and Tone burst ABR is mentioned in Table 3.1.

Table 3.1: *Stimulus and acquisition parameters for recording click evoked and TB-ABR*

Stimulus parameters	**Tone Burst ABR**	**Click ABR**
Type of stimuli	Tone burst	Click (100 microsecond)
Stimulus Frequency	500 Hz,1000 Hz, 2000 Hz & 4000Hz	Nil
Intensity	80 dB, 60 dB, 40 dB & 30 dBnHL	80 dB nHL
Repetition rate	11.1/sec	11.1/s & 90.1/s
Polarity	Alternating	Rarefaction
Total number of stimuli	1500	1500
Acquisition Parameters		
Analysis Time	12 ms	12 ms
Filter Setting	High pass: 100 Hz Low pass: 3000Hz	High pass: 100Hz Low pass: 3000Hz
Amplification	100000	100000
Number of channels	1	2
Number of recordings	2	2
Transducer	Insert ear phone (ER-3A)	Insert ear phone (ER-3A)
Electrode Montage	**Non inverting electrode (+):** upper forehead (Fz) or Vertex (Cz) **Inverting electrode (-):** mastoid / ear lobe of the stimulus side **Ground electrode:** Non-test ear mastoid	**Non inverting electrode (+):** upper forehead (Fz) or Vertex (Cz) **Inverting electrode (-):** Both ear mastoids **Ground electrode:** Lower forehead (Fpz)

2.5. Statistical Analyses

The data was analysed using statistical package for social sciences SPSS (Version 20). Shapiro-Wilks test of normality was performed to check whether the data was normally distributed or not. Descriptive statistics was performed to obtain mean and standard deviation of SNR of DPOAE and absolute wave latency for tone burst ABR. Mann-Whitney U test was performed to compare between groups for TB-ABR as well as for SNR of DPOAEs. Friedman test was done for within group comparisons. If there is any significant difference found in Friedman test, then Wilcoxon signed rank test was administered.

Chapter 4

Results

The aim of the present study was to check the effect of noise exposure on cochlea and auditory nerve functioning using Tone Burst ABR and DPOAEs in noise induced hidden hearing loss individuals. The parameters considered for analysis were wave Vth latency of tone burst ABR and the signal-to-noise ratio of DPOAEs at different frequencies. The TB ABR waves were recorded at four intensities (80, 60, 40 & 30 dB nHL) and four frequencies (500 Hz, 1000 Hz, 2000 Hz & 4000 Hz). The responses from the different intensities were compared between two groups i.e. individuals without occupational noise exposure in the age range of 30 to 45 years serve as the control group (group I) and those individuals with noise exposure serve as the experimental group (group II), all having normal hearing thresholds in both ears.

The SNR of DPOAE and wave latencies for the different tone burst frequencies and intensities were analysed using statistical tool SPSS. Shapiro-Wilks test of normality was administered and showed non-normal distribution of the data (p <0.05). Hence, non-parametric test was administered for both TB-ABR and DPOAEs. The statistical test administered was as follows.

1. Descriptive statistics was performed to obtain mean and standard deviation of SNR of DPOAE and absolute wave latency for tone burst at different frequencies (500 Hz, 1000 Hz, 2000 Hz & 4000 Hz) and intensities (80, 60, 40 & 30 dB nHL).
2. Mann-Whitney U test was performed between the two groups to compare the latency of TB-ABR as well as for SNR amplitude of DPOAEs.

3. Friedman test was done for within group comparisons i.e. to see the effect of different frequencies on DPOAE SNR amplitude as well as for TB-ABR latency.
4. If there is any significant difference found in Friedman test, then Wilcoxon signed rank test was administered to check the pair wise comparisons.

4.1. Signal-to-Noise Ratio (SNR) of DPOAEs

Descriptive statistics for mean and standard deviation (SD) was done for the measurement of SNR between group I and group II. The mean value of SNR in dB and standard deviation was obtained for DPOAE from 1000 Hz to 6000 Hz are mentioned in the Table 4.1 and represented in the Figure 4.1.

Table 4.1: *Mean and SD for SNR of DPOAEs amplitude across frequencies in group I and group II*

Frequency (Hz)	SNR			
	Group I (Control group)		Group II (Experimental Group)	
	Mean (dB)	SD	Mean (dB)	SD
1000	12.44	0.22	11.62	0.30
1500	12.64	0.36	11.86	0.42
2000	12.95	0.30	12.17	0.40
3000	12.12	0.37	10.92	0.47
4000	11.14	0.41	8.44	0.70
6000	10.56	0.52	7.86	0.84

The descriptive statistics results of SNR parameter indicated that mean SNR varies across the tested frequencies i.e. from 1000 to 6000 Hz in both the groups. The

mean SNR at low and mid frequencies (up to 3000 Hz) are more (better) in comparison to higher frequencies (above 3000 Hz) especially at 4000 Hz and 6000 Hz. However, the reduction (poorer) in mean SNR was statistically significant only for the experimental group. At relatively higher frequencies (above 3000 Hz) changes are minimal in the control group when compared to the experimental group. Further, standard deviation shows higher values at higher frequencies in comparison to lower frequencies particularly for the experimental group. These observations probably indicate that the noise exposure has an effect on DPOAEs amplitude, particularly for frequencies above 2000 Hz.

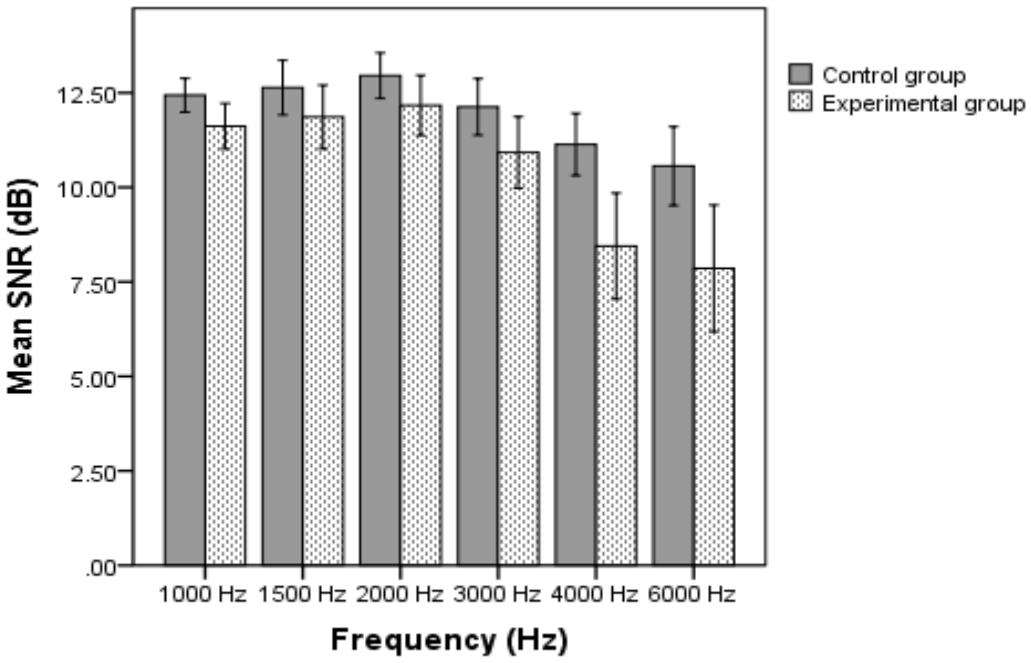

Figure 4.1: Mean value of DPOAE SNR amplitude across frequencies in group I and group II

Freidman test was done to compare the SNR differences across the frequencies within the group. Test results indicated that there is a statistically significant difference

across frequencies only for the experimental group [$x^2_{(5)}$ =13.367, p< 0.05] and for the control group there was no significant difference seen across frequencies [$x^2_{(5)}$ = 5.807, p > 0.05]. Further, Wilcoxon signed rank pair-wise comparison was administered for the experimental group, to see across different frequencies DPOAE SNR are different or not. The frequencies at which a statistically significant differences (p <0.05) seen in the experimental group are mentioned in Table 4.2.

Table 4.2: *Wilcoxon signed ranks test results for frequency pairs that exhibited a significant difference for Group II.*

Frequency Comparison	Z-value	*p*-value
1500-4000 Hz	-2.073	0.038*
1500-6000 Hz	-2.028	0.043*
2000-4000 Hz	-2.429	0.015*
2000-6000 Hz	-2.197	0.025*

*Note. 'p': Level of significance p <0.05: indicates significant difference; *p< 0.05.*

For the experimental group, Wilcoxon pair wise comparison revealed that there are statistical differences between SNR of low and mid frequencies (below 3000 Hz) and higher frequencies (above 3000 Hz). Whereas there are no significant differences within low and mid frequencies (1000-1500 Hz ,1000-2000 Hz,1000-3000 Hz, 1000-4000 Hz,1000-6000 Hz, 1500-200Hz, 1500-3000 Hz, 2000-3000 Hz) and within higher frequencies (3000-4000 Hz, 3000-6000 Hz and 4000-6000 Hz) at 0.05 level.

Mann Whitney U test was administered to evaluate the significant difference in SNR amplitudes of DPAOE across frequencies (from 1000 to 6000 Hz) between the

experimental group and control group. The statistical outcome of Mann Whitney U test is represented with Z-values in Table 4.3.

Table 4.3: *Z and p values of Mann Whitney test for both groups.*

Frequency	Z-value	p-value
1000 Hz	-1.69	0.091
1500 Hz	-2.46	0.014*
2000 Hz	-2.81	0.005**
3000 Hz	-2.62	0.009**
4000 Hz	-2.66	0.008**
6000 Hz	-2.68	0.010**

*Note: 'p': Level of significance p<0.05: indicates significant difference; *p< 0.05, **p<0.01.*

4.2. Latency of TB-ABR

Descriptive statistics for mean and standard deviation (SD) was done for the measurement of latency of waves I, III and V between control and experimental group across various Tone Burst frequencies (500 Hz,1000 Hz,2000 Hz & 4000 Hz) at different presentation levels ranging from 80 dB nHL to 30 dB nHL) are mentioned in the Table 4.4.

Table 4.4: *Mean and SD for latency of waves I, III and V across frequencies at different intensity levels in group I and group II.*

Frequency	Stimulation Level (dB nHL)	Waves I, III, V	Latency Parameter			
			Group I		Group II	
			Mean (ms)	SD	Mean (ms)	SD
500 Hz	80	V	7.07	0.07	7.60	0.12
	60		8.24	0.09	8.67	0.05
	40		9.44	0.14	9.64	0.13
1000 Hz	80	V	6.57	0.08	7.50	0.57
	60		7.32	0.09	7.89	0.49
	40		8.43	0.12	8.59	0.12
2000 Hz	80	I	2.28	0.08	2.48	0.08
	60		2.79	0.12	2.88	0.09
	80	III	4.22	0.08	4.44	0.11
	60		4.72	0.22	4.70	0.10
	80	V	6.28	0.06	6.70	0.06
	60		6.74	0.10	7.22	0.11
	40		7.50	0.11	8.20	0.13
4000 Hz	80	I	1.96	0.12	2.11	0.18
	60		2.19	0.08	2.27	0.06
	80	III	3.77	0.08	3.98	0.10
	60		4.22	0.07	4.32	0.06
	80	V	5.80	0.06	6.45	0.11
	60		6.34	0.10	6.95	0.24
	40		6.80	0.12	7.87	0.14

The descriptive statistical results of latency parameter indicated that there was an increase in the mean latency of ABR waves I, III and V at all the tested intensities for both the groups. Wave I, III latencies at 500 Hz, 1000 Hz and wave latency at 30 dB nHL for all frequencies were excluded from the analysis as very few number of

participants exhibited this response (N <5). It was observed that the prolongation of waves I, III and V were more (poorer) in the experimental group when compared to the control group. The same is represented in the Table 4.4 and depicted in the Figure 4.2. It was also noted that the mean latency of wave V was more for the experimental group especially for 4000 Hz, at higher presentation level i.e. at 80 dB nHL in comparison to lower presentation levels. In other words, a more pronounced difference was seen for wave V latency compared to wave I and wave III latency.

Freidman test was performed to compare the latency differences across different intensities and frequencies within the group. Test results indicated that there was a significant difference for both the experimental group[$x^2_{(15)}$ =30.000, p< 0.05] and for control group [$x^2_{(15)}$ = 89.536, p < 0.05]. Further, Wilcoxon signed rank pair-wise comparison was administered for both groups to see across different intensities and frequencies, latency of wave V are different or not. The intensities and frequencies at which there were statistically significant differences (p < 0.05) for group I and group II results are mentioned in Table 4.5 & Table 4.6.

Table 4.5: *Wilcoxon signed rank test across different intensities within the group for latency of TB-ABR*

Group I (Control group)				
80 vs 60 dB nHL	500 Hz**	1000 Hz**	2000 Hz **	4000 Hz**
80 vs 40 dB nHL	500 Hz*	1000 Hz*	2000 Hz *	4000 Hz*
60 vs 40 dB nHL	500 Hz*	1000 Hz*	2000 Hz *	4000 Hz*
Group II (Experimental group)				
80 vs 60 dB nHL	500 Hz*	1000 Hz*	2000 Hz **	4000 Hz**
80 vs 40 dB nHL	500 Hz	1000 Hz	2000 Hz *	4000 Hz**
60 vs 40 dB nHL	500 Hz	1000 Hz	2000 Hz *	4000 Hz*

*Note: 'p': Level of significance p <0.05: denotes significant difference; *p < 0.05, **p <0.01.*

The results indicated that for the control group there was a clear representation for the effect of intensity on wave latency across all the frequencies. But for the experimental group the effect of intensity on wave latency was clearly established only at higher frequencies (2000 Hz &4000 Hz) not for the lower frequencies (500 Hz & 1000 Hz).

Table 4.6: *Wilcoxon signed ranks test across frequencies at different intensity for group I and group II.*

	80 dB	60 dB
Group I	500 vs 2000 Hz**	500 vs 2000 Hz**
	500 vs 4000 Hz**	500 vs 4000 Hz**
	1000 vs 2000 Hz**	1000 vs 2000 Hz**
	1000 vs 4000 Hz**	1000 vs 4000 Hz**
	2000 vs 4000 Hz**	2000 vs 4000 Hz**
	80 dB	60 dB
Group II	500 vs 1000 Hz**	500 vs 1000 Hz*
	500 vs 2000 Hz**	500 vs 2000 Hz*
	500 vs 4000 Hz***	500 vs 4000 Hz**
	1000 vs 2000 Hz**	1000 vs 2000 Hz*
	1000 vs 4000 Hz**	1000 vs 4000 Hz**
	2000 vs 4000 Hz**	2000 vs 4000 Hz**

*Note: 'p': Level of significance p <0.05: denotes significant difference; *p < 0.05, **p <0.01, p*** < 0.001.*

Wilcoxon pair wise frequency comparison showed that there is statistical significant difference noticed in the absolute latency of wave V at 80 dB nHL and 60 dB nHL, but this difference was not there for 40 dB nHL. More noticeable difference was seen for the frequencies between 500 vs 4000Hz,1000 vs 4000 Hz and 2000 vs 4000Hz in clinical group. Whereas statistical differences noticed are very limited across these frequencies for the control group. Also a more pronounced statistical difference was seen for wave V latency at 4000 Hz when compared to other frequencies (500 Hz,1000 Hz & 2000 Hz).

Mann Whitney U test was done to evaluate the significant differences in latency of waves I, III and V at different frequencies and intensities between group I and group II. The statistical outcome is represented with Z-values and p-values for latency of TB-ABR across the frequencies at different intensities in Table 4.6.

Table 4.6: *Z and p values of Mann Whitney test for both groups.*

Frequency	Stimulation Level (dB nHL)	Waves I, III, V	Latency Parameter Z-value	*p*-value
500 Hz	80	V	-2.348	0.036*
	60		-0.402	0.688
	40		-2.200	0.028
1000 Hz	80	V	-2.507	0.010*
	60		-2.116	0.034*
	40		-1.502	0.133
2000 Hz	80	I	-2.507	0.012*
	60		-1.388	0.165
	80	III	-1.996	0.043*
	60		-0.178	0.859
	80	V	-3.878	0.000***
	60		-2.845	0.004**
	40		-2.608	0.013*
4000 Hz	80	I	-1.996	0.046*
	60		-2.55	0.011*
	80	III	-2.001	0.038*
	60		-2.593	0.010*
	80	V	-3.876	0.000***
	60		-3.778	0.000***
	40		-3.249	0.001**

*Note: 'p': Level of significance p <0.05: denotes significant difference; *p < 0.05, **p <0.01, p < 0.001.*

The results of Mann Whitney test indicated that there was a significant difference for most of the latency parameter between the two groups. A more pronounced statistical difference was seen for the Vth wave at 4000 Hz, when compared to other frequencies. It was also noted that there are statistical differences noticed for latency parameter at 80 dB nHL across all the frequencies between the two groups, whereas statistical differences are limited across frequencies at 60 and 40 dB nHL, particularly for 500,1000 and 2000 Hz. Whereas for 4000 Hz there was a significant difference even at 60 and 40 dB nHL. Also a more pronounced difference was seen for wave V latency when compared to wave I and wave III latency. Figure 4.2 & 4.3 shows waveforms across different Tone Burst frequencies at different presentation levels in group I and group II respectively.

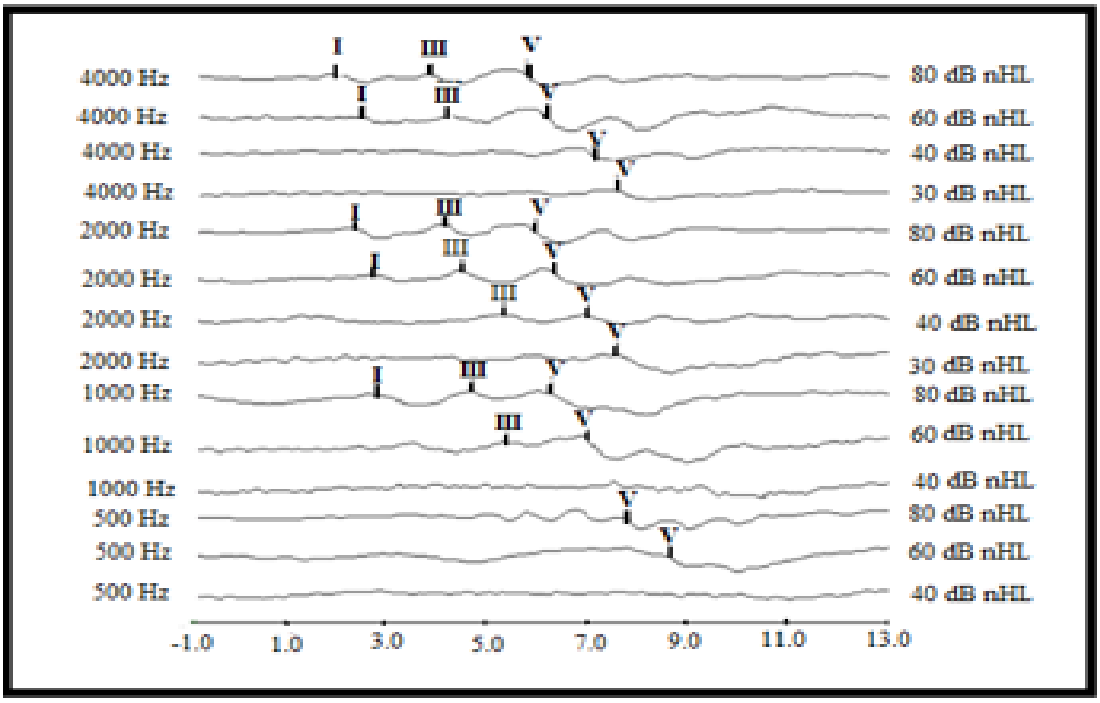

Figure 4.2: A sample TB-ABR waveforms at different frequencies and intensities from individuals without noise exposure (Group I).

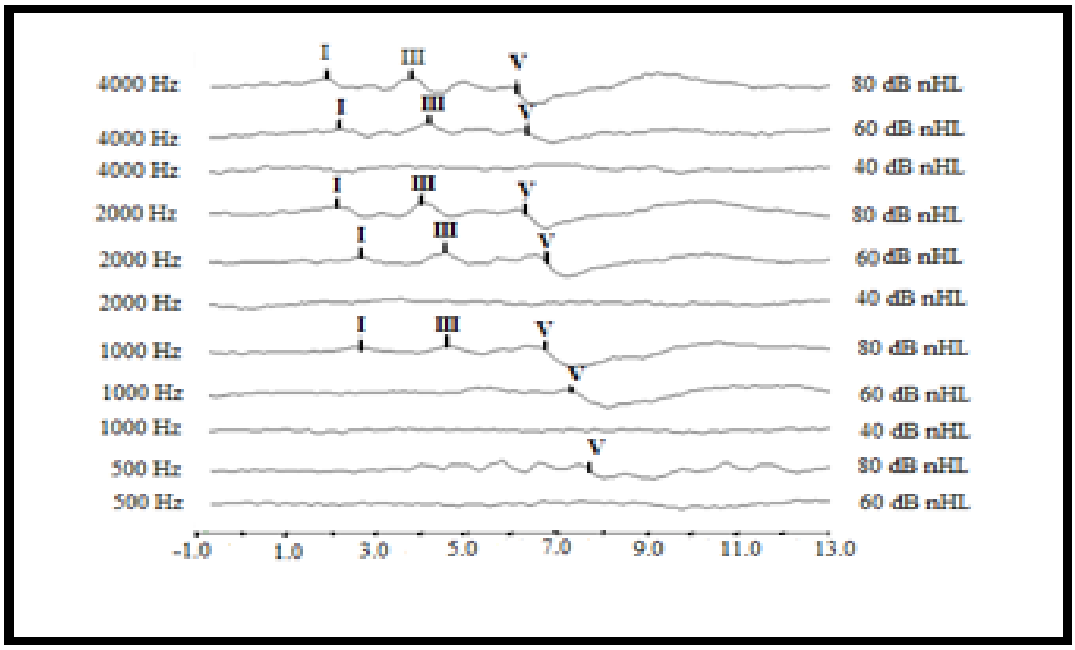

Figure 4.3: A sample TB-ABR waveforms at different frequencies and intensities from individuals with noise exposure (Group II).

From the figure 4.3 and 4.4 it can be infer that with respect to frequency, a more pronounced wave V latency shift was seen for 4000 Hz in comparison to other frequencies (500,10000 &2000 Hz). This difference was evident for 80 dB nHL than 60 and 40 dB nHL. Latency differences are limited across frequencies at 60 and 40 dB nHL, particularly for 500,1000 and 2000 Hz. Whereas for 4000 Hz there was a difference even at 60 and 40 dB nHL between the two groups. Figure 3.5 shows the mean absolute latency of wave V across frequencies at 80,60 and 40dB nHL in group I and group II.

4.3. Latency-Intensity graph of TB-ABR

Latency intensity graph of the TB-ABR was analysed for the individuals exposed to noise and compared with those individuals not exposed to noise. The analysis was done in relation to the different frequencies along with different intensity as the

variable. The below mentioned figure 4.4 shows the latency-intensity graph in control and experimental group.

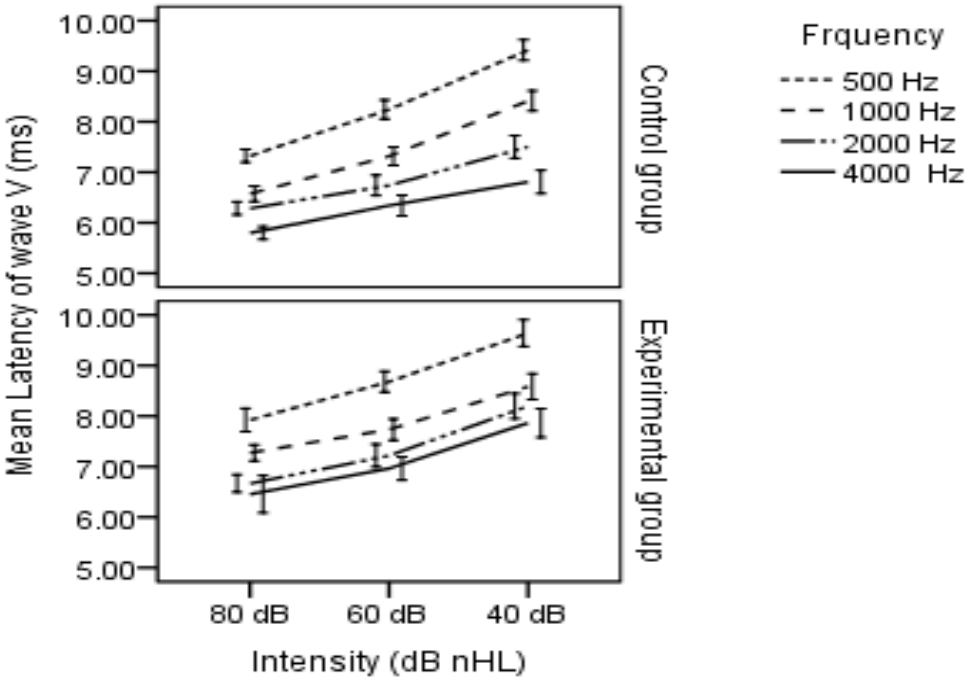

Figure 4.4: Latency-Intensity graph at different frequencies of wave V latency in group I and group II.

From the figure 4.4, it can be infer that for latency parameter a significant difference is observed for wave V between the control group and experimental group. With respect to frequency, there was a reduction in mean latency of wave V when the frequency shifts occurs from lower to higher frequencies (500 to 4000 Hz). There is a significant difference in the latency of wave V between the two groups especially at 4000 Hz in comparison to other frequencies. This could be because frequencies in the range of 4000 Hz to 6000 Hz are more vulnerable to the damages due to noise exposure. With respect to intensity, a more pronounced latency shift is seen at 80 dB nHL in

comparison to 60 and 40 dB nHL. With increase in intensity there was a decrease in absolute latency of wave V for both groups for all the frequencies (500,1000,2000 & 4000 Hz).

Chapter 5

Discussion

This study was taken up to compare the effect of noise exposure on cochlea and auditory nerve functioning using Tone Burst ABR and DPOAEs. The results of the study indicated that there was a significant effect of noise exposure on SNR amplitude of DPOAEs and latency of TB-ABR. The results of the study were discussed under the following heading:

5.1. Comparison of DPOAE SNR amplitude between individuals with and without noise exposure

Present study reported a statistical significant differences between group I and group II for SNR of DPOAE at each frequency except 1000 Hz. Also when comparison was done for DPOAE SNR across the frequencies within the group, only the group II showed a significant difference. There was a decrease (poorer) in DPOAE SNR among individuals exposed to noise in comparison to the control group and most significant decrease was seen for 3000 Hz, 4000 Hz and 6000 Hz. It might be because of the group II individuals may have subtle deficit in the cochlear region especially in the high frequency zone which is not identified in the pure tone audiometric thresholds but reflected in highly sensitive emission of distortion product. These observations are in agreement with studies done by other researchers (Attias et al., 1998; Balatotsouras, 2004). Seixas and colleagues reported that DPOAE amplitude deteriorated (poorer) at higher frequencies especially at 4, 6 and 8 kHz (Seixas et al., 2004). From the literature we can conclude that DPOAEs serve as a sensitive tool to evaluate damage to inner ear (hair cells) structures due to noise exposure. This is because the outer hair cells are

more sensitive to noise induced changes which are responsible for generation of DPOAEs.

The present study results indicated that there was a significant decrease (reduction) in SNR at higher frequencies and the reduction was more seen in group II in comparison to group I. Further, the difference was statistically significant for group II whereas not significant for group I. This is because as the duration to occupational noise exposure has been increased, the SNR amplitude of otoacoustic emission at 4000 Hz and 6000 Hz decreased (poorer) as the hair cells at these frequencies would be more vulnerable in comparison to the individual without occupational noise exposure. These observations from this study are in accordance with the studies done by other researchers (Corthals et al., 1999; Sexias et al., 2004). The sensitivity of DPOAE in monitoring the effects of TTS on outer hair cells was analysed in their study. They recorded DP-gram soon after the noise exposure and 6 hours post exposure. Results showed that the amplitude of DP-gram was significantly reduced in 4 kHz region. After 6 hours of cessation of exposure, amplitude in DP-gram reverted back to normal (Attias et al., 2001; Hughes et al., 2009). They compared the sensitivity of conventional pure tone audiometry and DPOAEs in monitoring the effects of temporary threshold shifts on OHCs and they found that the amplitude of DPOAEs did not recover completely to the pre-exposure level especially, in 4 kHz & 6 kHz frequency region. Study done by Korres et al in year 2009 in noise exposure individuals reported that the DPOAE amplitude decreases mainly in high frequencies. They also reported more effect especially in the range of 4 kHz to 6 kHz which is more vulnerable to damage in noise exposure. These findings suggest that OAEs are more sensitive when compared to conventional pure tone audiometry in detecting early cochlear changes. Moreover, it is

an objective method which doesn't require the active participation of an individual and it is less time consuming. Hence it can be used to identify early cochlear changes due to noise exposure. But few animal studies in literature do report that there was no decrease in amplitude of DPOAE after recovery from TTS and hence no permanent OHC damages occurred due to noise exposure (Kujawa & Liberman, 2009; Furman et al., 2013).

5.2. Comparison of latency of TB-ABR between with and without noise exposure

The response of TB-ABR showed that there was a significant difference between the two groups in terms of wave latency, especially for 4000 Hz in comparison to other frequencies. This prolongation was evident for all the waves I, III and V but most significant for wave V. More noticeable difference was seen for the frequencies between 500-4000 Hz,1000-4000 Hz and 2000-4000 Hz in experimental group. Whereas statistical differences noticed are very limited across these frequencies for the control group. This is because frequencies in the range of 4 kHz to 6 kHz which is more vulnerable to damages due to noise exposure. Similar results were obtained by Prendergast et al (2017) wherein, they found that wave V latency was prolonged (poorer) in normal hearing group with higher noise exposure in comparison to lesser noise exposure individuals. A study by Bharadwaj and colleagues used noise to find out amount of wave V latency shift in normal hearing individuals with greater noise exposure versus lesser noise exposure in backgrounds. Results of the study indicated that wave V latency shift was found to be more in individuals with noise exposure when compared to individuals without noise exposure (Bharadwaj et al., 2015).

5.3. Comparison of Latency-Intensity graph of TB-ABR between individuals with and without noise exposure.

The wave V latency at higher intensities to tone burst stimuli were significantly delayed in individuals with noise exposure when compared to control group without noise exposure. A more pronounced latency shift was seen at 80 dB nHL than 60 and 40 dB nHL. This prolongation in wave latency was seen for waves I, III and V, but this trend was well established for wave V in comparison to wave I and III. Similar findings were reported by other researchers (Ridley et al., 2018; Camera et al., 2019). They found that there was a delay in ABRs to supra threshold click stimuli high noise exposure group in comparison to the low noise exposure group. This prolongation was seen for all the waves I, III and V but was most significant for wave V. This prolongation in wave latency was seen at supra-threshold level, probably due to the cochlear synaptopathy which is specific for auditory nerve fibres with low spontaneous rate.

The results of the present study indicated that there was a clear shift in latency especially for wave V at higher intensities. And these findings are in consonance with the literature (Skoe et al., 2019; Ridley et al., 2018). They also reported that there was a delay in ABRs peaks to supra-threshold click stimuli in individuals with high noise exposure in comparison to low-exposure group and this prolongation was evident for all the waves I, III and V but most significant for wave V. In a similar line, study reported a significant shift in ABR Wave-V latency in increasing levels of background noise (Burkard & Hecox, 1987; Burkard & Sims, 2002). Attias and Pratt (1985) evaluated the changes in ABR in individuals exposed to occupational noise >90 dBA with normal hearing thresholds. They recorded ABR using click stimulus at 75 dB HL. They assessed waveform morphology, absolute latencies and interpeak latencies of

wave I, III and V. Results revealed that there was prolongation of waves I, III and V and also inter peak latencies. But few studies in literature do mention about that peak latencies and inter peak latencies were normal in individuals exposed to noise. Attias and colleagues reported that ABR peak latencies were within normal limits when they compared between with and without noise induced hearing loss individuals (Attias et al., 1993). Similar results were found by Almadori and group in year 1998 wherein they recorded ABR for clicks of alternating polarity in individuals with noise induced hearing loss. They assessed waveform morphology, absolute latencies and interpeak latencies of wave I, III and V. The results revealed absolute latencies and inter peak latencies were within normal limits.

Overall it can be concluded that the early cochlear and neural changes are difficult to monitor through conventional audiometry as thresholds might fall within normal limits. Hence, DPOAEs can be used to monitor the status of outer hair cells and its damage due to noise exposure, as it is more sensitive to any subtle changes in OHC's. In this connection, present study highlights the use of tone Burst ABR as an effective tool to identify and monitor early neural changes at the level of brainstem in individuals exposed to occupational noise. So, ABR along with otoacoustic emission might give an insight to early cochlear changes along with neural changes in individuals exposed to occupational noise which helps in early identification, monitoring and preventing issues related to auditory effects of occupational noise.

Chapter 6

Summary and Conclusion

The present study was conducted with the aim to check the effect of cochlear and auditory nerve functioning in industrial workers exposed to occupational noise. The objective of the study was to compare the cochlear and neural findings between the individuals exposed to noise and those who are not exposed to occupational noise on the following audiological tests: Distortion Product Otoacoustic Emission and Tone Burst ABR.

To attain the goal, two groups of participants in the age range of 30-45 years were included. Group I (control group) consists of 11 individuals with normal hearing thresholds who are not exposed to occupational noise and the group II (experimental group) with 10 individuals with normal hearing who are exposed to occupational noise. DPOAE SNR amplitude across frequencies (1000 Hz to 6000 Hz) was compared between the two groups. ABR was recorded using the 4 Tone Burst frequencies (500, 1000, 2000 & 4000 Hz) at four intensities (80, 60, 40 & 30 dB nHL).

The present study reported statistical significant differences between the two groups for DPOAE SNR amplitude, especially at 4000 and 6000 Hz. In group II, the mean of DPOAE SNR at lower and mid frequencies (up to 3000 Hz) was more (better) in comparison to the higher frequencies (above 3000 Hz). This is because frequencies in the range of 4 kHz to 6 kHz which is more vulnerable to damage due to noise exposure. For TB-ABR the latencies were found to be higher for group II in comparison to group I. This prolongation was evident for all the waves I, III and V but most significant for wave V. More noticeable difference was seen for the frequencies between 500-4000 Hz,1000-4000 Hz and 2000-4000 Hz in clinical group. This is

because frequencies in the range of 4 kHz to 6 kHz which is more vulnerable to damages due to noise exposure. Also a more pronounced difference was seen for wave V latency when compared to wave I and wave III latency of TB-ABR at different frequencies. This latency shift was more evident for 4000 Hz at 80 dB nHL in comparison to 60 and 40 dB nHL. This provides support to the hypothesis that noise induced synaptopathy is selective to low SR fibers which is indicated by increase in wave latency at higher compared to lower intensities.

Hence, present study concluded that the early cochlear changes due to noise exposure are seen mainly at the basal region of cochlea then extending to the apical part. Hence to detect these early cochlear changes and neural changes in individuals exposed with occupational noise, a test battery approach comprising of distortion product otoacoustic emissions and Tone Burst ABR to be used to identify, prevent, and monitor pathological changes in the auditory system due to occupational noise exposure.

5.1. Implications of the study

- This study would provide information regarding the diagnostic significance of combination of Tone Burst ABR & DPOAEs in noise induced hidden hearing loss individuals.
- The findings of the current study can be applied to see the effect of noise exposure on functioning of the cochlea in the early stages.
- The outcomes of the current study can be utilized for the possible management strategies in individuals with noise induced hidden hearing loss.

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