Audio-Vestibular findings in Bus drivers

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13AUD017

This Dissertation is submitted as partfullfillment

for the Degree of Master of Science in Audiology

University of Mysore, Mysore

May, 2015

All India Institute of Speech and Hearing, Mysore- 6

Certificate

This is to certify that this dissertation entitled **"Auido- Vestibular findings in Bus drivers"** has been prepared under my supervision and guidance. It is also been certified that this has not been submitted earlier to any other Universities for the award of any other Diploma or Degree.

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Declaration

This dissertation entitled "Audio- Vestibular findings in Bus Drivers" is the result of my own study under the guidance of Dr. Sujeet Kumar Sinha, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, and has not been submitted earlier in any other University for the award of any Diploma or Degree.

Register Number: 13AUD017

May, 2015

Abstract

Aim: This study was designed to evaluate the effect of noise exposure on vestibular system.

Method: Twenty bus drivers with history of noise exposure were the participants of experimental group. All the participants underwent pure tone audiometry, immitance, otoacoustic emissions and vestibular evoked myogenic potentials tests. Twenty participants with normal hearing and no history of noise exposure served as the control group.

Results: Out of 20, 6 participants had hearing loss > 40 dBHL in both ears and only one had asymmetrical hearing loss. Out of 6 participants with hearing loss, two had absent cVEMP and one absent oVEMP and one with reduced amplitude of cVEMP. There was no correlation found between the 4 kHz and vestibular evoked myogenic potentials. There was also no correlation drawn between the duration of driving and vestibular findings. Hence, the results of the present study revealed the effect of noise on cochlear function whereas the vestibular function remained intact in bus drivers.

Conclusion: These findings are consistent with studies which show that individuals who had symmetrical hearing loss, there are no evidence of vestibular symptoms and suggested the possibility of spontaneous recovery by central compensation and hence they have normal responses.

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Chapter 1

Introduction

Noise is an unwanted random signal that bears no useful information. The intensity of the noise is very random in time (Kryter, 1994). The exposure to high levels of noise for a prolonged duration causes damage to the hair cells in the cochlea and results in permanent noise-induced cochlear hearing loss. The main characteristics of noise induced hearing loss are sensorial, irreversible and in most cases, bilateral and symmetrical. Noise induced hearing loss happens majorly in the frequencies of 3, 4 or 6kHz and, it extends to the adjacent frequencies as the noise exposure duration continues, which takes a longer time to be involved. It is the most prevalent in the present work conditions, and one of the major problems found in factories, means of transportation and in social activities (Santos & Junior, 2009).

Exposure to noise causes adverse effects to the ear which are due to unpleasant sensations of loudness, pitch, duration and impulsiveness of noise. It also interferes with auditory communication, sleep, work performance and general behavior. High intensity sounds can cause temporary changes initially and later permanent change in the ear (Karimi, Jafari, Haghshenas 2010). Intense impulse sounds are capable of rupturing tympanic membrane and ossicles may dislocate. Middle ear exhibits non-linear characteristic response to high intensity sound and finally acoustical over stimulation causes traumatic injury to the sensori hair cells and destruction of most of organ of corti in the cochlea, causing noise induced hearing loss. It may present as partial or total hearing loss and its severity depends primarily on intensity of noise exposure and the duration of noise exposure. Besides the hearing loss which is caused by the prolonged noise exposure, the association of noise induced hearing loss with tinnitus is fully

described and it has been shown that tinnitus would be increased with increase of noise induced auditory damage (Azizi, 2010).

Similarly noise exposure can damage the vestibular structures as both cochlea and vestibular structures are in close proximity to each other and have the same mechano transduction properties. There are various anatomical changes that have been reported by many studies. Mc Cabe, Lawrence and Arbor, (1958) reported damage to the saccule due to noise exposure. Ylikoski, (1987) have reported displacement of the ampullary cristae, utricular and saccular maculae in the vestibular system due to noise exposure. These changes in the vestibular system have resulted in vestibular symptoms in individuals exposed to noise. Aantaa, Virolainen and Karskela, (1977) have reported abnormal caloric responses, spontaneous nystagmus and positional nystagmus in these individuals. The prolongation of cVEMP latencies p13, n23 latency, reduced peak to peak amplitude and abnormal amplitude ratio is also been reported in individuals exposed to noise (Wang & Young, 2007; Madappa, 2009).

The profession of driving is challenging in terms of occupation risks and health outcomes. The major factor to study the hearing and vestibular problems of individuals in the profession of driving are the engine sound that is present in the front of the vehicle and loud noise present in the environment and noise created by the people travelling in the buses. They reported the association between the hearing and balance with prolonged exposure to noise. They said that sound energy could somehow bypass the vestibular receptors and act directly on the central nervous system (Menninen, 1980). Many studies have reported noise induced hearing loss in bus drivers, which reported of 32.6% to 55.4% of prevalence. Fernandes et al., (2001) studied pure tone audiometry in bus drivers whose buses had front engine and reported that hearing loss was worse in 49.1% of the right ears and 62.8% of the left ears.

Balance is dependent on the information of signals through vestibular, visual and proprioceptive systems to generate the motor responses to maintain upright posture. The vestibular system comprises of semicircular canals and otolith organs. The semicircular canals help in angular rotation and otolith in linear acceleration. Vestibular evoked myogenic potential is a test to assess the integrity of otolith organs. VEMP consists of cervical and ocular vemp.

Cervical vemp is a muscle potential that is recorded for a high intensity sound. It is a biphasic potential. The Sternocleidomastoid muscle is responsible for the generation of positive peak. It is an inhibitory response that produces p13 and n23 peaks when a loud sound is presented which assess the integrity of the vestibulospinal reflex (Murofushi, Shimizu, Takegoshi & Cheng, 2001).

Ocular vemp is also a muscle potential that assess the vestibular system integrity. The inferior oblique muscle is responsible for the generation of n1, p1, n2 peaks. It is an excitatory response that generates n1, p1, n2 peaks for contra ear stimulation which assess the integrity of the vestibulooccular reflex.

<u>Need of the study</u>

❖ Bus drivers are exposed to high level of noise for 8 hours or more than 8 hours per day. The source of the exposure of noise comes from the engine noise, sometime some defects in engine noise or due to traffic congestion. This daily and prolonged exposure may lead to auditory and non auditory pathological symptoms (Portela & Zannin, 2010). Kumar and Jain (1994) reported that among the various modes of transport, noise levels are greatest in auto-rickshaws (81-96 dBA) followed by trucks (83-90 dBA) and buses (77-92 dBA). Noise levels in cars were appreciably lower (72-80 dBA). These high levels of

noise exposure may cause damage to the auditory/vestibular structures in the bus drivers.

❖ Various studies have reported a decline in audiological characteristics in bus drivers. For example, Durfresne et al, (1987) reported presence of hearing loss in all the truck drivers in whom they carried out audiological evaluations. The hearing loss was mainly centered in mid to high frequencies. Karimi, Nasiri, Kazerooni and Oliaei., (2010) reported that 12.6 % of truck drivers suffer from impaired hearing sense in left and right respectively (hearing threshold level greater than 25 dB) in mid frequencies (500, 1000, 2000 Hz) and 45% in high frequencies of both ears (4000 and 8000 Hz). However, there is a dearth of information regarding the vestibular findings in bus drivers, as it has been reported that the noise can damage the vestibular system also (Shilpashree, 2014).

❖ There is also dearth of information regarding the duration of hours the bus driven and its correlation with audiovestibular findings. Also, there is little information available regarding effects of other associated factor such as tobacco, alcohol etc on audiovestibular findings in these individuals.

Aim of the study

❖ To report the audiovestibular findings in bus drivers.

Objectives of the study

• To report the hearing related problems, if any in bus drivers.

• To find out the cVEMP and oVEMP findings in bus drivers.

• To find the effect of duration of driving on cVEMP and oVEMP findings in bus drivers.

• To correlate the 4k Hz threshold with vestibular findings in bus drivers.

Chapter 2

Literature Review

Hearing is one of the most important senses of human beings. It is one of the ways in which we communicate and interact with the society. There are a multitude of factors that can affect the hearing of an individual. Of the various factors, Noise is one of the major factors which have adverse effect on the auditory system. Based on the physical properties, it is defined as a sound, that is random in nature and spectrum which does not exhibit defined frequency composition (Behar, Chasin & Cheesman, 2000).

Noise has adverse effect on both the auditory and vestibular structures. In the auditory system the various physiological changes that are seen are greater loss of outer hair cell than inner hair cell in the 9mm to 13mm region of the cochlear duct (Mcgill, Harold & Schuknecht, 1976), displacement and detachment of the stereocilium from its rootlet (Tilney, Saunders, Egelman & Rosier, 1982), hair cell damage where in the sensory epithelium of outer hair cells, dieter cells, hensen cell were displaced from the basilar membrane (Hamernik, Turrentine, Roberto, Salvi & Henderson, 1984), loss of spiral ganglion cells and myelinated fibres within osseous lamina (Bohne, Yohman & Gruner, 1987), lesion confined to particular narrow region termed as 'focal region' (Bohne & Clark, 1990). There are reports indicating reorganization of neural activity in the central auditory pathway (Salvi, Saunders, Gratton, Arehole, & Powers, 1990), damage to the spiral ganglion cell whose central processes form the auditory nerve (Nadol & Xu, 1992). Due to noise exposure there is degeneration of central nervous system including the cochlear nuclei, superior olivary complex and inferior colliculus (Kim, Morest and Bohne, 1997).

These changes leads to changes in hearing sensitivity as reflected in pure tone audiometry (Fowler, 1929; Mantysalo, 1984; Hetu, Riverin, Lalande, Getty & Stcyr, 1988; Emmerich, Rudel & Richte, 2008). Other changes which are seen are changes in amplitude of otoacoustic emissions (Reshef, Attias & Furst, 1993; Robinette & Glattke, 2000; Attias, Horovitz, Hatib & Nageris, 2001), elevated auditory brainstem threshold (Attias et al., 1996; Attias, Perez, Freeman, Cokhen & Sohmer, 2002; Santos & Junior, 2009; Kujawa & Liberman, 2009).

Similarly exposure to noise can not only damage cochlea but can also damages vestibular structures as both the end organs are in close proximity to each other and have the same mechano transduction properties of the sensory cells. The various anatomical changes in vestibular system that have been reported are: damage to saccule (Mc Cabe, Lawrence & Arbor,1958), displacement of ampullary cristae, utricular and saccular maculae (Ylikoski, 1987), contusion of the labyrinth (Nageris, Attias & Feinmesser, 2000). Smith, Coffin, Miller and Popper (2006) reported loss of hair bundle density in the central saccule and in the caudal saccule.

These changes in the vestibular system have resulted in vestibular symptoms in individuals exposed to noise. Studies have reported of abnormal caloric responses, spontaneous nystagmus, positional nystagmus in these individuals (Aantaa, Virolainen & Karskela, 1977; Ylikoski, Juntunen, Matikainen, Ylikoski & Ojala, 1988; Wang & Young, 2007). The prolongation of p13, n23 latency, absence of responses, reduced peak to peak amplitude and abnormal amplitude ratio of cVEMP has also been reported (Perez, Freeman, Cohen & Sohmer, 2002; Wang & Young, 2007; Madappa, 2009; Kumar, Vivarthini and Bhat, 2010; Akin et al., 2012).

2.1 Anatomical changes in individuals with NIHL

2.1.1 Inner ear changes

Mcgill et al., (1976) reported the histopathological findings of temporal bone in 14 ears with noise induced hearing loss. They reported inner hair cell changes in the basilar membrane, majorly in the 9 mm to 13 mm region. There was a greater loss of outer hair cells than the inner hair cells. There was a correlation drawn between the permanent hearing loss and the anatomical lesion frequency scale.

Tilney et al., (1982) examined the effect of noise on Alligator lizards. The lizards were broad band noise of 105 dB intensity for duration of 24 hours. After the 24 hours of exposure the pure tone audiometry was carried out which revealed 33 dB of loss in hearing and there was a complete recovery of hearing loss after a span of 11 days. They reported lesions in the actin filament which accounts for the hearing loss. There was depolarization of the actin filament at the base of the stereocilium where it makes contact with the cuticular plate. This results in a detachment and displacement of the stereocilium from its rootlet, which affects the tallest stereocilium orientation.

Hamernik et al., (1984) studied the morphological changes for the exposure of blast waves at 160 dB peak SPL in the organ of corti of the Chinchilla group. A scanning electron microscopy was used for a period of 30 days to follow the development of lesion in the organ of corti. There was a lesion of complete separation of 5-7 mm strip in the sensory epithelium. The sensory epithelium consisted of lesion in the outer hair cells, deiter cells and hensen cells from the reticular lamina and the basilar membrane whereas the inner hair cells survived remarkably in the normal condition in the same area for several days. There observed the ciliary changes on the inner and outer hair cells that differed from most common studies reporting the effect of continuous noise exposure.

Bohne et al., (1987) studied the effect of an octave band of noise with a centre frequency of 4kHz and sound pressure level of 80 or 86 dB SPL in four group of chinchillas. The noise exposure was at interrupted schedules with 18, 42 or 16 hours of rest between successive 6 hour exposures. These ears were compared to that group who received continuous noise exposure which were equal in the energy. The results reported the pattern of cell loss found in ears exposed to continuous and interrupted exposures. However, the ears who received interrupted noise exposure the incidence and the size of lesion in the basal turn was reduced and they also reported that providing 18 hours of rest period is sufficient to provide protection from damage for the basal turn of cochlea.

Initially there was a degeneration of both outer and inner hair cells; however the outer hair cells are more sensitive to noise than the inner hair cells. With the continuous exposure along with hair cells, supporting cells are also damaged. (Bohne & Clark, 1990) 'focal' hair cell lesion is the term to describe the lesion confined to a narrow region. They defined a focal hair cell lesion as a region in which 50% or more of the OHCs and/or IHCs are missing over a distance of at least 0.03 mm. Other authors have termed it as 'cookie-bite' defects.

After the moderate loss IHC, there is loss of spiral ganglion cells and myleinated fibers within the osseous spiral lamina (Bohne et al., 1987). Eventually, there is complete loss of spiral ganglion cells and there is evidence of degeneration of central nervous system correspondingly in the cochlear nuclei, superior olivary complex and inferior colliculus (Bohne et al, 1998).

Salvi et al., (1990) measured evoked response in the group of chinchillas before and after exposure ton noise. The amplitude-level functions were measured from electrodes in the inferior colliculus. The chinchillas were exposed to a 2 kHz pure tone

of 105 dB SPL which produced approximately 20-30 dB of permanent threshold shifts in the region of 2 to 8 kHz and no hearing loss below and above this frequency range. There was a loss of less than 60% of outer hair cells in the region of hearing loss. The amplitude-level functions measured before and after of exposure revealed a loss of sensitivity at 2 kHz; however, the maximum amplitude was often greater than normal. Even though there was no loss in sensitivity at 500 Hz, there was a steeper amplitude-level function and the amplitude of evoked response was substantially larger in amplitude than normal. They reported that this enhancement of amplitude in the evoked response did not appear to originate from the cochlea, but reflected a neural reorganization in the central auditory pathway.

2.1.2   Vestibular system changes

Noise doesn't affect the auditory system only but it also affects the vestibular system. McCabe et al., (1958) studied the effect of noise of 136 dB and 150 dB in two groups of guinea pigs respectively. They examined the histological disturbances in the vestibular labyrinth. They reported that due to high level sounds the saccule was found to be the locus of damage, whereas the utricle and the semicircular canal remained normal.

Oosterveld, Polman and Schoonheyt (1982) exposure to noise does not only damage cochlea but damages the vestibular system. As the end organs of vestibular system and the cochlea have a common evolutionary origin and utilize the same basic principle of mechano-electric transduction with the sensory hair cells.

The effects of noise exposure can be explained physiologically. Both mechanical and acoustic trauma causes contusion of the labyrinth. Mechanical trauma can directly damage the vestibular system while the acoustic trauma damages vestibular system

through the round window of the cochlea (Nageris et al., 2000). Ylikoski (1987) reported the effects of noise exposure on guinea pigs with impulse noise of 1.1 kHz at 158 dBSPL, which resulted in structural damage in the vestibular system mainly in ampullary cristae, utricular and saccular maculae.

The studies have reported that fishes have the ability to regenerate lateral line and inner ear sensory hair cells that have been lost following exposure to ototoxic antibiotics. However, regenerative capabilities following the noise exposure have not been explored in fish. To assess this capacity of regeneration following a noise exposure in fishes a study was conducted by Smith et al., (2006) where Goldfish were exposed to white noise (170 dB) for 48 hours. The fishes were monitored for a period of 8 days after exposure. Using the auditory evoked potential technique, the auditory thresholds were determined and to visualize the hair cell bundles and nuclei, the morphological hair cell damage was analyzed using phalloidin and DAPI labelling. A significant temporary threshold shift (TTS; ranging from 13 to 20 dB) at all frequencies tested (from 0.2-2 kHz) was exhibited by the gold fish following the noise exposure and after 7 days post-exposure, hearing recovered significantly (mean TTS<4 dB) in them. And also the hair bundle density in the central saccule recovered by the end of the experiment (8 days post-exposure) while bundle density the caudal saccule did not return to control levels in this time frame.

The consequences of noise induced damage to the balance system were extensively studied by Hain, 2010 and he reported structures get damaged due to noise and animal studies also support the same.

2.2 Audiological findings in individuals with NIHL

2.2.1 Pure Tone Audiometry

Fowler (1929) conducted conventional pure tone audiometry in NIHL individuals. The pure tone audiometry was conducted from 250 to 8000 Hz for all the participants. The results revealed that exposure to long term noise for over a period of 8-10 years would result in a sensorineural hearing loss wherein the loss starts from high frequency (3-6kHz) region and symmetrical in both the ears. He reported that there was a tendency of threshold shift in 3 to 6 kHz region with more hearing loss at 4 kHz and was termed as 4kHz dip.

Flottorp (1973) examined the high frequency hearing in military noise exposure students. The students group consisted of 228 students in the average age of 20.7 years. In the frequency range of 125 to 12000 Hz, pure tone audiometry was carried out. The sensorineural hearing loss in the frequency range greater than 2kHz was found and this was considered as the effect of exposure to high intensity sounds. 67 participants (29%) exhibited less than 30 dB loss. Deviation of more than 30 dB was exhibited by 41 subjects (21%) called as larger deviation from threshold. Out of 41 cases 34 had unilateral and 16 had bilateral losses were reported.

Fausti, Erickson, Frey, Rappaport and Schechter (1981) compared high frequency audiometry in 36 participants (age between 20 to 29 year old) with histories of steady state or impulsive noise exposure. Mean threshold shift of 20 dB poorer than normal were seen for subjects exposed to steady state noise from 13 to 20 kHz and a symmetrical and smooth configuration were noticed. For the impulsive group, shifts were seen in the 2 to 20 kHz and the configuration of audiogram was jagged or asymmetrical.

Age matched western controls had a lower air conduction threshold above 8 kHz when compared to noise induced hearing loss individuals. Several authors have suggested that noise induced hearing loss can be detected with high frequency audiometry at an early stage (Osterhamrnel, 1979; Dieroff, 1982). Dieroff (1982) examined high frequency hearing in 200 weaving mill workers. They showed hearing threshold levels with a broad dip at about 4 or 6 kHz but with relatively good hearing at about 11 and 12 kHz. The greatest hearing threshold shifts were observed at 15 kHz and above in the same group of workers.

Mantysalo (1984) studied the effects of steady state and impulse noise on hearing. Three groups were exposed to three different condition of noise. The first group was exposed to impulse noise, second group was exposed to continuous steady state noise, and third group served as the control unexposed group. The pure tone audiometry was done to assess the hearing thresholds of the groups three times in two workdays. There were no significant differences in the hearing threshold measured in 3 different settings for all the three groups. Groups which were exposed to impulse noise had the highest threshold shifts in both the ears at 6000 Hz than the groups exposed to continous noise. Groups exposed to impulse noise for a longer duration had asymmetrical threshold shifts, 4khz in the left ear and 6Khz in the right ear. The group exposed to steady state noise continuously, also had asymmetrical threshold shifts at 6000 Hz in the left ear. Hence it was concluded that exposure to impulse noise for a longer duration, there will be wider area of frequency damage and shorter duration impulse noise results in permanent threshold shifts at 4000 and 6000 Hz than continuous steady state noise.

Hetu et al., (1988) reported hearing difficulties among noise-exposed workers. They investigated hearing difficulties by means of an interview and pure tone

audiometry. Of the 100 employees tested in earlier study by Hetu et al., 1987, results were explained to individual subjects and out of it 65 of them participated in the interview. The mean age of the final sample was 39.3 years and the length of occupational exposure to noise, 17.1 years. During the interview 43% of the workers reported of hearing difficulties and 34% of the workers had normal hearing, a significant hearing loss due to noise was found in 49% of them and a significant hearing loss not attributable to the sole effect of noise in 17% was seen.

Ylikoski et al., (1988) examined pure tone audiometry in 60 participants with varying degrees of noise induced hearing loss after exposure to intense impulse noise from firearms for a longer period. These participants who were exposed to noise were compared with 83 normal participants. For the statistical correlations, the thresholds at frequencies 500 Hz and 2k Hz was considered separately for both the ears and correspondingly 4 and 6 kHz were also considered. Audiometric examination revealed elevated thresholds, usually symmetrically in both ears in noise exposed participants.

Morata (1989) has reported noise induced hearing loss in rayon factory workers. The study included two groups. First group consisted of 60 workers who volunteered for testing in the age range of 22 to 53 years. The second group consisted of 205 workers who were randomly selected for testing. The pure tone audiograms of first group showed 71.7% hearing loss with 3.8% attributed to non occupational causes. For second group 66.7% of subjects showed hearing loss with 6.6% of them attributed to non occupational causes. They also reported that the occurrence of hearing loss increases from 46.7% for 2 years of exposure to 70.6% for 3 years exposure. The proportion of occupational hearing losses increased with age of the subject, from 52.3% at 18-29 years to 87.5% in the group of 50-60 years old.

Bartsch, Dieroff and Brueckner, (1989) investigated hearing level in the conventional hearing range and high frequency range. A total of 537 subjects working in a textile industry, at three different noise levels 80-84, 85-89, 90-94 dB(A) were tested. They reported that group of subjects working in the noise level of 90 dBA develop mainly high frequency loss and the conventional range remained unchanged. Noise induced hearing loss in the conventional range was noticed in the group who was exposed to noise above 90 dBA. Hence they concluded that noise level less than 90 dBA would not result in noise induced hearing impairment and considered 85 dBA as the auditory risk criteria.

Hallmo, Borchgrevink and Mair (1995) studied high frequency audiometry in 167 subjects in the age range of 18-59 years with a history of occupational noise exposure. Air conduction and bone conduction thresholds were determined in the ascending method. The results of the study revealed noise induced hearing loss bilaterally in 137 subjects and unilaterally in 30. They also reported that air conduction threshold elevation is seen in 3-6 kHz and throughout high frequency range of 9-18 kHz. The high frequency threshold elevation is in the same order of magnitude for all age groups.

Sułkowski et al. (2002) conducted pure tone audiometry on 61 subjects in the age range of 22- 58 years and with 2- 34 years of work experience in noise. 40 non noise exposed healthy workers were the control group, aged 25- 56 years. The results revealed hearing threshold within normal limits in 57% of subjects of noise exposed groups. Hearing loss of 16- 30 dB was seen in 16 % , loss 0f 31- 40 dB in 9.8%, loss of 41- 50 dB in 8.1% and 51- 60dB of loss in 8.1% of subjects in experimental group.

Chang et al. (2003) examined the simultaneous exposure of noise and carbon disulphide on hearing impairment in 131 participants exposed to noise of 80-90 dBA

and carbon disulphide. This group was compared with 105 participants who were exposed to only noise and one more group of 110 participants who served as the control group. All three groups were tested for hearing using an audiometer. The air conduction thresholds of 0.5, 1, 2, 3, 4, 6 kHz was carried out on all groups after 16 hours of noise exposure. The results revealed hearing loss was greatest for worker who was exposed to both Carbon disulphide and noise. Approximately >25 dBHL of hearing loss was reported in 80% in group one, whereas 32.4% of the group exposed to only noise. In the control group 23.6% of them had >25 dBHL. The impact of Carbon disulphide was greatest for individuals exposed for >20 years. The group exposed to both noise and CS_2 had higher hearing impairment in 0.5 to 2 kHz, the speech frequencies and the noise only group had stronger effect at 4kHz.

Noise related pure tone threshold shift has not been observed only because of occupation noise but also because of music. Emmerich et al., (2008) measured hearing thresholds in 109 professional musicians aged 30-69 years. Hearing loss of 15 dB and more was found in more than 50% of musicians. Higher threshold loss was found among the strings and the brass players. There was a significant hearing loss at 4 and 6 kHz in professional musicians aged older than 60 years than those aged 30-39 years. A dominant hearing deficit was seen for the string players in the left ear. They concluded that noise has more effect on older individuals.

Ciorba et al. (2011) described 460 individuals, the effect of age and noise exposure in the age of 70 years and older. 367 were affected by presbycusis alone and 93 affected by noise and presbycusis. The audiometric procedure was performed using headphones in ascending method using 5dB steps at 125, 250, 500, 1000, 2000, 4000 and 8000Hz frequencies. Bone conduction thresholds were assessed with white noise contralateral masking for 250 to 4000Hz. A slight significant difference between

thresholds was found between the two groups at only 4 kHz. The authors indicated that noise has more effect on individuals with presbycusis.

2.2.2   Oto Acoustic Emissions

The DPOAEs in subjects with NIHL were also investigated by Martin, Ohlms, Franklin, Harris and Lonsbury (1990) reported frequency specific reduction of DPOAE for stimulus frequencies corresponding to hearing impairment. DPOAEs are reduced or eliminated when two tone stimulus frequencies fall within a hearing impairment region, thus providing sensitive and frequency specific information about cochlear dysfunction.

Hotz, Probst, Harris and Hauser (1993) conducted a study on 117 military service men were training was provided for 17 weeks that included exposure to noise from fire arms. TEOAE's were measured pre and post training. Results revealed changes in the response amplitudes in the frequency range from 2 to 4 kHz significantly, whereas no significant changes in the frequency range from 0.5 to 2 kHz for either group. Amplitude was reduced by 84% and 90% in both the right and left ear respectively compared to initial level. They also compared TEOAE results with pure tone threshold and reported that TEOAE is a more sensitive than PTA in detecting early cochlear damage from noise.

Reshef et al., (1993) conducted a study on military personnel in the age range of 18-66 years who are exposed to hazardous military noise (mainly shooting). Click evoked otoacoustic emissions was used in a group of 72 ears with NIHL and 61 ears with normal hearing group less than 25 dB threshold. They reported that 95% of normal hearing individuals had a wide click evoked oto-acoustic emission spectrum while 91.5% of the NIHL individuals had narrow EOAE range.

Attias et al. (1995) examined otoacoustic emissions in 129 military personnel exposed to noise since 5 to 13 years. The 27 were randomly chosen from the new recruits normal having hearing thresholds less than 20 dB. They formed the group A. The experimental group was dived further into B to G group. Group B consists of 66 ears with normal hearing in the mean age of 29.9 years. The others had hearing loss and were divided into 5 groups. Click evoked oto acoustic emission were elicited with the ILO 88 Otodynamic analyser in the non linear mode. The results indicated a broader spectrum and increased emission level in group A when compared to group B even though the threshold difference between them was 5 dB. Thus the noise exposed individual displayed both reduction in the overall click evoked oto-acoustic emissions and spectral range inspite of normal thresholds. In the other hearing loss groups, the beginning of hearing loss frequency decreased, the spectral range of the emissions became correspondingly narrower. In 98.9% of the ears with NIHL emissions were not detected at frequencies were hearing thresholds exceeded 20 dBHL and this trend was marked in frequencies like 2, 3, 4 kHz.

A classical study by Attias, Bersloff, Reshef, Horowitz and Furman (1998) examined the efficacy of screening for NIHL with DPOAE. A total of 76 army personnel consisting of normal to various degrees of audiograms were studied. A group of normal act as control group. DPOAE was recorded with a flat frequency response between 0.5 to 6 kHz. Although very slight differences existed between the audiometric thresholds of the two groups they were not statistically significant at any frequency. In contrast, the DPOAE levels of the exposed ears were significantly reduced in amplitude as compared with non exposed ears at the test frequencies of 1 kHz, 2 kHz, 3 kHz and 4 kHz. They concluded that as the hearing loss increases, the amplitude and frequency

range of DPOAE decreased significantly. At least 25% noise exposed ears had absent OAEs at 1 and 6 kHz.

Attias et al., (2001) used Otoacoustic emissions for detection and clinical diagnosis of Noise induced hearing loss. The study group consisted subjects having normal audiogram in 283 noise exposed participants and 176 participants with a history of noise exposure. The findings were compared with the 310 young participants with normal audiogram and no history of noise exposure. On average, the hearing threshold was better than 25 dBHL and Click evoked oto acoustic emissions were recorded up to 2 kHz. There was a clear association observed between the severity of hearing loss and the OAEs response. As the severity of noise induced hearing loss increased, the emissions range became narrower and the amplitude smaller. Furthermore, OAE testing between ears with and without NIHL revealed a high sensitivity of 79 - 95% and specificity of 84 - 87%. This study shows that OAEs acts as a objective test with greater accuracy and supports the behavioral audiogram in the diagnosis and it also helps in monitoring of the cochlear status after the noise exposure.

Sulkowski et al., (2012) reported the effect of solvent exposure on 61 subjects with the mean age of 22 – 58 years. The subjects were selected based on the questionnaire survey and otolaryngological examinations. The duration of exposure to organic solvents ranged from 2 to 34 years. The noise level in the work environment was less than 85 dBA. The control group consisted of 40 non exposed healthy workers, aged 25 – 56 years. 42 % of workers had high frequency sloping hearing loss and 3% of non exposed workers. This was accompanied by the lower amplitudes of both TEOAE and DPOAE, or the absence if hearing loss exceeded 40-50 dB. They concluded that increase in dose of exposure caused highest threshold and lowered amplitudes of OAEs.

2.2.3   Auditory brainstem responses

Manabe, Kurokawa, Saito and Saito (1995) examined the effects of noise on Electocochleography. The subjects were divided into two groups based on the presence and absence of vertigo. The vertigo group included 39 ears of 20 subjects and the non vertigo group included 26 ears of 16 subjects. The transtympanic electocochleography was performed on all subjects. The results revealed significantly no differences in the hearing level between the two groups and the noise exposure duration. A significant correlation was found between the hearing level at lower frequencies and the SP/AP ratio. And a near significant correlation was observed between the hearing level at 4 kHz and SP/AP ratio. There was a significantly larger SP/AP ratio in the vertigo group than the non vertigo group suggesting the effect of noise on the vestibular structures.

Attias et al., (1996) performed auditory brainstem response in patients suffering from tinnitus due to noise exposure. 13 noise exposed males in the age range of $21 - 45$ years suffering from tinnitus were the experimental and 11 controls without tinnitus but exposed to noise, age and hearing thresholds matched. Both ipsi and contra responses of ABR was recorded. On a grand average both ipsilateral and contralateral ABR waveforms was present in both the groups and peak I, III, V were identified in all groups. Post hoc analysis showed that ipsilaterally recorded wave III was significantly larger for the tinnitus group. Wave III amplitude were normalized by calculating the III-I and V-III amplitude ratios separately. The ipsilateral III-I ratio was significantly larger for the tinnitus group. All other ratio did not differ significantly. The grand averaged ABR power spectrum recorded ipsilaterally and contralaterally was enhanced in tinnitus group when compared to controls but was not statistically significant.

Perez et al., (2002) performed ABR in sand rats after exposing them to 160dBSPL of impulse gunshot noise. The evoked potential was repeatedly measured

after 2 hours, 4 hours, 1 week and 6 weeks after the exposure. The thresholds of ABR were elevated up to 60 dB for a long period. The latency prolonged at the recording of 2 to 4 hours after exposure in comparison to baseline. During the follow up measurements, there noticed a persistent latency prolongation, but in terms of amplitude they showed a partial recovery. They concluded that impulse noise damages the cochlea.

Santos and Junior (2009) studied the brainstem evoked auditory potentials in noise induced hearing loss bus drivers to show the effect of noise on the neuronal involvement.. The study included 50 bus drivers with mild to moderate NIHL in the age range of 27 to 40 years with mild to moderate NIHL and 20 normal individuals in the age range of 29 to 40 years. In the NIHL group, the auditory thresholds were significantly higher in the left ear in 3, 4 and 6 kHz frequencies. In the NIHL group, wave I, III and V were not present in a small number of the individuals. They observed a statistically significant increase in wave I, III and V absolute latencies, I-III interpeak latencies, bilaterally and I-V in the left ear. They concluded that besides sensorial injury, changes in ABRs latency response suggest an early functional injury of the first auditory pathway afferent neuron.

Kujawa and Liberman (2009) studied noise exposure on CBA strain Mice because they show good sensitivity of cochlea and limited age related threshold variations. Male mice were noise exposed at 16 weeks of age and controls were age and gender matched. The acoustic over exposure stimulus was an octave band of noise at 100 dBSPL, for 2 hours. Auditory brainstem responses were recorded via sub dermal needle electrode. Stimulus were 5ms tone pips with a 0.5ms rise-fall time delivers at 30/s. At 24 hour post exposure, 100 dBSPL produced a 40 dB elevation in ABR at high frequencies and lower response in DPOAE, substantially indicating OHC damage and

the neural damage. Indeed, following the exposure there was swelling seen in the peripheral terminals of IHC area of the cochlear nerve fibers. By 2 weeks post exposure the response came back to baseline.

2.3 Vestibular tests findings in individuals with noise induced hearing loss

Aantaa, Virolainen and Karskela (1977) studied noise effects on vestibular system in 49 workers aged 20 to 52 years. They had been exposed to noise and vibration since 6 months to 10 years. The rotatory chair test and caloric tests are done on all the subjects. They reported symptoms of vestibular dysfunction in the form of spontaneous nystagmus 4.1%, lowered caloric excitability or pathology in rotational tests as high as 44.9% in a group of 49 male workers who had been too exposed to extreme noise and vibration between 6 months to 10 years. They also reported the effect of low frequency vibration as the cause for lesion in the peripheral vestibular system.

Oosterveld, Polamn and Schoonheyt (1982) carried out vestibular examination on 29 individuals exposed to noise for more than 5 years in the age range of 22 to 63 years. 10 subjects complained of occasional spells of dizziness and sensations of being off the balance. The vestibular examination included observation for the presence and absence of spontaneous nystagmus and positional nystagmus in four positions i.e, supine, prone, left lateral and right lateral. Results revealed spontaneous nystagmus in 18 subjects, positional nystagmus in 24, cervical nystagmus in 17 and nystagmus preponderance of more than 20% in 7 subjects. All subjects showed pathology in one or more tests but there was no correlation drawn about hearing loss and vestibular dysfunction.

Ylikoski et al., (1988) conducted a study on 60 subjects with varying degree of noise exposure. They were tested for body sway. The force platform technique was used

to measure the body sway in the participants. The participants were given practice trials before the actual testing. Three recordings were performed on each subject: first one with visual control of the deviations both along X and Y axis from the screen, second one with visual control by a fixed spot 2m behind the platform, and third one with eyes closed condition. Each testing 30 s interval was registered after about 30 s of adaptation time. The results showed that NIHL participants exposed to impulse noise of high intensity had more body sway which suggests an exposure-effect relationship. It also suggests the presence of sub clinical vestibular pathology in NIHL participants. They also reported that individuals who had severe NIHL had more severe sway movement.

The vestibular function was assessed in 22 NIHL individuals and 21 age matched controls using electronystagmography and the smooth harmonic acceleration. The results showed symmetrical hearing loss was associated with centrally compensated decrease in the vestibular end organ responses. The ENG recordings revealed spontaneous horizontal nystagmus in 27 % in study group and 9.5% in control group. In the study group 4.5% had positional nystagmus, and 13.7% of them had positioning nystagmus. The slow phase eye velocity for each of the caloric response was indeed lower in the noise induced hearing loss group but did not reach statistical significance. (Shupak et al., 1994).

Manabe et al (1995) studied vestibular functioning in subjects exposed to noise in the occupational setting for more than 5 years. The subjects were divided into two groups based on the presence or absence of vertigo. The vertigo group included 39 ears of 20 subjects and non vertigo group included 26 ears of 16 subjects. Air caloric tests were performed on them. The results revealed reduced caloric responses in 47.1% of ears where a maximum slow phase velocity less than 10^0/s.

Golz et al. (2001) reported vestibular damage in 258 military professionals exposed to intense noise. The subjects were divided into two groups based on hearing; 134 subjects having symmetrical loss and 124 subjects had asymmetrical losses, were the two groups. Further, within the group they were sub divided based on the vestibular complaints. All the subjects underwent complete electronystagmographic evaluation. They reported that subjects who had asymmetrical hearing loss had abnormalities or absence of ENG responses. They also reported a strong correlation between the subjects complaint and results of vestibular function tests. However, the correlation between the severity of hearing loss and vestibular problem did not show any significance. They concluded that subjects exposed to noise will have vestibular symptoms only, if they have asymmetrical hearing loss.

Perez et al., (2002) performed VsEPs on sand rats after exposing them to 160dBSPL of impulse gunshot noise. The evoked potentials were repeatedly measured after 2 hours, 4 hours, 1 week and 6 weeks after the exposure. The results for the recording at 2 to 4 hours post exposure revealed reduction in amplitude of the first wave of VsEPs and prolonged latency in comparison to baseline. The latency prolongation persisted after follow up recordings also, whereas there was a partial recovery noticed for amplitude for the linear acceleration. The first wave of VsEPs in response to angular acceleration was unchanged long term and ABR thresholds were elevated in the long term by 60 dB. From this they concluded that impulse noise not only damages the cochlea, but also causes clear functional impairment mainly in the otolith organs.

Sulkowski et al (2012) conducted a battery of ENG tests which included saccadic, eye- tracking, spontaneous and positional nystagmus, optokinetic test, rotatory and bithermal caloric tests on 61 subjects exposed to noise since 2 to 34 years. The ENG tests revealed the presence of vestibular disorders of mild or advanced degree in

47.5% of workers. The parameters of vestibular oculomotor induced reactions revealed significantly decreased duration, amplitude and slow phase angular velocity of nystagmus in these individuals when compared to normal counterparts.

Wang and Young (2007) investigated the effect of chronic noise exposure on vestibular system. They performed caloric tests on a group of 20 subjects exposed to chronic noise having bilateral notch audiogram. The results revealed abnormal caloric responses in nine (45%) of subjects. The hearing threshold of 4kHz did not correlate with caloric results.

Akin et al., (2012) studied vestibular function tests in 43 participants with a history of noise exposure (military noise exposure) and 14 age-matched controls. The horizontal semicircular canal function was tested using slow harmonic acceleration on a rotatory chair. To assess the functioning of the central vestibular system in NIHL participants, ocular motor and vestibular suppression tests were performed. The results revealed a normal functioning of the horizontal semicircular canal function in NIHL participants in the rotatory chair test. All the participants had a normal phase, gain, and asymmetry during slow harmonic acceleration (SHA). Ocular motor function in all the noise exposed participants was within normal limits and none of the subjects had spontaneous or positional nystagmus. 21(49%) of the 43 noise exposed individuals reported of dizziness and described as vertigo by 8 (35%), 15 (65%) as imbalance, 20 (87%) described it as lightheadedness.

2.3.1   Vestibular Evoked Myogenic Potentials in noise induced hearing loss

Wang, Hsu and Young, (2006) investigated VEMP in 20 patients (29 ears) in the age range of 22 to 67 years with acoustic trauma. The study revealed normal VEMP test results in 18 ears, whereas abnormal responses were obtained in 11 ears (38%). After 3

months of treatment, there was a complete recovery noticed in 4 ears and improvement in hearing was noticed in 4 ears and in 21 ears there was no recovery seen. There was hearing improvement in 8 ears out of 18 ears that had normal VEMP responses. However, in all 11 ears who had absent or delayed VEMPs hearing loss remained unchanged. Hence the results of the study indicated a significant relationship between VEMp response and the hearing outcome. Thus, VEMP test can predict hearing outcome after acute noise exposure with a sensitivity of 44% and a specificity of 100%.

Wang and Young (2007) investigated the effect of chronic noise exposure on VEMP. They performed audiometry and VEMP tests on a group of noise exposed subjects. The noise exposed group had 20 subjects who had bilateral notched audiogram at 4 kHz. The results revealed abnormal VEMP responses in 10 (50%) subjects. The results revealed significant association between the hearing threshold of 4kHz and vestibular evoked myogenic potential response i.e., subjects who had higher degree of hearing loss had abnormal VEMP responses. Hence they concluded that subjects with bilateral 4 kHz notched audiogram and hearing threshold of > 40 dB showed abnormal VEMP response, indicating damage in the vestibular part, the sacculocolic reflex pathway.

Vivarthini, Kaushal and Bhat (2008) studied the effect of noise on vestibular evoked myogenic potentials. A total of 30 subjects were tested with VEMP. Out of 55 ears, VEMP was absent in 16 ears (29%). VEMP was normal in 20 (36.4%), latency prolonged and peak to peak amplitude reduced in 19 (34.4%). Hence VEMP was either absent or prolonged in 67% of individuals which indicating noise causing damage to the sacculocollic pathway.

Madappa (2009) examined the functioning and susceptibility of the saccule in 30 NIHL subjects in the age range of 25 to 50 years. They reported that p13 latency

measure is more sensitive than the n23 latency. They reported abnormal VEMP in 61.4% cases with significant prolongation of p13 and reduced amplitude of p13-n23 complex and absent responses in 38.6% of cases. They reported that TEOAE was absent in most of the clinical group and the most clinical condition was that those who had absent TEOAE had the presence of VEMP responses. They found correlation of VEMP with the presence or absence of vestibular symptoms in more than 50 % of the clinical group. There was a positive correlation between the degree of hearing loss and TEOAE response but there was no correlation between the VEMP response and degree of hearing loss.

Kumar et al., (2010) studied the VEMP in NIHL on 30 subjects with the age range of 30-40 years. They reported of increased pure tone thresholds, prolonged VEMP latencies and peak to peak amplitude was reduced in NIHL subjects. Out of 55 ears, VEMP was absent in 16 ears, latency was prolonged and peak to peak amplitude was reduced in 19 ears. VEMP was found to be normal in 20 ears also. Hence VEMP is abnormal in 67% of NIHL cases and there is a high chance of probability saccular pathway dysfunction in them.

Akin et al., (2012) studied vestibular function tests in 43 participants with a history of noise exposure (military noise exposure) and 14 age-matched controls. cVEMPs were carried out on all individuals. The cVEMPs responses were present at 120 dBpeak SPL in 37 better hearing ears and 29 of the poorer-hearing ears. cVEMPs were absent bilaterally in 6 participants. cVEMPs were absent unilaterally for 11 of the 43 noise exposed participants (26%) and 10 of the 11 participants with the poorer-hearing ear. 29 of the 43 participants with better hearing had normal amplitude ratio indicating normal and symmetrical cVEMPs. 10 of them had 100% amplitude ratio indicating an absent cVEMP in the poorer hearing ear and present response in the better

hearing ear. The results of the test indicated significantly greater signed amplitude ratio in noise exposed group than the control group.

Shilpashree (2014) studied cVEMP and oVEMP in noise induced hearing loss industrial workers. 15 individuals in the age range of 25 – 50 years with greater than 2 years of noise exposure and age matched normal were considered for the study. Results revealed prolonged oVEMP and cVEMP latencies and reduced peak to peak amplitude. They did not find any significant correlation between duration of noise exposure and prolongation of latencies and reduction of amplitude for both cVEMP and oVEMP in NIHL subjects. They also did not find correlation between hearing threshold and cVEMP and oVEMP results.

Emami (2014) studied the effect of Daf music on vestibular system. The control groups included 20 healthy individuals and the experimental group had 18 musicians who had atleast 5 years history with the Daf drum. The subjects were selected based on normal tympanometric results as it could affect the VEMP response. cVEMP was performed on all the participants. The results revealed abnormal cVEMPs in 11 subjects, which showed an absence of both responses in one and prolonged latencies with decreased peak to peak amplitudes in 10 subjects. On comparison of mean latencies of p13, n23 and peak to peak amplitude was significantly affected in the experimental group when compared to control group.

Thus the review of literature VEMP is useful in identifying the pathology of vestibular systems. VEMP is also useful in identifying the involvement of vestibular system in noise exposed individuals. But the different types of noise can have different types of effect on the vestibular system. There is dearth of information in literature regarding vestibular condition of the bus drivers.

Chapter 3

Method

The study was conducted with an aim of studying hearing and vestibular related problems in bus drivers. The study also aimed in correlating the duration of exposure of noise on hearing and vestibular test findings and also to correlate the audiological and vestibular findings in bus drivers.

3.1 Participants

The participants were divided into experimental group and control group. The experimental group consisted of 20 adult participants in the range of 40-60years (bus drivers) and the control group consisted of 20 normal adults in the same age range.

3.1.1 Participant selection criteria

3.1.1.1 Experimental group

1. The study included participants having normal hearing and threshold shifts as it is reported in a number of studies that individuals exposed to noise exhibit threshold shift majorly in the 3-6KHz region. They were 6 individuals with normal hearing and 14 individuals with threshold shifts majorly in the 4kHz region which varied from 30 to 60 dB of loss.

2. Immittance evaluation revealed A/$_{As}$ type tympanogram with ipsi and contra reflexes present/absent/ or elevated depending upon the amount of hearing loss for all the participants.

3. The participants did not have any history of ear pain, ear discharge or any other otological problem.

4. The participants did not have obvious complaints of any vestibular symptoms like vertigo, nausea and vomiting, swaying, imbalance.

5. The participants did not have diabetics, blood pressure.

6. There was evidence of no retrocochlear pathology based on auditory brainstem response in all the participants.

7. None of the participants reported any history of medical and surgically treated ear diseases.

8. None of the participants reported of any uncomfortable loudness level problem.

3.1.1.2 Control group

1. Participants had normal hearing sensitivity (within 15 dBHL) at octave frequencies between 250 Hz and 8000 Hz for air conduction and between 250 Hz and 4000 Hz for bone conduction.

2. The participants had normal A/A$_s$ type tympanogram with ipsi and contra reflexes present.

3. The participants did not report of any otological problem.

4. The participants did not have vestibular symptoms like vertigo, nausea, vomiting, swaying, imbalance.

5. The participants did not have any history of neurological disorder.

6. The participants did not have diabetes and hypertensive disorder.

7. The participants did not report of uncomfortable loudness tolerance problem.

3.2 Instrumentation

1. Calibrated two channel Piano Inventis diagnostic audiometer with Telephonics TDH-39 supra aural headphones housed in MX-41 AR ear cushions and Radioear B-71 bone vibrator was used for pure tone threshold estimation.

2. Calibrated GSI TYMPSTAR Immittance meter with a 226Hz probe tone frequency was used for tympanometry and ipsi-contra reflexometry was carried out using 500Hz, 1000Hz, 2000Hz, 4000Hz reflex eliciting stimulus.

3. Biologic Navigator Pro EP was used for recording of the click evoked auditory brainstem responses and Cervical and ocular vestibular evoked myogenic potentials.

4. Calibrated ILO92 OAE instrument was used for recording click evoked TEOAE.

3.3 Test Environment: All the testing was carried out in an acoustically and electrically shielded room where the levels was within the permissible limits (ANSI S3.1; 1991).

3.4 Procedure

Informed consent was signed from all the participants. A detailed case history for all the participants was carried out about the otological, vestibular and neurological history. Questions related to the type of driving like hilly area or flat area, the duration of driving per day, the type of bus/truck they drive was also asked for the experimental participants.

3.4.1 Pure tone audiometry

Pure tone thresholds were obtained for frequencies 250 to 8000Hz for air conduction and 250 to 4000Hz for bone conduction thresholds using modified Hugson-westlake procedure (Carhart & Jerger, 1959). The mid octave frequency threshold was also determined in case of a difference exceeding 20dB HL between the adjacent octave frequencies.

3.4.2 Immittance evaluation

Tympanometry and reflexometry were carried out for all subjects using 226Hz probe tone to know the status of the middle ear. The participants were seated comfortably and were told not swallow and avoid any head movements during the

testing period. Initially tympanometry was done using 226Hz probe tone at 85dBSPL by varying pressure from +200 to -400daPa. The reflexometry was carried out using same probe tone frequency along with reflex eliciting stimulus of 500, 1000, 2000, 4000 Hz in both ipsi and contralateral conditions.

3.4.3 Transient evoked otoacoustic emissions

The subject was instructed to sit and restrict the body movements. The probe was fit firmly in the ear canal until a good seal was achieved. The otoacoustic emissions were recorded using non linear clicks trains presented at 80dBSPL for both the group of subjects. The probe tip of appropriate size was placed in the ear canal and was adjusted to obtain a flat spectrum. The two averaged TEOAE waveforms of each buffer composed of 260 trains of click stimulus and were automatically cross correlated and used to determine the reproducibility of the measured TEOAEs by the software. An SNR of +6dB were accepted as the response with >80% reproducibility. Care was taken to ensure that the position of probe was not altered during the whole recording.

3.4.4 Auditory brainstem response

ABR was acquired using double channel recording. This was done to rule out any retrocochlear pathology. The subjects were made to sit on a reclining chair and were instructed to relax while the testing is done. The skin surface at the higher forehead, lower forehead and mastoid of both the ears was cleaned using skin abrasive to achieve an impedance of less than 5Kohms. The disc shaped electrodes were placed using conduction paste and surgical plaster for firm attachment.

Auditory brainstem response for site of lesion testing was measured with the repetition rates of 11.1 and 90.1/s at the intensity level of 90dBnHL in rarefaction polarity. The obtained response were filtered between 100- 3000 Hz. Conventional 2 channel electrode montage of non-inverting-high forehead, inverting-mastoid of both

the ears, ground-lower forehead as per International 10/20 system for electrode placement (Jasper, 1958).

3.4.5 Cervical VEMP (CVEMP)

The VEMP was recorded for all the participants in both control and experimental group. The Participants were instructed to sit straight and turn their head to the opposite side of the ear in which stimulus were presented, so as to activate the ipsilateral sternocleidomastoid muscle as it gives reliable and greater amplitude. The participants were instructed to maintain the tonicity of the sternocleidomastoid muscle for the cervical VEMP. A reference point was given and they were instructed to move their head to right or left direction as to keep the muscle in contracted position and to maintain the equal strength. They were also instructed to avoid head, neck and jaw movements during the testing phase. Biologic instrument was used with unrectified EMG recording procedure. The sites of electrode placement were prepared with skin preparing gel, for getting good skin impedance of less than 5Kohms. Absolute electrode impedances and inter electrode impedance should not exceed 5 KΩ and 2 KΩ respectively. The electrode placement were the non inverting on the upper 2/3 rd of the Sternoclediomastoid muscle, inverting on the Sternoclavicular joint and ground on the lower forehead.

An ER-3A insert earphone with an appropriate ear tip size was used for stimulus presentation. The stimulus protocol included a 500 Hz tone burst stimulus of duration of 2-0-2 cycles. The stimulus was presented at an intensity of 125dBSPL with rarefraction polarity at a rate of 5.1/sec. The obtained responses were analysed for 75ms in which pre stimulus recording is for 15ms and post stimulus recording for 60ms were used. The response was band pass filtered between 10 to 1500 Hz and amplified by a factor of 5000. A total of 150 averages were obtained per recording and twice the recording was

repeated to assess for reproducibility. The artifact rejection mode was switched of throughout the recording.

3.4.6 Ocular VEMP

Ocular VEMP was recorded for all the participants in the study for an upper gaze direction. Participants were instructed to maintain the same upper gaze direction throughout the stimulus presentation. The recordings are done in contra lateral ear recording being stimulated. The site of electrode placement was prepared with skin preparing gel, for getting good skin impedance of less than 5Kohms. Absolute electrode impedances and inter electrode impedance should not exceed 5 KΩ and 2 KΩ respectively. The electrode sites for O-VEMP were non-inverting on 1-2 cm under eye on inferior oblique, inverting just below the inferior oblique muscle and ground on lower forehead.

An ER-3A insert earphone with an appropriate ear tip size was used for stimulus presentation. The series of rarefraction 500 Hz tone bursts stimulus is presented at an intensity of 125dBSPL at a rate of 5.1/s at an 2-0-2ms cycle. The response is band pass filtered between 1 to 1500 Hz and amplified by a factor of 5000. The response window was set at 75msec in which the pre recording is 15ms and post recording is 60ms. A total of 150 averages presented twice for a test retest reliability.

3.5 Response analysis

a) The pure tone threshold for both the groups from 250 to 8000Hz for air conduction testing was analyzed.

b) The signal to noise ratio values of Oto-acoustic emissions was analyzed for both the groups.

c) Cervical VEMP
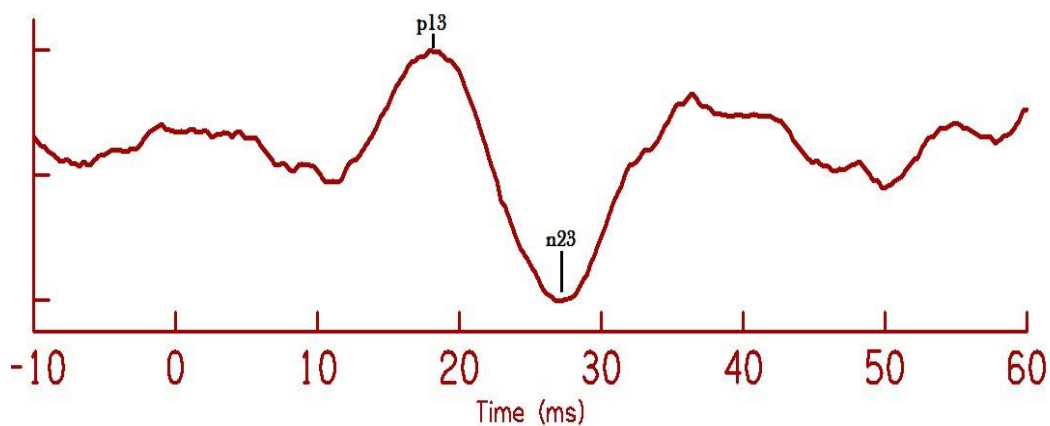
Figure: 3.1 *cVEMP response waveform*

    i)   Latency of p13 and n23 for both the groups was analyzed.

    ii)   Amplitude of p13- n23 amplitude complex was analyzed.

d) Ocular VEMP
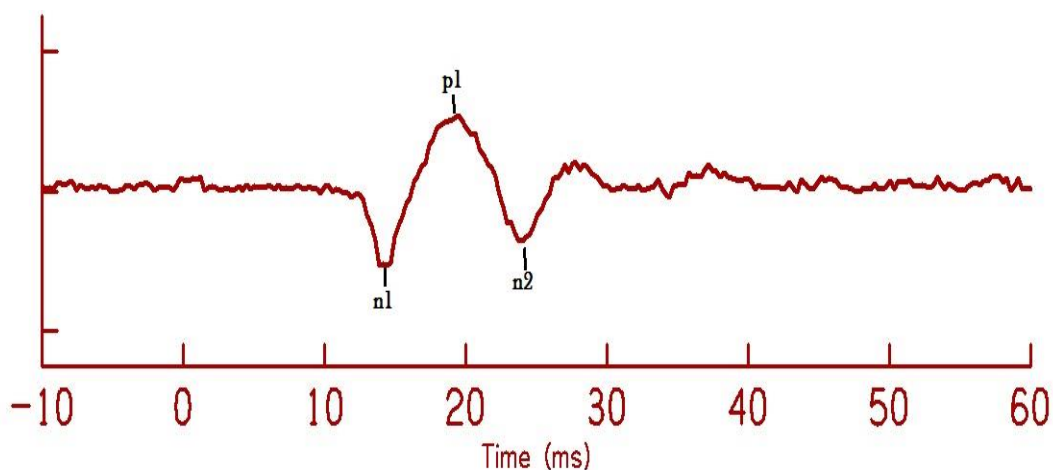

Figure: 3.2 *oVEMP response waveform*

    i)   Latency of n1, p1 and n2 were analyzed.

    ii)   Amplitude of n1-p1 complex and p1-n2 complex were analyzed.

e) Correlation between the effect of duration of driving and the audiovestibular findings was analyzed.

f) Correlation between the hearing thresholds sand the VEMP responses were analyzed.

Chapter 4

Results

The study was conducted with an aim of reporting the audiovestibular findings in bus drivers. To achieve this aim pure tone audiometry, cVEMP and oVEMP tests were administered. The obtained data were subjected to following statistical analysis.

a) Descriptive statistics to find out mean and standard deviation of p13, n23 latency of cVEMP for both the groups.

b) Descriptive statistics to find out mean and standard deviation of amplitude of p13-n23 complex for both the groups.

c) Descriptive statistics to find out mean and standard deviation of n1, p1, n2 latency of oVEMP for both the groups.

d) Descriptive statistics to find out mean and standard deviation of amplitude of n1-p1, p1-n2 complex for both the groups.

e) Kruskal Wallis test to compare the cVEMP latency and amplitude between control and experimental group.

f) Mann- Whitney U test to compare the cVEMP latency and amplitude between control and experimental group.

g) Kruskal Wallis test to compare the oVEMP latency and amplitude between control and experimental group.

h) Mann- Whitney U test to compare the oVEMP latency and amplitude between control and experimental group.

4.1 Audiological findings in control and experimental group

Table.4.1 and 4.2 shows the air conduction thresholds, tympanogram, reflexometry and otoacoustic emissions for right and left ear in control and experimental group respectively.

Table 4.1: *Audiological test results in control group*

Sl.No		PTA(Air conduction thresholds)						Immittance		OAE	
	Ear	250	500	1K	2K	4K	8K	Tymp	Reflexes	TEOAE	DPOAE
C 1	Right	10	10	5	5	10	5	A	+	+	
	Left	5	10	15	10	10	15	A	+	+	
C 2	Right	10	5	10	10	5	5	A	+	+	
	Left	10	5	5	10	10	10	A	+	+	
C 3	Right	10	15	15	20	25	20	A	+	+	
	Left	20	25	15	20	25	20	A	+	+	
C 4	Right	10	15	20	25	30	15	A	+	+	
	Left	15	20	15	25	25	15	A	+	-	-
C 5	Right	15	10	10	5	10	10	A_s	+	+	
	Left	10	5	15	10	10	5	A	+	-	+
C 6	Right	20	20	10	10	5	10	A	+	+	
	Left	20	20	15	5	10	15	A	+	+	
C 7	Right	10	20	20	25	20	10	A	+	-	+
	Left	20	10	20	20	20	15	A	+	-	+
C 8	Right	10	15	15	20	15	20	A	+	-	+
	Left	15	20	15	15	15	15	A	+	-	+
C 9	Right	15	10	15	10	15	10	A	+	-	-
	Left	15	15	10	5	10	10	A	+	+	
C 10	Right	10	15	20	15	15	30	A_s	+	+	
	Left	15	10	20	20	25	25	A_s	+	+	

C 11	Right	15	20	15	15	15	10	A	+	-	+
	Left	20	15	15	20	15	15	A	+	-	-
C 12	Right	15	20	20	25	20	20	A	+	+	
	Left	15	20	20	15	15	20	A	+	+	-
C 13	Right	20	15	15	15	30	20	A_s	+	-	+
	Left	10	15	10	15	25	15	A	+	+	
C 14	Right	15	10	20	25	20	20	A	+	-	+
	Left	10	15	20	30	25	20	A	+	-	+
C 15	Right	15	20	15	15	10	15	A_s	+	-	+
	Left	15	10	15	10	10	10	A_s	+	-	+
C 16	Right	20	15	15	20	20	30	A	+	-	-
	Left	20	15	15	20	20	30	A_s	+	-	-
C 17	Right	5	15	10	15	20	20	A	+	-	+
	Left	15	15	10	15	20	15	A	+	-	+
C 18	Right	20	20	15	20	15	20	A	+	-	+
	Left	15	20	20	20	15	20	A	+	-	+
C 19	Right	10	20	15	10	15	15	A_s	+	-	+
	Left	15	20	15	10	15	10	A	+	-	+
C 20	Right	20	15	20	10	15	10	A	+	-	-
	Left	20	10	10	10	15	15	A	+	-	-

* + indicates response present

- indicates response absent

Table 4.2: *Audiological test results in experimental group*

Sl.No	Ear	PTA(Air conduction thresholds)						Immittance		OAE	
		250	500	1K	2K	4K	8K	Tymp	Reflexes	TEOAE	DPOAE
E 1	Right	10	15	15	10	10	5	A_s	+	+	
	Left	10	15	15	10	15	5	A_s	+	+	
E 2	Right	20	15	10	10	30	10	A_s	+	+	
	Left	15	20	15	15	20	15	A_s	+	+	
E 3	Right	20	15	15	10	35	10	A_s	+	+	
	Left	10	10	10	10	35	10	A_s	+	+	
E 4	Right	20	20	15	15	45	20	A	+	+	
	Left	25	20	20	15	50	35	A	+	+	
E 5	Right	15	15	15	15	30	15	A	+	+	
	Left	15	15	20	15	30	25	A	+	+	
E 6	Right	10	5	5	5	10	15	A_d	+	+	
	Left	10	5	5	5	10	15	A	+	+	
E 7	Right	20	15	20	30	35	20	A	+	-	-
	Left	10	10	20	25	40	25	A	+	-	-
E 8	Right	20	15	15	10	30	15	A	+	+	
	Left	15	15	15	25	50	25	A	+	-	-
E 9	Right	20	10	20	10	30	10	A	+	+	
	Left	15	15	25	15	25	10	A	+	+	
E 10	Right	10	20	20	20	20	5	A	+	-	+
	Left	10	20	15	20	30	10	A	+	-	+

E 11	Right	10	15	15	15	35	45	A	+	+	
	Left	10	20	10	20	40	55	A	+	-	-
E 12	Right	15	10	15	15	10	5	A	+	+	
	Left	25	20	15	20	20	20	A	+	-	-
E 13	Right	15	15	10	20	40	15	A	+	+	
	Left	20	15	10	20	50	10	A	+	+	
E 14	Right	20	15	20	20	20	10	A	+	-	+
	Left	20	20	20	20	15	5	A	+	-	+
E 15	Right	25	20	20	20	40	15	A	+	+	
	Left	30	25	25	35	45	45	A	+	-	-
E 16	Right	15	20	15	35	40	15	A	+	-	-
	Left	15	20	15	20	50	10	A	+	-	+
E 17	Right	15	15	15	30	50	15	A	+	-	-
	Left	20	15	15	20	60	10	A	+	-	-
E 18	Right	10	25	30	35	35	10	A	+	-	+
	Left	15	25	25	50	30	10	A	+	-	+
E 19	Right	10	5	5	10	15	10	A	+	-	+
	Left	10	5	10	10	20	15	A	+	-	-
E 20	Right	20	25	25	15	40	30	A	+	-	+
	Left	20	15	30	20	30	30	A	+	-	+

* + indicates response present

  - indicates response absent

4.2 cVEMP results in control and experimental group

cVEMP was recorded for 20 participants each in control and experimental group. Out of 20 participants in control group cVEMP was present in 18 participants, whereas cVEMP was present in 17 participants in experimental group.
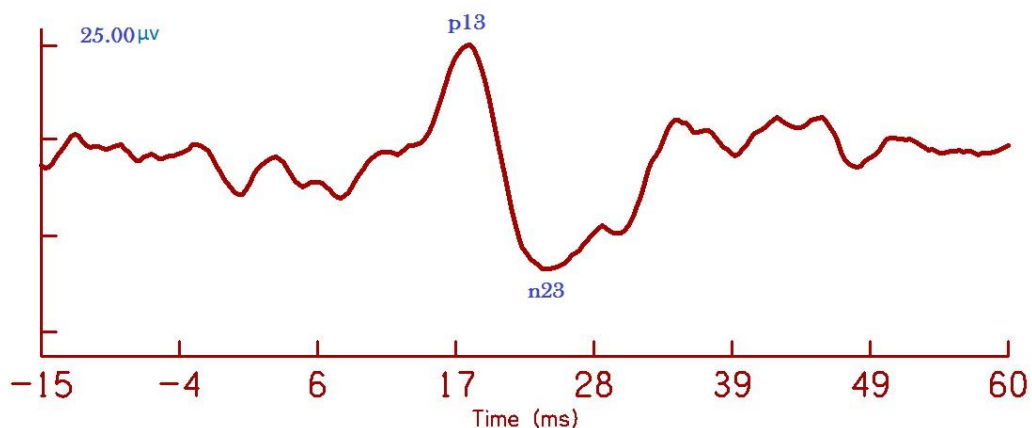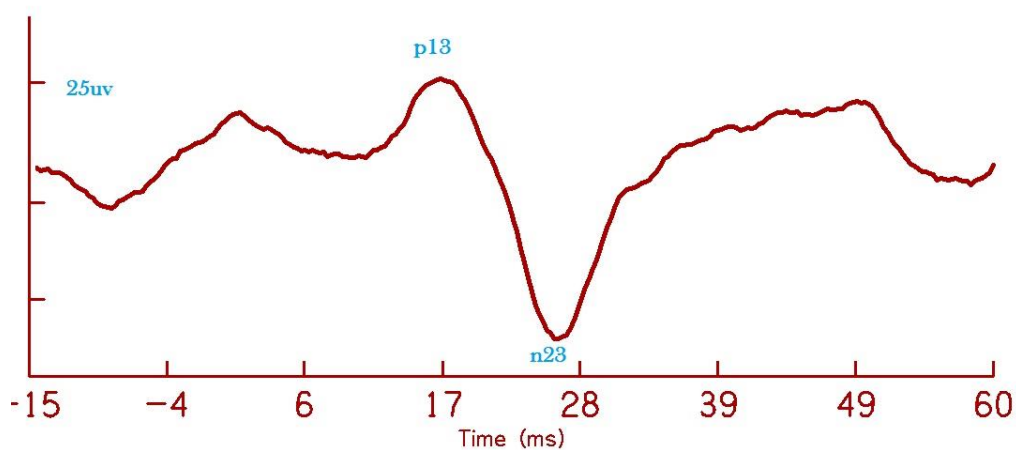

Figure 4:1 *cVEMP response of the control group*

Figure 4:2. *cVEMP response of the experimental group*

Figure 4:1 and 4.2 shows the cVEMP response of the control group and experimental group respectively.

Descriptive statistics was done to find out the mean and standard deviation of latency of p13, n23 and amplitude of p13-n23 complex of cVEMP for control and

experimental group. Table.3 and 4 shows mean and standard deviation of latency of p13, n23 and amplitude of p13-n23 complex for the control group and experimental group respectively.

Table 4.3: *Mean and standard deviation of latency and amplitude of cVEMP in control group*

			N	Mean	Std Deviation
p13	Latency (msec)	Right	18	16.54	1.83
		Left	18	17.61	2.30
n23	Latency (msec)	Right	18	24.01	2.16
		Left	18	24.89	2.45
p13-n23	Amplitude(µV)	Right	18	56.93	48.16
		Left	18	60.30	35.81
Asymmetry ratio (%)			18	26.11	22.39

It can be seen from Table 4.3 that mean latency of p13 and n23 peak is longer for the left ear compared to the right ear in control. Also the amplitude of p13- n23 complex is higher for left ear compared to the right ear.

Table 4.4: *Mean and standard deviation of latency and amplitude of cVEMP in experimental group*

			N	Mean	Std Deviation
p13	Latency (msec)	Right	17	16.49	1.19
		Left	17	18.00	2.71
n23	Latency (msec)	Right	17	24.01	1.65
		Left	17	24.60	2.55
p13-n23	Amplitude(µV)	Right	17	60.25	49.63
		Left	17	63.36	44.36
Asymmetry ratio (%)			17	21.08	27.55

It can be seen from Table.4.4 that mean latency of p13 and n23 peak is longer for the left ear compared to the right ear in the experimental group. Also, the amplitude of p13- n23 complex is higher for the left ear compared to right ear.

On comparing the mean latency of p13, n23 between the two groups, mean latency of p13 peak for right ear is almost same for both the groups. Mean latency of p13 peak for left ear is prolonged for experimental group compared to control group. It can also be seen that mean latency of n23 peak for right ear is same for both the groups, whereas latency of n23 peak for left ear in experimental group is more compared to control group. Also the mean asymmetry ratio is higher in control group than the experimental group. The same can be seen in graph 4.1 and graph 4.2.
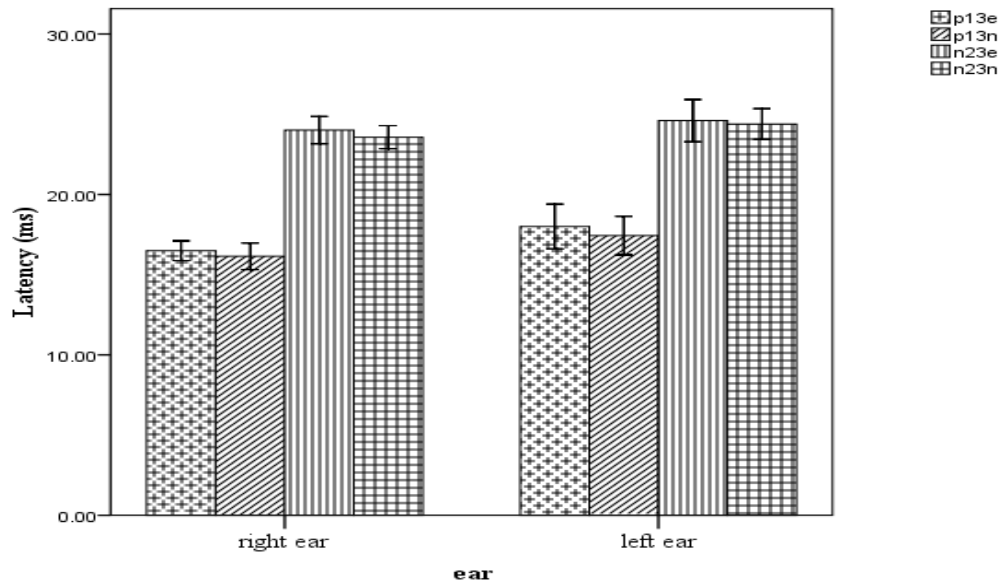
Graph 4.1: *p13, n23 latency of right and left ear for control and experimental group*
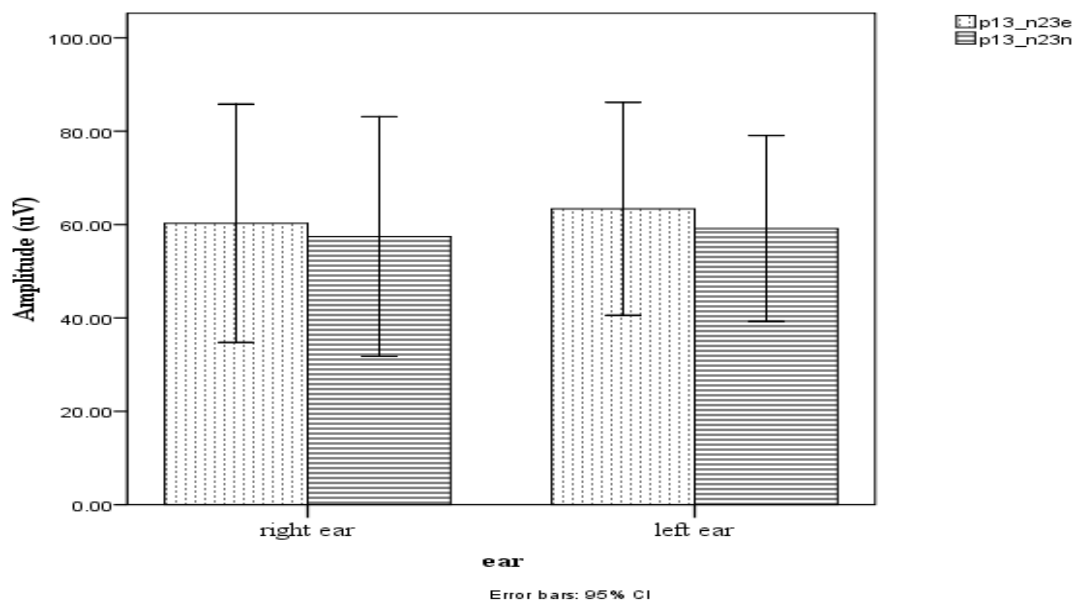
Graph 4.2*: p13- n23 amplitude of right and left ear for control and experimental group*

The data was subjected further to the test the normality. The results of the tests of normality are given in Table 4.5.

Table 4.5: *Normality test results for cVEMP in control and experimental group*

Parameters	Group	Kolmogorov –Smirnov			Shapiro-Wilk		
		Statistic	Df	Sig.	Statistic	Df	Sig.
p13 Right	Control	0.119	18	0.200	0.944	18	0.333
	Experimental	0.161	17	0.200	0.962	17	0.674
p13 Left	Control	0.211	18	0.033	0.906	18	0.72
	Experimental	0.212	17	0.041	0.835	17	0.006
n23 Right	Control	0.151	18	0.200	0.864	18	0.014
	Experimental	0.102	17	0.200	0.981	17	0.966
n23 Left	Control	0.163	18	0.200	0.898	18	0.054
	Experimental	0.210	17	0.044	0.855	17	0.013
p13-n23 Right	Control	0.209	18	0.036	0.798	18	0.01
	Experimental	0.190	17	0.103	0.826	17	0.005
p13-n23 Left	Control	0.180	18	0.126	0.940	18	0.292
	Experimental	0.238	17	0.011	0.806	17	0.002

It can be seen from Table 4.5 that some of the data falls under normality and some does not fall under the normality curve. Hence a non parametric test was done to compare the data between the two groups.

Kruskal Wallis test was done for comparison of latency and amplitude of cVEMP measures between the control and the experimental group. Kruskal Wallis test revealed statistically no significant differences between the experimental and control group latencies of p13 of right ear [$\chi^2 (1) = 0.004$, p> 0.05], p13 of left ear [$\chi^2 (1) = 0.132$, p> 0.05], n23 of right ear [$\chi^2 (1) = 0.145$, p>0.05], n23 of left ear [$\chi^2 (1) = 0.185$, p>0.05], amplitude of p13- n23 of right ear [$\chi^2 (1) = 0.184$, p>0.05], p13-n23 of left ear

$[\chi^2 (1) = 0.10,\ p>0.05]$, amplitude ratio $[\chi^2(1)=2.833,\ p>0.05]$. Thus Kruskal Wallis result shows no significant differences between the two groups.

Mann – Whitney U test was done to compare latency and amplitude of cVEMP between the two groups. Mann – Whitney U test revealed statistically no significant differences between the two groups. The latency of p13 of right ear $[Z = 0.06,\ p>0.05]$, p13 of left ear $[Z= 0.363,\ p>0.05]$, n23 of right ear $[Z = 0.380,\ p>0.05]$, n23 of left ear $[Z = 0.43,\ p>0.05]$, amplitude of p13-n23 of right ear $[Z = 0.42,\ p>0.05]$, p13-n23 of left ear $[Z = 0.09,\ p>0.05]$, amplitude ratio $[Z= 1.68,\ p>0.05]$. Thus the result of Mann Whitney U test indicates no significant differences between the two groups.

Thus the results suggest that cVEMP parameters were same for both the groups.

4.3 oVEMP results

oVEMP was recorded for 20 participants each in control and experimental group. Out of 20 participants in control group oVEMP was present in 17 participants, whereas oVEMP was present in 17 participants in experimental group.
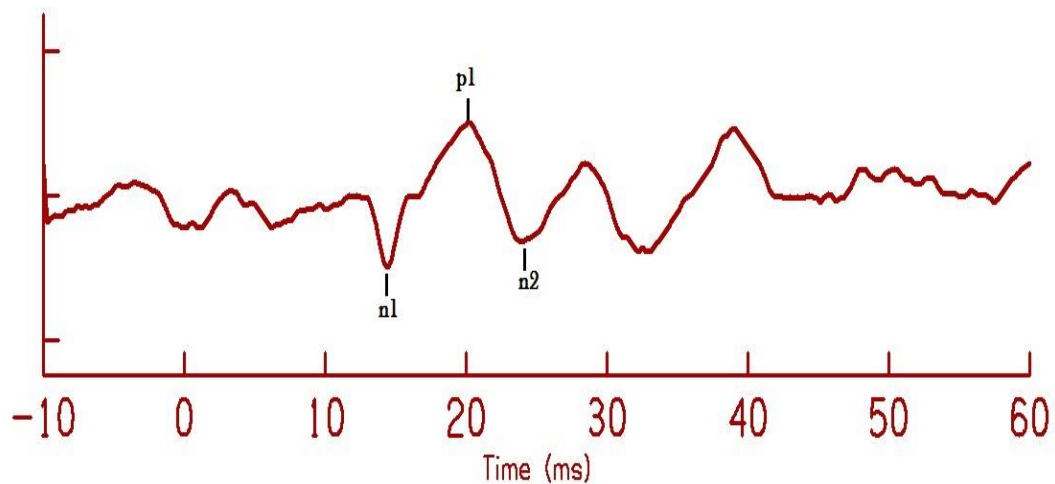
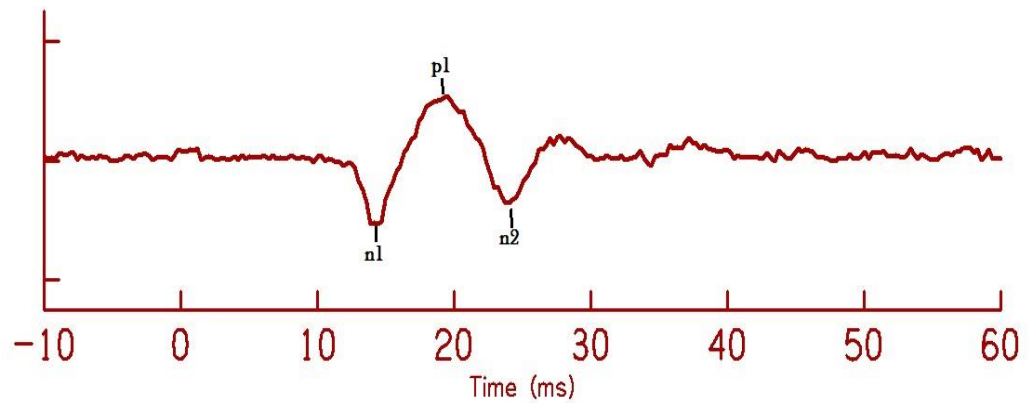Figure 4:3 *oVEMP response of control group*

Figure 4:4 *oVEMP response of experimental group*

Figure 4:3 and 4:4 shows oVEMP response of the experimental and control group respectively.

Descriptive statistics was done to find out the mean and standard deviation of latency n1, p1, n2 and amplitude of n1-p1, p1-n2 complex of oVEMP in control and experimental group. Table.6 and 7 shows mean and standard deviation of latency n1, p1, n2 and amplitude of n1-p1, p1-n2 complex of oVEMP for the control group and experimental group respectively.

Table.4.6: *Mean and standard deviation of latency and amplitude of control group*

		N	Mean	Std Deviation
n1 latency (msec)	Right	17	11.46	0.84
	Left	17	11.52	0.78
p1 Latency (msec)	Right	17	16.71	1.05
	Left	17	16.90	1.31
n2 Latency (msec)	Right	17	21.28	1.25
	Left	17	21.64	1.39
n1-p1 Amplitude(μV)	Right	17	4.02	2.89
	Left	17	3.00	1.60
p1-n2 amplitude (μV)	Right	17	3.71	2.43
	Left	17	3.45	2.02
Asymmetry ratio (%)	n1-p1	16	30.92	22.71
	p1-n2	17	27.52	19.80

It can be seen from Table 4.6 that mean latency of n1, p1, n2 peak is longer for the left ear compared to the right ear in control. Also the amplitude of n1-p1 and p1-n2 complex is higher for right ear compared to the left ear.

Table 4.7: *Mean and standard deviation of latency and amplitude of experimental group*

		N	Mean	Std Deviation
n1 latency (msec)	Right	17	11.36	0.84
	Left	17	10.80	0.78
p1 Latency (msec)	Right	17	16.43	1.05
	Left	17	16.12	1.31
n2 Latency (msec)	Right	17	21.14	1.25
	Left	17	20.89	1.39
n1-p1 amplitude (µV)	Right	17	6.38	2.89
	Left	17	6.41	1.60
p1-n2 amplitude (µV)	Right	17	6.67	2.43
	Left	17	5.10	2.02
Assymetry ratio (%)	n1-p1	17	26.45	24.26
	p1-n2	17	25.35	18.36

It can be seen from Table 4.7 that the mean latency of n1, p1 and n2 peak is longer for the right ear compared to the left ear in the experimental group. Also, the amplitude of n1-p1 and p1-n2 complex is higher in the right ear than the left ear.

On comparing the mean latency of n1, p1 and n2 peak between the two groups, mean latency of n1, p1, n2 peak for right and left ear is longer for experimental group compared to control group. The mean amplitude of n1-p1, p1-n2 complex for right and left in experimental group is higher than the control group. Also the asymmetry ratio of n1-p1 and p1-n2 is higher in control group than the experimental group. The same can be seen in graph 4.3 and graph 4.4.
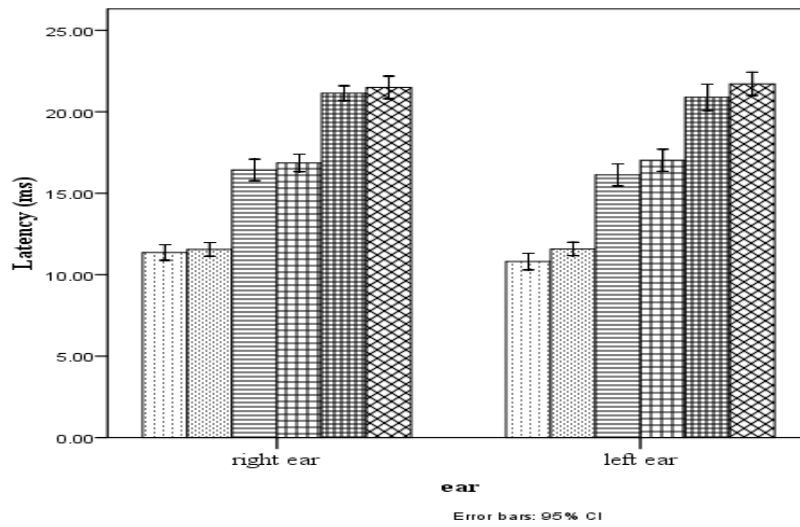
Graph 4.3: *n1, p1, n2 latency of right and left ear for control and experimental group.*
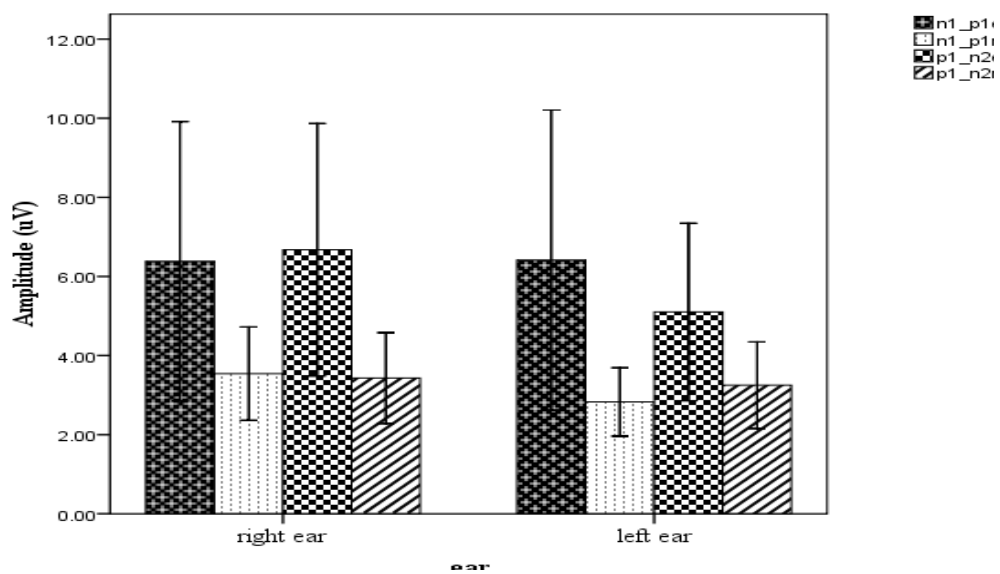
Graph 4.4: *n1-p1, p1-n2 amplitude complex of right and left ear for control and experimental group.*

The data was subjected further to the test of normality. The results of the tests of normality are given in Table 4.8.

Table 4.8: *Normality test results for oVEMP in control and experimental group*

Parameter	Group	Kolmogorov- Smirnov			Sharpiro Wilk		
		Statistic	Df	Sig.	Statistic	df	Sig.
n1 Right	Control	0.194	17	0.088	0.950	17	0.450
	Experimental	0.254	17	0.005	0.870	17	0.022
n1 Left	Control	0.139	17	0.200	0.887	17	0.042
	Experimental	0.206	17	0.054	0.902	17	0.075
p1 Right	Control	0.183	17	0.135	0.913	17	0.113
	Experimental	0.144	17	0.200	0.917	17	0.132
p1 Left	Control	0.140	17	0.200	0.957	17	0.579
	Experimental	0.200	17	0.068	0.927	17	0.191
n2 Right	Control	0.128	17	0.200	0.963	17	0.692
	Experimental	0.133	17	0.200	0.916	17	0.127
n2 Left	Control	0.156	17	0.200	0.962	17	0.673
	Experimental	0.121	17	0.200	0.965	17	0.719
n1-p1 Right	Control	0.280	17	0.001	0.742	17	0.000
	Experimental	0.186	17	0.122	0.907	17	0.088
n1-p1 Left	Control	0.290	17	0.001	0.784	17	0.001
	Experimental	0.163	17	0.200	0.922	17	0.159
p1-n2 Right	Control	0.262	17	0.003	0.817	17	0.003
	Experimental	0.098	17	0.200	0.970	17	0.820
p1-n2 Left	Control	0.304	17	0.000	0.790	17	0.001
	Experimental	0.113	17	0.200	0.951	17	0.479

It can be seen from Table 4.8 that some of the data falls under normality and some does not fall under the normality curve. Hence a non parametric test was done to compare the data between the two groups.

Kruskal Wallis test was done for comparison of latency and amplitude of oVEMP measures between the control and experimental group. Kruskal Wallis test revealed statistically no significant differences between the latencies of n1 of right ear [$\chi^2(1)= 0.37$, p>0.05] , n1 of left ear [$\chi^2(1)= 6.81$, p>0.05], p1 of right ear [$\chi^2(1)= 0.72$, p>0.05], p1 of left ear [$\chi^2(1)= 2.41$, p>0.05], n2 of right ear [$\chi^2(1)= 0.07$, p>0.05], n2 of left ear [$\chi^2(1)= 2.05$, p>0.05], amplitude of n1-p1 of right ear [$\chi^2(1)= 0.21$, p>0.05], n1-p1 of left ear [$\chi^2(1)= 0.18$, p>0.05], p1-n2 of right ear [$\chi^2(1)= 1.29$, p>0.05], p1-n2 of left ear [$\chi^2(1)=0.34$, p>0.05], asymmetry ratio of amplitude complex n1-p1 [$\chi^2(1)=0.57$, p>0.05], p1-n2 [$\chi^2(1)=0.18$, p>0.05]. Thus the results revealed no significant differences between the two groups.

Mann- Whitney U test was done to compare latency and amplitude of oVEMP measures between the control and experimental group. Mann- Whitney U test revealed statistically no significant differences between the latencies of n1 of right ear [Z=0.191, p>0.05] , n1 of left ear [Z= 2.61, p>0.05], p1 of right ear [Z=0.84, p>0.05], p1 of left ear [Z=1.55, p>0.05], n2 of right ear [Z=0.08, p>0.05], n2 of left ear [Z=1.43, p>0.05], amplitude of n1-p1 of right ear [Z= 0.46, p>0.05], n1-p1 of left ear [Z=0.43, p>0.05], p1-n2 of right ear [Z=1.13, p>0.05], p1-n2 of left ear [Z=0.58, p>0.05], asymmetry ratio of amplitude complex n1-p1 [Z=0.75, p>0.05], p1-n2 [Z=0.43, p>0.05].

Thus the results suggest that oVEMP parameters were same for both the groups.

4.4 Correlation of the effect of duration of driving on audiovestibular findings

4.4.1 cVEMP latency and amplitude correlation with duration of driving

To understand the correlation between the duration of driving on cVEMP latency and amplitude Spearman Correlation analysis was done. Also to understand the correlation between the two variables a scatter plot was plotted which is shown in figure-4:5.
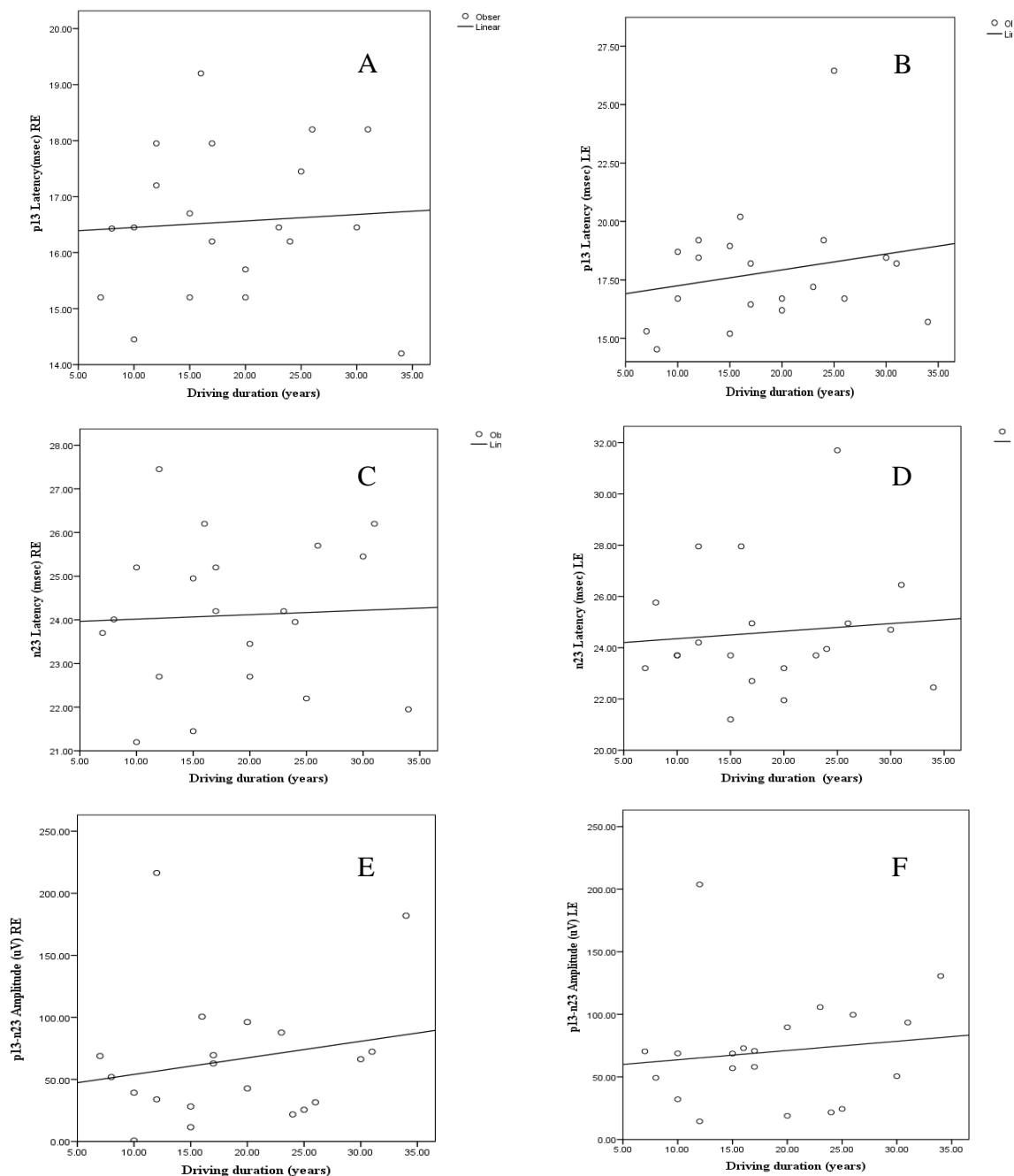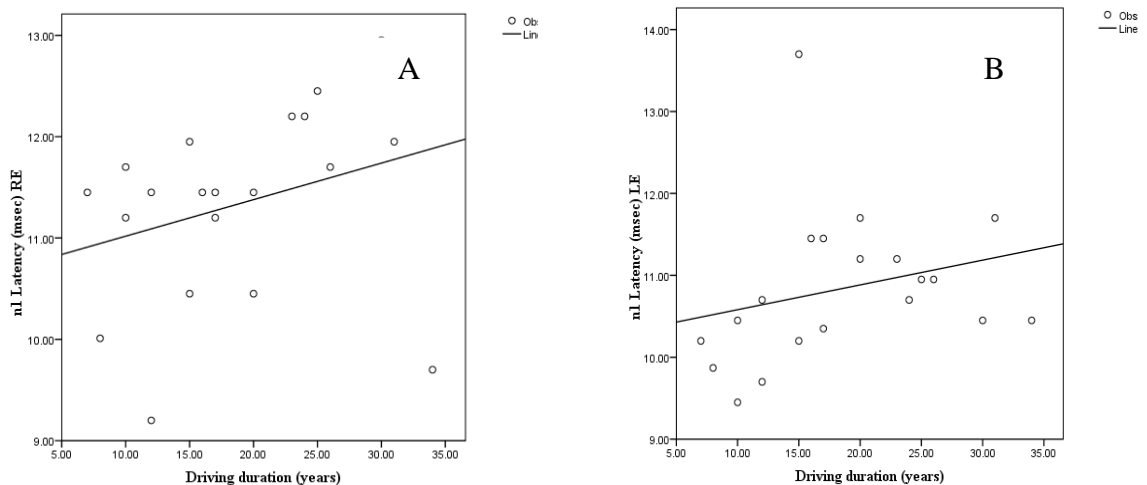

Figure 4:5. *A, B, C, D, E, F is the scatter plots for the correlation between cVEMP parameters and duration of driving*
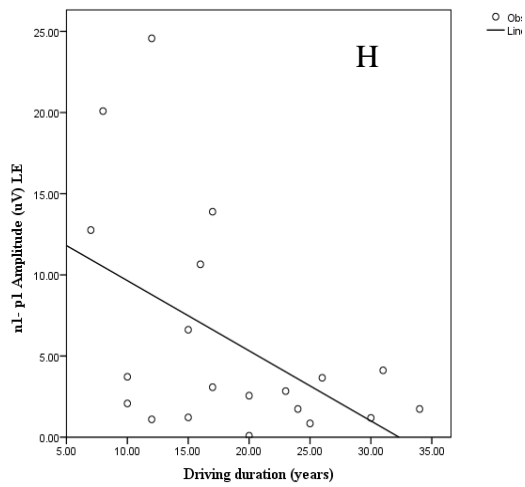
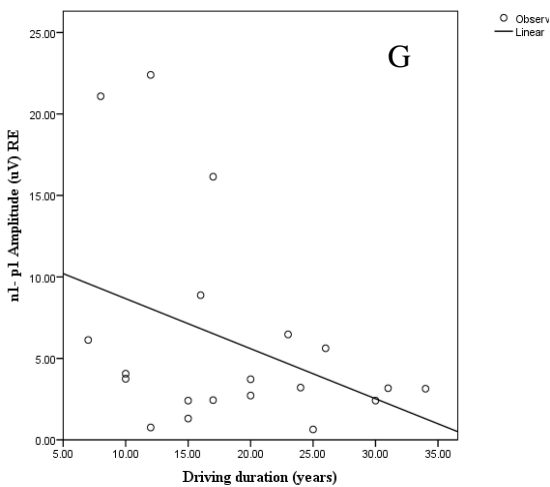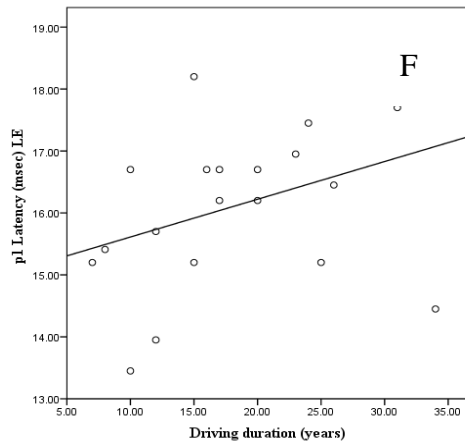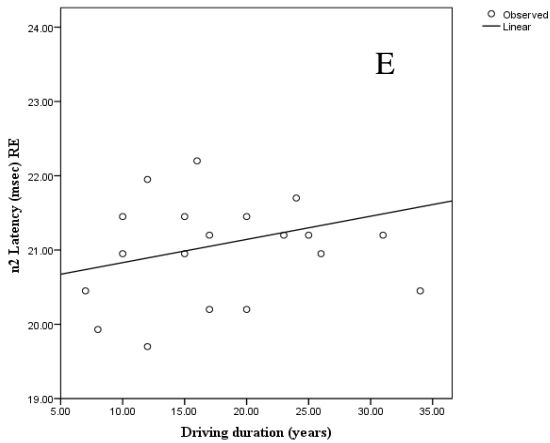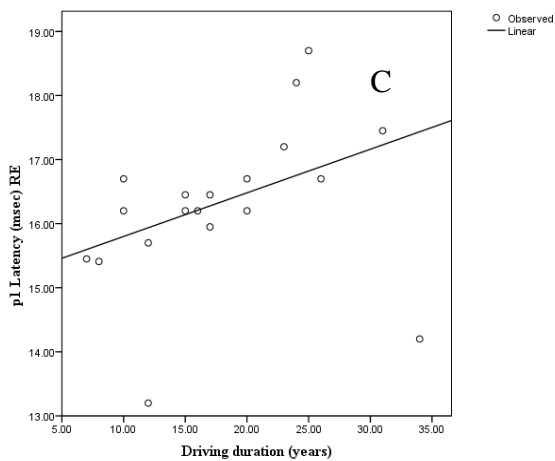It can see from the above scatter plots, as the duration of exposure increases there is prolongation of the p13, n23 latencies in both the ears. Also the amplitude complex of p13-n23 is increasing with the duration of exposure. These findings are present consistently but it is not statistically significant.

Spearman correlation test revealed statistically no significant correlation between the duration of driving on p13 latency of right ear [r= 0.159, p>0.05], p13 latency of left ear [r=0.156, p>0.05], n23 latency of right ear [r= 0.112, p>0.05], n23 latency of left ear [r= 0.080, p>0.05], on amplitude of p13-n23 complex of right ear [r= 0.190, p>0.05], p13-n23 complex of left ear [r= 0.234, p>0.05] in the experimental group.

4.4.2 oVEMP latency and amplitude correlation with the duration of driving.

To understand the correlation between the duration of driving on oVEMP latency and amplitude Spearman Correlation analysis was done. Also to understand the correlation between the two variables a scatter plot was plotted which is shown in figure-4:6.
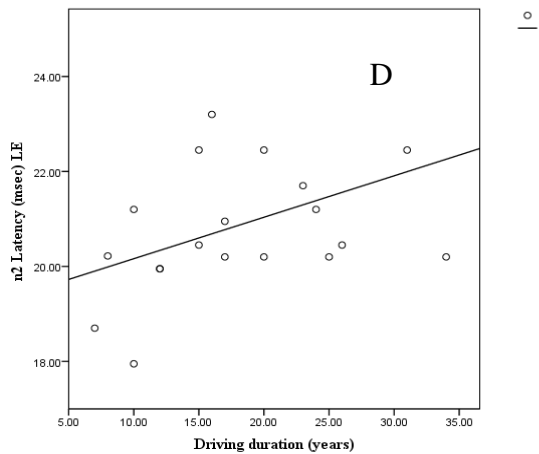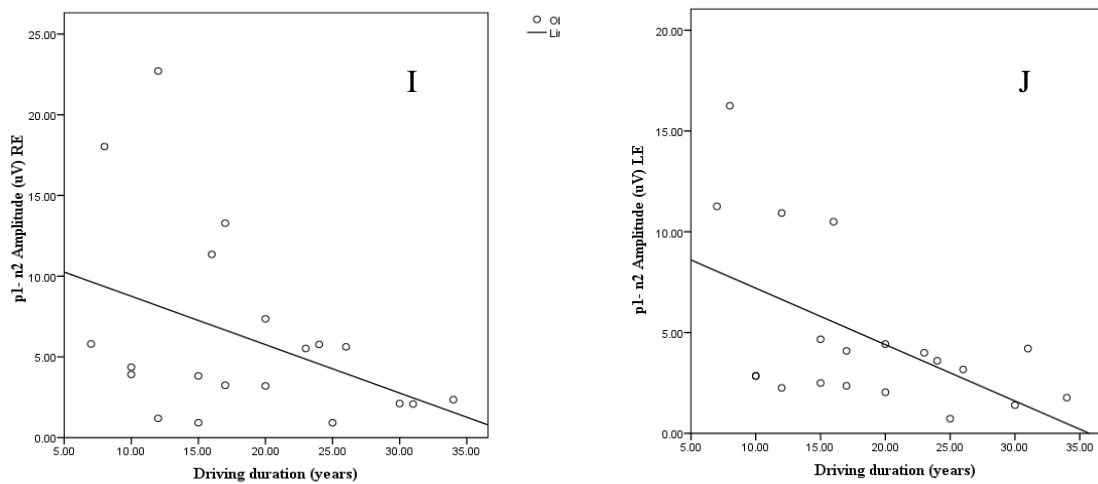
Figure 4:6. *A, B, C, D, E, F, G, H, I, J are the scatter plots for the correlation between the oVEMP parameters and the duration of driving*

From the above scatter plots we can infer that as the duration of exposure is increasing the latency of n1, p1, n2 prolonged and the amplitude complex of n1-p1, p1-n2 reduced but the results are not statistically significant.

Spearman correlation test revealed statistically no significant differences between the duration of driving on oVEMP latency and amplitude. Statistically no significant correlation between the duration of driving and n1 latency of right ear [r= 0.413, p>0.05], n1 latency of left ear [r=0.477, p>0.05], p1 latency of right ear [r= 0.553, p>0.05], p1 latency of left ear [r= 0.383, p>0.05], n2 latency of right ear [r= 0.222, p>0.05] , n2 latency of left ear [r= 0.421, p>0.05], on amplitude of n1- p1 complex of right ear [r= 0.327, p>0.05], n1-p1 complex of left ear [r= 0.430, p>0.05], p1-n2 complex of right ear [r= 0.378, p>0.05], p1-n2 complex of left ear [r= 0.477, p>0.05] in the experimental group.

4.4.3 Correlation between the duration of driving with 4kHz threshold

To understand the correlation between the duration of driving on 4kHz threshold Spearman Correlation analysis was done. Also to understand the correlation between the two variables a scatter plot was plotted which is shown in figure 4:7.
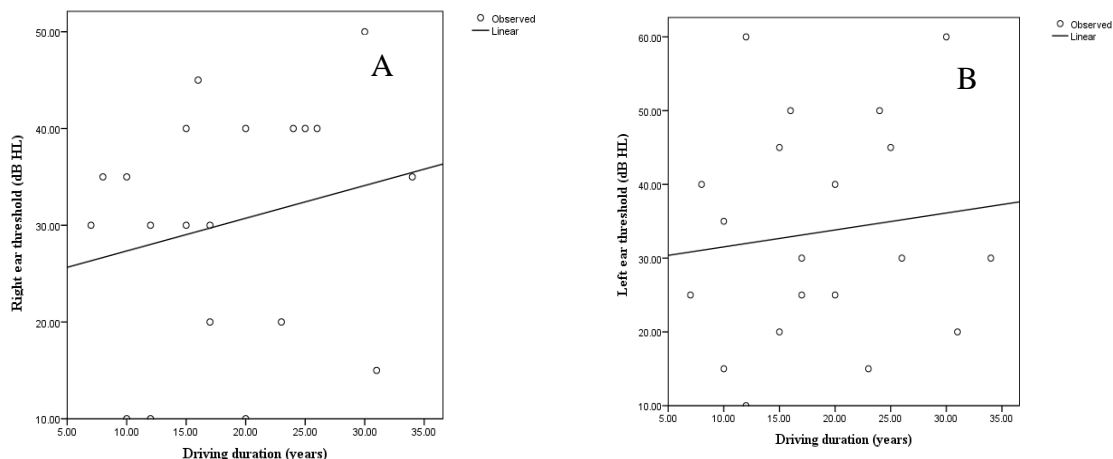

Figure 4:7. *A, B are the scatter plots for the correlation between the 4kHz threshold and the duration of driving.*

The above scatter plots represent correlation between the duration of driving with 4kHz threshold. As we can see there is slight threshold shift along with increase in duration of exposure but this is not statistically significant.

Spearman correlation revealed statistically no significant correlation between the duration of driving on 4kHz threshold. Statistically no significant difference between the duration of driving on 4kHz threshold of right ear [r= 0.278, p>0.05], 4kHz threshold of left ear [r= 0.131, p>0.05] in the experimental group.

4.5 Correlation between the audiological and vestibular findings

4.5.1 cVEMP latency and amplitude correlation with 4kHz threshold

To understand the correlation between the cVEMP latency and amplitude on 4kHz threshold Spearman Correlation analysis was done. Also to understand the

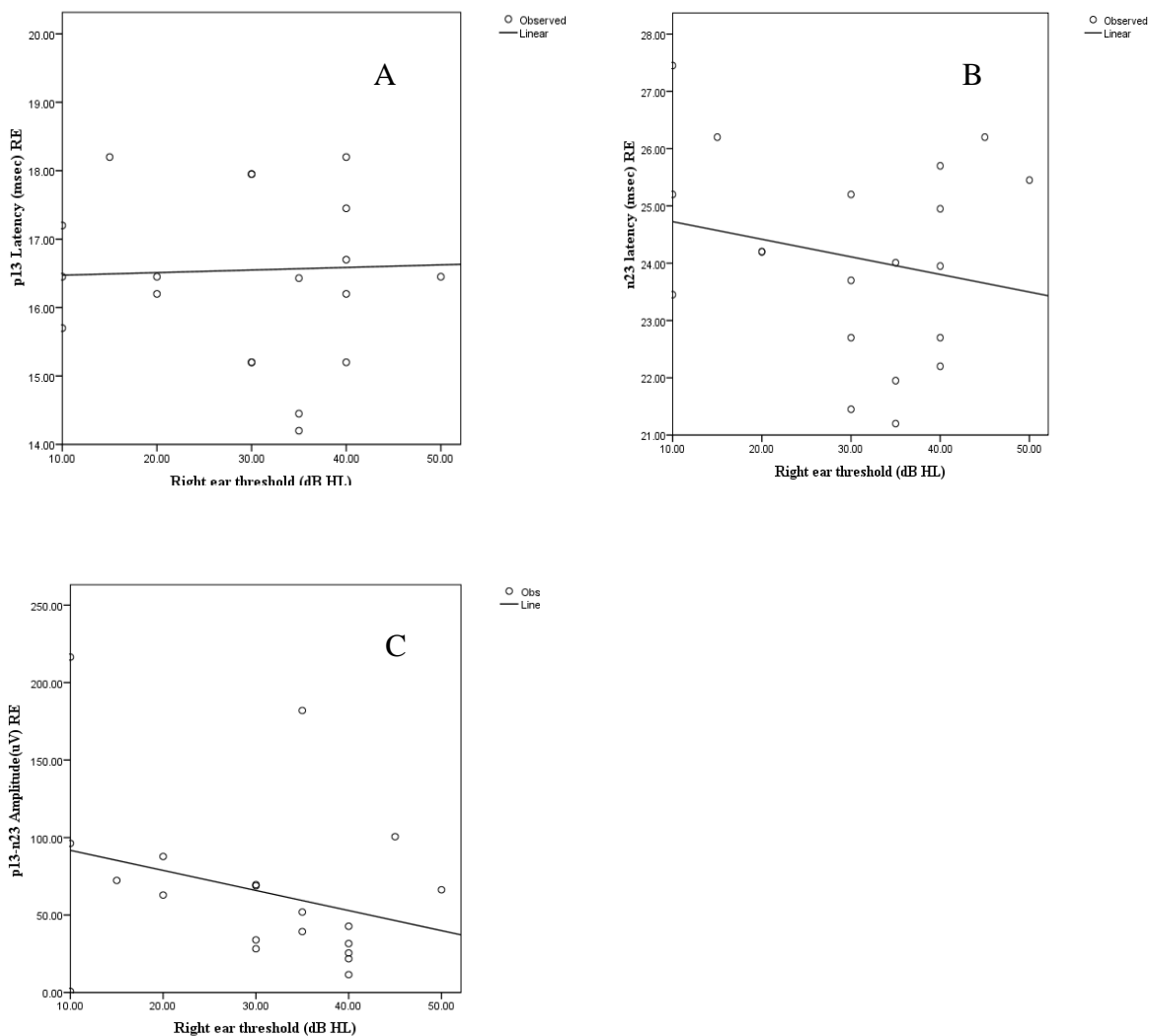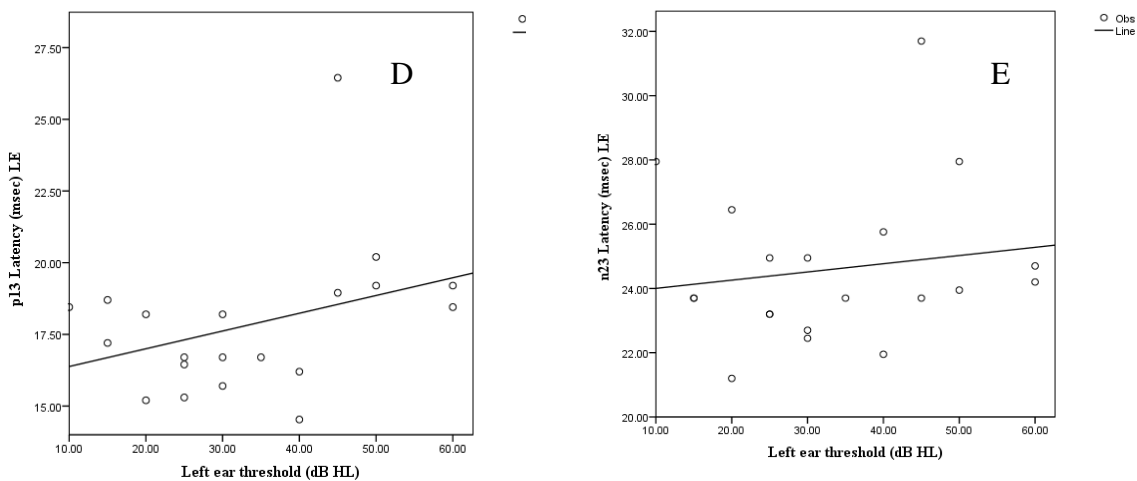correlation between the two variables a scatter plot was plotted which is shown in figure 4:8 and 4.9.

Figure 4:8. *A, B, C is the scatter plots for the correlation between cVEMP parameters and the 4kHz thresholds for the right ear.*
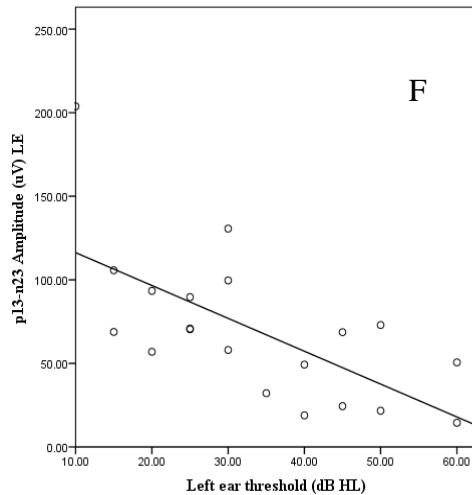
Figure 4:9. *D, E, F is the scatter plots for the correlation between cVEMP parameters and the 4kHz thresholds for the left ear.*

As we can see from the above scatter plots, there is no statistically significant correlation between the hearing threshold shifts and the cVEMP responses. The amplitude of p13-n23 of right and left ear is reducing with the increase in the threshold shift but not statistically significant.

Spearman correlation test revealed statistically no significant correlation between the cVEMP latency and amplitude and pure tone thresholds. There was no significant correlation for right ear of p13 latency on 4kHz threshold [r= 0.082, p>0.05], n23 latency on 4kHz threshold [r= 0.078, p>0.05], on amplitude of p13-n23 complex on 4kHz threshold [r= 0.273, p>0.05], for left ear of p13 latency on 4kHz threshold [r=0.400, p>0.05], n23 latency on 4kHz threshold [r= 0.184, p>0.05], p13-n23 complex on 4kHz threshold [r= 0.659, p>0.05] in the experimental group.

4.5.2 oVEMP latency and amplitude correlation with 4kHz threshold

To understand the correlation between the oVEMP latency and amplitude on 4kHz threshold Spearman Correlation analysis was done. Also to understand the

correlation between the two variables a scatter plot was plotted which is shown in figure 4:10 and 4:11.
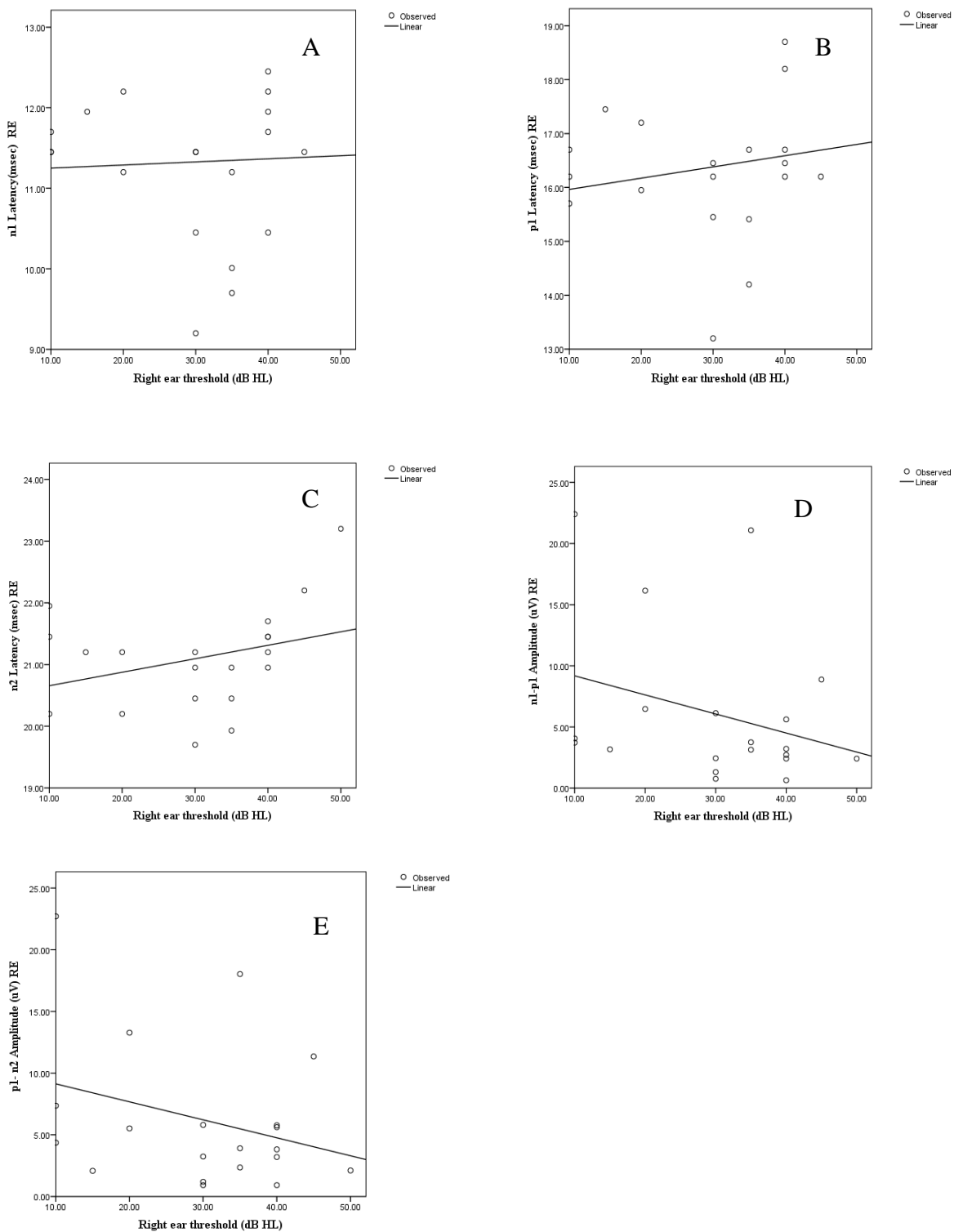

Figure 4:10. *A, B, C, D, E is the scatter plots for the correlation between the oVEMP parameters and the 4kHz thresholds for the right ear.*
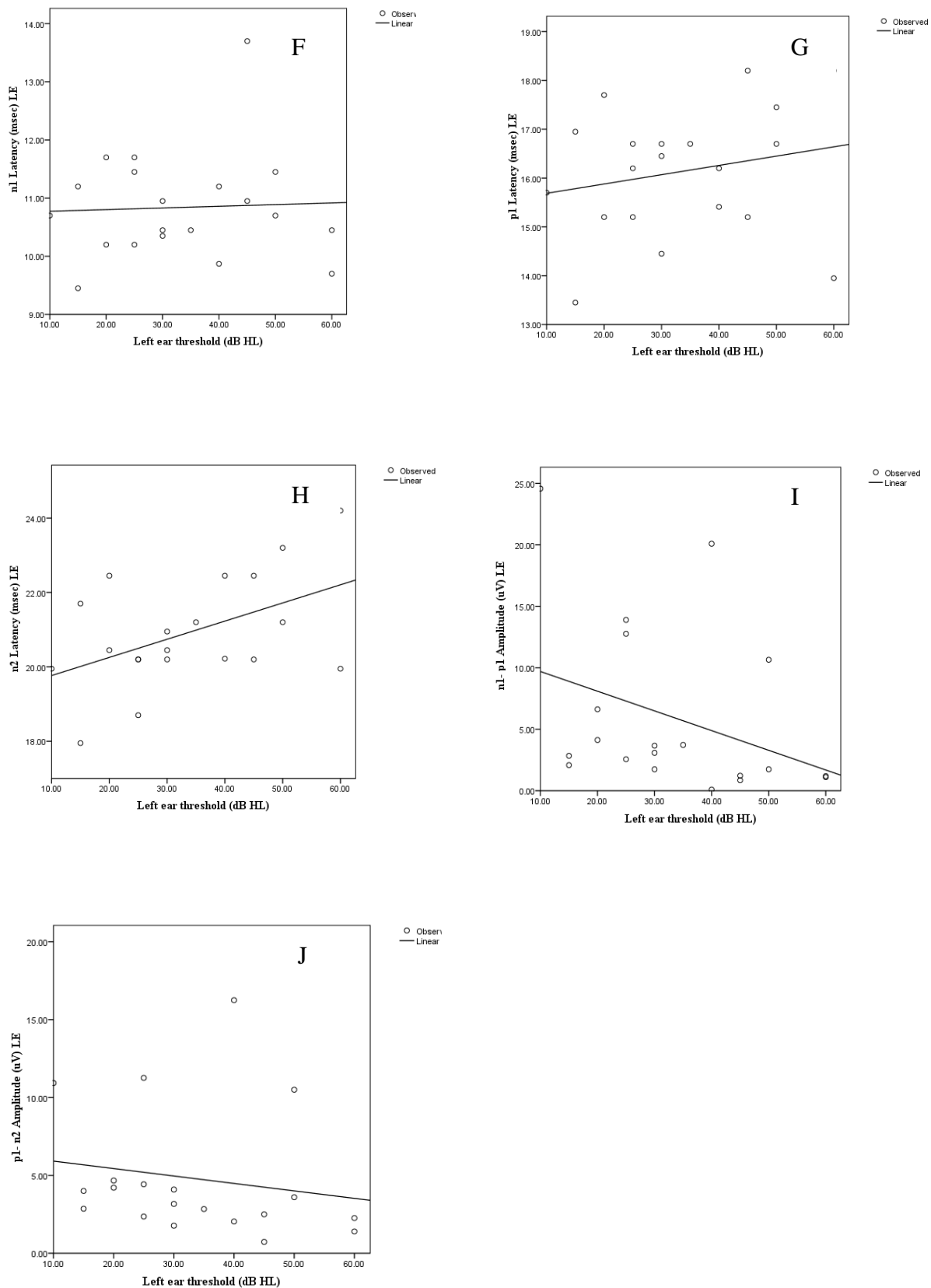
Figure 4:11. *F, G, H, I, J is the scatter plots for the correlation between the oVEMP*

parameters and the 4kHz thresholds for the left ear.

As we can infer from the above scatter plots that there is no significant correlation between the oVEMP response and the 4kHz threshold. The amplitude of n1-p1, p1-n2 complex of right and left ear is decreasing with the increase in threshold shift at 4kHz but the results are not statistically significant.

Spearman correlation test revealed statistically no significant correlation between the oVEMP latency and amplitude and pure tone thresholds. No statistically significant correlation between the right ear 4khz threshold on n1 latency [r= 0.186, p>0.05], p1 latency [r= 0.266, p>0.05], n2 latency [r= 0.351, p>0.05], on amplitude of n1- p1 complex [r= 0.318, p>0.05], p1-n2 complex [r= 0.216, p>0.05], the left ear threshold on n1 latency [r=0.011, p>0.05], p1 latency [r= 0.209, p>0.05], n2 latency [r= 0.398, p>0.05], n1-p1 complex [r= 0.517, p>0.05], p1-n2 complex [r= 0.437, p>0.05] in the experimental group.

Chapter - 5

Discussion

The aim of the present study was fulfilled by collecting data from both the control and the experimental group by administrating a test battery which included a detailed case history, pure tone audiometry, immitance and reflexometry, transient evoked otoacoustic emissions, and vestibular evoked myogenic potentials. The results of pure tone audiometry and vestibular evoked myogenic potentials were subjected to various statistical analysis and the results are discussed in this chapter.

5.1 cVEMP & oVEMP results in control and experimental group

In the present study the cVEMP responses was found to be present in 18 participants in the control group and 17 participants in the experimental group. There was no difference in latency and amplitude of cVEMP and oVEMP between control group and experimental group in whom the responses were present.

Previous studies have reported equivocal findings regarding the effect of noise on vestibular system. For example, Wang, Hsu and Young (2006) reported 18 ears out of 29 ears presenting normal cVEMP responses in individuals exposed to acute acoustic trauma. Madappa (2009) also examined the effect of noise on functioning and susceptibility of the saccule in individuals exposed to noise. Maddapa (2009), reported normal cVEMP latency responses in 54.29% and normal amplitude in 48.57% of individuals exposed to noise. Akin et al. (2012) recorded cVEMP in 43 individuals with a history of noise exposure. Akin et al. (2012) reported absence/ abnormal cVEMP in only 33 percent of the subjects, whereas in 67% of the participants cVEMP was normal. Emami (2014) reported 11 subjects with mild hearing loss due to noise exposure in whom the cVEMP responses were absent in all the subjects.

Compared to the cVEMP studies in NIHL individuals, the oVEMP studies in individuals with NIHL is relatively less. Shilpashree (2014) reported a significant decrement in amplitude of oVEMP in individuals with NIHL. In contrast, Emara and Gabr (2014) reported no significant difference in latency or amplitude of oVEMP between individuals exposed to or not exposed to noise.

It has been reported that individuals with NIHL in whom the auditory thresholds are poorer the vestibular evoked myogenic potentials are absent whereas in individuals with NIHL with relatively normal hearing have a normal vestibular evoked myogenic potentials (Kumar, Vivarthini, Bhat, 2010). Also, Wang and Young (2007) reported abnormal/absent VEMP responses in individuals with NIHL for whom the puretone thresholds were more than 40 dB HL.

When looked upon the data for the participants in the present study only 6 participants had threshold which was higher than 40 dB. Out of 6 participants two participants (3ears) had absence of cVEMP responses, and one participant had oVEMP responses which were absent, whereas another participant had reduced amplitude of cVEMP. Ideally, the cVEMP or the oVEMP responses should have been absent in all these 6 participants. But it has been reported that the presence or absence of cVEMP and oVEMP is independent of the cochlear function (Bickford et al. 1964; Colebatch et al. 1994; Ozeki et al. 1999; Bansal, Sahni & Sinha, 2014). Hence, the results of the present study indicate that although the cochlear function might be affected in these individuals, the vestibular function might be intact in these individuals.

Another study by Shupak et al (1994) reported that individuals with NIHL who have asymmetrical hearing loss, only those participants have abnormality of the vestibular function. Golz et al. (2001) also reported a similar finding. Looking at the data of the present study, only one participant had an asymmetrical hearing loss rest of

the participants had symmetrical hearing sensitivity (thresholds within 10 dB for both the ears). This could be one of the reasons wherein the cVEMP and oVEMP responses are present in the experimental group participants.

Intensity of the noise generated in the bus could be another variable for the presence or absence of cVEMP and oVEMP in the bus drivers. A study by Wang et al (2006) noted that an intensity level lesser than 90 dB SPL will not result in any damage to cochlea and vestibular structures. The noise levels of 90 to 130 dB SPL, the degree of damage majorly depends on the individual variations and susceptibility. And the levels greater than 130 dB SPL will result in a direct mechanical injury to the cochlea which is inevitable and irreversible. Wang et al (2006) reported a otolithic membrane damage and saccular collapse in the guinea pigs for an intensity of 136 to 150 dB SPL continuously for 20 minutes and concluded that, only if the noise intensity is high there will be damage to the cochlea and the saccule damage could be anticipated.

Patwardhan et al. (1991) reported that the bus drivers are typically exposed to 89-106 dB of noise. In another study, Mondal, Dey and Datta (2014) reported that Indian bus drivers are typically exposed to 88-104 dB(A) noise. Oosterveld, Polman, Schoonheyt (1982) reported that any noise level above 80 dB (A) would result in vestibular damage in individuals with NIHL. However, there are studies which indicate that for a continuous noise exposure the vestibular damage is more compare to a non continuous noise (Akdogan et al. 2009; Hsu et al. 2008). In the present study, the bus drivers had breaks in between their driving after every one hour for 10-15 minutes. Hence, they were not exposed to the bus noise for 8 hours in a row. Hence the bus drivers are not exposed to high level of noise continuously and thus may not have a damage to the saccule and the utricle. This could be another reason for the presence of cVEMP and oVEMP responses in these bus drivers.

5.2 Correlation between the duration of noise exposure and VEMP findings in Experimental group

It has been reported that duration and intensity of noise exposure has an effect on blood supply to cochlea and saccule as they are supplied from the common labyrinthine artery (Kumar et al., 2010). Also Wang and Young (2007) concluded that as the duration of noise exposure increases, there is decrement in the blood flow which may lead to permanent hearing threshold and abnormal VEMP responses. However, Manabe et al (1995) reported no correlation between the duration of noise exposure and vestibular responses. There was no correlation found between the cVEMP and oVEMP latencies and amplitude responses with duration of noise exposure in individuals with noise induced hearing loss (Emara & Gabr, 2014).

The data of the present study also did not show a significant correlation between duration of noise exposure and vestibular evoked myogenic potential. Emara and Gabr (2014) reported that individuals exposed to noise for a prolonged duration will have insults to both inner ear and the otolith organs but will demonstrate only permanent threshold shifts with no abnormality in VEMP responses which indicates a spontaneous recovery of the otolith organs due to central compensation. It has also been reported that the presence or absence of cVEMP and oVEMP is independent of the cochlear function (Bickford et al. 1964; Colebatch et al. 1994; Ozeki et al. 1999; Bansal, Sahni & Sinha, 2014). Hence a correlation between duration of driving and the cVEMP and oVEMP findings does not have a correlation.

5.3 Correlation between the 4kHz threshold and VEMP responses

Akin et al (2012) found a significant correlation between the abnormal or absent cVEMP responses and the degree of hearing loss in individuals with noise induced hearing loss. Akin et al (2012) reported that noise exposed individuals who had abnormal cVEMPs had poorer high frequency hearing sensitivity and greater high frequency threshold differences were seen between individuals who had normal and abnormal cVEMPs.

Another study by Wang and Young (2007) reported a significant association between the 4kHz threshold cVEMP responses. They said that individuals who had higher thresholds at 4kHz had abnormal VEMP responses and as the degree of hearing loss increases there is prolongation of latencies and decrement in amplitude of cVEMP responses. Also Wang et al (2007) noted a significant correlation between the hearing outcome and VEMP responses and they reported that VEMP responses can indirectly estimate the noise level and its effect on hearing outcome with a sensitivity of 44% and specificity of 100%.

Looking into the present study data, there was no significant correlation obtained between 4kHz threshold and the cVEMP responses. Out of 20 individuals with noise induced hearing loss, only 6 had high frequency hearing loss greater than 40dB HL. Out of 6 participants, two had (3 ears) absence of responses in cVEMP and one oVEMP response absent and one with reduced amplitude of cVEMP response. Ideally all the 6 participants with high frequency hearing loss should have absent or abnormal VEMP responses. But it has been reported that the presence or absence of cVEMP and oVEMP is independent of the cochlear function (Bickford et al. 1964; Colebatch et al. 1994; Ozeki et al. 1999; Bansal, Sahni & Sinha, 2014). Hence there was no correlation obtained between the conditions.

Shupak et al (1994) and Manabe et al (1995) found that as the hearing sensitivity reduces symmetrically there is symmetrical reduction of vestibular end organ responses. There was no correlation reported between the hearing sensitivity and vestibular responses. They suggested that there is less incidence of clinical symptoms noticed in these individuals exposed to noise as there is compensation that would have occurred by the central nervous system.

Another factor for no correlation between the 4kHz thresholds and VEMP responses would be the effect of noise only on cochlea and not on otolith organs. For example a study by Wit et al (1981) reported that due to presence of round window, the sound stimuli (pressure) entering the inner ear by the oval window would be majorly towards the cochlea than the vestibule.

Perez et al (2002) examined the effect of noise on vestibular end organs for the exposure of 113 dBSPL of broad band noise for 60 minutes in the normal ear and found no significant effect of noise and noted that there is an effect of the same noise on vestibular end organs when there is a fenestration in the semicircular canal. Perez et al reported that these findings may be the result of round window acting as the pressure release in the perilymphatic channel of cochlea. Therefore the sound pressure through the stapes footplate is transmitted towards the cochlea than the vestibular channels. Hence this can be a supporting factor for the present study showing no correlation between the 4kHz threshold and vestibular evoked myogenic potentials in bus drivers.

Chapter 6

Summary and Conclusions

The noise induced hearing loss is one of the most common causes of sensorineural hearing loss (second to presbycusis). It is the most prevalent causes of Occupational hearing loss. The cochlear damage in NIHL individuals is a well established phenomenon. Various studies have reported symptoms of vestibular damage in individuals with NIHL. The pure tone audiometry and the otoacoustic emissions act as a sensitive tool to assess the cochlear damage in individuals with NIHL. The Vestibular Evoked Myogenic Potential is an efficient tool in evaluating the functioning of otolith organs. The Cervical evoked myogenic potential assess the Saccular and Inferior Vestibular nerve functioning whereas Ocular evoked myogenic potential assess the Utricle and Superior Vestibular nerve functioning. Hence pure tone audiometry, otoacoustic emissions, cVEMP, oVEMP provides a complete picture about the audiological and vestibular findings in bus drivers. Hence, the present study was aimed :

- To report the audiological findings in bus drivers.

- To evaluate the functioning of Saccule and Utricle in bus drivers.

- To correlate the duration of noise exposure with VEMP findings.

- To correlate the 4kHz thresholds with VEMP findings.

To achieve the aim of the study, 20 participants with normal hearing which were the control group and 20 participants with noise induced hearing loss which were the experimental group in the age range of 40 – 60 years were considered in the study. All the participants underwent a detailed case history, pure tone audiometry, immitance and reflexometry, otoacoustic emissions, auditory brainstem response (for site of lesion testing), cVEMP and oVEMP tests.

The waveform of cVEMP and oVEMP responses were obtained from both the groups and it was analysed for cVEMP parameters of latency p13, n23 and amplitude of p13-n23 complex. Similarly for oVEMP parameters of latency n1, p1, n2 and amplitude of n1-p1, p1-n2 complex were analyzed. From the data the mean and standard deviation were calculated and the following statistical analysis was done.

- ✓ As the data obtained did not fall under the normality curve, non parametric tests were carried out.

- ✓ To compare the control and the experimental group, Kruskal Wallis test and Mann Whitney U test were carried out for cVEMP and oVEMP measures.

- ✓ To correlate between the duration of driving and VEMP responses, spearman correlation test was carried out. Also for the correlation between the 4kHz threshold and the VEMP responses spearman correlation was carried out.

The results obtained from the above statistical measures are as follows:

cVEMP in Control group

- – The mean latency of p13, n23 is longer in the left ear than the right ear.
- – The amplitude of p13-n23 complex is higher in the left ear than the right ear.

cVEMP in Experimental group

- – The mean latency of p13 and n23 peak is longer for the left ear compared to the right ear in the experimental group.

- – Also, the amplitude of p13- n23 complex is higher for the left ear compared to right ear.

cVEMP comparison between the control and the experimental group

- – There was statistically no significant difference between the two groups for latency of p13 and n23 peak for right ear.

- There was statistically no significant difference between two groups for p13 and n23 latency for left ear.

- There was statistically no significant difference in amplitude of p13-n23 amplitude complex between the two groups.

- Also, there was statistically no significant difference in amplitude asymmetry ratio between the two groups.

oVEMP in control group

- The mean latency of n1, p1, n2 peak is longer for the left ear compared to the right ear in control group.

- Also the amplitude of n1-p1 and p1-n2 complex is higher for right ear compared to the left ear.

oVEMP in experimental group

- The mean latency of n1, p1 and n2 peak is longer for the right ear compared to the left ear in the experimental group.

- Also, the amplitude of n1-p1 and p1-n2 complex is higher in the right ear than the left ear.

oVEMP comparison between the control and the experimental group

- There was statistically no significant difference between two groups for latency of n1, p1 and n2 peaks for left ear.

- There was statistically no significant difference between two groups for latency of n1, p1 and n2 peaks for Right ear.

- There was statistically no significant difference between two groups for amplitude of n1-p1, and p1-n2 amplitude between the two groups.

Correlation between the duration of noise exposure and cVEMP and oVEMP responses

- To understand the correlation between the duration of noise exposure and cVEMP and oVEMP responses, Spearman's correlation coefficient was done and scatter plot were drawn. The results revealed statistically no significant correlation between the duration of driving and cVEMP and oVEMP responses.

Correlation between the 4kHz threshold and cVEMP and oVEMP responses

- To find the correlation between the 4kHz thresholds and VEMP responses, Spearman's correlation coefficient was carried out and scatter plots were drawn. The results revealed statistically no significant correlation between the 4kHz threshold and cVEMP and oVEMP responses.

Conclusion

Both cVEMP and oVEMP provides complimentary information about the saccule, utricle and its innervating structures, and hence these tests can be utilised to assess various vestibular pathology. In the present study both the tests were utilised to assess the utricule, saccule and its innervating structure in bus drivers. Previous studies have reported the effect of noise on auditory and vestibular system but the present study did not show any significant effect of noise on saccule and utricle of vestibular system. Thus, it can be concluded that not every type of noise will affect the saccule or the utricle. Further, the study can be conducted on a larger population and could be checked for any significant difference.

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