Vestibular Evoked Myogenic Potential (VEMP) in individuals with Noise Induced Hearing Loss (NIHL)

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MAY, 2009.


Dedicated to
APPAJI
AMMA

CERTIFICATE

This is to certify that this dissertation entitled "Vestibular Evoked Myogenic Potential (VEMP) in individuals with Noise Induced Hearing Loss (NIHL)" is a bonafide work in part of fulfillment for the degree of Master of Science (Audiology) of the student Registration number: 07AUD008.This has been carried under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award of any diploma or degree.

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CERTIFICATE

This is to certify that this dissertation entitled “Vestibular Evoked Myogenic Potential (VEMP) in individuals with Noise Induced Hearing loss (NIHL)” has been prepared under my supervision & guidance. It is also certified that this dissertation has not been submitted earlier to any other university for the award of any diploma or degree.

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DECLARATION

This is to certify that this master's dissertation entitled "Vestibular Evoked Myogenic Potential (VEMP) in individuals with Noise Induced Hearing loss (NIHL)" is the result of my own study and has not been submitted earlier to any other university for the award of any degree or diploma.

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Table of Contents

LIST OF TABLES

LIST OF FIGURES

Chapter 1

Introduction

Hearing is one of the most important senses in human beings. It is one of the channels through which we communicate and interact with the society. Unfortunately, there are a multitude of factors that can affect the hearing of an individual. Of the various factors, the common most factor which can have an adverse effect on our hearing is ‘noise’. The word noise is derived from the Latin word ‘nausea’ meaning annoyance (Ward, 1980). It can be defined as a sound- generally random in nature, the spectrum of which does not exhibit clearly defined frequency components (Behar, Chasin & Cheesman, 2000).

Since the Industrial Revolution, an increasing number of ears have been injured by noise via two ways. One is acute acoustic trauma, which is defined as a sudden change in hearing as a result of a single exposure to a sudden burst of sound. Other is the Noise-Induced Hearing Loss (NIHL), which is cumulative and insidious, growing slowly over years of exposure and commonly associated with occupational noise (Ylikoski, Juntunen, Matikainen, Ylikoski, & Ojala, 1988).

Occupational noise-induced hearing loss, as opposed to occupational acoustic trauma, is hearing loss that develops slowly over a long period of time (several years) as the result of exposure to continuous or intermittent loud noise (American College of Occupational and Environmental Medicine, 2002). It is estimated that 1.1 million people

are exposed to excessive noise at work and of these 1 lakh 70 thousand will suffer from significant ear damage as a direct result of noise exposure (South, 2004).

Noise is the insidious of all industrial pollutants, involving every industry (Trivedi & Raj, 1992). Noise has both auditory and non auditory effects. Noise levels exceeding 75 dB(A) to 85 dB(A) begin to stress the auditory system (Kvaerner, Engdahl, Arnesen, & Mair,1995). Extreme noise can clearly damage hair cells in the cochlea, leading to temporary or permanent threshold shifts in hearing (Rosler, 1994). There are reports of the spiral ganglion cells being damaged whose central processes form the auditory portion of the eighth nerve (Nadol & Xu, 1992). The degeneration of the central nervous system including the cochlear nuclei, superior olive and inferior colliculus (Kim, Leonard, Smurzynski, & Jung, 1997; Morest, Kim, Potashner & Bohne, 1998) has been reported as a result of noise exposure. Also, negative reactions (Fields, 1994; Hatfield & Job, 1998; Job, 1988), sleep disturbances (Ohrstrom, Brorkman & Rylander, 1990; Pearsons, Barber, Tabachnick & Fidell, 1995) and detrimental effect on cardiovascular health (Talbott et al., 1996) have been reported resulting from noise exposure.

There are battery of audiological tests for evaluating the auditory effects of noise and the early diagnosis of NIHL of which Otoacoustic Emissions (OAEs) provide objectivity and greater accuracy, complementing the behavioral audiogram in the diagnosis and monitoring of the cochlear status following noise exposure (Attias, Abrovitz, Hatib, & Nageris, 2001). The Transient Evoked Otoacoustic Emissions's (TEOAEs) are known to be abolished by sensorineural hearing loss and absent once the

hearing threshold exceed 30 dB HL (Hall, 2000). The sensitivity of TEOAEs has been reported to be as high as 90% at 2 kHz and 4 kHz (Hall & Lutman, 1999).

Noise exposure not only damages the cochlea, but threatens the vestibular organs too. (Oosterveld, Polman, & Schoonheyt, 1980). Oosterveld, Polman, & Schoonheyt (1982) reported that noise-exposed individuals could be disabled because of vertigo or balance disorder; an important and perhaps neglected aspect of noise-induced hearing damage. Similar reports of vestibular involvement leading to various vestibular symptoms in individuals exposed to noise have been studied using various test procedures for assessing the vestibular system (Barr, 1886; Rodger, 1915; Chadwick, 1966; Aantaa, Virolainen & Karskela, 1977; Pulec, 1972; Paparella & Mancini, 1983).

Typical tests, which are used since past in the evaluation of vestibular disorders such as Electronystagmography (ENG), Caloric test and other tests actually assess only semicircular canals and superior vestibular nerve but not the saccule and inferior vestibular nerve. Thus, VEMP plays an important role in the vestibular test battery as a non-invasive measure of saccular function (Hall, 2006).

Vestibular evoked myogenic potentials (VEMPs) were first described by Bickford, Jacobson & Cody (1964). It has been proposed as a reliable clinical tool to assess the saccular or the inferior vestibular nerve function (Colebatch, 2001). The neurophysiologic and clinical data indicate that the VEMPs are mediated by a pathway that includes the saccule, macula, inferior vestibular nerve, lateral vestibular nucleus,

lateral vestibulospinal tract, and motor neurons of the ipsilateral sternocleidomastoid muscle (Halmagyi & Curthoys, 2000).

The VEMP waveform consists of two components (an early positive-negative p13- n23 component and a later negative-positive n34- p44 component), of which only the first component (p13- n23) is generated by activation of saccular afferents (Colebatch, Halmagyi & Skuse, 1994). VEMP testing may provide useful, non-invasive method for the assessment of otolith function and the functional integrity of inferior vestibular nerve (Clarke, Schonfed & Helling, 2003).

VEMP has a wide clinical applicability. VEMP has been reported to be useful in the assessment of various peripheral and central vestibular disorders such as endolymphatic hydrops (Murofushi, Matsuzaki & Takegoshi, 2001), vestibular schwannoma (Matzsuzaki, Murofushi & Mizuno, 1999) and vestibular neuritis (Colebatch & Halmagyi, 1994), Superior canal dehiscence syndrome (Brantberg, Bergenuis & Tribukait, 1999) and Vestibular hypersensitivity (Streubel, Cremer & Carey, 2001).

In a recent study, Wang & Young (2007) reported that patients with bilateral NIHL (bilateral 4 kHz notched audiogram with hearing threshold of 4 kHz > 40 dB) may show abnormal VEMP indicating that vestibular part especially, the sacculocollic reflex pathway has also been damaged. Christina, Kumar, & Bhat (2008) also observed

abnormal VEMP in 82%, out of which, 36% were having absent VEMP and 46% were having abnormal VEMP, in a total of 6 subjects with noise induced hearing loss.

Need of the present study

The vestibular end organs and the cochlea both utilize the same basic principle of mechano-electric transduction with the help of the sensory hair cells (Eisen & Limb, 2007). Also, the bony labyrinth is stimulated in response to high levels of occupational noise. Hence, balance system could also have negative effects secondary to long term noise exposure, along with the hearing sensitivity. However, attempt has not been made to find out the causation factors and or the vestibular structures involved which leads to the observed vestibular symptoms.

The anatomical dimensions of the various structures associated with vestibular system and the force required for the breakage of each of these structures may reveal some information about the susceptibility to damage of these structures due to over stimulation.

The saccule has been reported to be the thinnest membrane (0.015mm) after Reissener's Membrane (0.014mm). Also, saccule can withstand is much lesser force (0.57gf/mm) before breakage as against the Reissener's membrane which can withstand a force of 0.84gf/mm (Tetsuo, Nobukazu, & Terufumi, 1990). Furthermore, the distance of the utricle and saccule from the stapes are 0.65mm and 0.4mm respectively which in turn adds to the probability of the balance system getting affected due to noise. It is also reported that saccular maculae among the vestibular structures, are the most sensitive

structure to sound stimulation (Goldbeg, 2000). Hence, it can be speculated that long-term exposure to noise could also affect the functioning of the vestibular system.

The possible vestibular involvement in patients with NIHL is relatively new and there is dearth of information regarding the same. Individuals exposed to noise either for short or long duration might exhibit giddiness or sensation of giddiness or may have Tullio phenomenon or may not exhibit any other vestibular symptoms. It is clear that early peaks of VEMP (p13 and n23) are sensitive for the assessment of saccular and inferior vestibular nerve. Thus, VEMP recording might help to unfold the saccular involvement in individuals who are exposed to noise with or without any vestibular symptoms.

Aims of the present study

- ✓ To evaluate the functioning of the saccule and the inferior vestibular nerve in individuals with Noise Induced Hearing Loss.
- ✓ To assess the susceptibility of cochlea or saccule to noise exposure, based on Transient Evoked Otoacoustic Emission (TEOAE) and Vestibular Evoked Myogenic Potential (VEMP) test results.
- ✓ To know whether the vestibular system damage is associated with saccular dysfunction in individuals with NIHL, by correlating the vestibular symptoms and VEMP response.
- ✓ To know whether there is any relationship between degree of hearing loss and saccular dysfunction in individuals with NIHL.

Chapter 2

Review of literature

The impact of noise upon the auditory system has become a major problem in today's highly technological society. Hearing loss due to occupational noise exposure is the most prevalent industrial malady and has been recognized since industrial revolution. Occupational hearing loss can be defined as a partial or complete hearing loss in one or both the ears as a result of an individual's employment (Nandi & Dhatrak, 2008). It includes acoustic trauma which results in sudden change in hearing resulting from single exposure to a sudden burst of sound (ACOEM, 2003) and Noise Induced Hearing Loss (NIHL).

"Noise Induced Hearing Loss (NIHL) results from damage to the ear from sounds of sufficient intensity and duration that produces a temporary or permanent sensorineural hearing loss. The hearing loss may range from mild to profound, may result in tinnitus and is cumulative over a lifetime" (Noise and Hearing Loss Consensus Conference, 1990). NIHL is a specific condition with established symptoms and objective findings (Morata, 2007). It refers to SNHL in subjects exposed to environmental noise when other causes of hearing loss are excluded (Pyykko, Toppela, Zou & Kentala, 2007).

Studies have shown that people who are exposed to continuous noise levels higher than 85dB-90dB(A) suffered from NIHL (Rabinowitz & Rees, 2005; Suter, 1998). Noise induced hearing loss depends on an individual's susceptibility, amount and duration of

noise exposure and initially it results in high tone hearing defect and later it spreads to speech frequency region (Schwetz, Doppler, Schewezik, & Wellesxhik, 1980).

Exposure to excessive noise is one of the major causes of permanent hearing impairment worldwide and it is the second most common cause for sensorineural hearing loss (Rabinowitz & Rees, 2005). Worldwide, 16% of the hearing disability in adults is attributed to occupational noise and it ranges from 7 to 21% in the various sub regions (Nelson, Nelson, Barrientos, & Fingeruhut, 2005).

Noise exposure can cause two kinds of health effects; non auditory effects and auditory effects.

Non auditory effects

Evidence shows that exposure to excessive noise over prolonged periods produces both physiological as well as psychological changes in human beings. The physiological changes includes vasoconstriction of the peripheral blood vessels and minor changes in the heart rate, changes in galvanic skin response, slow deep breathing and changes in skeletal muscle tension have been reported (Davis, Buchwald, & Frankman, 1955). Changes in gastrointestinal motility (Davis, Buchwald, & Frankman, 1955; Davis & Berry, 1964; Stern, 1964), chemical changes in the blood and urine from glandular stimulation have also been reported (Hales, 1952; Levi, 1967).

Noise exposure also affects psychological condition of an individual. It promotes negative psychological reaction (Job, 1988; Fields, 1994 and Job & Hatfield, 1998) and psychological stress (Office of Noise Abatement and Control, 1981; Weiten, 1992;

Evans, Hygye & Bullinger, 1995). Also, momentary insufficiencies tend to be more likely to occur in conditions of loud noise (Broadbent, 1979). Noise can delay sleep and shift the sleep stages upward (Thiessen, 1978). It can cause annoyance, aggression, reduce helping behavior, influence judgment and subsequently lead to several psychological perturbations (Kryter, 1985) that can seriously affect the quality of one's life and their family members. It has been reported that noise may negatively impact on the interpersonal relationships (Katz, 1994).

Auditory effects

Due to noise exposure, there are two functional consequences to hearing namely TTS (temporary threshold shift) and PTS (permanent threshold shift) (Plontke and Tubingen, 2004).

TTS refers to a transient sensorineural hearing loss lasting for hours to a few days. Hearing thresholds are depressed until the metabolic activity in the cochlea recovers. Hence prior to audiometric testing subjects should be out of noise for at least 24 hours if not 48 hours to avoid the effects of TTS on hearing (Bohne & Harding, 1999). NIHL causes damage to the outer hair cells of the cochlea resulting in a reduction of the amplification ability of the cochlea (Reshef, Attias, & Furst, 1993). Shortly after a damaging exposure, the cells and tissues of the inner ear are in a dynamic state of injury, degeneration and/or repair. This has been termed as the acute phase of noise damage.

With intense exposures (≥140 dB SPL), a portion of the organ of Corti is displaced from its position on the basilar membrane and is often found floating within the scala media (Lurie, 1942). Swollen hair cells are found at the edges of the lesion and

signs of damage are apparent in the non myelinated nerve fibers of the organ of Corti both within and adjacent to the displaced portion (Bohne, 1976). Nordmann, Bohne & Harding (1999) reported that cochlea had the outer hair cell (OHC) stereocilia which were not embedded in the tectorial membrane in the region of the TTS. Buckling of the pillar bodies was also reported which correlated with TTS region.

Exposure of noise extending from the TTS may lead to permanent shift in threshold referred to as Permanent threshold shifts. PTS refers to a permanent loss of sensorineural hearing which is the direct result of irreparable injury to the organ of Corti (Bohne & Harding, 1999). In case of permanent noise damage, initially there is degeneration of both types of hair cells; however, outer hair cells (OHCs) are more sensitive to noise than the inner hair cells (IHCs). With longer exposures or a more intense noise, there is further loss of OHCs, IHCs, and supporting cells. The cell loss is confined to a narrow region of the organ of Corti which develops into a 'focal' hair-cell lesion (Bohne & Clark, 1990).

Once IHC reaches moderate proportions of damage, there will be loss of myelinated nerve fibers which are the peripheral processes of the spiral ganglion cells within the osseous spiral lamina (Bohne, Yohman, & Gruner, 1987). Hair cell and supporting cell losses within a focal lesion gradually progresses and involves 100% of the cells over a variable length of the organ of Corti. Eventually, the spiral ganglion cells are progressively lost, including their central processes which form the auditory portion of the eighth nerve (Nadol & Xu, 1992). There is evidence to suggest that once the degeneration of the spiral ganglion cells has begun; there is a corresponding degeneration within the central nervous system including the cochlear nuclei, superior olive and

inferior colliculus (Kim et al., 1997; Morest, Kim, Potashner, Bohne, 1998). Nordmann, Bohne & Harding (1999) reported that the PTS was associated with focal losses of inner and outer hair cells and afferent nerve fibers at the corresponding frequency location of noise exposure.

Tests available for evaluating the auditory effects of noise

Occupational hearing loss is a specific disease with established symptoms and objective findings. The hearing loss is of sensorineural type with damage to the cochlear hair cells with a history of long term exposure to intense noise levels and it develops gradually over a period of years, specifically during the first 8-10 years of noise exposure. The hearing loss initially starts in the higher frequencies (generally 3-6 kHz) and it is equal in both the ears with the speech discrimination scores generally good (over 75%) and hearing loss stabilizes if the patient is removed from noise exposure (Cited in Sataloff & Sataloff, 1987).

Pure tone air conduction threshold assessment is the most useful audiological measurement with a typical 4 kHz notch or hearing loss at higher frequencies. Luxon (2003) reported that the 4 kHz notch reflects the structural damage to the spiral organ of corti at the point of 10mm distant from its basal end. But the puretone audiometry has fallen short of detecting the NIHL sufficiently early in order to prevent from developing and moreover, it is difficult to distinguish between NIHL and other cochlear diseases using pure tone audiometry (Kowalska & Kotyla, 1997). Impedance audiometry is a useful test to exclude a middle ear component of hearing loss. Stapedial reflex assessment is crucial in confirming the cochlear site of the lesion and in differential diagnosis of

NIHL and retro-cochlear pathologies. ABR is important in the reconstruction of an audiogram especially while evaluating medico-legal subjects when accounting for the compensation claims. It is also useful in differential diagnosis from retro-cochlear pathology. The ABR can be used in monitoring the threshold shift as a result of exposure to short duration noise (Hsu, Wang, Lue, An-Shiou & Young, 2008)

Oto acoustic emissions (OAE) are highly sensitive to cochlear dysfunction as they are generated by and reflect vital biochemical activity within the normal cochlea (Katz, 1994; Lonsbury, Martin, & Whitehead, 1997) and the unique quality of the OHC's produces the OAE's. (Kemp, 1982; Kowalska & Sulkowski, 1997). OAE's are used in monitoring of the cochlear function in subjects with NIHL, and this improves the sensitivity of the otoacoustic emission to be assessed directly.

Transient Evoked Otoacoustic Emissions (TEOAE) and Noise Induced Hearing Loss (NIHL)

Noise exposure for a short duration alters the frequency spectrum of otoacoustic emission and increases the threshold (Rossi, Solero, Rolando, Olina, 1991; Avan, Bonfils, Loth, Teyssou, Menguy, 1993). A positive correlation between TEOAE and hearing threshold shifts have been reported in a group of soldiers exposed to white noise at 90dBSL for 10 minutes (Attias & Bresloff, 1996). However, in some of the subjects, TEOAE changes were not accompanied by pure tone threshold shift; which supports the hypothesis that otoacoustic emissions are a sensitive measure of minor changes in cochlear function.

Kvaerner et al., (1995); Sliwinska-Kowalska et al., (1999) found a significant reduction of the mean TEOAE and elevation of the puretone thresholds in employees after 3 consecutive days of 7-hours of noise exposure at 85-90dB(A). They did not demonstrate any correlation between TEOAE changes and TTS and also there was no reduction of OAE's at high frequencies.

Attias & Bresloff (1996) reported decreased otoacoustic emissions with no change in pure tone thresholds in some subjects and temporary threshold shift with no changes in otoacoustic emissions was also found in few subjects exposed to noise. The comparative studies on occupational noise by Kvaerner et al., (1995); Kowalska, Kotylo, & Hendler (1999) and Attias & Bresloff (1996) indicate greater pure tone threshold shifts that were accompanied by greater decrease of TEOAE responses.

Vinck, Van Cauwenberge, Leroy, & Corthals, (1999) carried out two experiments in order to evaluate the sensitivity and applicability of TEOAEs and DPOAEs to assess the functional integrity of OHC during TTS. In the first experiment, a group of volunteers were exposed to broad band noise at 90dBSPL for 1 hour. The TEOAE and DP- gram responses were measured both before and after 6 hours of noise exposure. The results revealed a significant decrease of OAE amplitude at 4 kHz and higher and decreased repeatability of TEOAE, although there was no shift in pure tone threshold. Decrease of otoacoustic emissions due to noise appears prior to hearing impairment measured with pure-tone audiometry. OAEs seem to be more sensitive to temporal changes in cochlear function after acute noise exposure than conventional methods. In the second set of experiments, eight young healthy adults were exposed to disco-tech music for five consecutive hours. A significant TTS was observed with pure-tone audiometry with a

greater decrease in OAEs. The time course of recovery for DPOAE and TEOAE was very similar to behaviorally measured temporary threshold shift.

Monitoring of early changes in OAE's caused by prolonged exposure to industrial noise is scarce. Hotz et al (1993) reported a significant reduction of TEOAE amplitude in 2-4 kHz frequency range among soldiers after 17-week exposure to impulse noise. Amplitude was reduced by 84% and 90% in both the right and the left ear respectively compared to initial level of OAE's. Although, pure-tone audiometry changes were not monitored, the comparison of extrapolated data with pure-tone audiometry results of another soldier group, exposed to comparable noise level, suggested higher sensitivity of TEOAE than conventional methods.

Kowalski & Kotylo (2001) monitored hearing loss in a group of metal factory workers, who were exposed to noise 85-97dB(A) for 0.5-6years and weaving mill employees who were exposed to 88-92dB(A) for more than 6years using TEOAE, DPOAE and pure-tone audiometry for 2 years. There was a gradual decrease in TEOAE, although there was no hearing loss using PTA during 2 years of observation. Compared to controls, the decrease of TEOAE was significantly higher in both the groups. However, there were no consistent changes in DPOAE pattern suggesting that TEOAE can be a better tool to detect progressive sub clinical cochlear dysfunction associated with NIHL.

The above literature makes it evident that, in the test battery for the evaluation and monitoring of individuals exposed to noise, inclusion of OAEs and especially TEOAEs provides important information and is very sensitive to the OHC damage which is the structure primarily involved in individuals exposed to noise. Also, it is clear that identification of early subclinical cochlear lesions is possible even before the changes are noticeable in the audiometric threshold.

Vestibular symptoms in individuals with Noise Induced Hearing Loss

The noise exposure not only damages the cochlea, but threatens the vestibular organs too (Oosterveld, Polman & Schoonheyt, 1980). The vestibular end organs and the cochlea have a common evolutionary origin and utilize the same basic principle of mechano-electric transduction with the help of the sensory hair cells (Eisen & Limb, 2007). An association between the noise exposure and vestibular function has long been suspected. Noise which has been recognized as a cause of cochlear damage resulting in hearing loss; however, its role in vestibular dysfunction remains unclear and a cause effect relationship has not been accepted (Nageris, Attias & Feinmesser, 2000).

Tullio (1929) has referred to the activation of the vestibular system due to auditory stimulation to one of the ears as Tullio phenomenon. However, when auditory stimulus is presented to both the ears, Tullio phenomenon does not occur since the reflexes equalise each other. There have been a few studies reported in the literature showing the audio-vestibular phenomenon (Oosterveld, Polman & Schoonheyt, 1980; Oosterveld, Polman & Schoonheyt, 1982; Ylikoski et al, 1988; Manabe et al, 1995; Cassandro et al, 2003).

The noise exposure which affects vestibular functions can be explained physiologically. Both mechanical and acoustic trauma causes contusion of the labyrinth. Mechanical trauma can directly damage the vestibulum while the acoustic trauma damages the vestibular system through the round window of the cochlea (Nageris, Attias & Feinmesser, 2000). Lesions in the bony walls of the vestibular system resulting in vestibular reactions when exposed to weak sounds have been reported by Roggensen & Van Dischock (1950).

Mc Cabe & Lawrence (1958) reported that guinea pigs exposed to sound of 136 to 150 dBSPL for 20 minutes, demonstrated that the saccule and cochlea (pars inferior) are the structures which are most readily damaged, whereas the utricle and semicircular canals (pars superior) remain free of structural changes. This difference indicates a functional dichotomy between pars superior and inferior, possibly attributable to the existence of membrana limitans, which serves as a barrier between two partitions, leading to differential sensitivity of cochlear and vestibular sensory cells in the presence of noxious substances (Hara & Kimura, 1993). In other words, in the vestibular labyrinth, the saccule rather than the utricle or the semicircular canals is the locus of predilection for noise damage.

Barr (1886) reported that 15 of the 100 individuals exposed to occupational noise had "some sensation of giddiness". Rodger (1915) reported that vestibular system was stimulated in presence of noise and also found that 10% of patients with noise induced hearing loss (NIHL) complained of giddiness. Chadwick (1966) reported that out of 1800 patients with NIHL, eight had the fluctuating hearing loss, tinnitus and attacks of vestibular dysfunction.

Aantaa, Virolainen & Karskela (1977) reported symptoms of vestibular disturbances (exhibited in the form of spontaneous nystagmus, lowered caloric excitability, or pathology in rotatory tests) as high as 44.9% in a group of 49 male workers (mean age 30 years) who had been exposed to extreme noise and vibration between 6 months - 10 years. The lesions were believed to have arisen in the peripheral vestibular organ as a consequence of the low frequency vibrations.

Pulec (1972) and Paparella & Mancini (1983) reported that some patients with Meniere's disease had a history of exposure to hazardous noise, and they speculated that the noise exposure may in fact have caused the disease.

Oosterveld, Polman & Schoonheyt (1982) carried out vestibular examination in a group of 29 noise-exposed individuals. Results revealed spontaneous nystagmus in 18 subjects, positional nystagmus in 24, cervical nystagmus in 17 and nystagmus preponderance of more than 20% in the rotation test in 7 individuals. A difference in excitability between the labyrinths of more than 20% was shown by 7 subjects. There was no correlation between the degree of the hearing loss and the vestibular symptoms. All subjects showed pathology in one or more of the vestibular tests. In consequence, individuals with noise-exposure could be disabled because of vertigo or balance disorder which is an important and perhaps neglected aspect of noise-induced hearing damage.

Ylikoski, et al., (1988) reported that sixty patients with varying degrees of noise-induced hearing loss (NIHL) did not manifest any clinical symptoms of vestibular pathology after long-term exposure to intense impulse noise from firearms. They were tested for body sway using a stable platform. The results indicated that subjects with NIHL showed significantly more body sway than the control group. This sway was estimated as movement of the centre of gravity in the horizontal plane. It was also observed that subjects with more severe NIHL showed more sway than subjects with milder acoustic trauma and also the body sway is increased in patients with NIHL from exposure to impulse noise of high intensity. This suggests subclinical disturbances of the vestibular system in individuals with noise induced hearing loss.

Shupak, et al., (1994) evaluated vestibular function in a group of 22 men suffering from NIHL and 21 matched controls using Electronystagmography (ENG) and the smooth harmonic acceleration (SHA). The results showed a symmetrical centrally compensated decrease in the vestibular end organ response which was associated with the symmetrical hearing loss**.** In addition statistically significant correlations were found between the average hearing loss, the decrement in the average vestibulo-ocular reflex gain and ENG caloric lateralization. These correlations might indicate a single mechanism for both cochlear and vestibular noise-induced injury. The results can be implicated as subclinical, well compensated malfunction of the vestibular system associated with NIHL.

Manabe, et al., (1995) recorded Ecogh and PTA in thirty-six NIHL patients who were divided into two groups; vertigo group and non vertigo group based on the presence or absence of vestibular symptoms. Electrocochleograms were recorded from all the subjects after the pure tone audiometry. Results revealed a higher incidence of increased summating potential (SP)/action potential (AP) ratio in the vertigo group than in the non-vertigo group. Caloric tests performed in the vertigo group revealed reduced response in 47.1% of ears. Since the -SP/AP ratio is a useful indicator of endolymphatic hydrops, it can be speculated that the episodic vertigo in NIHL patients may result from a pathophysiological mechanism similar to that of Meniere's disease. Okuno et al., (1996) reported that out of 475 soldiers tested, 1.4% had Meniere's disease and 32.5% had experienced dizzy spells.

Golz et al (2001) reported vestibular damage in 258 military subjects who were exposed to intense noise. They found that vestibular damage was either absent or abnormal in subjects with asymmetrical hearing loss. There was a strong correlation between the subjects' complaints and the results of the vestibular function tests. But there was no correlation between the severity of the hearing loss and the vestibular symptomatology and pathology. They concluded that subjects exposed to intense noise may have evidence of vestibular pathology only when there is an asymmetrical hearing loss.

Unlike the auditory system, which can be objectively and effectively tested by the audiogram, the vestibular system has no single primary objective test with which single diagnoses can be made. Rather, a cadre of tests are available that are used in different combinations to yield a vestibular evaluation and diagnosis. The most commonly used

tests are ENG, caloric irrigation, rotatory chair, posturography, Videonystagmography (VNG). These tests assess the vestibular disorders of semicircular canals and superior vestibular nerve origin. These would help in differentiating the vestibular and the non vestibular causes of dizziness, also between central and peripheral sites of vestibular disorders. These tests assess the vestibular reflex and hence provide valuable information about the integrity of the brainstem. ENG assess only the function of lateral semicircular canal and superior vestibular nerve and not the other parts of vestibular apparatus in humans such as saccule and inferior vestibular nerve. This lacuna in vestibular evaluation is filled by a test namely Vestibular Evoked Myogenic Potential (VEMP).

Vestibular evoked myogenic potential (VEMP)

VEMP's are short latency EMG (electromyogram) which could assess saccular or inferior vestibular nerve disturbances (Colebatch, 2001). VEMP's are mediated by the pathway that includes saccular maculae, inferior vestibular nerve, the lateral vestibular nuclei, lateral vestibular spinal tract and motor neurons of ipsilateral Sternocliedomastiod (SCM) muscle (Halmagyi & Curthoys, 2000). VEMP testing may provide useful, non-invasive method for assessment of otolith function and the functional integrity of the inferior vestibular nerve (Clarke, Schonfed & Helling, 2003; Hall, 2006). It reflects a vestibulocollic reflex that is quick reflexive change in muscle tone (flexor or extensor depending on the muscle group) that occurs to stabilize the head following an unexpected translation (Zapala & Brey, 2004). VEMP also provides additional information about disturbances of vestibular function as a result of their dependence upon different vestibular receptors (Neck muscles via the medial vestibulospinal tract (MVST) and the

leg muscles via the Lateral Vestibulo Spinal Tract (LVST). (Colebatch & Halmagyi, 1992; Wilson, Fukushima, Rose, & Shinoda, 1995; Murofushi, Halmagyi, Yavor, & Colebatch, 1996; Uchino et al., 1997).

VEMP reflects vestibular system activity that is elicited by high intensity sounds and detected as a change in muscle potentials within the neck. Specifically it is recorded as a change in activity within SCM muscle secondary to stimulation of the vestibular system with acoustic signals at intensity levels of about 90dBHL and higher**.** VEMP appears as a biphasic (positive and negative) response in the latency region of 10 to 25ms. The response is actually a reflection wave form of transient inhibition (reduction) in SCM muscle activity secondary to acoustic stimulation of the saccule. Yoshie and Okudaira (1969) reported that the first positive–negative complex is often labeled as p13–n23. “p” for positive or “n” for negative and numbers 13 and 23 depict their latencies respectively. Amplitude has been reported to be a variable measure (from few microvolts to hundreds of microvolt) by majority of the researchers and it is attributed to muscle tension and intensity of stimulus. (Cheng & Murofushi, 2001a, 2001b; Colebatch, Halmagyi, & Skuse, 1994; Li, Houlden & Tomlinson, 1999; Ochi, Ohashi, & Nishino, 2001; Pyykko, Aalto, Gronfors, Starck, & Ishizaki, 1995; Versino, Colnaghi, Callieco, & Cosi, 2001; Wu & Murofushi, 1999 and Wu, Young, & Murofushi, 1999).

Anatomy and physiology of VEMP

The peripheral vestibular system includes 5 end organs: 3 (lateral, superior & posterior) semi circular canals, the saccule and the utricle. The semi circular canals respond to angular acceleration and the otoliths sense linear acceleration. The lateral and

superior semi circular canals are innervated by the superior vestibular nerve, while the posterior semi circular canal is innervated by inferior vestibular nerve. Moreover, the macula of the utricle is innervated by superior vestibular nerve and majority of macula of the saccule is innervated by the inferior vestibular nerve.

Displacement of the stapes footplate, as a result of sound or bone vibration causes the endolymph of the inner ear at the level of the oval window to vibrate. These vibrations are primarily dispersed to the endolymph in the cochlea but also a bit to the endolymph in the vestibular organ. Because all vibrations first enter the saccule before they reach the utricule and semi circular canals, the saccule is most sensitive to sound (Akin, 2004; Welgampola & Colebatch, 2005). It has been demonstrated that VEMP in humans originate from the saccular region and that only the Inferior Vestibular Nerve (IVN) contribute to the generation of VEMPs (Basta, Todt, Eisenschenk & Ernst, 2005).

Reflex pathway of VEMP

The pathway of the sacculo-collic reflex underlies the VEMP response. The sacculo-collic reflex is the saccular part of the vestibulo-collic reflex (VCR). The VCR is subserved by relatively direct, as well as indirect pathways linking vestibular nerve activity to cervical motor neurons innervating the neck musculature (Uchino et al, 1997 ; Takemura & King, 2005).

VEMP is a reflex arc which includes the receptor, the afferent pathway, central neurons, the efferent pathway and the end muscles (SCM). The receptor of VEMP is being mediated by the saccule (Townsend & Cody, 1971; Sheykholeslami & Kaga, 2002). The afferent pathway includes the inferior vestibular nerve and then to the

vestibular nuclei (Murofushi, Matsuzaki & Mizuno, 1998; Komatsuzaki & Tsunado, 2001).The efferent pathway is the vestibulospinal tract (Bickford, Jacobson, & Cody, 1964). The vestibular nuclei have descecnding connections to the spinal motor neurons. The lateral vestibulospinal tract (LVST) and the medial vestibulospinal tract (MVST) are considered as the possible efferent pathways for the SCM. Both LVST and MVST are found to project to the anterior horn cells of the cervical cord, which control all the neck muscles including SCM.

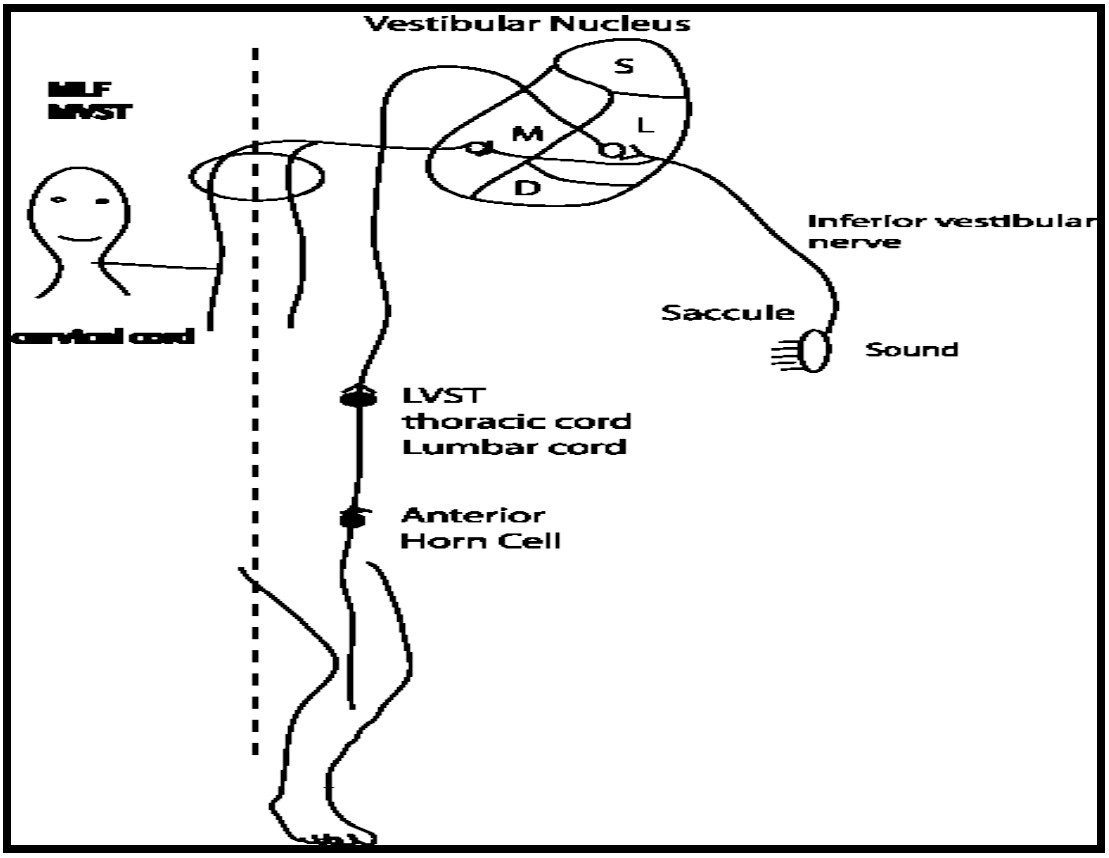

Figure 1. Reflex Pathway of the VEMP

More specifically, the VEMP response pathway originates in the saccular macula, followed by respectively the neurons of the vestibular (Scarpa's) ganglion, inferior vestibular nerve (IVN), lateral vestibular nucleus (LVN) and medial vestibule-spinal tract (MVST), and finally the motor neurons of some neck flexor, extensor and rotator muscles. The saccule has an excitatory effect on ipsilateral and contralateral neck extensor muscles and an inhibitory effect on ipsilateral and contralateral neck flexor and ipsilateral rotator SCM muscles (Uchino, Sakaki, Sato, Bai & Kawamoto, 2005 and Su et al, 2005)

In humans both the MVST as well as the lateral vestibule-spinal tract (LVST) project to the upper cervical spinal cord. The LVN is primarily connected to the LVST and therefore it is possible that in humans the LVST is involved into the pathway of the VEMP response. In cats a small population of saccular-neck-muscle pathways are scattered through the brain. It is possible that the VEMP response can be influenced by stimulation of the utricule and canals. Uhino et al., (2005) reported that besides main pathway there are probably more pathways involved in the VEMP response. In humans only the involvement of the inferior vestibular nerve and the motor neurons of some neck muscles are proven (Basta et al, 2005; Ferber-Viart, Dubreuil & Duclaux, 1999; Colebatch & Rothwell, 2004). In cats it has been shown that the utricule has both excitatory effect and inhibitory effect on neck rotator muscles, neck extensor and flexor muscles. So, it is possible that VEMP responses can be influenced by stimulation of the utricle and canals. In conclusion, the precise pathways that are involved in the generation of the VEMP response are still unknown.

Factors affecting VEMP

a. Stimulus related factors

A number of factors related to stimulus affect the VEMP response. Various method of stimulus delivery has been used to elicit VEMP response. This includes bone conduction (Sheykholeslami, Kermany, & Kaga, 2001, Sheykholeslami & Murofushi, Kermany, & Kaga, 2000), skull taps (Yang & Young, 2003 and Brantberg, Tribukait & Fransson, 2003), galvanic stimulation (Watson & Colebatch 1998) and air conduction (Wu, Shiao, Yang & Lee, 2007; Todd, Cody & Banks, 2000 and Akin, Murnane & Profitt, 2003). Sheykholeslami, Kermany, & Kaga (2000, 2001) reported that the largest amplitude of the bone conducted VEMP occurred in response to the 200Hz tone burst. Similarly, for the air conduction low frequency tone burst elicits larger VEMP amplitude and lower thresholds (Akin, Murnane & Proffitt, 2003). With the tone burst elicited VEMP, the p13 and n23 latency increases with increase in the rise time and fall time (Cheng & Muroushi, 2001a), and or plateau time (Cheng & Murofushi, 2001b). The suggested optimal plateau time for a 500 Hz tone burst with a 1-ms rise time is 2ms (Cheng & Murofushi, 2001a). Also there is increase in amplitude and no change in latency with increase in intensity (Ochi, Okashi & Nishino, 2001 and Akin, Murnane & Proffitt, 2003). The recommended use of rate is of 5Hz to decrease the test time and to increase signal to noise ratio (Wu and Murofushi, 1999). However, Brantberg & Frannson (2001) reported that the sensitivity was 100% at 4 per/sec repetition rate.

B. Recording related factors

Ferber-Viart et al (1997) recorded VEMP from both trapezius and SCM using clicks and reported that the latencies of the responses from the trapezius muscle were on average 3.8ms longer and response amplitude was 7.1 µV larger than those recorded from the SCM. Vijayshankar & Basavaraj (2008) evaluated the effect of Mode of SCM excitation. The different modes used were Subject's body rotated to one side for measurement on the opposite side in sitting position, Subject's body rotated to one side for measurement on the opposite side while lying in supine position and instructing the subject to press the forehead against a soft surface. Results indicated no difference in terms of latency of p13 & n23, amplitude of p13-n23 and between males and females. Stable latencies and greater amplitudes are best obtained with non inverting input with a large surface area (Zapala & Brey, 2004) and by placing non inverting electrode at the middle part of the muscle (Sheykholeslami, Murofushi & Kaga, 2001). VEMP requires a band pass filter of 10 -30 Hz to approximately 1000 Hz- 3000 Hz.

C. Subject related factors

Wang, Chen, Hseih & Young (2008) reported that the latencies were prolonged for both p13 and n23 for preterm babies as compared to full term babies. With advancing age there was a decrease in the p13-n23 amplitude and increase in the threshold of detection (Ochi & Ohashi 2003). In terms of gender, amplitude of VEMP across males and females is constant (Ochi & Ohashi, 2003; Brantberg & Fransson, 2001). However, p13 latency occurs on average 0.73ms earlier for women than men (Brantberg & Fransson, 2001). There is linear relationship between the amplitude of the response and

the mean level of EMG activity (Ochi, Ohashi & Nishino, 2001). Welgampola, Rosengren, Halmagyi, & Colebatch (2003) reported high correlation between average muscle tonus and the peak-to-peak amplitude of p13-n23**.**

Clinical applicability of VEMP

Disruption of any one or more of the vestibular organs could lead to dizziness or vertigo. The pathologies related to abnormal vestibular dysfunction are sometimes difficult to identify due to the insensitivity of current diagnostic tools. Although the caloric test is helpful in identifying dysfunction of the lateral semi circular canal, other procedures which are easy and reliable are needed to evaluate other peripheral vestibular organs (Zhou & Cox, 2004). VEMP is a valuable tool which helps in assessing inferior vestibular nerve and saccule. Its clinical significance can be briefed as below.

Central vestibular disorders

Vestibular Schwannoma/Acoustic Neuroma

VEMP is useful for detecting dysfunction of inferior vestibular nerve in patients with acoustic neuroma and can provide useful information in diagnosing acoustic tumors. (Matsuzaki, Murofushi, & Mizuno, 1999). Murofushi, Matsuzaki & Mizuno (1998) reported that, out of 21 patients with Vestibular Schwannoma, 80% of the patients had abnormal VEMP on the side of Vestibular Schwannoma and 3 patients had normal caloric response but had abnormal VEMPs and other 3 had abnormal caloric responses with normal VEMP responses. Similar results were reported by Murofushi, Matsuzaki & Mizuno (1999). Yang et al (2005) stated that Inter aural latency of VEMP can be used as a tool for diagnosing Vestibular Schwannoma. Tsutsumi, Tsunoda, Noguchi &

Komatsuzaki (2000) reported that none of the tests of VEMP, Pure Tone Audiometry, MRI and Caloric testing correlated with the size of tumor. However, absent VEMP's were obtained in patients with tumors arising from inferior Vestibular Nerve. It was demonstrated that VEMP results were not always correlated with the tumor location; also there is no correlation between VEMP and the tumor size.

Multiple sclerosis

Measurement of VEMPs could be helpful in detecting sub clinical vestibulospinal lesions in suspected multiple sclerosis. The latencies of a vestibulo spinal reflex can be prolonged in individuals with multiple sclerosis (MS). Shimizu, Murofushi, & Sakurai, (2000) reported delayed VEMP which they attributed to demyelination either of primary afferent axons at the root entry zone or secondary vestibulo spinal tract axons rather than to lesion involving vestibular nucleus. Versino et al (2002) administered VEMP, ABR and Saccadic tests in 70 patients with multiple sclerosis and confirmed brainstem and/or cerebellar signs, VEMP were abnormal in 31.4% of patients.

Brainstem disease

VEMP may be useful in differentiation of brainstem lesions affecting the rostral and caudal brainstem. Itoh et al (2001) reported that in 13 patients with various brainstem lesions and results indicated that mid brain lesion yielded normal VEMP, whereas pontine and medullary lesions involvement yielded abnormal VEMP. Chen & Young (2003) reported VEMP results in 5 patients with ischemic infarction and 2 with hemorrhage. Delayed or absent VEMPs were seen in 71% of the subjects. Chen, Young

& Seng (2002) described VEMP in 9 patients with CP angle tumors and results revealed that in 89% of the patients the VEMP was abnormal/absent on the affected side.

Auditory Neuropathy

Sheykholeslami, Schmerber, Kermany & Kaga (2005) studied 3 auditory neuropathy patients associated with balance disorders. Results revealed that VEMP responses were absent in the affected ear. They concluded that, in patients with isolated auditory neuropathy, the vestibular branch of the 8th cranial nerve and its innervated structures may also be affected. Similar findings were reported by Kumar, Sinha, Kumar, Barman (2007) who reported absent or prolonged latency and reduced amplitude of VEMP responses in 16 out of 20 ears. Whereas, Sheykholeslami, Schmerber, Kermany & Kaga (2005) observed absence of VEMP on left ear stimulation and a biphasic response with normal latency and amplitude on right-ear stimulation in a case with bilateral auditory neuropathy (AN).

Peripheral vestibular disorders

Meniere's disease (Endolymphatic hydrops):

VEMPs are useful in diagnosing Meniere's disease which results from endolympahtic hydrops (De waele, Hay, Diard, Freyss, & Vidal, 1999; Shojaku, Takemori, Kobayashi, Watanabe, 2001 and Robertson & Ireland, 1995) reported absence of VEMPs in 54% of the patients with MD. Murofushi, Shimizu, Takegoshi, and Cheng (2001) studied 43 patients with Meniere's disease and reported that VEMPs were absent or decreased in 51% of patients with Meniere's disease. Ohki, Matsuzaki, Sugasawa, &

Murofushi (2002), reported absence of or abnormal VEMPs in contra lateral ears that may have delayed enadolymphatic hydrops. Ribeiro, Almeida, Caovilla, & Gananca (2005) reported absence of VEMP responses in 7 cases, and increase in interaural amplitude difference ratios in one case in patients with unilaterally defined Meniere's disease. Seo, Node, Yukimasa & Sakagame (2003) investigated to see whether endolymphatic hydrops in Meniere's disease could be diagnosed comparing VEMP before and after furosemide administration (F-VEMP). Results showed positive findings in all the three subjects with furosemide administration. Murofushi, Matsuzuki, and Takegoshi (2001) reported improvement in VEMPs in some patients with unilateral Meniere's disease after oral administration of glycerol. This result suggests that abnormal VEMPs in patients with unilateral Meniere's disease could result from endolymphatic hydrops.

Superior semicircular canal dehiscence (SCD)

Minor (2000) stated that SCD is a condition in which the bone of the middle fossa overlying the superior semicircular canal is very thin or dehiscent, third window formed ensues in the labyrinthine system that results in increased compliance of the endolymph-containing compartment. Vestibular activation in response to auditory stimulation (Tulio phenomenon, Tullio, 1929) is reported to be present in SCD (Minor, Soloman, Zinreich & Zee, 1998). Vestibular evoked myogenic potential is highly sensitive and specific for Superior Canal Dehiscence, possibly better than CT scan. Brantberg, Bergenius, & Tribukait, (1999), reported abnormally large amplitudes and low thresholds of VEMP for 3 patients with SCD syndrome. Similar findings were reported by Streubel, Cremer, &

Carey (2001); Sven-Olrik, Carey, Weg & Minor (2001) and Brantberg, Bergenius, & Tribukait, (2004).

Vestibular hypersensitivity

VEMPs are indicative of a pathological increase in the normal vestibular sensitivity to sound. Colebatch, Rothwell, Bronstein & Ludman (1994) studied VEMP's in a patient with tulio phenomenon and found that the response elicited from the symptomatic side were larger in amplitude and had abnormally low thresholds but retained normal waveform configuration. Watson, Halmagyi & Colebatch (2000) also studied VEMP threshold on 4 patients with the tulio phenomenon and reported that the threshold of click evoked VEMPs were low for all affected ears (fair at 65 dB, one at 35 dB HL) and normal (70-90 dB nHL) for three unaffected ears.

Vestibular neuritis

VEMP has been employed in the assessment of individuals of inferior vestibular nerve in patients with vestibular neurolabyrinthitis. Absence of VEMP is reported in patients with Vestibular Neuro labyrynthitis which indicates the involvement of inferior vestibular nerve (Murofushi et al., 1996). Halmagyi, Aw, Karlberg, Curthoys & Todd (2002) reported that VEMPs were absent on the affected side for 2 patients with acute vertigo but normal lateral semicircular canal function as indicated by the caloric test. Halmagyi & Colebatch (1995) administered VEMP and caloric tests in individuals with vestibular neuritis. They reported that patients who did not have caloric responses on the affected sides indicated dysfunction of the lateral semicircular canal. VEMPs were normal in 6

patients, reduced in 5 patients and absent in 11 patients. Similar findings were also reported by Ochi & Ohashi & Watanabe (2003).

Conductive hearing loss

Interference of sound transmission due to some disorders such as chronic otitis media (COM) may lead to absent VEMPs (Young, Wu & Wu, 2002; Yang & Young, 2003; and Halmagyi, Colebatch, & Curthoys, 1994). Myogenic potentials may be evoked with the tapping method to elicit the absent VEMPs that result from middle ear. Yang & Young (2003) compared VEMP evoked by the tone burst and tapping in 22 ears with conductive hearing loss due to chronic otitis media. Results showed that 13 (59%) of the 22 ears showed positive VEMPs using the tone burst method whereas 20 ears (91%) displayed positive VEMPs by the tapping method. They concluded that while stimulating, sound is attenuated by middle ear pathology, VEMPs are expected to be poorly elicited under such conditions. Halmagyi, Colebatch, and Curthoys (1994) reported similar results in subjects with otosclerosis. They reasoned that as conduction across the middle ears ossicular chain is defective, VEMPs are attenuated or absent.

Degree of hearing loss

Townsend and Cody (1971) in view to prove VEMP as of saccular origin, stated that VEMP responses are preserved despite SNHL. Takegoshi and Murofushi (2003) reported that VEMP is present in patients who have severe deformation or absence of the cochlea but a functioning saccule. Rosengren and Colebatch (2006) reported normal

VEMP potentials can be recorded from patients with profound bilateral hearing loss which suggests VEMP is dependent purely upon vestibular activation.

VEMP in NIHL

Vestibular symptoms are evident in individuals with NIHL and VEMP is effective in evaluating a part of vestibular system (inferior vestibular nerve, saccule and sacculocculic reflex pathway). In this part of the review VEMP findings in NIHL is discussed.

Wang, Hsu & Young (2006) investigated the VEMP responses in 20 patients (29 ears) with acute acoustic trauma. Eighteen ears presenting normal VEMPs revealed hearing improvement in eight ears (44%) and unchanged hearing in ten ears (56%). However, hearing loss remained unchanged in all 11 ears (100%) with absent or delayed VEMPs, exhibiting a significant relationship between VEMP results and hearing outcome. Thus, VEMP test can predict the hearing outcome after acute acoustic trauma with a sensitivity of 44% and a specificity of 100%.

Wang & Young (2007) investigated the effect of chronic noise exposure on vestibular-evoked myogenic potentials. Twenty patients with chronic noise-induced hearing loss, with bilateral notched at 4 kHz, underwent audiometry, caloric, and vestibular evoked myogenic potential tests. Caloric and vestibular-evoked myogenic potential test results revealed abnormal responses in nine (45%) and 10 (50%) patients, respectively. However, when both results were considered together, the abnormal rate

reached 70% (14 of 20). The hearing threshold of 4 kHz significantly associated with vestibular-evoked myogenic potential results (i.e, vestibular-evoked myogenic potential was abnormal in patients with greater degrees of hearing loss), but not with caloric responses. so it was concluded that Patients with bilateral 4-kHz notched audiogram and hearing threshold of 4 kHz _ 40 dB may show abnormal(absent or delayed) vestibular-evoked myogenic potentials, indicating that the vestibular part, especially the sacculocollic reflex pathway, has also been damaged. Christiana, Kaushal & Bhat (2008) studied the deviancy of vestibular evoked myogenic potential in subjects with noise induced hearing loss. A total of 30 subjects (55 ears) with noise induced hearing loss were considered. Results indicated that, Out of the 55 ears, VEMP was absent in 16 (29.0%) ears. The latency was prolonged and the peak to peak amplitude was reduced in 19 (34.6%) ears. VEMP results were normal in 20 (36.4%) ears. So, VEMP was either abnormal or absent in 67% of NIHL subjects. It can also be concluded that the possibility of vestibular dysfunction, specially the saccule pathway is high in individuals with NIHL. However, Sohmer (1999) reported that at the higher intensity (113dB SPL), there was a clear affect on cochlea and not on vestibular end organs.

Thus, from the above mentioned review of literature, it is evident that individuals exposed to noise exhibit various vestibular symptoms suggesting vestibular involvement in these individuals. VEMP is an efficient tool to evaluate vestibular disorders associated with Inferior vestibular nerve and specifically with the saccular damage. There are reports of saccular damage in individuals exposed to excessive noise, but there is dearth of research in evaluating this saccular damage using the VEMP. So the present study was taken to evaluate the saccular function in individuals with noise induced hearing loss.

Chapter 3

Method

The present study was taken up with the aim of evaluating the involvement of the vestibular system (saccular function) in individuals with Noise Induced Hearing Loss (NIHL); to investigate which among the two structures - Cochlea and Saccule; is more susceptible to noise based on the results of Transient Evoked Otoacoustic Emission (TEOAE) and Vestibular Evoked Myogenic Potential (VEMP) tests. The study also aimed to evaluate the correlation between the vestibular symptoms and the VEMP results and to correlate the VEMP and TEOAE results with the degree of hearing loss.

Subjects:

To accomplish the objectives of the study, two groups of subjects were taken in the age range of 25 – 50 years. The control group consisting of individuals with normal hearing sensitivity with no exposure to noise and the clinical group consisting of individuals either having normal hearing sensitivity with 3-6 kHz notch or any degree of hearing loss with history of exposure to noise.

Control Group

The control group comprised of 30 individuals (60 ears) with normal hearing sensitivity. The subjects in this group were in the age range of 25-50 years with a mean age of 38.66 years.

Clinical Group

The clinical group consisted of 30 individuals (57 ears) with Noise Induced Hearing Loss (NIHL). The clinical group was further subdivided into two groups based on the vestibular symptoms:

Group I: This group included 15 subjects (28 ears) in the age range of 26 – 50 years with a mean age of 39.33 years. All the individuals in this group exhibited atleast one of the vestibular symptoms which is given in Appendix I. The duration of noise exposure for this group ranged from 4 – 28 years with a mean of 20.93 years.

Group II: This group included 15 subjects (29 ears) in the age range of 29 – 49 years with a mean age of 42.40 years. All the individuals in this group did not exhibit any of the vestibular symptoms. The duration of noise exposure for this group ranged from 8-26 years with a mean of 19.47 years.

Subject selection criteria:

Control group

Subjects fulfilling the following criteria were included in the control group:

1. Normal hearing sensitivity in both the ears with air conduction (AC) and bone conduction (BC) thresholds within 15 dB HL at frequencies from 250 Hz to 8000 Hz and 250 Hz to 4000 Hz in octaves and mid octaves respectively.
2. 'A' type tympanogram with normal ipsilateral and contralateral acoustic reflexes in both the ears.
3. Good speech identification scores i.e., $\geq$ 80%.
4. No history or presence of any middle ear pathology.

5. No history or presence of any neurological symptoms.
6. Uncomfortable level (UCL) was ≥ 95 dB HL for speech.
7. Presence of TEOAE responses with a SNR of +6 dB and the response reproducibility and stimulus stability of ≥80%.
8. No reports of high blood pressure and/or spondilitis.

Clinical group

Subjects fulfilling the following criteria were included in the clinical group:

1. Normal hearing sensitivity or sensorineural hearing loss with air borne gap not exceeding 10 dB HL with air conduction notch between 3- 6 kHz with any degree of hearing loss in the ear that was considered for the study.
2. Noise exposure for a duration of 8hrs per day, atleast for more than 2 yrs.
3. Speech identification scores were ≥ 80% or proportionate to the degree of hearing loss.
4. No history or presence of any middle ear pathology.
5. 'A' type tympanogram with presence/elevated or absence of ipsilateral and contralateral acoustic reflexes in both the ears.
6. Absence of space occupying lesions which was ruled out based on the Auditory Brainstem Response (ABR) test results and/or neurological reports.
7. Uncomfortable level (UCL) was ≥ 95 dB HL for speech.
8. TEOAEs showing either normal (in individuals with 3-6 kHz notch), abnormal or absent responses (in individuals having hearing loss indicating cochlear pathology).
9. No reports of high blood pressure and/or spondilitis.

Instrumentation

1. The puretone thresholds for both air conduction and bone conduction, speech identification scores and UCL for speech was obtained using a calibrated 2-channel diagnostic MADSEN ITERA audiometer.
2. A calibrated GSI- Tympstar immitance meter was used for both tympanometry and acoustic reflexometry.
3. A calibrated Otoacoustic Emission system ILO-V6 was used for the measurement of Transient Evoked Otoacoustic Emission (TEOAE).
4. The Vestibular Evoked Myogenic Potential (VEMP) and Auditory Brainstem Response (ABR) were recorded using IHS smart EP version 3.94 US Bez (Intelligent hearing system, Florida, USA) instrument. An Eartone 3A insert earphone was used to deliver the stimuli.

Test environment

All the audiological tests were carried out in a sound treated double room situation. The noise levels were within the permissible limits as per ANSI S3.1 (1991).

Procedure

1. A detailed case history was taken for all the individuals in the clinical group by administering the questionnaire developed by Tharmar (1990) which is given in Appendix-I.

   To obtain information about the presence or the absence of vestibular symptoms, the IInd section of dizziness questionnaire developed at Maryland hearing and balance center was used which is given in Appendix-II.

2. Puretone thresholds were obtained between 250 Hz to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction at all the octaves and mid octave frequencies, using the Modified Hughson and Westlake procedure (Carhart & Jerger, 1959).

3. Along with the conventional pure tone average (PTA) of three frequencies (500 Hz, 1 kHz and 2 kHz), PTA2 (average of 1 kHz, 2 kHz and 4 kHz) was also calculated to account for hearing sensitivity at high frequencies. This high frequency average of 3 frequencies was considered for the statistical analysis.

4. The speech identification scores were obtained at 40 dB HL above the speech recognition threshold using monosyllable list developed by Vandana (1998).

5. The uncomfortable loudness levels (UCLs) were determined by presenting the running speech through the headphones (TDH-39) at different intensities using ascending method. The UCL for speech was defined to be the hearing level at which the subjects considered the speech to be uncomfortably loud.

6. Immittance audiometry was carried out with a low probe tone frequency of 226 Hz. The ipsilateral and contralateral acoustic reflex thresholds were measured for 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz tones.

7. ABR testing was carried out to rule out any space occupying lesions. Initially the electrode site was cleaned with the help of skin preparing gel. Electrodes were then placed on the recording site with the conduction paste and then fixing them with the help of a surgical tape. The electrode impedance was checked and it was ensured that the impedance at each electrode site was ≤ 5 kΩ and the inter electrode impedance was within 2 kΩ. The Neuro-diagnostic ABR was carried out with the following protocol given in Table 1:

Table 1.

Parameters used to Record ABR

Stimulus Parameters	Stimuli	Click
	Stimulus duration	100 µsec
	No. of stimuli	1500
	Intensity	90 dBnHL
	Repetition rate	11.1/sec, 90.1/sec
	Polarity	Rarefaction
	Transducer	Insert ear phone (ER-3A)
Acquisition Parameters	Analysis time	10 msec
	Filter setting	100 Hz -3000 Hz
	Electrode placement	Cz-Non-inverting (positive) Test ear mastoid- Inverting (negative) Forehead- Ground
	Notch filter	On
	Artifact rejection	50 µV
	Number of channels	Single

The Subjects who had both the absolute latencies and the inter-peak latencies within the normal range, with good waveform morphology for both low and high repetition rates were considered as devoid of any space occupying lesions and were considered for the study.

8. The Otoacoustic emissions evoked by clicks trains presented at 84±3 dB pe SPL for the non linear clicks were recorded for subjects in both the control and the clinical group. The probe with an appropriate sized tip was positioned in the external ear canal and was adjusted to give a flat stimulus spectrum across the frequency range (500 Hz - 6000 Hz). The response was acquired using the averaging method. The two averaged TEOAE waveforms of each memory buffer composed of 260 accepted click trains, were automatically cross-correlated and used to determine the reproducibility of the measured TEOAEs by the software. Responses were accepted with a SNR of +6 dB and response reproducibility of ≥ 80%. A total of two responses were recorded to ensure the stability of the response. A minimum of one minute gap was given between any two recordings to reduce the influence of the one recording over another recording. Care was taken to ensure that the position of probe was not altered.

9. The VEMP was recorded for all the subjects in both the control and the clinical group. The subjects were instructed to sit straight and turn their head to the opposite side of the ear in which the stimulus was presented, so as to activate the ipsilateral Sternocleidomastiod (SCM) muscle, as it gives reliable and greater amplitude. Subjects were instructed to maintain the same throughout the test run. They were also instructed to avoid any extraneous movements of head, neck and jaw to elude muscle artifacts. The electrode site was cleaned with the help of a skin preparation gel. Silver chloride disc electrodes were placed on the recording site with a conducting gel. The absolute electrode impedances were ≤ 5 kΩ and inter-electrode impedances were ≤ 2 kΩ. While recording the VEMP, the tonic EMG level was maintained for each of the subject between 100 to 200 micro volts. A visual feed back which was available in the instrument was provided to each of the subject to monitor tonic EMG level of sternocleidomastoid muscle.

The protocol proposed by Wang & Young (2007) was used in the present study to record the VEMP which is given in Table 2.

Table 2.

Parameters used to Record VEMP

Stimulus Parameters	Type of stimuli	Tone burst
	Stimulus frequency	500 Hz
	Stimulus duration	2-1-2 cycle
	Intensity	95 dBnHL
	Repetition rate	3.1/sec
	Polarity	Rarefaction
	Transducer	Insert ear phone (ER-3A)
	Total number of stimuli	200
Acquisition Parameters	Analysis time	60 msec
	Filter setting	30 Hz -1500 Hz
	Notch filter	Off
	Electrode placement	Non- inverting (positive) – Midpoint of SCM muscle Inverting(negative) - Sternoclavicular junction Ground – Forehead
	Artifact rejection	40 µV
	Amplification	5000
	Number of channels	Single

The initial positive and negative polarity of waveform with peaks termed p13 and n23 on the basis of their latency was used to determine the presence or absence of VEMP response. Two consecutive runs were performed on the same ear to verify the reproducibility and averaged as the final response. The Subjects for whom the waveform replicability was good (for 2 waveforms) were considered for the study.

Analysis

- Statistical analysis was done to calculate the mean and the standard deviation for both p13 and n23 latencies, p13-n23 complex amplitude, TEOAE amplitude and PTA 2.
- Analysis was done to study the ear and group differences for both the control and the clinical group.
- Analysis was done further to find the correlation between the following:
  - ✓ TEOAE responses and VEMP responses.
  - ✓ Vestibular symptoms and VEMP results
  - ✓ VEMP results with the degree of hearing loss.
  - ✓ TEOAE results with the degree of hearing loss.

Chapter 4

Results

The present study was taken up with the aim of evaluating the saccular function in individuals with Noise Induced Hearing Loss (NIHL). The susceptibility of the cochlea and the saccule to noise was evaluated based on the results of the Transient Evoked Otoacoustic Emission (TEOAE) and Vestibular Evoked Myogenic Potential (VEMP) tests. The study also aimed to assess the correlation between the vestibular symptoms and the VEMP responses in individuals with Noise Induced Hearing Loss. Further, the correlation between the VEMP and the TEOAE results with the degree of hearing loss was also studied.

To accomplish the objectives, the VEMP latencies of p13 and n23, the p13-n23 complex amplitude, TEOAE amplitude and Pure Tone Average 2 (PTA2) were measured. Comparison of VEMP latency and amplitude and TEOAE amplitude between the groups and within the groups were carried out. To analyze the data, Statistical Package for the Social Sciences (SPSS) version 16 software was used. The following statistical analyses were carried out:

- Descriptive statistics to obtain the mean and the standard deviation for p13 and n23 latency, p13- n23 complex amplitude and the TEOAE amplitude measures.
- Mixed Analysis of Variance (Mixed ANOVA) to study the group effects, ear effects and interaction between the group and ear effect for p13 and n23 latency, p13- n23 complex amplitude and TEOAE amplitude measures.
- Paired sample t test to compare the ear effects for p13, n23 latency, p13- n23 complex amplitude and TEOAE amplitude measures.

- Since the data available for all parameters in the clinical group was less, Mann Whitney t test was done to cross check the results of Mixed Analysis of Variance for group comparisons for p13, n23 latency, p13- n23 complex amplitude and TEOAE amplitude measures.
- Pearson's correlation to see the correlation between the VEMP responses and the TEOAE responses with the degree of hearing loss.
- Cross tabulation for obtaining the frequency of the presence or absence of VEMP responses with the TEOAE responses and the presence or absence of the vestibular symptoms with the VEMP responses.

Results obtained from the different statistical analysis are discussed below for both the control and the clinical group:

A. VEMP results in the Control and the Clinical group

VEMP results for the Control group

VEMP was recorded from 30 subjects (60 ears) with normal hearing sensitivity. Out of the 60 ears, the VEMP response was present in 51 ears while it was absent in 9 ears. So, it can be inferred that in the control group, the response rate for the VEMP was 85%.

The VEMP response recorded from an individual with normal hearing sensitivity is given below:

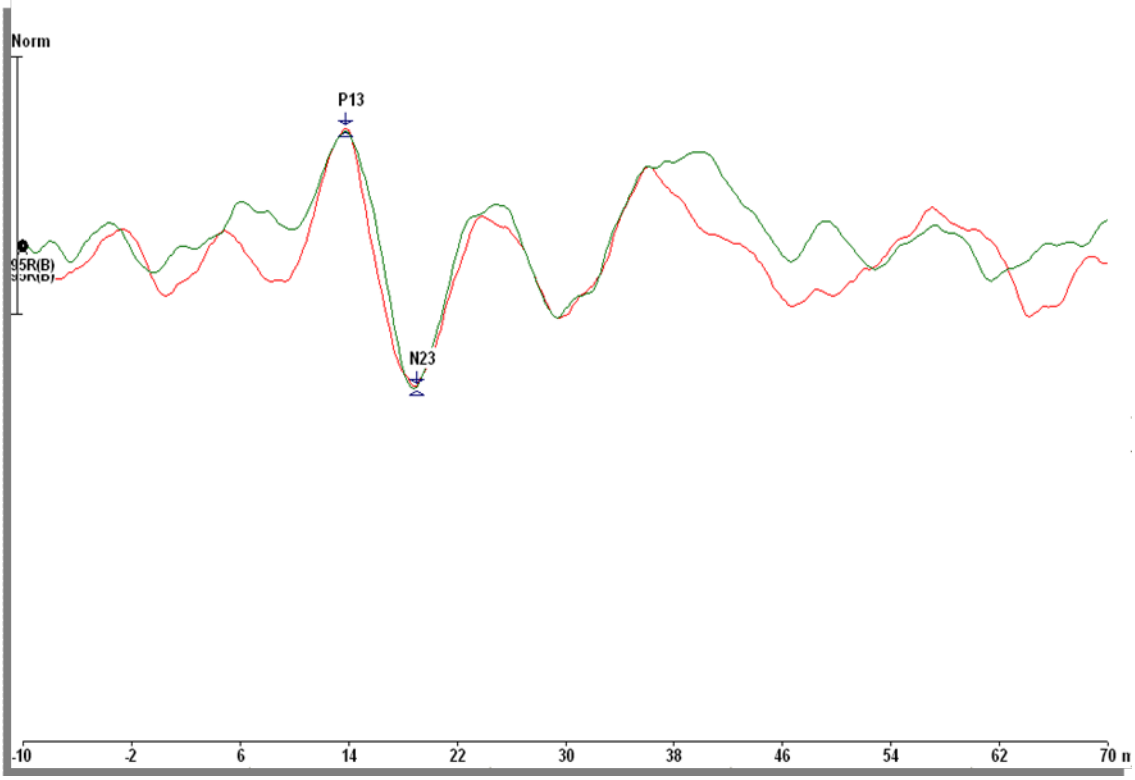

Figure 2. VEMP response showing p13 and n23 latency recorded for a 500 Hz tone burst presented at 95 dB nHL in an individual with normal hearing sensitivity.

The mean and the standard deviation for p13, n23 latency and p13- n23 complex amplitude and paired t test results obtained in individuals with normal hearing were calculated and the results are outlined in Table 3.

Table 3.

Mean, Standard Deviation (SD) and t-values with Level of Significance of p13, n23 Latency and p13- n23 Complex Amplitude of VEMP in the Control Group.

Parameter	*Right ear*		*Left ear*		*t-value*	*Significance*
	Mean	*SD*	*Mean*	*SD*	*(df=33)*	*level*
p13 latency	13.42	1.10	13.29	1.02	0.52	0.60
n23 latency	21.40	2.08	21.33	2.30	0.14	0.88
p13- n23 amplitude	55.75	16.45	55.59	18.90	0.05	0.95

From the Table 3, it can be inferred that the mean latencies of p13 and n23 was longer for the right ear as compared to the left ear. The variability for the p13 latency measure was higher for the right ear, while for the n23 latency, it was higher for the left ear. Overall, the variability for the n23 latency was greater as compared to the p13 latency. For the p13- n23 complex amplitude, the mean value was larger for the right ear than the left ear while the variability was higher for the left ear. Paired t test was administered to see the significant difference in VEMP parameters between the two ears. It was found that there was no significant difference between right and left ears for the p13, n23 latency and amplitude of p13- n23 complex which can be seen in Table 3.

VEMP results for the Clinical group

VEMP was recorded from 30 subjects (57 ears) with noise induced hearing loss. The VEMP response was present in 35 ears and was absent in 22 ears. So, it can be inferred that in the clinical group, the response rate for the VEMP was 61.4%.

Response patterns of VEMP latency in the clinical group

In the clinical group, the responses for VEMP latency measure had variations. The response patterns showed normal, prolonged or shortened latency. The different patterns observed for the latency measure is given in Table 4.

Table 4.

Response Patterns of the Latency Measures for the Clinical Group

Latency measure	*Responses (no. of ears)*		
	Normal	*Prolonged*	*Shortened*
p13	19	14	2
n23	20	12	3

It can be inferred from the Table 4 that in the clinical group for the p13 latency measure, 54.29% had normal responses, 40% had prolonged latency and 5.71% had shortened latency. For the n23 latency, 57.14% had normal responses, 34.29% had prolonged latency and 8.57% had the shortened latency.

The VEMP response recorded from an individual with noise induced hearing loss is given below:

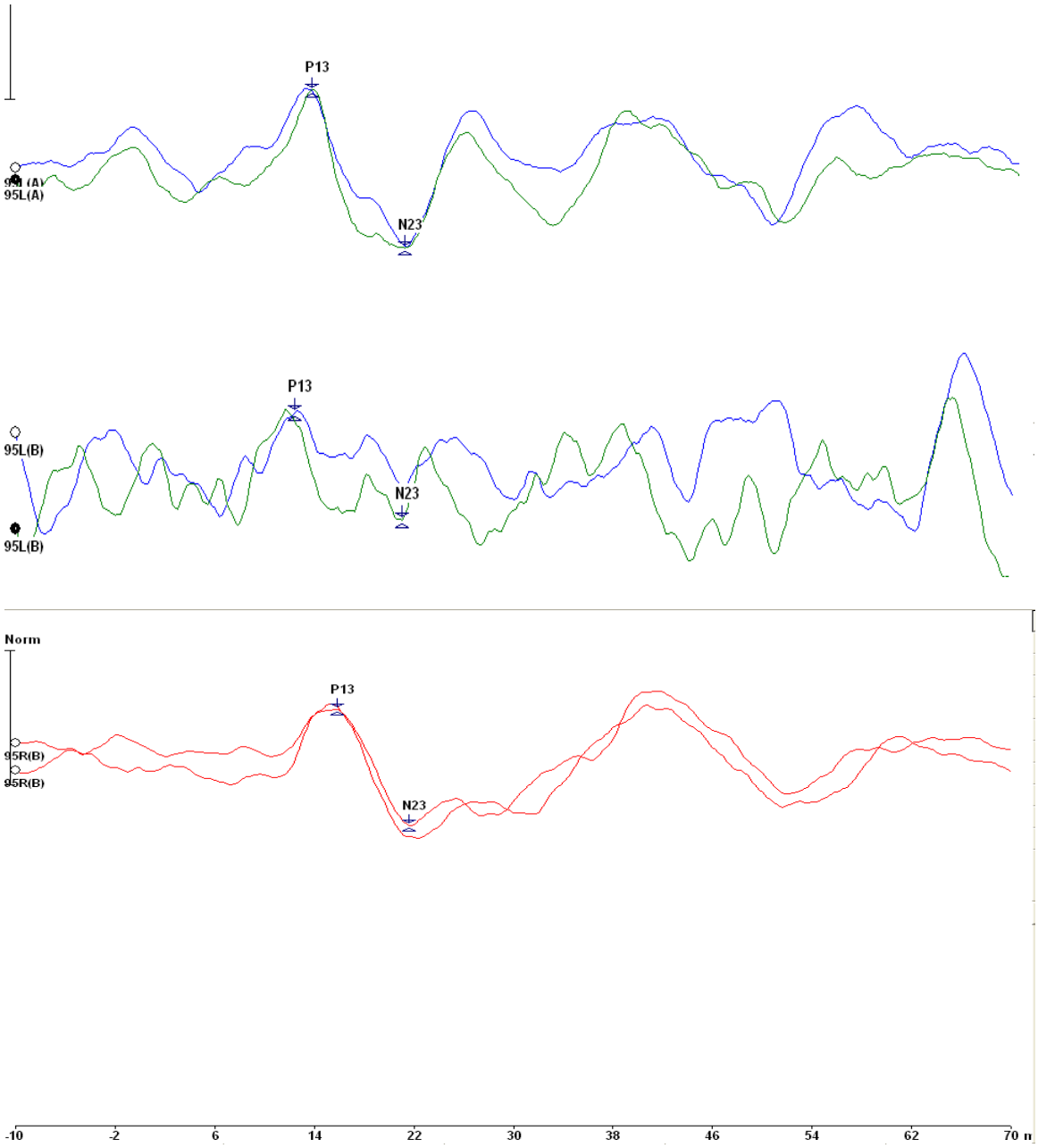

Figure 3. Waveform recorded from 3 subjects in the clinical group indicating normal latency, reduced amplitude and prolonged latency.

Response patterns of VEMP amplitude in the clinical group

In the clinical group, the responses for VEMP amplitude measure had variations. The response patterns either exhibited normal or reduced amplitude. The different patterns observed for the amplitude measure is given in Table 5.

Table 5.

Response Pattern for VEMP Amplitude Measure for the Clinical Group

Parameter	*Responses*	
p13 - n23 complex amplitude	*Normal*	*Reduced*
	17	18

It can be inferred from the Table 5 that in the clinical group for p13 - n23 complex amplitude**,** 48.57% had normal amplitude values while 51.43% had reduced amplitude.

The mean and the standard deviation for p13, n23 latency and the p13- n23 complex amplitude and the paired t test results obtained in individuals with noise induced hearing loss was calculated and the results are tabulated in Table 6.

Table 6.

Mean, SD and t- values with Level of Significance of p13, n23 Latency and p13- n23 Complex Amplitude in the Clinical Group.

Parameter	*Right ear*		*Left ear*		*t- value*	*Significance*
	Mean	*SD*	*Mean*	*SD*	*(df= 22)*	*level*
p13 latency	14.95	2.68	14.78	1.56	0.17	0.86
n23 latency	21.33	2.30	22.48	3.82	0.17	0.86
p13-n23 amplitude complex.	40.10	17.45	39.60	19.18	0.08	0.93

From the Table 6, it can be speculated that the mean latency value as well as the variability of p13 was larger for the right ear as compared to the left ear. For the n23 latency, the mean value was smaller for the right ear but the variability was higher for the left ear. For the p13- n23 complex amplitude, mean value was larger for right ear while the variability was higher for the left ear. Paired t test was administered to see significant difference in VEMP parameters between the two ears. It was found that there was no significant difference between right and left ears for the p13, n23 latency and p13- n23 complex amplitude which can be seen in Table 6.

B. Comparison of VEMP latency and amplitude measures across the control and the clinical group

Mixed Analysis of Variance (ANOVA) was done to evaluate the group effects, ear effects and interaction between the group and ear effect for p13, n23 latency, and p13- n23 complex amplitude .

Comparison of p13 latency between the control and the clinical group

The mean and the standard deviation for p13 latency in ms for both the control and the clinical are depicted in Figure 4.

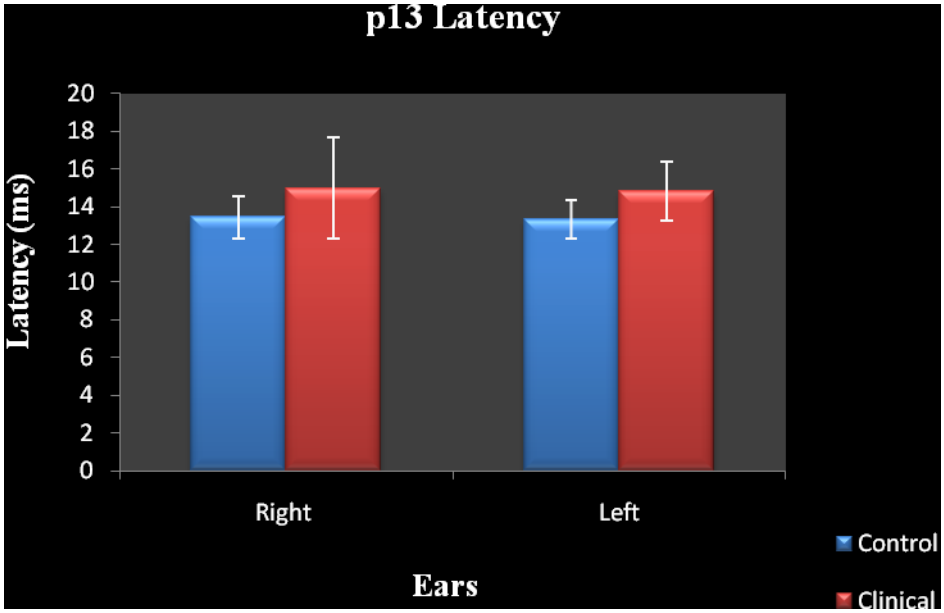

Figure 4. Mean and SD of p13 latency for both ears obtained in the control group and the clinical group.

It can be seen from the Figure 4 that the p13 latency value obtained for the control group is shorter than the clinical group for both right ear and left ear. The variability obtained for the p13 latency was more for the clinical group than the control group.

Mixed Analysis of Variance was done to evaluate group effect, ear effect and interaction between the group & ear for the p13 latency. The results revealed that there was a statistically significant difference in p13 latency values obtained between the control and the clinical group [$F(1, 33) = 14.08$, $p < 0.05$]. For within subjects, there was neither ear effect [$F(1, 33) = 0.15$, $p > 0.05$] nor the interaction effect between the group and ear [$F(1, 33) = 0.00$, $p > 0.05$].

Comparison of n23 latency across the control and the clinical group

The mean and the standard deviation for n23 latency for both the control and the clinical are shown in figure 5.

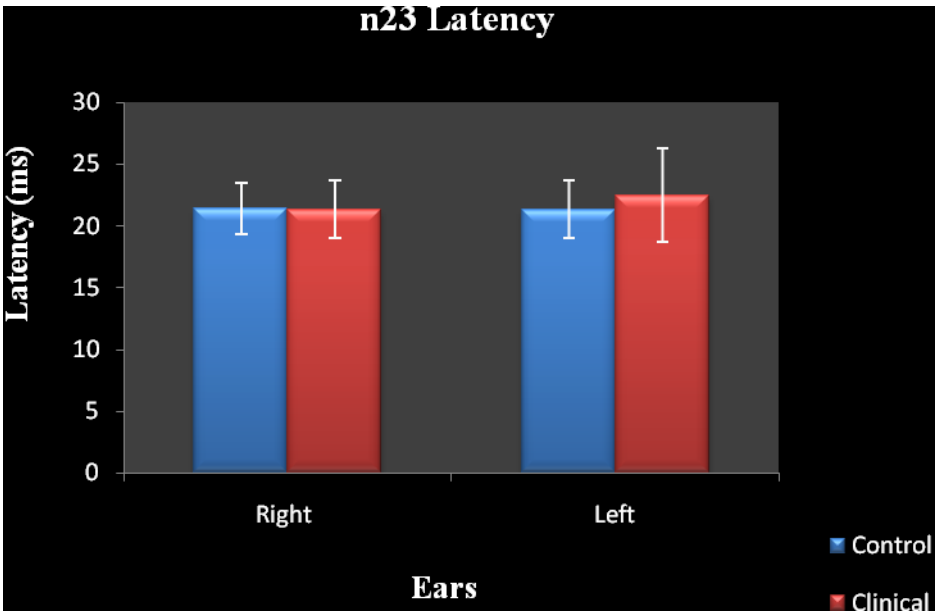

Figure 5. Mean and SD of n23 latency for both the control group and the clinical group.

It can be seen from the Figure 5 that the n23 latency value for the clinical group is longer than the control group for both the right and left ear. Greater variability was noticed for the clinical group for both right and left ear.

Mixed Analysis of Variance was done to evaluate the group effect, ear effect and interaction between the group & ear for the n23 latency. The results revealed that there was no statistically significant difference in n23 latency values between the control and the clinical group [$F(1, 33) = 2.10$, $p > 0.05$]. For within subjects there was neither ear effect [$F(1, 33) = 0.01$, $p > 0.05$] nor the interaction effect between the group and ear [$F(1, 33) = 0.06$, $p > 0.05$].

Comparison of p13- n23 complex amplitude for both the control and the clinical group

The mean and the standard deviation for p13- n23 complex amplitude for both the control and the clinical are presented graphically in Figure 6.

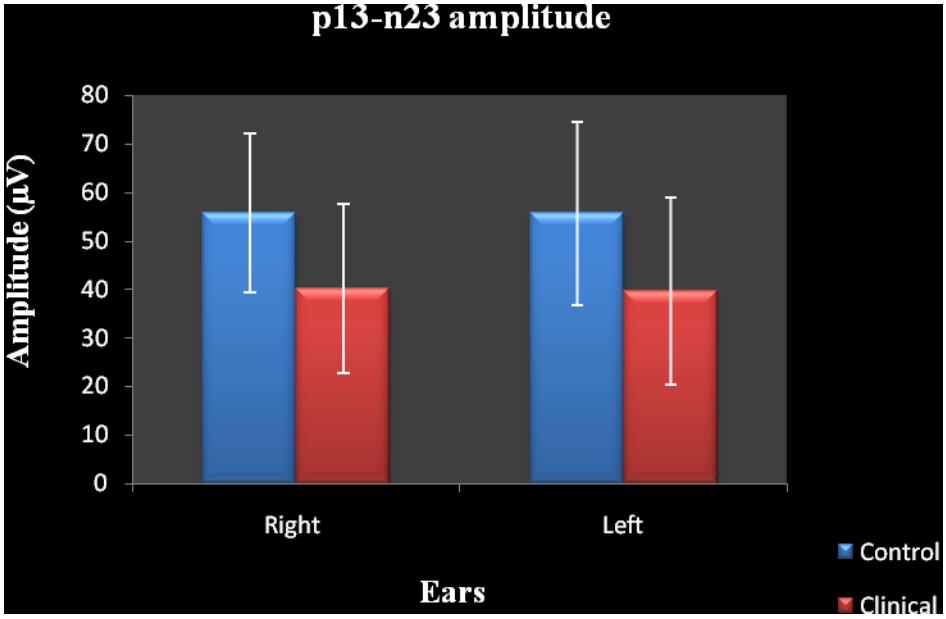

Figure 6. Mean and SD of p13- n23 complex amplitude for both the control group and the clinical group.

It can be seen from the Figure 6 that the p13- n23 complex amplitude for the clinical group in both the right and the left ear is smaller than the mean p13- n23 complex amplitude for the control group. The variability seen for the clinical group for both the ears is greater than that of the control group.

Mixed ANOVA results revealed that there was a statistically significant difference in p13- n23 complex amplitude values between the control and the clinical group [F (1, 33) = 8.60, $p < 0.05$]. For within subjects, there was neither ear effect [F (1, 33) = 0.01, $p > 0.05$] nor the interaction effect between the group and ear [F (1, 33) = 0.00, $p > 0.05$].

Because of the unequal sample size owing to the absence of response in many of the ears considered in the clinical group, Mann Whitney test was done for the group comparisons of p13, n23 latency and p13- n23 complex amplitude between the two groups. The results of Mann-Whitney test for p13, n23 latency and p13- n23 complex amplitude across the two groups are tabulated in Table 7.

Table 7.

Results of Mann-Whitney test for p13, n23 *Latency and* p13- n23 *Complex Amplitude across the Two Groups*

Parameter	*Z value*	*Asymp. Sig. (2-tailed)*
p13 right ear	-2.74	.00**
n23 right ear	-1.45	.14
p13 left ear	-2.29	.02*
n23 left ear	-.45	.64
p13- n23 amplitude right ear	-3.03	.00**
p13- n23 amplitude left ear	-3.69	.00**

Note. *p< 0.05, **p< 0.01

As seen from the Table 7, results of the Mann-Whitney test showed a significant difference between the clinical and the control group for p13 latency and p13- n23 complex amplitude for both the ears. While there was statistically no significant difference for the n23 latency between the two groups for both the right and left ears. This result is in accordance with the mixed ANOVA results indicating statistically significant difference for p13 latency and p13- n23 complex amplitude and statistically no significant difference for the n23 latency between the two groups.

C. TEOAE response in the Control and the Clinical group

TEOAE amplitude for the Control group

The TEOAE was recorded from 30 subjects (60 ears) with normal hearing sensitivity. The TEOAE being one of the criteria for the selection of subjects in the control group, the response rate for TEOAE was 100% response.

The mean and the standard deviation for TEOAE amplitude in individuals with normal hearing were calculated for both the ears separately and the results are shown in Table 8.

Table 8.

Mean, SD and t-values with Significance Level of TEOAE Amplitude in the Control Group

Parameter	*No. of ears*	*Right ear*		*Left ear*		*t- value (df=29)*	*Significance level*
		Mean	*SD*	*Mean*	*SD*		
TEOAE amplitude	60	17.07	4.17	14.57	4.07	2.73	0.08

From the Table 8, it can be observed that the mean amplitude value for the right was larger than the left ear. Also, the variability was higher for the right ear as compared to the left ear. Paired t test was done to see the significant difference between the two ears. It was found that there was no significant difference between right and left ears which can be seen in Table 8.

TEOAE amplitude for the Clinical group

The TEOAE was recorded from 30 subjects (57 ears) with noise induced hearing loss. The TEOAE was present in 20 ears while it was absent in 37 ears. So, it can be inferred that in the control group, the response rate for the VEMP was 35.09%.

The mean and the standard deviation for TEOAE amplitude in individuals with noise induced hearing loss were calculated for both the ears separately and the results are outlined in Table 9.

Table 9.

Mean, SD and t-values with Significance Level of TEOAE Amplitude in the Clinical Group

Parameter	*No. of ears*	*Right ear*		*Left ear*		*t- value (df=6)*	*Significance level*
		Mean	*SD*	*Mean*	*SD*		
TEOAE amplitude	57	9.14	2.17	10.05	2.12	1.05	0.33

From the Table 9, it can be seen that the mean amplitude value for the right ear was larger than the left ear; whereas the variability for the left ear was higher compared to the right ear. Paired t test was done to see significant difference between the two ears. It was found that there was no significant difference between right and left ears which can be seen in Table 9.

D. Comparison of TEOAE response across the Control and the Clinical group

Mixed Analysis of Variance was done to evaluate the group effects, ear effects and interaction between the group and ear effect for TEOAE amplitude. The mean and the standard deviation for TEOAE amplitude for both the control and the clinical as shown in figure 7.

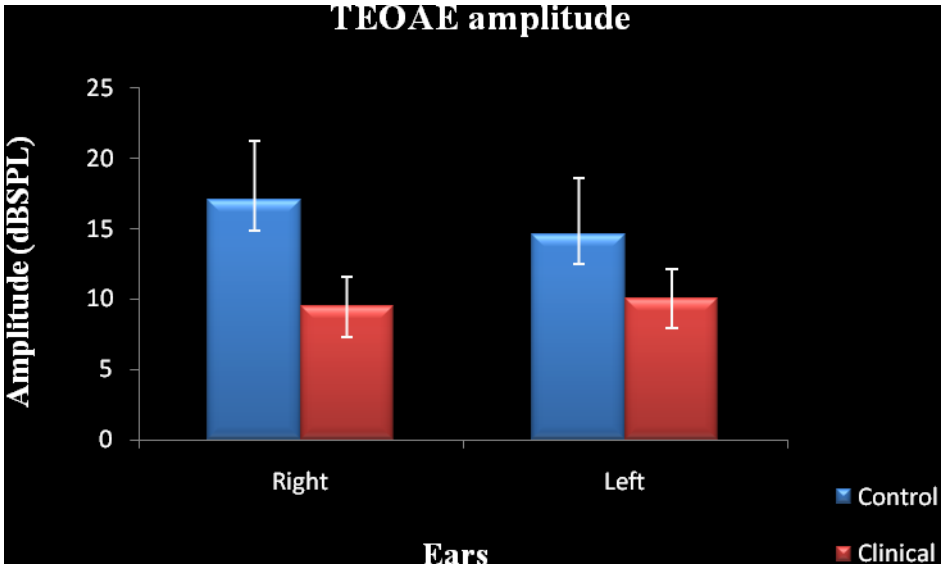

Figure 7. Mean and SD of the TEOAE amplitude for both the control group and the clinical group

It can be evident from the Figure 7 that the TEOAE amplitude is lesser for the clinical group than the control group for both the right and the left ear. The variability is less for the clinical group than for the control group.

Mixed ANOVA results revealed that there was a statistically significant difference in TEOAE amplitude values between the control and the clinical group [$F (1, 35) = 23.22$, $p < 0.05$]. For within subjects, there was neither ear effect [$F (1, 35) = 0.65$, $p > 0.05$] nor the interaction effect between the group and ear [$F (1, 35) = 3.04$, $p > 0.05$].

Because of the unequal sample size, Mann Whitney t test was done to cross check the results of mixed ANOVA for group comparisons of TEOAE amplitude across the two groups. The results of Mann-Whitney test for TEOAE amplitude across the two groups are tabulated in Table 10.

Table 10.

Results of Mann-Whitney test for TEOAE Amplitude for Both the Ears Between the Two Groups.

Parameter	*Z-*	*Asymp. Sig. (2-tailed)*
TEOAE amplitude Right ear	-4.15	.00**
TEOAE amplitude Left ear	-2.73	.00**

Note. **$p < 0.01$

As seen from the Table 10, results of the Mann-Whitney test showed a significant difference between the clinical and the control group for TEOAE amplitude in both the ears. This result is in accordance with the mixed ANOVA results indicating statistically significant difference for TEOAE amplitude between the two groups.

C. Comparison of TEAOE and VEMP responses in the Clinical group

To evaluate the susceptibility of the cochlea versus the saccule, the VEMP responses were compared with the TEOAE responses. This was done using the cross tabulations wherein comparison of the frequency of the presence or the absence of the responses for both VEMP and TEOAE were made. The frequency of presence and absence of VEMP and TEOAE responses in the clinical group are tabulated in table 11.

Table 11.

Frequency of Presence and Absence of VEMP and TEOAE Responses in the Clinical Group

Conditions	*Number of ears (57)*	*Percentage of occurrence (%)*
TEOAE present and VEMP present	14	24.56
TEOAE present and VEMP absent	5	8.77
TEOAE absent and VEMP present	21	36.84
TEOAE absent and VEMP absent	17	29.82

It can be observed from the Table 11 that the condition in which the TEOAE being absent with VEMP present was more prevalent, followed by both TEOAE and VEMP absent whereas, ears with both TEOAE and VEMP present had intermediate occurrence. It is also evident that only a small percentage has TEOAE present with VEMP being absent.

D. Comparison of VEMP responses with the vestibular symptoms in the clinical group

To compare the VEMP responses with the presence or absence of any vestibular symptoms, cross tabulations where done. Here the frequency of the presence or the absence of VEMP was correlated with the presence or absence of vestibular symptoms.

The Table 12 depicts the number of individuals exhibiting the vestibular symptoms in correlation with the absence of the VEMP responses (in %). The subjects exhibited either one or more than one symptom listed below. Two symptoms (Tullio phenomenon and walking in dark) which were not present in the questionnaire are listed in the table as it was reported by the subjects.

Table 12.

Vestibular Symptoms and the VEMP Response in the Clinical Group

Serial no.	*Vestibular symptoms*	*Number of subjects (N)*	*% of the absent VEMP*
1	Lightheadedness or swimming sensation in the head	3	66.66%
2	Blacking out or loss of consciousness	3	33.33%
3	Tendency to fall	3	33.33%
4	Objects spinning or turning around you	-	-
5	Sensation that you are turning or spinning inside	1	100%
6	Headache	5	80%
7	Pressure in the head	3	66.66%
8	Nausea or vomiting	1	100%
Additional symptoms not present in the questionnaire			
9	Walking in dark	3	66.66%
10	Tullio phenomenon	2	50%

It can be observed from the Table 12 that the correlation of VEMP in hierarchical order was maximum for symptom 5 and 8, followed by symptom 6. Further on, VEMP correlated equally for symptom 1, 7 and 9, followed by symptom 10. VEMP responses correlated least with symptom 2 and 3. The frequency of presence or absence of the vestibular symptoms and the VEMP responses in the clinical group are tabulated in Table 13.

Table 13.

Frequency of Presence or Absence of the Vestibular Symptoms and the VEMP Responses in the Clinical Group

Conditions	*Number of ears (57)*	*Percentage of occurrence (%)*
Vestibular symptom present and VEMP present	15	26.32
Vestibular symptom present and VEMP absent	13	22.81
Vestibular symptom absent and VEMP present	20	35.09
Vestibular symptom absent and VEMP absent	9	15.79

It is evident from the table 13 that the condition in which vestibular symptom was absent with VEMP response being present is most prevalent. Also the percentage of occurrence of the vestibular symptom being present with VEMP absent is higher. So, it can be inferred that out of 57 ears tested in the clinical group VEMP correlated with vestibular symptoms in 33 ears (57.89%).

E. Correlation between VEMP responses with the degree of hearing loss in the clinical group

Pearson's correlation analysis was done to evaluate the correlation between the VEMP & the degree of hearing loss for the clinical group. The results of the correlation analysis for the latency and amplitude measures of VEMP for the clinical group are outlined in Table 14.

Table 14.

r Value and Significance Level for p13 , n23 Latency and p13- n23 Amplitude w.r.t Degree of Hearing Loss for the Clinical Group.

Measure	*Parameter*	*r-*	*Significance level*
Latency	p13 right	-0.07	0.79
	p13 left	-0.18	0.72
	n23 right	-0.09	0.43
	n23 left	-0.26	0.26
Amplitude	p13- n23 right	-0.36	0.18
	p13- n23 left	0.08	0.71

It can be seen from the Table 14 that both the latency as well as amplitude measures are not correlated with the severity of the hearing loss for both right and left ear.

The VEMP responses across the different degrees of hearing loss are tabulated in Table 15.

Table 15.

VEMP Responses Across the Different Degrees of Hearing Loss

Severity of hearing loss	*Response (No. of ears)*	*No response (No. of ears)*	*% of present response*	*% of absent response*
Normal hearing with 3-6 kHz notch	3	1	75	25
Minimal	15	5	75	25
Mild	12	7	63.16	36.48
Moderate	3	2	60	40
Moderately severe	2	3	40	60
Severe	0	4	0	100

It is evident from the table 15 that in ears with normal hearing with 3-6 kHz notch and those with minimal degree of hearing loss showed equal percentages of presence and absence of VEMP responses. For degree of hearing from mild to severe loss, the frequency of presence of VEMP response decreased and occurrence of absence of response increased and for the severe degree of hearing loss none of the ears showed presence of VEMP.

F. Correlation between TEOAE responses with the degree of hearing loss in the clinical group

Pearson's correlation analysis was done to evaluate the correlation between TEOAE and the degree of hearing loss for the clinical group. The results of the correlation analysis for the TEOAE amplitude measures for the clinical group are outlined in Table 16.

Table 16.

r- Value and Significance Level for TEOAE Amplitude for the Clinical Group.

Parameter	*r-*	*Significance level*
TEAOE ampl. right	.55**	.00
TEAOE ampl. Left	.40*	.03

Note. *p< 0.05, **p< 0.01

It can be seen from the table 16 that the TEOAE amplitude is highly correlated with the severity of the hearing loss for both right and left ear.

The TEOAE responses across the different degrees of hearing loss are tabulated in Table 17.

Table 17.

TEOAE Amplitude Responses Across Different Degrees of Hearing Loss

Severity of hearing loss	*Response (No. of ears)*	*No response (No. of ears)*	*% of present response*	*% of absent response*
Normal hearing with 3-6 kHz notch	5	0	100	0
Minimal	9	11	45	55
Mild	6	13	31.58	57.89
Moderate	0	6	0	100
Moderately severe	0	5	0	100
Severe	0	4	0	100

It can be seen from Table 17 that percentage of presence of TEOAE response reduced as the degree of hearing loss increased. Also, it can be observed that from moderate degree of hearing loss, there was absence of TEOAE response.

It can be concluded from the obtained results that:

- The VEMP responses were abnormal in the clinical group (individuals with NIHL) as compared to the control group. The p13 latency measure is more sensitive than the n23 latency. Also, p13- n23 complex amplitude was found to be significantly reduced in the clinical group.
- TEOAE was found to be absent in most of subjects in the clinical group.
- The most prevalent condition was absence of TEOAE and presence of VEMP in the clinical group.
- VEMP correlated with the presence or absence of vestibular symptoms in more than 50% of the evaluated ears in the clinical group.
- There was a positive correlation between degree of hearing loss and TEOAE response was positive whereas no correlation between degree of hearing loss and VEMP responses.

Chapter 5

Discussion

The aims of the present study were fulfilled by collecting data from both the control and the clinical group using various test battery which included detailed case history, pure tone audiometry, immitance and reflexometry, transient evoked otoacoustic emissions, auditory brainstem responses and vestibular evoked myogenic potentials. Results from each of these tests were subjected to various statistical analysis and the results obtained are discussed in this chapter.

VEMP results for the Control group

In the present study the response rate for VEMP was found to be 85% for the control group. The overall response rate is consistent studies by Cody & Bickford (1969), Townsend & Cody (1971) and Vijayashakar (2008).

The mean p13 and n23 latency was 13.42 & 21.40 for right ear and 13.29 & 21.33 for the left ear. The mean values of p13 and n23 latencies of VEMP response in present study are almost in agreement with the studies on VEMP by various authors such as Murofushi, T., Shimiyu, K., Takegoshi, H., Cheng (2001), Akin, Murnane & Proffitt (2003), Kaushal (2006), Wu, Shiao, Yang & Lee (2007) and Vijayashakar (2008).

In the present study the mean p13-n23 complex amplitude was 55.75 µV with SD of 16.45 for right ear and 55.59 with SD of 18.90 for the left ear. This finding is in accordance with Vijayashankar (2008) wherein the amplitude value was around 50 µV and SD was about 25 µV. The amplitude in the control group is slightly greater and the variation is less in this present study as compared to the study by Vijayashankar (2008).

The reason for this could be that the EMG level in the present study was controlled in the range of 100-200 micro volts as compared to Vijayashankar (2008) wherein the EMG maintained was lower (30-50 micro volts). It is possible that the EMG level greater than 50 micro volts would have raised the mean amplitude value of p13-n23.

VEMP results for the Clinical group

The response rate in the clinical group for the occurrence of VEMP was found to be 61.4% and was absent in 38.6%. The present VEMP responses for the clinical group are inclusive of prolonged p13 latency (40%) and reduced p13-n23 complex amplitude (51.43%) response.

The results of the present study are in consonance with Christiana, Kumar and Bhat (2008). They reported that VEMP was abnormal or absent in 35 ears (67%) and normal in 20 (36.4%) ears out of 55 NIHL ears evaluated. Out of the 35 ears, VEMP was absent in 16 ears. The latency was prolonged and the peak to peak amplitude was reduced in 19 ears. They concluded that the possibility of vestibular dysfunction, specially the saccule pathway is high in individuals with NIHL and that VEMP can be employed in these individuals to assess sacculo-collic reflex.

Wang & Young (2007) carried out VEMP testing in individuals with NIHL and reported abnormal VEMP responses in 50% of the subjects, which included absent VEMPs in eight and delayed VEMPs in three (one patient had absent in right ear and delayed response in left ear) subjects. The absence of VEMP reflects a lesion affecting the sacculocollic reflex pathway, whereas the delayed VEMP latencies are indicative of a

retro-labyrinthine or brainstem lesion, especially in the vestibule-spinal tract (Wang & Young, 2006).

There are discrepancies seen in the quantitative measures of each of the considered parameter, and this can be attributed to the number of subjects, years of exposure to noise and other recording parameters adopted in different studies.

Comparison of VEMP latency and amplitude measures across the control and the clinical group

p13 and n23 latency

The p13 latency was found to be significantly prolonged in the clinical group as compared to the control group. However, there was statistically no significant difference in the n23 latency though it was prolonged in the clinical group. Also the SD was more for n23 latency measure for both the control and clinical group in comparison to the p13 latency. The SD was greatest for the n23 latency measure in the clinical group.

The results of the present study are in close agreement with the study by Wang and Young (2007) and Christiana, Kaushal & Bhat (2008). Wang and Young (2007) reported specific prolongation of p13 latency, but in Christiana, Kaushal & Bhat (2008) the prolongation was reported for both the peak latencies.

Another speculation of the p13 being prolonged with n23 within normal limits may be reasoned due to the standard deviation value. The SD of n23 was greater than that of p13, resulting in a wider normal range of n23 than p13. Also, the literature on the response consistency of VEMP which was reviewed by Ferber, Dubreuil, & Duclaux (1999) based on the studies done by Cody & Bickford (1969), Townsend & Cody (1971),

Colebatch, Halmagyi, & Skuse (1994) and Robertston & Ireland (1995) which suggested that the response consistency was not 100% in all the subjects for both the waves of VEMP. However, consistency is more for p13 and less for n23 of VEMP response.

Thus, p13 was found significantly prolonged in the clinical group suggesting saccular dysfunction. However, n23 was found to be not much different between two groups which are attributed to greater inter-subject variation in both control and clinical group with maximum variation for clinical group.

p13- n23 complex amplitude

The mean of p13- n23 complex amplitude in the clinical group was 40.10 with a SD of 17.45 for right ear and 39.60 with a SD of 19.18 for left ear. In the clinical group, 48.57% had normal amplitude values while 51.43% had reduced amplitude. The VEMP amplitude (p13-n23 complex) was found to be significantly reduced as compared to the control group in the current study.

Christiana, Kaushal and Bhat (2008) reported the amplitude being reduced in 19 ears accounting for 34.6% of the abnormal responses. The percentage of the reduced amplitude was higher (51.43%) in the present study which may be because of the difference in the duration as well as the intensity of noise exposure in the study group in the two studies. Also, the variation in the amplitude measure may be due to the mean level of the electromyographic activity (Colebatch, Halmagyi, & Skuse, 1994). It has also been reported in the literature that there are variations in VEMP amplitudes, from a few microvolts to several hundred microvolts, depending on the muscle tension and the intensity of stimuli (Cheng & Murofushi, 2001a, 2001b; Colebatch, Halmagyi, & Skuse,

1994; Li, Houlden, Tomlinson, 1999; Ochi, Ohashi, & Nishino, 2001; Pyykko, Aalto, Gronfors, Starck, & Ishizaki, 1995; Versino, Colnaghi, Callieco, & Cosi, 2001; Wu & Murofushi, 1999; Wu, Young, & Murofushi, 1999).

Hence, it could be concluded that although reduced VEMP amplitude does indicate abnormality, it cannot be conclusive as long as the intensity of the signal and more importantly the muscle tension is controlled.

Comparison of TEOAE response across the Control and the Clinical group

The TEOAE response was present in all the individuals in the control group accounting for 100% response rate. The TEOAE amplitude was greater for right ear than the left ear. This is in consonance with the literature where prevalence of TEOAE response is reported to be 96%-100% in individuals with normal hearing sensitivity (Probst, Lonsbury, Martin & Coats, 1987). They also reported that right ear OAE's were much greater than the left ear OAE's. Similar findings of right ear OAE's being much greater than the left ear OAE's were reported by Moulin, Collet, Veuillet and Morgan (1993).

In the clinical group TEOAE's were present in only 35.09% of 57 ears evaluated and the amplitude for both right and left ear was significantly reduced compared to the individuals with normal hearing sensitivity, also amplitude for the right ear was lesser than the left ear. The findings of the study are similar to as reported by Shupak et al (2007). They reported of reduced TEOAE amplitudes in individuals during the first two years of occupational noise exposure.

Kowalska and Kotylo (2007) reported that changes in OAE's exactly follow the changes in audiogram related to noise exposure and that patients with NIHL show amplitude reduction and or complete absence of OAE's. They stated that the rationale for using OAE's in patients with NIHL includes the clinical aspect that is confirmation of cochlear lesion.

Comparison of TEAOE and VEMP responses in the Clinical group

The results of the present study revealed that condition in which the TEOAE absent with VEMP present was more prevalent, followed by both TEOAE and VEMP being absent. The condition in which TEAOE present and VEMP absent was least prevalent. So, it can be concluded that it is the cochlea which is more susceptible to noise exposure compared to the saccular part of the vestibular system.

The findings of the present study are well supported by the anatomical positioning of the cochlea and saccule wherein the cochlea is at more proximity to the stapes than the saccule. When the ear is exposed to noise cochlea will be more susceptible. Hence, the outer hair cells of the cochlea would be affected before the macula of the saccule resulting in abnormal TEOAE's prior to abnormal VEMP responses. Ceranic. B (2007) stated that owing to mechanical force of noise exposure, the most extensive morphological changes are expected to be in the cochlea.

Wang & Young (2007) reported abnormal VEMP responses in NIHL subjects and explained that the mechanism of noise-induced hearing loss can be classified either as direct mechanical injury or metabolic damage to the organ of Corti. Talasaka & Schacht (2007) reported that the direct mechanical damage is mostly caused due to acoustic

trauma and chronic noise exposure. This leads to metabolic overstimulation, which produce toxic reactions resulting in cell death. The metabolic damage includes ischemia, generation of reactive oxygen species (ROS), toxic free radicals, metabolic exhaustion, and ionic imbalance in the inner ear fluid. The extent of noise effect on cochlear blood flow appears to be heavily influenced by the duration and intensity of the noise exposure (Lamm & Arnold, 2000). Although the cochlea receives its blood supply mainly from the common cochlear artery, the saccule is supplied by anterior and posterior vestibular arteries; all these arteries originate from the labyrinthine artery. Therefore, as the duration and intensity of the noise exposure increases, there is reduction in blood flow which leads to permanent hearing threshold shifts and abnormal VEMP responses.

Comparison of VEMP responses with the vestibular symptoms in the clinical group

In the present study "headache" was the most prevalent vestibular symptoms and correlation with VEMP was found to be good. Although, there were other vestibular symptoms that were in good correlation with VEMP, the numbers of subjects exhibiting these particular symptoms were less. Also, some individuals exhibited multiple symptoms and abnormal VEMP findings making it difficult to precisely point out the vestibular symptom best correlating with VEMP. This finding is in close relation with the Kumar and Barman (2006). In their study they correlated the different dizziness symptoms with VEMP responses and reported that VEMP can be associated with symptoms like "objects spinning/turning around you", tendency to fall, loss of balance when walking, nausea or vomiting. They concluded that subjects who complain these symptoms are likely to have saccular pathway lesions. But, they did not correlate VEMP

responses with multiple symptoms, as many would have more than one symptoms of dizziness.

Thus, it can be concluded that vestibular symptoms that would originate from saccular origin and or inferior vestibular nerve pathologies may result in abnormal VEMP responses.

Correlation between VEMP responses with the degree of hearing loss in the clinical group

The results of the present study revealed that the degree of hearing loss did not correlate with the VEMP results. Similar findings have been reported by Hsu, et al., (2008) who assessed the saccular functioning in guinea pigs that were exposed to noise. They also evaluated the hearing sensitivity using ABR and attempted to correlate the ABR results with the VEMP results. They found that with short duration of noise exposure to noise there was abnormality in VEMP (prolonged or absent) and shift in the thresholds on ABR (temporary Threshold Shift). Both the VEMP and ABR responses returned to normal values within 2-4 days, however, recovery of VEMP response to normalcy was faster than recovery of threshold on ABR. In terms of long term exposure to noise in these animals, permanently absent VEMP responses were observed in 78% on the 30^{th} day of exposure, whereas 100% absent VEMP responses were seen on the day 0 of noise exposure. Permanent Threshold Shift was also confirmed with elevated threshold found on ABR for 83% on the 30^{th} day. They compared the abnormal percentages between VEMP and ABR and found statistically no significant differences on post–noise

exposure days 0 through 30. They concluded that the saccule can exhibit temporary or permanent functional loss resembling hearing threshold shifts in guinea pigs following noise exposure. Recovery of VEMP precedes restoration of hearing threshold after damage from short-term noise exposure. Conversely, permanent VEMP loss after long-term noise exposure may reflect permanent hearing threshold shifts. Wang, Hsu and Young (2006) reported that VEMP test may provide another clue for assessing the hearing outcome. He concluded that VEMPs in patients after acute acoustic trauma showed absent or delayed VEMP responses which indicate poor prognosis with respect to hearing improvement. Young and Cheng (2007) reported that in NIHL absent VEMP responses were observed with increasing degree of hearing loss.

In the present study though there was no correlation between the VEMP responses and degree of hearing loss, the trend of response suggested that as the degree increased the frequency of presence of VEMP response decreased and occurrence of absence of VEMP response increased and for the severe degree of hearing loss none of the ears showed presence of VEMP. Wang and Young (2007) reported that in patients who were exposed to noise with bilateral 4 kHz notched audiogram and hearing threshold of 4 kHz $\geq$ 40 dB showed abnormal (absent or delayed) vestibular-evoked myogenic potentials, indicating that the vestibular part, especially the sacculocollic reflex pathway, has also been damaged. Hara and Kimura (1993) attributed the abnormal VEMP findings to the differential sensitivity (possibly because of membrana limitans) of cochlea and saccule from that of other vestibular structures (utricle and saccule).

It can be concluded that in general, VEMP does not correlate with degree of hearing loss, but in cases of noise exposure (acoustic trauma and chronic noise exposure) higher degree of hearing loss may affect the VEMP response and thus may be indicative of saccular involvement.

Correlation between TEOAE responses with the degree of hearing loss in the clinical group

The results of the present study revealed that there was a high correlation between degree of hearing loss and TEOAE response (r=0.55), with majority of individuals in the clinical group having absence of TEOAE's (64.91%). Findings of the present study are in consonance with literature. Probst, LonsBury, Martin & Coats (1987) demonstrated that noise induced high frequency hearing loss was associated with a reduction in the number of prominent peaks in the spectra of TEOAE's and that TEOAE's were absent for hearing loss above 25-30 dB. Corge et al, (1997) reported that evoked OAE's are reduced in amplitude by cochlear hearing loss and hearing losses exceeding 40-60 dB usually show no detectable emission.

Desai, Reed, Cheyne, Richards and Prasher (1999) reported that in 56% of the subjects with noise induced hearing loss, transient evoked otoacoustic emissions (TEOAEs) were absent as compared to controls (0%). They concluded that the reduction in incidence of OAEs in the noise exposed group may be associated with sensory cell damage to localized cochlear regions sub-serving specific frequencies.

From the above it can be concluded that noise exposure have severe effect on the OHC's and that TEOAE's are very sensitive to any damage to the OHC's. Hence, the strong correlation between TEOAE and degree of hearing loss is rightly justified.

Chapter 6

Summary and Conclusion

Noise Induced Hearing Loss is one of the most prevalent causes of Occupational hearing loss. Cochlear damage in individuals with Noise Induced Hearing Loss (NIHL) is well established. Transient Evoked Oto-Acoustic Emissions are well known as a sensitive tool for cochlear damage, but along with cochlear damage there are reports of vestibular involvement. There are numerous reports of the presence of vestibular symptoms in individuals who are exposed to noise. The saccule, among the vestibular structures can be considered as most susceptible to noise due to its thinness and the least ability to withstand the force and or pressure. Also it's distance from the stapes makes it's the probable structure to be damaged by noise. Vestibular Myogenic evoked potential is an efficient tool in evaluating saccular and Inferior Vestibular Nerve functioning. Hence, it may provide useful information about saccular and inferior vestibular nerve functioning in individuals with NIHL. Hence, the present study was aimed:

- ✓ To evaluate the functioning of the saccule in individuals with Noise Induced Hearing Loss.
- ✓ To assess the susceptibility of cochlea or saccule to noise exposure, based on Transient Evoked Otoacoustic Emission (TEOAE) and Vestibular Evoked Myogenic Potentials (VEMP) results.

- ✓ To know whether the vestibular system damage is associated with saccular dysfunction in individuals with NIHL, by correlating the vestibular symptoms and VEMP.
- ✓ To know whether there is any relationship between degree of hearing loss and saccular dysfunction in subjects with NIHL.

To arrive at the objectives, 30 (60 ears) individuals with normal hearing sensitivity (control group) and 30 (57 ears) individuals with noise induced hearing loss (clinical group) in the age range of 25-50 years were taken. All the individuals in both the control and clinical group were tested on a test battery including case history, pure tone audiometry, immittance & reflexometry, Oto-Acoustic Emissions (TEOAEs), ABR (Site of lesion testing) and Vestibular evoked Myogenic potential (VEMP). Two questionnaires were administered to obtain information about noise exposure and presence or absence of vestibular symptoms. The VEMP was recorded with a 500 Hz tone burst stimuli at 3.1 repetition rate at 95 dBnHL for both the groups. The waveform obtained from both the groups was analyzed by three experienced judges for p13, n23 latencies and p13-n23 complex amplitude. From the obtained data the mean, standard deviation and range were calculated and following statistical analysis were done.

- Mixed Analysis of Variance (Mixed ANOVA) to study the group effects, ear effects and interaction between the group and ear effect for p13 and n23 latency, p13- n23 complex amplitude and TEOAE amplitude measures.
- Paired sample t test to compare the ear effects for p13, n23 latency, p13- n23 complex amplitude and TEOAE amplitude measures.

- Since the data available for all the parameters in the clinical group was less, Mann Whitney t test was done to cross check the results of Mixed ANOVA for group comparisons for p13, n23 latency, p13- n23 complex amplitude and TEOAE amplitude measures.
- Pearson's correlation to see the correlation between the VEMP and the TEOAE responses with the degree of hearing loss.
- Cross tabulation for obtaining the frequency of the presence or absence of VEMP responses with the TEOAE responses and the presence or absence of the vestibular symptoms with the VEMP responses.

The results obtained from the various statistical procedures are as follows:

Control group:

VEMP

- The response rate for VEMP was 85%.
- The mean latencies of p13 and n23 were longer for the right ear as compared to the left ear. The variability for the p13 latency measure was higher for the right ear, while for the n23 latency, it was higher for the left ear.
- The variability for the n23 latency was greater as compared to the p13 latency.
- The mean p13- n23 complex amplitude was larger for the right ear than the left ear while the variability was higher for the left ear.
- No significant difference between the right and the left ears for the p13, n23 latency and p13- n23 complex amplitude.

TEOAE

- The TEOAE had a response rate of 100% with the mean amplitude value for the right ear larger than the left ear. Also, the variability was higher for the right ear as compared to the left.

Clinical group:

VEMP

- The response rate for VEMP was 61.4%. There was prolongation of p13 latency in 40% of the ears, while for n23 latency it was 34.29%. For the p13- n23 complex amplitude, 51.43% of the ears exhibited reduced amplitude.
- The mean latency value as well as the variability of p13 was larger for the right ear as compared to the left ear.
- For the n23 latency, the mean value was smaller for the right ear but the variability was higher for the left ear.
- For the p13- n23 complex amplitude, mean value was larger for the right ear while the variability was higher for the left ear.
- No significant difference between right and left ears for the p13, n23 latency and p13- n23 complex amplitude.

TEOAE

- The response rate for TEOAE was 35.09%. The mean amplitude value for the right ear was larger than the left ear; whereas the variability for the left ear was higher compared to the right ear.

Comparison of VEMP and TEOAE responses across control and clinical group:

- The p13 latency value obtained for the control group was shorter than the clinical group for both the right ear and the left ear. The variability obtained was more for the clinical group than the control group.
- The n23 latency value for the clinical group was longer than the control group for both the right and left ear. Greater variability was noticed for the clinical group for both right and left ear.
- The p13- n23 complex amplitude for the clinical group in both the right and the left ear was smaller than the mean p13- n23 complex amplitude for the control group. The variability seen for the clinical group for both the ears was greater than that of the control group.
- The TEOAE amplitude was lesser for the clinical group than the control group for both the right and the left ear. The variability was less for the clinical group than for the control group.
- Statistically significant difference was observed for p13 latency, p13- n23 amplitude and TEOAE amplitude between the clinical and the control group but no significant difference for the n23 latency.

Comparison of TEAOE and VEMP responses in the Clinical group

The condition in which the TEOAE was absent with VEMP present was more prevalent, followed by both TEOAE and VEMP absent. Whereas, ears with both TEOAE and VEMP present had intermediate occurrence.

Comparison of VEMP responses with the vestibular symptoms in the clinical group

- The correlation of VEMP being absent was maximum for symptom of "Sensation that you are turning or spinning inside" and "Nausea or vomiting" followed by the symptom "headache". VEMP responses correlated least with symptom of 'Blacking out or loss of consciousness" and "tendency to fall".
- The VEMP responses correlated with the vestibular symptoms in 33 ears (57.89%).

Correlation between VEMP and TEOAE responses with the degree of hearing loss in the clinical group

- Both the latency as well as amplitude measures are not correlated with the severity of the hearing loss for both right and left ear.
- However, for degree of hearing loss from mild to severe, the frequency of presence of VEMP response decreased and occurrence of absence of VEMP response increased and for severe degree of hearing loss none of the ears showed presence of VEMP.
- The TEOAE amplitude is highly correlated with the severity of the hearing loss for both right and left ear.

- For hearing losses of moderate and higher, there was absence of TEOAE response.

Conclusion

The p13 latency and p13- n23 complex amplitude parameters of VEMP could be considered to show the effect of noise on saccular system which was obtained significantly different. VEMP is expected to be affected or absent in clients with the dysfunction of the vestibular system, as in the current study, all the individuals with "Sensation that you are turning or spinning inside" and "Nausea or vomiting" had absent VEMP responses indicating saccular involvement in NIHL group. It is also evident that the cochlea is more susceptible to noise in these individuals with NIHL as the TEOAE was absent in most of the client with NIHL.

Implication of the study

- ✓ This study would give an insight into the involvement of saccular system in individuals with noise induced hearing loss.
- ✓ VEMP could be used as a tool to identify the susceptibility of vestibular dysfunction in occupational health hazard individuals.
- ✓ Study also suggests that which structure among cochlea and saccule is more susceptible to noise.
- ✓ The knowledge about susceptibility of a particular structure would help us to understand better the hazardous effects of noise.

- ✓ Abnormal VEMP responses (if found) would suggest an immediate need to include VEMP testing in test battery for individuals who are exposed to noise for monitoring the vestibular dysfunction, just like currently available test battery to monitor the auditory dysfunction.
- ✓ The specific vestibular symptom indicates saccular involvement in individuals with NIHL.

Future Research

- ♦ VEMP was the objective test carried out to evaluate vestibular system in this study, a more detailed study including other vestibular tests may help in better understanding of the vestibular system functioning in individuals with noise induced hearing impairment.
- ♦ Research is required to compare the VEMP results obtained in individuals with NIHL and other individuals with sensori-neural hearing loss subjects.
- ♦ Research can be carried by correlating the later VEMP potentials (n33-p44) with the TEOAE response individuals with NIHL.

Chapter 7

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APPENDIX - I

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MANASAGANGOTHRI, MYSORE - 570 006

DEPARTMENT OF AUDIOLOGY

Name : Age : Sex : M/F

Address :

Occupation : Education :

Working Since / No. of years of Service :

1. Name of your Industry :
2. Type of products manufactured
3. Do you feel the place where you work is too noisy ? Yes/No
4. Do you have to raise your voice when you talk to the person next to you ? Yes/No
5. Do you use more gestures (signs) instead of talking in order to be understood ? Yes/No
6. Do you find difficult in understanding others' speech, at the distance of 3 feet, because of noise ? Yes/ No
7. Do you feel that you can hear only the machine sound or noise when it is on ? Yes/ No
8. Do you understand better when a person is using gestures (signs) also while talking to you ? Yes/ No
9. Do you have to raise your voice for others to hear better ? Yes/ No
10. Do others have to raise their voice, so that you can hear better ? Yes/ No
11. Do you find difficulty in understanding even when others shout because the noise is very loud. Yes/ No
12. Do you face above problem through out the day or only for a few hours ? Yes/ No
13. Were you working in noisy environment before you joined here ? Yes/ No
14. If yes, specify the number of hours that you were exposed to noise in a day. hrs./day
15. Specify the number of hours that you are exposed to this environment in a day. hrs./day
16. Do you get irritated or frustrated working in the noisy situation. Yes/ No
17. Is noise present in only your section or entire factory (strike-out which is not applicable) Your Section/Entire Factory
18. Do you feel any fluctuation in your hearing capacity, i.e., difficulty in hearing sounds clearly, immediately after coming out from work. Yes/ No
19. Do you find difficulty in understanding speech when noise is present ? Yes/ No
20. Do you feel that you can understand speech better when the surrounding is quite ? Yes/ No
21. Do you feel that your hearing becomes better when you are away from work for a few days or weeks ? Yes/ No
22. Do you get tired of work easily ? Yes/ No

23. If yes, what is the reason. Yes/ No
    - Because of loud noise
    - Tension
    - Any other, specify

24. Do you hear ringing sounds in the ear ? Yes/ No

25. Do you get ear ache when you are working ? Yes/ No

26. Do you find any change in your voice ? i.e., Does your voice become hoarse ? Yes/ No

27. If yes, since when ? _____

28. Did you have your hearing tested before ? Yes/ No

29. If yes,

    When ? _____

    Where ? _____

    What was the finding ? _____

    Treatment, if any _____

30. Do you have any report of increase in blood pressure when you are exposed to noise. Yes/ No

31. Are you using any ear protective devices ? Yes/ No

32. If yes, State :

    The type of ear protective device/s _____

    Was it given by your management Yes/ No

    Is it your own Yes/ No

33. Are you exposed to chemicals / fumes while working ? Yes/ No

34. If yes, specify.

35. Have you taken any medicines Yes/ No

    (Antibiotics like quinine, streptomycin, gentamycin) for any illness / fever Yes/ No

36. If yes, Specify

37. Does any one in your family have hearing problem ? Yes/ No

38. What type of noise is made by the operation of machine where you work ?
    (Tick the one which is applicable)
    Continuous noise (Eg: noise made by refrigerator)
    Intermittent noise (Eg: noise made by time-piece)
    Impact noise

39. Any other problem you think that you face on account of working in noisy environment :

APPENDIX-II

Dizziness questionnaire
Maryland Hearing and Balance Center

Name_____ Date_____

I. **Which of these best describes your dizziness? Circle only one**.

A sensation of movement of yourself or the room: spinning, tilting, or wave-like movement

Lightheadedness or feeling that you are going to faint

Loss of balance

Disassociation or disorientation with the world

II. When you are "dizzy" do you experience any of the following sensations? You may circle as many yes responses as necessary.

Yes	**No**	**1.**	**Lightheadedness or swimming sensation in the head.**
Yes	**No**	**2.**	**Blacking out or loss of consciousness.**
Yes	**No**	**3.**	**Tendency to fall.**
Yes	**No**	**4.**	**Objects spinning or turning around you.**
Yes	**No**	**5.**	**Sensation that you are turning or spinning inside.**
Yes	**No.**	**6.**	**Loss of balance when walking**
Yes	**No**	**7.**	**Headache**
Yes	**No**	**8.**	**Pressure in the head.**
Yes	**No**	**9.**	**Nausea or vomiting.**

III. Please fill in the blanks or circle appropriate answer

A. When did the dizziness first occur?_____

B. Is the dizziness CONSTANT or does it come in ATTACKS?

C. If the dizziness comes in attacks, how often do these attacks occur?

_____times per day / week / month / year.

D. If the dizziness comes in attacks, how long do the attacks last?

_____ seconds / minutes / hours / days.

E. What factors provoke the dizziness or make the dizziness worse?

_____

F. What makes the dizziness better?

_____

G. Does your hearing change when the dizziness occurs? Yes / No

How?_____

Which Ear? Right / Left

H. Are there any other symptoms associated with the dizziness, such as visual changes, numbness or tingling in the arms or legs, weakness in the arms or legs, changes in speech? _____

I. Are you completely free of dizziness between attacks? Circle Yes / No

J. Have you ever been diagnosed with a head or neck injury? Circle Yes / No

K. Do you have any history of a neurological disease such as migraine, multiple sclerosis or stroke? Circle Yes / No

Explain _____

IV. Do you have any of the following symptoms? Please circle Y*es* or No and circle Ear involved.

Yes	No	1. Difficulty in hearing?	Right	Left
Yes	No	2. Noise in your ears?	Right	Left
Yes	No	3. Does noise change during the dizziness? How? _____		
Yes	No	4. Fullness or stuffiness in your ears?	Right	Left

V. Have you experienced any of the following symptoms?

Yes	No	1. Double vision, blurred vision or blindness.
Yes	No	2. Numbness of face.
Yes	No	3. Numbness of arms or legs.
Yes	No	4. Weakness in arms or legs.
Yes	No	5. Clumsiness of arms or legs.
Yes	No	6. Confusion or loss of consciousness.
Yes	No	7. Difficulty with speech.
Yes	No	8. Difficulty with swallowing.
Yes	No	9. Pain in the neck or shoulder.