cVEMP & oVEMP findings in Noise induced hearing loss individuals

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CERTIFICATE

This is to certify that this dissertation entitled "**cVEMP & oVEMP findings in Noise Induced hearing loss Individuals** " is a bonafide work submitted in part fulfilment for the Degree of Master of Science (Audiology) of the student (Registration No.: 12AUD027). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any of the University for the award of any other Diploma or Degree.

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CERTIFICATE

This is to certify that this dissertation entitled "**cVEMP & oVEMP findings in Noise Induced hearing loss individuals**" has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in other University for the award of any Diploma or Degree.

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DECLARATION

This is to certify that this dissertation entitled "**cVEMP & oVEMP findings in Noise Induced hearing loss individuals**" is the result of my own study under the guidance of Dr. Sujeet Kumar Sinha, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier in other University for the award of any Diploma or Degree.

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Chapter-I

INTRODUCTION

Hearing loss can be caused by exposure to harmful noise, either very loud impulse sound(s) or repeated exposure to sounds over 90 decibel levels over an extended period of time that damage the sensory structures of the inner ear. Noise damages the ear structures in two ways. One is noise-induced hearing loss, which is cumulative and insidious, growing slowly over years of exposure and commonly associated with occupational noise. The other is acute acoustic trauma. Two types of mechanisms are involved in the destruction of the end organs by noise: direct mechanical destruction, and metabolic decompensation with subsequent degeneration of sensory elements (Oosterveld, Schoonheyt & Polman, 1982). Hence, it is likely that individuals, who have NIHL causedby exposure to intense noise, in addition to cochlear lesion, will have damage to the vestibular end organs as well (Ylikoski, Juntunen & Matikainen, 1988).

Several auditory problems have been reported in individuals with noise induced hearing loss .Auditory problems found in these individuals are tinnitus, sensori neural hearing loss, temporary and permanent threshold shift. Auditory effects or any other health effects may develop to any person at any frequency level depending upon the individual exposure to noise situation. Low frequency noise, up to100 Hz may cause non-aural physiological and psychological effects below the individual hearing threshold. Leventhal *et al,* also emphasized that 10-200 Hz frequency noise is an environmental noise sensitive to people. This generates many complaints and is generally seen to the people of middle age. It may also occur to the subjects working in industry, but generally found at levels well above threshold.

Auditory finding which is findings in individuals with noise induced hearing loss is mild to profound degree of hearing loss depending on the type of stimulus they are exposed to, middle ear muscle reflexes would be present or absent depending on the type and degree of hearing loss, Oto Acoustic Emission (OAE) amplitude will be reduced or it will be absent. Auditory brain stem responses (ABR) will be present in few individuals or may be present in few individuals depending on the type and degree of hearing loss.

The human inner ear contains the end organ for hearing (cochlea) and the end organs for balance (the semicircular canals and the otolith organs (saccule and utricle). The vestibular system's response to sound has not always been clearly understood. It was first suspected to be sensitive to sound in the early twentieth century. Pietro Tullio (1929) hypothesized that loud sounds generate vestibular symptoms in patients. Postulation was further developed by Georg von Békésy (1935), who hypothesized that high intensity sounds greater than 125 dB SPL would affect the vestibular system.

Various tests are used to assess the functioning of the different parts of the vestibular system. The function of the semicircular canal is assessed by ENG, whereas the functioning of otolith organs are assessed by vestibular evoked myogenic potentials. Cervical vestibular-evoked myogenic potentials (cVEMPs) have been successfully recorded from tonically contracted cervical muscles using loud sound stimulation (Colebatch et.al., 1994; Welgampola & Colebatch, 2005). cVEMPs test assesses the descending vestibular pathway as an ipsilateral sacculocollic reflex (Uchino et.al, 1997). It has become common in daily clinical practice over the past decade, providing another method of testing saccular function (Young, 2006). Recent investigations have demonstrated that VEMP can also be recorded from extra-ocular muscles in response to

loud sound (Rosengren et al., 2005; Todd et.al, 2007). This variation of VEMP is termed ‘‘ocular” VEMP (oVEMPs).

oVEMPs test evaluates the ascending vestibular pathway as a crossed vestibulo-ocular reflex (Iwasaki et.al, 2008). Both cVEMPs and oVEMPs has been found as a useful tool in the diagnosis of various vestibular disorders such as vestibular schwanomma (Matsuzaki, Murofushi & Mizuno, 1999), multiple sclerosis (Shimizu, Murofushei, Sakurai, & Halmagyi, 2000), brainstem disease(Chen & Young, 2003), auditory Neuropathy (Sheykholeslami, Schmerber, Keamany & Kaga, 2005), Menier’s disease (Murofushi, Shimizu, Takegoshi & Cheng, 2001), Superior Semicircular Canal Dehiscence (Minor, 2000), Vestibular hypersensitivity (Watson, Halmagyi & Colebatch, 2000), Vestibular Neuritis (Ochi, Ohashi & Watanabe, 2003).

Need of the study:

- Vestibular evoked myogenic potentials have an important role in assessing the functioning of the otolith organ of the vestibular system. The electronystagmography traditionally evaluates only the semicircular canal. Hence the inclusion of VEMPs will provide information about otolith dysfunction in individuals with NIHL.
- oVEMP is a new variation of VEMP responses, which assess the utriculooccular pathway. Combining oVEMP and cVEMP result will provide complementary information about Utricular otolith function. Thus, there is a need to study both the cVEMPs as well oVEMPs in individuals with NIHL.

- Earlier studies have confirmed saccular dysfunction in NIHL based on cVEMPs results (Akin, Murnane et.al, 2012). However there is dearth of information on oVEMPs studies in NIHL subjects.
- There is also dearth of information on association of different test results with audiological findings and sign and symptoms exhibited by individuals with NIHL. Hence it is needed to associate the findings.

Aim of the study

- To study the results of cVEMP and oVEMP in noise induced hearing loss individuals.

Objectives of the study

- To study cVEMP in noise induced hearing loss individuals.
- To study oVEMP in noise induced hearing loss individuals.
- To find out correlation between the duration of noise exposure with cVEMP & oVEMP findings in noise induced hearing loss individuals.
- To find out correlation between puretone thresholds at 4 KHz and cVEMPs & oVEMP test results in noise induced hearing loss individuals.

Chapter-II

REVIEW OF LITERATURE

Noise induced hearing loss (NIHL) results from damage to the ear from high intensity sounds that produces a temporary or permanent sensorineural haring loss. Along with the hearing loss the exposure to high intensity noise may cause damage to the vestibular structures. Since the vestibular systems the cochlear system shares the same cavity it is possible that noise damages the vestibular structures also. Hence it is important to assess the vestibular system.

VEMP testing may provide a useful, non-invasive method for assessment of otolith function and the functional integrity of the inferior vestibular nerve (Akin et.al, 2003). Clinically, the test is relatively easy to perform and can be performed with most evoked potentials recording systems. There are two variants of the VEMP test: cervical vestibular evoked myogenic potentials (cVEMP) and ocular vestibular evoked myogenic potentials (oVEMP). The cVEMP test, which assesses the descending vestibular pathway as ipsilateral sacculo-ocollic reflex, the oVEMP test has been validated to evaluate the ascending vestibular pathway as crossed vestibulo-ocular reflex (Iwasaki et al, 2008).

In recent years, short latency myogenic potentials, oVEMPs (ocular VEMPs), produced by synchronous activity in the extraocular muscles in response to different stimuli including sound has been introduced. It has been reported that oVEMPs can provide another diagnostic tool for assessing the vestibule-ocular reflex. Analogous to cVEMPs, stimulation by loud sound can be via air or bone conduction. In human, studies demonstrated that bone conduction (BC) vibration stimulation efficiently evokes both

oVEMPs and cVEMPs. oVEMP's are excitatory responses that may be elicited more easily than cVEMPs even in elderly or disabled subjects who fail sustained neck contraction.oVEMP is characterized by a biphasic peak where first peak appears at a latency of 10 msec and second peak appears at a latency of 14msec.

Clinical Utility of Ocular vestibular evoked myogenic potentials (oVEMP)

MENIERS'S DISEASE

Winters, Campschroer, Grolman and klis (2011) studied oVEMP in 37 individuals with Meniere's disease. The recorded oVEMP responses were compared with that of 55 healthy individuals. The results revealed that the amplitude of oVEMP were lower in individuals with Menier's disease compared to the normal hearing individuals. The study also concludes that the air conducted oVEMP can be useful addition to the currently used battery of test which is used for the assessment of Menier's disease.

Bao, Xu and Guo (2013) evaluated the reliability of oVEMP in 27 participants with Meniere's disease. The responses obtained were compared with 30 healthy normal individuals. They could observe abnormal responses in 8 subjects in the normal healthy individuals, and presence of normal responses in only 19 subjects in individuals with Meniere's disease. The authors concluded that oVEMP test is brief, safe, objective test which could be included in the audiological test battery.

Wen, cheng and Young (2012) studied oVEMP in 40 individuals with Meniere's disease. The objective of the study was to explain the mechanism behind augmented oVEMP in these individuals. All the participants underwent audiometry, caloric testing,

cVEMP through bone conducted stimuli and oVEMP through bone conducted stimuli. Results were compared with the stages of Meniere's disease. The individuals with augmented responses had earlier n1 and p1 latencies and also larger amplitude for n1-p1 complex oVEMPs. They concluded that oVEMPs have earlier latencies and larger amplitude compared with the reduced oVEMPs which indicated large amount of utricular afferents are affected during the early stage on Meniere's disease.

Huang, Wang and Young (2011) recorded oVEMP for 30 subjects with Meniere's disease. They could observe abnormal responses in these cases. Abnormal response rate was about 65% in the affected ear. Authors concluded that by advocating the inclusion of oVEMP in the test battery for the diagnosis of Meniere's disease.

Abdeltawwab (2013) reported abnormal oVEMP responses in subjects with Meniere's disease. In the study 30 healthy volunteers and 31 subjects with Meniere's disease were included. Contralateral recording of oVEMP was done for all the participants. They could observed significant lower mean amplitude for the contralateral recording of oVEMP and also the mean latencies were significantly longer in Meniere's disease compared to that of normals. The authors concluded that oVEMP can be included in the test battery of Meniere's disease.

Jerin, Berman, Krause, Wagner & Gurkov (2014) reported that the oVEMP 500/1000 Hz amplitude ratio may be a valuable diagnostic tool for Meniere's disease. The study included 39 subjects with certain Meniere's disease and also 19 healthy controls who were aged matched.500 and 1000 Hz air conducted tone burst was used as a stimulus for oVEMP recording and also 500/1000 Hz amplitude ratio were also calculated. Results revealed that 500/1000 Hz amplitude ratio is significantly smaller in

the affected ear for the Meniere's disease participants compared to that of unaffected ear of the same subjects and also with that of the control group. The study concluded that oVEMP can also be included in the test battery of Meniere's disease.

It is also reported in the literature that Vestibular Migraine and Meniere's disease behave similarly for most of the VEMP test battery. These responses can be due to a link in their pathophysiology which was reported by Zuniga, Janky, Schubert and Carey, 2012).they also reported that use of 500 Hz tone burst as a stimulus will help in differentiating Meniere's disease with that of Vestibular Migraine.

Zuniga, Janky, Schubert and Carey, (2012) recorded both cVEMP and oVEMP using different stimulus. Stimulus used were click, 500 Hz tone burst and midline tap stimuli (reflex hammer and mini shaker). For the study 20 subjects with Meniere's disease, 21subjects with Vestibular Migraine and 28 age matched normals were considered. They found that amplitude was reduced for both cVEMP & oVEMP (relative to controls/normals) when clicks were used as a stimulus. Only Meniere's disease group revealed reduction in tone evoked for the oVEMP amplitude. Authors also reported that no difference in oVEMP with the midline tap stimuli. Authors concluded that using 500 Hz tone burst which helps in differentiating oVEMP reponses from controls(normals) and from Vestibular Migraine.

BENIGN PAROXYSMAL POSITIONAL VERTIGO

Talaat et al.,(2013) reported abnormal responses in subjects with BPPV. For 32 subjects with BPPV both cVEMP and oVEMP was recorded.80 subjects with non recurrent BPPV and 100 healthy Volunteers who were aged matched and gender matched

were considered. They found prevalence of abnormalities for both cVEMP and oVEMP with BPPV. It was about 20.5%. 40.3% of subjects with recurrent BPPV showed abnormal oVEMP and cVEMP, while 12.5% had abnormal VEMP in subjects with non recurrent BPPV. Absent VEMP, delayed VEMP and asymmetrical VEMP were the forms of VEMP abnormalities. The authors concluded that VEMP abnormalities were detected more in recurrent BPPV subjects suggesting that it may be indicative of the risk of BPPV recurrence (Talaat et al.,2013).

VESTIBULAR NEURITIS & LABYRINTHITIS

oVEMP responses are found to be abnormal in individuals with Vestibular neuritis & Labyrinthitis.Few of the supporting studies are Moon, Lee, Park (2012) recorded both cVEMP and oVEMP using 500 Hz tone bone as a stimulus on individuals with vestibular Neuritis and Acute Viral labyrinthitis. Results revealed that there was about 20% abnormal responses for the cVEMP for Vestibular Neuritis subjects and all the subjects with acute viral labyrinthitis had abnormal responses (100%).For the oVEMP , 90% and 100% abnormal responses were present for vestibular Neuritis and Acute Viral Labyrinthitis respectively. The authors also found a positive correlation of oVEMP with caloric test and subjective visual vertical in subjects with Vestibular neuritis and Labyrinthitis.

Manzari et al.,(2012) studied 59 subjects with probable inferior vestibular neuritis for both cVEMP and oVEMP. They found asymmetrical p1-n2 component in cVEMP while symmetrical n1 component in oVEMP. They concluded that sense organ of cVEMP and oVEMP cannot be same, as one response was normal and other was not.

Kim et al., (2013) recorded CVEMP and oVEMP using air conducted sound (ACS)and bone conducted vibration (BCV) for 30 subjects with vestibular neuritis and 45 normals. Both ACS and BCV had abnormal responses for vestibular neuritis subjects. Response rate was about 80% for ACS and 73.3% at forehead and 76.7% at mastoid for BCV.In contrast, cVEMPs were mostly normal with both ACS and BCV stimulation mode. Results suggested that oVEMP induced by either ACS or BCV appears to depend on the integrity of the superior vestibular nerve, possibly due to the utricular afferents travelling in it. In contrast cVEMP elicited by either ACS or BCV may reflect function of the saccular afferents running in the inferior Vestibular nerve

Shin et al.(2012) reported abnormal oVEMP responses in individual with Vestibular Neurities.41 subjects with acute neuritis an d 60 normal healthy individuals were recorded with cVEMP and oVEMP responses. Out of 41 subjects with Vestibular neuritis 30 subjects had superior vestibular nerve involvement, 3 had inferior involvement and 8 had both inferior and superior damaged vestibular nerve. They found 30 subjects with superior vestibular neuritis had normal cVEMPs and abnormal oVEMPs in all 30 with superior vestibular neuritis. The subjects with inferior vestibular neuritis showed normal oVEMP and abnormal cVEMP. The authors concluded that abnormalities of oVEMP and cVEMP in subjects with vestibular neuritis selectively involve the superior or inferior vestibular nerve suggest that the origin of the vestibular nerve afferents of oVEMP differ from those of cVEMP.

SUPERIOR SEMICIRCULAR CANAL DEHISCENCE SYNDROME (SCD)

SCD is caused by the loss of the bony covering overlying the superior semicircular canal. In SCD, oVEMPs are characterized by significantly larger amplitude and with lower thresholds (Rosebgren et al.,2008). In the study air conducted stimulation had larger amplitudes than 5mV in 7 to 10 individuals with SCD ears but in none of the healthy controls, revealing a sensitivity of 0.7 and a specificity of 1.0 in this small group of SCD subjects. Thresholds differentiate SCD subjects from healthy subjects especially when using air conducted stimulation. Enlarged amplitude for the contralateral ear was present for both oVEMP and cVEMP evoked by different stimulus modes that is the Air conducted stimulation and the bone conducted vibrator. They concluded that there is a significant correlation between the size of the dehiscence and oVEMP amplitudes(Manzari et al., 2012).

AUDITORY NEUROPATHY

It is a disorder which is characterized by abnormal 8^{th} nerve functioning, with the normal outer hair cell functioning. Sinha, Shankar and Sharanya(2013) reported high percentage of absent responses of oVEMP in these individuals. 11 subjects with auditory neuropathy were considered for the study cVEMP and oVEMP responses were recorded for these individuals. Results revealed 100% absent response for oVEMP ang 90.90% for cVEMP. The authors concluded that there is high incidence of vestibular involvement in persons with auditory neuropathy. They also advocated the necessity of inclusion of both cVEMP and oVEMP in vestibular test battery which would be helpful in assessing the persons with auditory Neuropathy.

CLINICAL APPLICATIONS OF CERVICAL VESTIBULAR EVOKED MYOGENIC POTENTIALS

MENIERE'S DISEASE (MD)

MD is a common disorder characterized by fluctuating hearing loss, tinnitus, aural fullness, and episodic rotary vertigo. The aetiology is still unclear, although histopathology studies have indicated the presence of endolymphatic hydrops. Specific sites of lesion are observed most often in the cochlea, followed by the saccule and utricle. Clinical diagnosis of MD relies mainly on symptoms, electrocochleography (EcochG), and ENG/caloric testing. Recent work indicates that VEMP testing may bring to the table a new tool for the diagnosis of MD.

Study done by De Waele et al. (1999) studied cVEMP on 59 individuals with unilateral Meinere's disease with the age range of 18-74 years.Results revealed that saccular responses wereabsent on the affected side in 54% of the subjects of Meinere's disease.This absence was correlated with the degree of low frequency haring loss but not with canal paresis.Subjects with Meniere's disease had absence response of 54%. The authours concluded that VEMP testing is useful in detecting the individuals who are at risk in saccular lesion.

Shojaku, Takemori, Kobayashi Watanabe (2001) recorded cVEMP response to one burst stimulus with glycerol for 5 healthy young adults with normal hearing, 15 subjects with unilateral definite Meniere's disease (UMD) and 7 subjects with delayed endolymphatic hydrops (DEH). Using the GVEMP test, 8 of the 15 subjects (53%) with UMD were evaluated as being abnormal. In addition, a greater number of subjects (67%)

were judged to be abnormal when the results of the GVEMP test were combined with those from a glycerol dehydration test, trans-tympanic electrocochleography (ECochG) or furosemide vestibulo-ocular re• ex test (FVOR). Four of the 7 subjects with DEH (57%) showed abnormal results in the GVEMP test. In particular, in subjects with the ipsilateral type of DEH, only the GVEMP test was able to detect the affected side. These findings suggest that the GVEMP test is a new and useful test for EH, and that a test battery comprising the GVEMP test together with one of the other three tests is useful for diagnosing endolymphatic hydrops of the inner ear

Murofushi et al.(2001) studied VEMP on 134 subjects. In which 43 were diagnosed as Meniere's disease, 62 as auditory neuropathy, and 23 as vestibular neuritis. Results revealed that there was prolonged latencies with decreased amplitude or absent responses in 51% in individuals with Meniere's disease and prolonged latencies were present in individuals with auditory neuropathy and vestibular neuritis which was beyond the normal range.

Seo (2003) conducted Furosemide VEMPs on Twenty-five affected ears of subjects with unilateral Meniere's disease. 22 ears of 11 normal healthy volunteers were considered as control group. The amplitude of the p13–n23 biphasic wave was significantly enlarged in 7 of 18 cases in which it could be detected before diuretic loading. The biphasic waves appeared after diuretic loading in 3 of 7 cases in which it could not be recorded before loading. Thus, the positive ratio of F-VEMP for unilateral Meniere's Disease was considered to be 40% (10 of 25). The ratio was similar to that of the conventional examinations for endolymphatic hydrops such as the glycerol test,

furosemide test, and electrocochleogram. The authors concluded that F-VEMP test is a useful tool in the diagnosis of endolymphatic hydrops

VESTIBULAR SCHWANNOMA

Since the neural pathway of VEMPs involves the vestibular nerve, VEMP testing could be useful in the evaluation of vestibular nerve function. VEMP has been found to be reduced in subjects with vestibular Schwannoma. Murofushi et al. (1998) recorded VEMP in 17 subjects. The authors reported that VEMP was abnormal in 80% of the subjects. In another study done by Matsuzaki et al. (1999) found abnormal VEMPs in 2 subjects with vestibular schwannoma while ABR data were normal.

Ochi et al.(2001) also reported 3 vestibular schwannoma cases with abnormal VEMPs, including elevated thresholds, abnormal interaural differences of thresholds, and abnormal p13–n34 amplitude ratios between left and right sides. Although VEMP testing may provide valuable information for the diagnosis of vestibular schwannoma, it is not appropriate to use VEMPs in isolation to document the nerve origin of the vestibular schwannoma.

Tsutsumi et al.(2000) demonstrated that VEMP results were not always correlated with the nerve where the tumour was located. Moreover, no correlation was found between the VEMPs and tumour size.

SUPERIOR CANAL DEHISCENCE SYNDROME (SCD)

Minor and colleagues (2001) identified a previously unrecognized vestibular entity called superior canal dehiscence (SCD). Subjects with SCD usually have sound

and/or pressure-induced vertigo and nystagmus. Minor et al. explained that the dehiscence would create an additional mobile window (a third window) in the labyrinth. Normally, volume displacements within the labyrinth in response to stapes movements are not strong enough to stimulate the vestibular end organs. However, the existing dehiscence will decrease the impedance and allow additional volume displacements within the labyrinth and deflections of vestibular sensors in response to sound.

VEMP testing has been reported to be sensitive to SCD. Brantberg, Bergenius, and Tribukait (1999) studied cVEMPs on 3 subjects with SCD. They showed abnormally large responses with low thresholds, particularly in the frequency range of 500–1000 Hz on the affected side.

Brantberg et al. (2001) studied 8 subjects with SCD. In all subjects, cVEMPs were present with extremely low thresholds and abnormally large amplitudes on the affected side. In contrast, 4 of the 8 subjects had normal hearing, and 6 subjects had normal findings with caloric testing.

Study done by Streubel, Cremer, Carey, Weg, and Minor (2001) tested 10 subjects with superior canal dehiscence (SCD) were evaluated. For the 8 subjects without prior middle ear disease, the cVEMP threshold from the affected side was compared to the threshold from normal participants . In the 2 remaining subjects with conductive hearing loss, cVEMPs were present from the affected side. Given that cVEMPs should not be expected in ears with conductive hearing loss, the Streubel et al. (2001) findings are compelling with regard to the sensitivity of cVEMPs in diagnosing SCD in a variety of different hearing conditions.

VESTIBULAR NEURITIS AND DIFFERENTIAL DIAGNOSIS

The use of VEMPs has also been applied to evaluate function of the saccule and inferior vestibular nerve. Halmagyi and Colebatch (1995) studied VEMPs in 22 subjects with reported vestibular neuritis. All subjects had no caloric responses on the affected sides, indicating dysfunction of the lateral semicircular canal. In contrast, VEMPs were normal in 6 subjects, reduced in 5 subjects, and absent in 11 subjects. Their results not only suggested that VEMPs were not of lateral canal origin but also revealed different pathologies involved in vestibular neuritis.

BENIGN PAROXYSMAL POSITIONAL VERTIGO(BPPV)

Heide et al. (1999) investigated VEMPs in the differential diagnosis of acute vertigo. These authors evaluated 40 subjects with acute vertigo: 26 with acute peripheral vestibulopathy, 5 with MD, 3 with BPPV, and 6 with psychogenic vertigo. These authors found 12 of 29 subjects had normal VEMPs with abnormal caloric tests. Results of the study revealed that all the subjects with BPPV had normal VEMPs.

Acute vestibular neuritis is usually caused by viral infection (Schuknecht & Kitamura, 1981). The inflammation caused by the viral infection can affect superior or inferior vestibular nerves. Halmagyi, Aw, Karlberg, Curthoys, and Todd (2002) recently reported 2 subjects with acute vertigo but normal lateral semicircular canal function as indicated by the caloric test. It was reported that these 2 subjects had selective inferior vestibular neuritis since VEMPs were absent on the affected side for both cases.

In a similar study, Murofushi, Halmagi, Yavor, and Colebatch (1996) found that in a population of subjects with vestibular neuritis, presence or absence of VEMPs would

predict subsequent BPPV occurrence. In 47 subjects with acute vestibular neuritis, 10 had subsequent BPPV posterior canal on the same side as the neuritis. All 10 subjects with BPPV had VEMPs in spite of the vestibular neuritis, whereas 16 subjects revealed absent VEMPS. The authors concluded that if VEMPs are absent at the time of the acute neuritis, the patient is unlikely to develop consequential BPPV.

AUDITORY NEUROPATHY

Sheykholeslami, Schmerber, Kermany & kaga (2005) studied 3 auditory neuropathy subjects associated with balance disorders. Results revealed that VEMP responses were absent in the affected ear. They concluded that, in subjects with isolated auditory neuropathy, the vestibular branch of the 8^{th} cranial nerve and its innervated structures may also b affected.

Similar findings were reported by Kumar, Sinha, Bharti, Singh, & 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).

Kumar, Sinha, Bharti, Singh and Barman (2007) describe cVEMP in 10 subjects with auditory neuropathy. 10 subjects were considered for the study out of which 9 subjects showed absent cVEMP responses. They also observed there was no one to one correlation between the vestibular symptoms reported by the subject with that of the responses got. 80% of the ears with Auditory neuropathy showed abnormal cVEMP

results giving an indication that there is high involvement of Vestibular system in these population. This study provides for involvement of the vestibular branch of the VIIIth cranial nerve in a high percentage of the auditory neuropathy individuals.

Study done by Sazgar et al. (2010) studied cVEMP on 8 subjects who are diagnosed as having auditory neuropathy. For the control group 30 normal subjects were considered with no history of any neurological or auditory disorders. Normal responses were obtained from 3 ears and abnormal responses in all others including non replicable waves in 4 ears and absent responses in 9 ears.

Study done by Akdogan et. al(2008) investigated the vestibular functions in children with Auditory neuropathy. Different tests were carried out like caloric, magnetic Resonance Imaging. cVEMP responses were recorded in these children. Results obtained were normal cochlear nerve structure, with no abnormalities in caloric test. They concluded that it would be valuable if both caloric testing and cVEMP is carried out for the vestibular evaluation in children with auditory neuropathy.

VESTIBULAR FINDINGS IN INDIVIDUALS WITH NOISE INDUCED HEARING LOSS

The consequences of excessive noise exposure on the balance system were studied extensively, those investigations reported that due to this the structures get damaged (Hain, 2010). These results were supported by the animal studies. Ylikoski (1987) investigated the effect of noise exposure in guinea pig with impulse noise of 1.1kHz at 158 dB SPL, which found that the excessive noise level exposure leading to the severe

structural changes in the vestibular systems mainly in ampullary cristae, utricular and saccular maculae.

Similarly,Ylikoski, Juntunen, Matikainen,Ylikoski, and Ojala, (1988) investigated the excessive noise exposure effect on subjects with different magnitude of hearing loss due to the noise exposure. Their results suggest that the subclinical symptoms occured priory than the exact occurrences of clinical symptoms associated with the abnormal vestibular issues. The sway movement is predominantly higher in severe NIHL subjects when compared to milder subjects.

NIHL subjects showed variety of vestibular tests results like reduction in the caloric exicitation, unprompted nystagmus and abnormal rotatory tests as reported by Aantaa, Virolainen, &Karskela (1977). Oosterveld, Polman, & Schoonheyt (1982) investigation suggested the similar vestibular tests results.

Electro-nystagmography(ENG) and smooth harmonic acceleration(SHA) tests results showed lower in vestibule- ocular reflex gain and reduction in the caloric responses in NIHL (Shupak, Bar, Podoshin, Spitzer, Gordon, & David, 1994). In SHA and ENG tests results they didn't find in any asymmetry in the parameters which tested in those tests. The authors concluded these results by saying that the central compensation due to the vestibular issues might be symmetrical with symmetrical hearing loss. And also found that there was inverse correlation in magnitude of hearing loss with vestibule-ocular reflex gain as well as with ENG caloric lateralization.

Electrophysiological tests results in NIHL also proposed some amount of problems on vestibular system. Manabe, Kurokawa, Saito, &Saito (1995) didelectrocochleaogram in NIHL subjects with and without vestibular symptom (vertigo).

In the vertigo group they found presence of increased summating potential (SP)/action potential (AP) than in the non-vertigo group. They suggested that the mechanism of pathophysiology in NIHL and meneires disease was same.

Dizziness questionnaire was administered by Raghunath, Suting, and Maruthy (2012) in 20 factory workers who is exposed to noise more than ten years. They found difference in the vestibular symptoms in NIHL group compared to control group. And also postulated that the vestibular symptoms are occurring first than the evident of hearing loss in them.

Perez, Freeman, Cohen & Sohmer (2002) performed ABR and short latency vestibular evoked potentials (VsEPs) in Sand rats after these animals exposed to 10 gunshots generating impulse noise at an intensity of approximately 160 dB SPL. Repeated measurements of the evoked potentials were conducted 2 to 4 hours, 1 week, and 6 weeks after the exposure. They compared the amplitude and latency of the first wave of VsEPs in response to linear and angular acceleration stimuli between baseline and post-exposure measurements. The results showed that the amplitude of the first wave of the VsEPs in response to linear acceleration was significantly reduced and the latency significantly prolonged 2 to 4 hours after the exposure in comparison to baseline measurements. The latency prolongation persisted in follow-up measurements also, whereas the amplitude showed a partial recovery. The first wave of VsEPs in response to angular acceleration was unchanged long-term and ABR thresholds were elevated in the long-term by 60 dB. From this it is revealed that impulse noise not only damages the cochlea, but also causes clear functional impairment to the vestibular end organs, mainly the otolith organs.

Wang and Young (2007) investigated the effect of chronic noise exposure on vestibular-evoked myogenic potentials. They performed audiometry, caloric, and vestibular-evoked myogenic potential tests in twenty subjects with chronic noise-induced hearing loss with bilateral notched audiogram at 4 kHz. 70 % of the cases were found to have abnormal VEMP and caloric responses. They also found a significant association between hearing threshold at 4 kHz and VEMP.

Madappa (2009) evaluated the functioning and susceptibility of the saccule in 30 individuals with NIHL within the age range of 25 to 50 years. Abnormal VEMP responses were obtained for 61.4% cases with significant prolongation of p13 and reduced amplitude of p13-n23 complex and VEMP responses were absent in 38.6% of cases.

Kumar et al. (2010) studied Vestibular evoked myogenic potential in noise-induced hearing loss on 30 subjects with the age range of 30-40 years. Results revealed that as the average pure tone hearing threshold increased, the VEMP latencies were prolonged and peak to peak amplitude was reduced in NIHL subjects. 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. Therefore, VEMP was abnormal or absent in 67% of NIHL y. Hence it can be concluded that the possibility of vestibular dysfunction, specially the saccular pathway, is high in individuals with NIHL. VEMP, a non-invasive and user friendly procedure can be employed in these individuals to assess sacculo-collic reflex.

Akin, Murnane, Tampas, Clinard, Byrd, and Kelly (2012) investigated the effects of noise exposure on the cervical vestibular evoked myogenic potential in 43 individuals

with asymmetric noise-induced sensorineural hearing loss. They also found abnormal cVEMP in ears with higher threshold. The subjects with greater degree of NIHL with abnormal cVEMPs were shown poorer high-frequency pure-tone thresholds and greater interaural high-frequency pure-tone threshold differences than the noise-exposed participants with normal cVEMPs.

From these evidences it is clear that noise exposure can results in vestibular damage and the sacculo-collic pathway susceptible to noise-related damage and the severity of NIHL is associated with the presence or absence of cVEMPs.

From the above stated investigations it is evident that the excessive exposure to noise creates problems in hearing as well as in vestibular system also. The vestibular symptoms are more evident in the asymmetrical NIHL subjects. Most of the studies did not support with the evident correlation between magnitude of hearing loss and problems in vestibular system.

Chapter-III

METHOD

Present study was conducted with an aim of assessing the sacculocollic and utriculo-ocular pathway using cVEMPs and oVEMPs respectively in individuals who are exposed to noise for 8 hours a day. The aim of the study was also to find a correlation between duration of noise exposure and cVEMP and oVEMP finding, correlation between threshold at 4 KHz, cVEMPs and oVEMPs findings.

Participants

Two groups of participants were taken for the study.

Clinical group: The clinical group consisted of 15male individuals in the age range of 25-50 years(Mean age 38 yrs) with history of noise exposure at the work place.

Control group: 15males participants with normal hearing sensitivity in the age range of 25-50 years(Mean age 38 yrs) with no history of noise exposure.

Participant selection criteria

Clinical group

- Normal hearing sensitivity or sensori neural haring loss with the air bone gap not exceeding 10 dB HL.
- Noise exposure for a duration of 8 hours per day, at least for more than 2years.
- Speech identification scores proportionate to the degree of hearing loss.
- No history or presence of any middle ear pathology.
- A or As type of tympanograms with presence/elevated or absence of ipsi lateral and contra lateral acoustic reflexes in both the ears.

- Absence of space occupying lesions which was ruled out based on the auditory brainstem response (ABR) test results and or a report from neurologist.
- Uncomfortable level (UCL) was greater than or equal to 95 dB HL for speech for all the particpants.
- Presence or absence of TEOAE
- No reports of presence of diabetes.

Control group

- Normal hearing sensitivity in both the ears with air conduction and bone conduction thresholds within 15dB HL at frequencies from 250 – 8000Hz and 250 Hz to 4000Hz with no previous history of noise exposure.
- A or As type tympanograms with presence of ipsilateral and contralateral acoustic reflexes in both the ears.
- Good speech identification scores i.e., greater than 80%.
- No history or presence of any middle ear pathology.
- A type tympanograms with presence of ipsi lateral and contra lateral acoustic reflexes in both the ears.
- No history or presence of any otological problems.
- No history or presence of any neurological symptoms
- Uncomfortable level greater than 95 dB HL for speech.
- No history of exposure to noise at their work place or home.
- Presence of TEOAE responses with a SNR of +6 dB and the response reproducibility and stimulus stability of greater than or equal to 80%.
- No reports of presence of diabetes.

Instrumentation

- A calibrated 2 channel audiometer Madsen Orbiter OB-922, with TDH-39 supra-aural earphones housed in MX-41/AR ear cushions and Radioear B-71 bone vibrator was used to obtain air-conduction and bone-conduction thresholds. The same audiometer in air conduction modality alone was used for speech audiometry and uncomfortable level testing.
- Tympanograms along with ipsilateral and contralateral acoustic reflex thresholds was obtained using GrasonStadler Incorporated- Tympstar (GSI Tympstar) middle ear analyser (Version 2.0.0) with default probe assembly and insert earphone.
- Transient evoked oto-acoustic emission (TEOAE) was acquired using Otodynamics ILOV6 with default transducers.
- A Biologic Navigator Pro auditory evoked potential unit (Version 7.0.0) with Etymotic ER- 3A insert earphones was used to record click evoked auditory brainstem responses and air-conducted tone burst evoked oVEMP.
- The cervical vestibular evoked myogenic potential and auditory brainstem response (ABR) was recorded using IHS smart EP Version 2.5 instrument. An Eartone 3A insert earphone was used to deliver the stimuli.

Test Environment

All the audiological tests was conducted inside well illuminated air conditioned, sound treated rooms with ambient noise levels within permissible limits (ANSI S 3.1, 1999). Evaluations using the audiometer were carried out in a double room set-up whereas immittance evaluation, TEOAE, ABR, and VEMP recordings were obtained in a single room set-up.

Procedure

- A detailed case history was taken for all the individuals in the clinical and control group.
- Pure tone thresholds was obtained between 250 Hz to 8000 Hz for air conduction and between 250-4000 Hz for bone conduction at all the octaves and mid octaves frequencies, using the modified Hughson and Westlake procedure (Carhart & Jerger, 1959).
- The speech identification scores was obtained using PB word list developed by Yathiraj and Vijayalakshmi (2005).
- Uncomfortable loudness levels was determined by presenting the running speech through the headphone (TDH -39) at different intensities using ascending method. The UCL for speech was defined as the level at which the subjects considered the speech to be uncomfortably loud.
- Immittance audiometry was carried out with a low probe frequency of 226Hz. The ipsilateral and contralateral acoustic reflex thresholds was measured for 500Hz, 1000Hz, 2000Hz & 4000Hz tones for both the ears.
- 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 was 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 is less than or equal to 5KΩ and the inter electrode impedance within 2 KΩ. ABR was carried out with 100 usec click presented at 90 dBn HL, with 11.1/sec, 90.1/sec repetition rate in

rarefraction polarity with the filter setting of 100Hz to 3000Hz .Total 1500 stimulus were used for recording. The analysis time was kept at 10msec. Single channel recording was done by placing the non inverting electrode on the Cz, inverting electrode on the mastoid and forehead as the ground.

oVEMP test:

The recording site was cleaned with a commercially available abrasive gel to obtain acceptable electrode impedances. The gold plated electrodes was placed using adequate amount of Ten20 conductive paste and secured in place with surgical tape. Absolute and inter electrode impedance was maintained below 5kΩ and 2KΩ respectively for both the recordings.The subject was made to sit in a chair and relax. During recording, the subject was instructed to look upward at a small fixed target >2 m from the eyes. oVEMPs were recorded for the contralateral stimulation. The recording protocol for oVEMP are given in table-3.1

Table 3.1

oVEMP recording protocol

Type of stimuli	Tone Burst
Stimulus frequency	500 Hz (Blackman window)
Stimulus duration	2-0-2 cycle
Intensity	125 dB SPL
Repetition rate	5.1/sec.
Polarity	Rarefaction
Transducer	Insert ear-phone
Total number of stimuli	150
Analysis window	70 msec including 10 msec pre-stimulus recording
Filter setting	High pass: 1 Hz Low pass: 1000 Hz
Notch filter	Off
Amplification	30,000
Number of channel	1
Electrode montage	Inverting electrode (-):- inferior of lower eyelids (1 cm below the centre of each lower eyelid). Non-inverting electrode (+):- immediately inferior to the inverting electrode (1 cm to inverting electrode). Ground electrode:- lower forehead.
Mode of recording	Contralateral

cVEMP test:

The active electrodes was placed on the upper half of the sternocleidomastoid (SCM) muscles, with a reference electrode on the suprasternal notch, and a ground electrode on the forehead. The sites of electrode placement was prepared using a skin preparation gel. Silver chloride disc electrodes was used for the recording. Individual electrode impedance and inter electrode impedance was maintained (5KΩ & 2KΩ.) During the recording the participant was instructed to sit straight and turn their head to the other side of the ear in which the stimulus was presented. A visual feedback was provided for the participant to maintain correct posture (LED light with red and green light). The recording protocol for cVEMP recording is given in table 3.2.

Table 3.2

Recording Protocol for cVEMP

Stimulus Parameters	
Type of stimulus	Tone burst
Stimulus frequency	500 Hz (Blackman Window)
Stimulus duration	2-0-2
Stimulus intensity	125 dB SPL
Repetition Rate	5.1/sec
Polarity	Rarefaction
Total stimulus presentation	150
Acquisition Parameter	
Analysis time	70 msec including 10 msec pre stimulus
Filter settings	High pass: 30Hz, Low pass:1500Hz
Notch filter	Off
Amplification	5000
No. of channels	1
No. of recording	2
Tranducer	Insert Earphone (ER-3A)
Electrode Montage	Inverting electrode(-): Sternoclavicular junction, Non-inverting electrode (+): Mid point of SCM of the side being stimulated Ground electrode: Lower forehead

Analysis of the data:

The recorded cVEMPs & oVEMPs responses were analyzed. For cVEMPs latency of P13, N23 and amplitude of P13-N23 complex was analysed for both the groups. For oVEMPs latency of n1, p1 and n2 peaks and amplitude of n1-p1 complex and p1-n2 complex was analysed for both the groups.

Chapter-IV

RESULTS

The aim of the study to characterize the cVEMP and oVEMP findings in individuals with NIHL. The aim of the study was also to correlate the cVEMP and oVEMP findings with the duration of noise exposure, threshold at 4 kHZ . Further the correlation of cVEMP and oVEMP findings were also studied in the both the groups. SPSS version 16.0 was utilized for statistical analysis. Following statistical analyses were done

a. Descriptive statistics to obtain mean and standard deviation for P13 latency, N23 latency, and amplitude of P13-N23 amplitude complex of cVEMP for normal hearing individuals and individuals exposed to noise.
b. Repeated Measure ANOVA was done to see the overall effect of noise on cVEMP latency and amplitude parameters with group and ear as between subject factors.
c. Multiple analysis of variance (MANOVA) was done to see the group differences for various latency and amplitude parameters of cVEMP.
d. Independent sample‘t’ test was done to compare the cVEMP latency and amplitude parameters between normal hearing individuals with individuals exposed to noise.
e. Pearson correlation test was done to find out a correlation between cVEMP findings with duration of noise exposure, threshold at 4 kHZ in individuals exposed to the noise.
f. Descriptive statistics to obtain mean and standard deviation for n1 latency, p1 latency, n2 latency and amplitude of n1-p1 amplitude complex and p1-n2

amplitude complex oVEMP for normal hearing individuals and individuals exposed to noise.

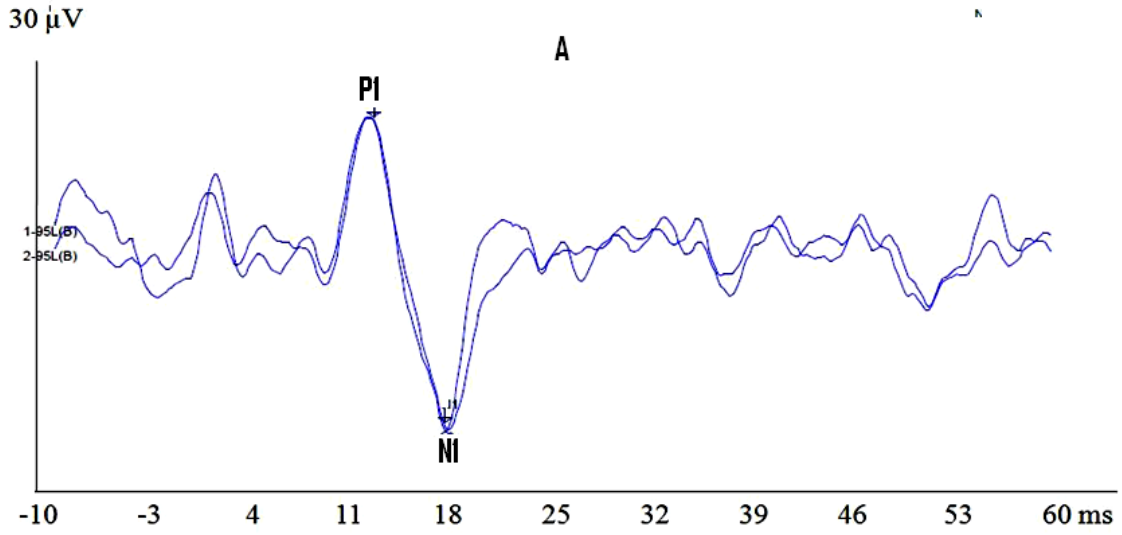
g. Repeated Measure ANOVA was done to see the overall effect of noise on oVEMP latency and amplitude parameters with group and ear as between subject factors.

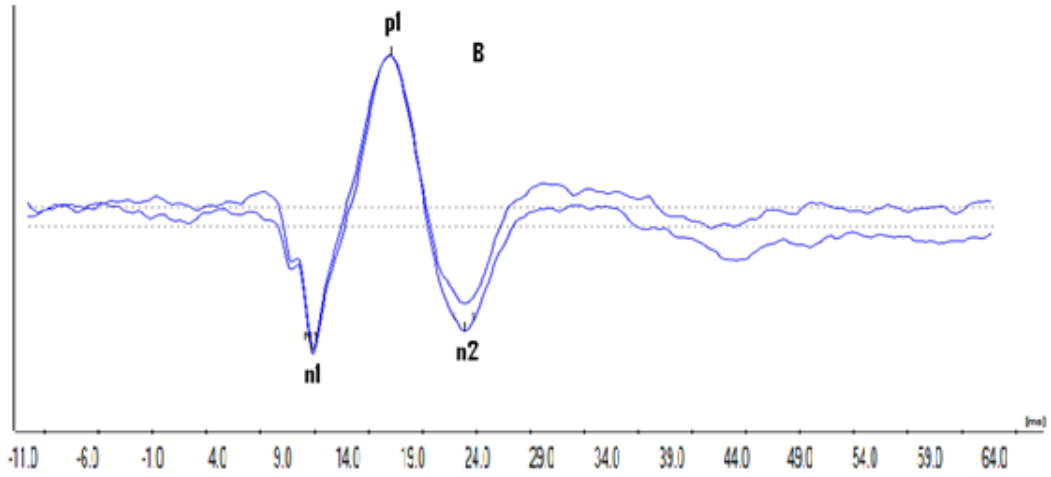
h. Multiple analysis of variance (MANOVA) was done to see the group differences for various latency and amplitude parameters of oVEMP.

i. Independent sample‘t’ test was done to compare the oVEMP latency and amplitude parameters between normal hearing individuals with individuals exposed to noise.

j. Pearson correlation test was done to find out a correlation between oVEMP findings with duration of noise exposure, threshold at 4 kHZ in individuals exposed to the noise.

The representations of waveform 0f cVEMP and OVEMP for control and experimental group are shown in figure-4.1





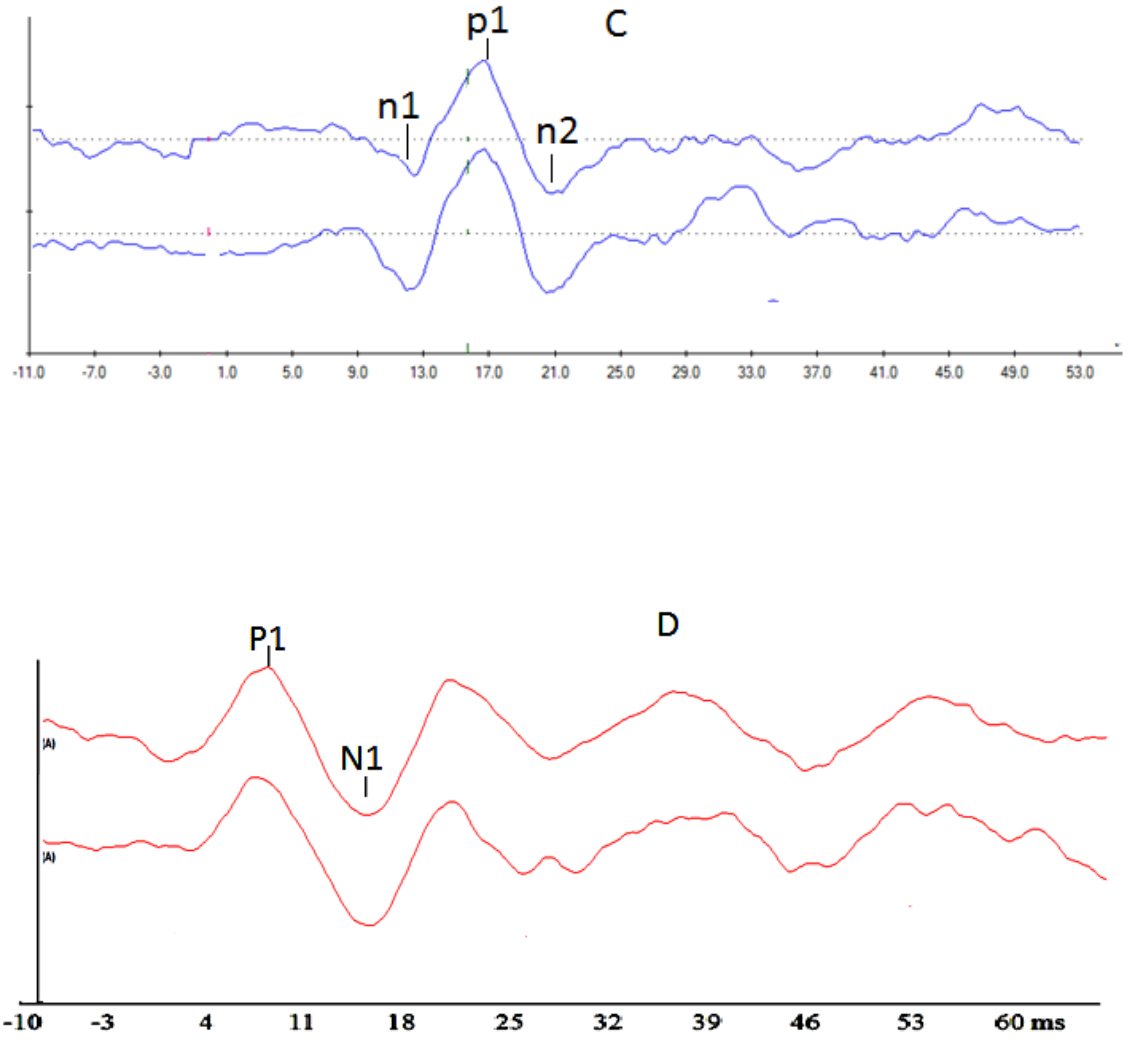


Figure-4.1. A. cVEMP waveform in normal hearing individual B. oVEMP waveform in normal hearing individual C. oVEMP waveform in NIHL individual D. cVEMP waveform in NIHL individual

Latency of cVEMP

The cervical VEMP responses could be recorded from both the ears of all the participants from for both the groups. The latency of P13 peak, N23 peak was measured for both the groups. Descriptive statistics was done to find out the mean and standard deviation (SD) for latency of P13 peak and N23 peak. The descriptive results of latency is given in table 4.1

Table 4.1

Mean and standard deviation of latency of peaks P13, N23 peak of the control group and the experimental group

Parameter	Control group				Experimental group			
	Right ear		Left ear		Right ear		Left ear	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
P13	14.00	1.76	14.04	0.77	16.31	1.77	14.40	1.04
N23	19.80	2.56	19.95	1.28	22.96	2.50	22.96	2.72

It can be seen from the table 4.1 that the mean latency of P13 peak and N23 peak is more for the experimental group compared to the normal hearing individuals for both the left and the right ear. To understand the overall effect of noise on various peak of cVEMP repeated measure ANOVA was done with group and ear as between subject factor. Repeated measure ANOVA revealed a significant main effect for peaks [$F(1, 36) = 321.82$, $p < 0.05$], Repeated measure ANOVA also showed a main effect for the groups [$F(1, 36) = 15.28$, $p < 0.05$], but it failed to show a main effect for the ear [$F(1, 36) = 2.09$, $p > 0.05$]. Further Repeated measure ANOVA showed no significant interaction

between peaks and the groups [F(1, 36)= 2.71, p>0.05], failed to show significant interaction between peaks and the ears [F(1, 36)= 0.38, p>0.05], failed to show interaction for peaks, ear and groups [F (1, 36) = 0.21, p > 0.05]. Repeated measure ANOVA also failed to show any interaction between groups and the ears [F(1, 36)= 2.68, p>0.05].

Since the ear did not show a significant main effect and also there was no interaction between the groups and the ears, data from two ears were combined. The mean and SD of the combined data were calculated in descriptive statistics and the result is shown in table 4.2

Table-4.2

Mean and standard deviation (SD) combined of the two ears

Parameter	Control group		Experimental Group	
	Combined			
	Mean	SD	Mean	SD
P13	14.02	1.32	15.36	1.72
N23	19.87	1.97	22.39	2.61

It can be seen from table-4.2 that the mean latency for both P13 and N23 peak are more for the experimental group compared to the control group.

Multiple analysis of Variance (MANOVA) was done to see overall group effect for latency of each peak separately. MANOVA showed significant main effect between groups for P13 peak latency [F (1, 38) = 7.59, p<0.05)], it also showed significant main effect for the latency of N23 peak latency [F (1, 38) = 11.87, p <0.05].

Also an Independent sample‘t test’ was done to understand the significant difference between the latency of P13 and N23 peaks between the two groups. Independent sample‘t’ test revealed a significant main difference between the P13 peak between the two groups ($t(38)=2.75$, $p<0.05$), also showed significant difference between the two groups for N23 latency ($t(38)=3.44$, $p>0.05$).

Amplitude measures of cVEMP

The cervical VEMP responses could be recorded from both the ears of all the participants from for both the groups. The P13- N23 amplitude was measured. Descriptive statistics was done to find out the mean and standard deviation for P13-N23 amplitude. The descriptive results of amplitude is given in table 4.3

Table 4.3

Mean and standard deviation of P13- N23 amplitude of the control group

	Control group				**Experimental group**			
	Right ear		Left ear		Right ear		Left ear	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Amplitude of p1n1	139.78	46.96	133.91	37.55	112.75	38.05	103.42	21.53

It can be seen from the table 4.3 that the mean amplitude of P13-N23 is more for the control group than experimental group for both the left and the right ear. To understand the overall effect of noise on amplitude of various peak of cVEMP repeated measure ANOVA was done with group and ear as between subject factor. Repeated measure ANOVA revealed a significant main effect for peaks [$F(1, 36) = 291.09$, $p <$

0.05], Repeated measure ANOVA also showed a main effect for the groups [F (1, 36) = 4.20, $p < 0.05$], but it failed to show a main effect for the ear [F (1, 36) = 0.27, $p > 0.05$]. Further Repeated measure ANOVA showed significant interaction between peaks and the groups [F (1, 36) = 6.76, $p<0.05$], failed to show significant interaction between peaks and the ears [F (1, 36) = 0.71, $p>0.05$], failed to show interaction for peaks, ear and groups [F (1, 36) = 0.06, $p > 0.05$]. Repeated measure ANOVA also failed to show any interaction between groups and the ears [F (1, 36) = 0.018, $p>0.05$].

Since the ear did not show a significant main effect and also there was no interaction between the groups and the ears, data from two ears were combined. The mean and SD of the combined data were calculated in descriptive statistics and the result is shown in table 4.4

Table-4.4

Mean and standard deviation (SD) combined of the two ears

Parameter	Control group		Experimental group	
	Mean	SD	Mean	SD
p1n1	136.85	41.49	108.08	30.47

It can be seen from table-4.4 that the mean amplitude of P13- N23 is more for the control group compared to the experimental group.

Multiple analysis of Variance (MANOVA) was done to see overall group effect for amplitude of P13-N23 complex. MANOVA showed significant main effect between groups for amplitude of P13-N23 complex [F (1, 38) = 8.30, $p<0.05$)]. Also an

Independent Sample‘t test’ was done to understand the significant difference between the amplitude of P13-N23 complex. Independent sample‘t’ test revealed a significant main difference between amplitude of P13-N23 complex between the two groups ($t(38)=2.88$, $p<0.05$).

Correlation of cVEMP results with the duration of exposure to noise

Pearson correlation was done to see if any correlation existed between the duration of exposure with that of the cVEMP results. Pearson correlation test revealed no significant correlation between duration of exposure to noise and P1 latency [$r(18) = 0.097$, $p > 0.05$], Pearson correlation also revealed no significant correlation between duration of noise exposure and N1 latency [$r(18) = 0.69$, $p > 0.05$], but it revealed a significant correlation between the duration of noise exposure and the P13-N23 amplitude complex [$r (18) = - 0.64$, $p<0.05$].

Correlation of cVEMP results with the threshold at 4 k Hz

Pearson correlation was done to see if any correlation existed between the thresholds at 4 kHz with that of the cVEMP results. Pearson correlation test revealed no significant correlation between threshold at 4 kHz and P1 latency [$r (18) = 0.40$, $p > 0.05$]. Pearson correlation also revealed no significant correlation between duration of noise exposure and N1 latency[$r(18) = 0.87$, $p > 0.05$], and revealed no significant correlation between the P13-N23 amplitude and cVEMP results[$r (18) = - 0.43$, $p > 0.05$]

Latency of oVEMP

The ocular VEMP responses could be recorded from both the ears of all the participants from for both the groups. The latency of n1 peak, p1 peak and n2 peak were measured. Descriptive statistics was done to find out the mean and standard deviation for

latency of n1 peak, p1 peak and n2 peak. The descriptive results of latency is given in table 4.5

Table 4.5

Mean and standard deviation of latency of peak n1, p1 and n2 of the control group

Parameter	**Control group**				**Experimental group**			
	Right ear		Left ear		Right ear		Left ear	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
n 1 latency	12.47	0.84	11.62	1.39	13.01	2.89	14.30	4.47
p 1 latency	17.95	0.76	16.84	2.04	18.56	3.36	19.35	4.71
n 2 latency	23.05	2.33	22.13	2.33	25.05	2.21	25.01	3.19

It can be seen from the table 4.5 that the mean latency of n1 peak is more for the experimental group for both the left and the right ear. It can be seen that the mean latency of p1 peak is more for the experimental group compared to the normal group for both the left and the right ear and it can also seen that the mean latency of n2 peak. To understand the overall effect of Noise on various peak of oVEMP, Repeated measure ANOVA was done with group and ear as between subject factor. Repeated measure ANOVA revealed a significant main effect for peaks [$F (1, 36) = 442.48$, $p < 0.05$], Repeated measure ANOVA failed to showed a main effect for the groups [$F (1, 36) = 2.445$, $p < 0.05$], also failed to show a main effect for the ear [$F (1, 36) = 0.125$, $p > 0.05$]. Further Repeated measure ANOVA showed a significant interaction between peaks and the groups [$F(1, 36)= 7.32$, $p<0.05$], showed no significant interaction between peaks and ears [$F(1, 36) =$

0.070, p<0.05] also it showed no interaction for peaks, ear and groups [F (1, 36) = 1.52, p > 0.05].

Since the ear did not show a significant main effect, data from two ears were combined. The mean and SD of the combined data were calculated in descriptive statistics and the result is shown in table 4.6

Table 4.6

Mean and standard deviation (SD) combined of the two ears

Parameter	Control group		Experimental group	
	Right ear	Left ear	Right ear	Left ear
	Mean	SD	Mean	SD
n1	12.04	1.20	13.65	3.72
p1	17.40	1.61	18.96	4.00
n2	22.59	2.32	25.03	2.67

It can be seen from table-4.6 that the mean latency for n1, p1 and n2 are more for the experimental group compared to the control group. The same can be seen in figure-3

Multiple analysis of Variance (MANOVA) was done to see overall group effect for latency of each peak separately. MANOVA showed no significant main effect r n1 peak latency [F (1, 38) = 2.18 p<0.05)], MANOVA also showed no main effect for the p1 latency [F(1, 38) = 1.96, p>0.05] respectively, whereas it showed significant main effect for the latency of n2 peak latency [F (1, 38) = 7.34, p <0.05].

Also an Independent sample‘t test’ was done to understand the significant difference between the latency of n1, p1 and n2 peaks between the two groups.

Independent sample 't' test revealed significant main difference between the n2 peak between the two groups ($t(38)= 2.71$, $p<0.05$), but it failed to show any significant difference between the two groups for n1 ($t(38)=-1.47$, $p>0.05$), and p1 latency ($t(38) = 1.39$, $p >0.05$).

Amplitude measures of oVEMP

The ocular VEMP responses could be recorded from both the ears of all the participants from for both the groups. The n1-p1 and p1-n2 amplitude were measured. Descriptive statistics was done to find out the mean and standard deviation for n1-p1 and p1-n2 amplitude. The descriptive results of amplitude is given in table 4.7

Table 4.7

Mean and standard deviation of n1-p1, p1-n2 amplitude of the control group

	Control group				**Experimental group**			
	Right ear		Left ear		Right ear		Left ear	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Amplitude of n1p1	5.40	1.04	5.08	0.98	4.42	0.62	4.08	0.54
Amplitude of p1n2	5.04	0.74	5.17	0.51	4.30	0.36	4.02	0.51

It can be seen from the table 4.7 that the mean amplitude of n1-p1 and p1-n2 is more for the control group than experimental group for both the left and the right ear. To understand the overall effect of Noise on various peak of oVEMP, repeated measure ANOVA was done with group and ear as between subject factor. Repeated measure

ANOVA revealed a significant main effect for peaks [F (1, 36) = 442.48, p < 0.05], Repeated measure ANOVA failed to showed a main effect for the groups [F (1, 36) = 2.445, p < 0.05], also failed to show a main effect for the ear [F (1, 36) = 0.125, p > 0.05]. Further Repeated measure ANOVA showed a significant interaction between peaks and the groups [F(1, 36)= 7.32, p<0.05], showed no significant interaction between peaks and ears [F(1, 36) = 0.070, p<0.05] also it showed no interaction for peaks, ear and groups [F (1, 36) = 1.52, p > 0.05]. Since the ear did not show a significant main effect, data from two ears were combined. The mean and SD of the combined data were calculated in descriptive statistics and the result is shown in table 4.8

Table-4.8

Mean and standard deviation (SD) combined of the two ears

Parameter	Control group		Experimental group	
	Mean	SD	Mean	SD
n1p1	5.24	0.99	4.25	0.59
p1n2	5.11	0.62	4.16	0.45

It can be seen from table-4.8 that the mean amplitude of n1-p1 and p1-n2 is more for the control group compared to the experimental group.

Multiple analysis of Variance (MANOVA) was done to see overall group effect for latency of each peak separately. MANOVA showed significant main effect for n1-p1

amplitude complex [F (1, 38) = 15.13, p<0.05)], MANOVA also showed significant main effect for the p1-n2 amplitude complex [F(1, 38) = 29.64, p<0.05].

Also an Independent sample 't test' was done to understand the significant difference between the amplitude of n1-p1 complex, p1-n2 complex between the two groups. Independent sample 't' test revealed significant main difference between the n1-p1amplitude complex between the two groups (t(38)= 3.89, p<0.05), it also showed significant difference between the two groups for p1-n2 amplitude complex (t(38)=-5.44, p>0.05).

Correlation of oVEMP results with the duration of exposure to noise:

Pearson correlation was done to see if any correlation existed between the duration of exposure with that of the oVEMP results. Pearson correlation test revealed no significant correlation between duration of exposure to noise and n1 latency [r (18) = -0.22, p > 0.05], no significant correlation between duration of noise exposure and p1 latency [r (18) = 0.043, p> 0.05], no significant correlation between duration of noise exposure n2 latency [r(18) = -0.301, p > 0.05]. Pearson correlation revealed no significant correlation between duration of noise exposure n1-p1 amplitude [r(18) =0.552,p>0.05] and no significant correlation between duration of noise exposure p1-n2 amplitude respectively and [r(18)=0.059, p > 0.05].

Correlation of oVEMP results with the threshold at 4 k Hz:

Pearson correlation was done to see if any correlation existed between the threshold at 4 kHz with that of the oVEMP results. Pearson correlation test revealed no

significant correlation between threshold at 4 kHz and n1 latency [r (18) = -0.061,p >0.05], Pearson correlation test revealed no significant correlation between threshold at 4 kHz and p1 latency [r(18) = -0.86, >0.05], Pearson correlation test revealed no significant correlation between threshold at 4 kHz and n2 latency [r (18) = -0.252, p >0.05]. Pearson correlation test revealed no significant correlation between threshold at 4 kHz and n1-p1 amplitude [r (18) = -0.072, p >0.05] Pearson correlation test revealed no significant correlation between threshold at 4 kHz and p1n2 amplitude [r (18)= -0.117, p>0.05].

To summarise the results, for cVEMP there was a significant main difference between the latency of P13 peak and N23 peaks between the control and the experimental groups, there was also a significant main difference between amplitude of P13-N23 complex between the two groups. For oVEMP latency, significant main difference was observed between the latency of n2 peak between the two groups; however, significant difference could not be seen between the two groups for n1 and p1 latency. A significant difference could also be observed between the n1-p1amplitude complexes between the two groups also showed significant difference between the two groups for p1-n2 amplitude complex for oVEMP. Further Pearson correlation did not reveal any significant correlation between duration of noise exposure and latency parameters of cVEMP or oVEMP findings, but Pearson correlation revealed significant correlation between duration of noise exposure and amplitude parameters of cVEMP and oVEMP. Pearson correlation analysis also did not reveal any significant correlation between threshold at 4KHZ and cVEMP or oVEMP findings.

Chapter-V

DISCUSSION

Present study was conducted with an aim of studying the results of cVEMP and oVEMP in noise induced hearing loss individuals. The objectives of the study were, studying cVEMP in noise induced hearing loss individuals, studying oVEMP in noise induced hearing loss individuals, studying the correlation between the duration of noise exposure with cVEMP & oVEMP findings in noise induced hearing loss individuals, and studying the correlation between puretone thresholds at 4 KHz and cVEMPs & oVEMP test results in noise induced hearing loss individuals. The discussion for the results are given below:

1. Cervical vestibular evoked myogenic potentials

There was a significant main difference between the control group and experimental group for P13 and N23 latency for cVEMP. There was a significant main difference between the control group and the experimental group for the amplitude of P13-N23 complex for cVEMP.

Cervical evoked myogenic potentials represent the summed responses from the saccule and its innervating structures (Kumar et al. 2010; Kumar et al. 2011; Akin et al. 2012). Previous studies which have reported findings of cervical evoked myogenic potentials have also reported a affected cVEMP findings in individuals with NIHL (Wang et al. 2006; Wang & Young 2007; Wu & Young 2009; Kumar et al. 2010; Akin et al. 2011). However there are equivocal findings regarding the effect of noise on latency of cVEMP in individuals with NIHL.

Present study reported that there was a significant delay in latency of cVEMP in individuals with NIHL. However, some of the previous studies (Wang et al. 2006; Wang & Young 2007; Wu & Young 2009: Akin et al. 2012) have not reported any latency delay in cVEMP results in individuals with NIHL. Results of the present study are in good agreement with Kumar et al (2010), where Kumar et al. (2010) also reported a delay in latency of cVEMP in individuals with NIHL.

The damage to the inner ear system due to the noise could be due to the either the direct mechanical injury to the inner ear or due to the metabolic changes in the inner ear (Lamm, & Arnold, 2000). The metabolic changes in the inner ear could be due to ischemia, generation of reactive oxygen species, toxic free radicals, metabolic exhaustation and ionic imbalance in the inner ear fluid (Lamm, & Arnold, 2000). These changes in the inner ear could lead to a damage to both the cochlear as well as vestibular system damage. However the damage to the peripheral damage could lead to the reduction in the amplitude of the vestibular evoked myogenic potentials and will not cause any delay in the latency of vestibular evoked myogenic potentials (Huang, Wang &Young, 2011; Jerin, Berman, Krause, Wagner & Gurkov 2014).

However in the present study, there was a delay in latency of vestibular evoked myogenic potentials in individuals with NIHL. Some of the previous studies on effect of cervical vestibular system have revealed that individuals who have a central lesion have a prolonged latency of cVEMP (Itoh et al. 2001; Pollack et al. 2006; Tseng & Young 2010). Since in the present study also there was a significant delayed cVEMP in individuals with NIHL, it is hypothesized that there could be a secondary damage to the vestibular nerves attached to the saccular system.

There was no correlation between the latency of cVEMP and duration of noise exposure, however there was a significant correlation between cVEMP amplitude and duration of noise exposure.

Previous studies which have evaluated the auditory system in individuals with NIHL have also reported a significant correlation between the duration of noise exposure and the damage to the inner ear. For example, Ahmed (2001) reported a significant correlation between duration of noise exposure and the high frequency thresholds, Morata (1989), also reported a decline in hearing threshold as the years of noise exposure increased, also Hotzet al. (1993) reported a decline in OAE amplitude with increase in years on noise exposure. Since, the inner ear cavity contains both the cochlear and vestibular systems, it can be hypothesized that with the increase in years of exposure to the noise there could be a more damage to the vestibular system. However, there was a correlation between amplitude of cVEMP and years of noise exposure but there was no correlation between the cVEMP latency and duration of noise exposure. This could be due to the fact that the saccule system might get affected faster compared to the neuronal system of the vestibular system. Hence, there could be a more reduction in the amplitude of the CVEMP compared to the increase in latency of cVEMP.

Also, t*here was no significant correlation between latency or amplitude parameters of cVEMP and 4 KHz thresholds in experimental group*.

The changes observed in the results of the vestibular evoked myogenic potentials are more likely due to the damage of the saccule and its innervating structures than related to the hearing loss as it has been well established that the presence/absence of

VEMP is independent of the cochlear functions (Bickford et al. 1964; Colebatch et al. 1994; Ozeki et al. 1999).

2. Ocular vestibular evoked myogenic potentials

There was no significant difference between control group and experimental group for n2 latency of oVEMP. However, there was no significant difference between control group and experimental group for latency of n1 peak and p1 peak. However, there was a significant difference in amplitude of the oVEMP between the normal hearing individuals and individuals with NIHL.

The effect of noise exposure on vestibular evoked myogenic potentials have just started to appear and these studies have reported a damage to the saccule and its innervating structures utilizing the cervical vestibular evoked myogenic potentials (Wang et al. 2006; Wang & Young 2007; Wu & Young 2009; Kumar et al. 2010; Akin et al. 2011). However, there is dearth of information on how the noise exposure affects the otolith ocular pathway. This is the first study describing the damage of the otolith ocular pathway in individuals with NIHL. The possible reason for the damage to the otolith ocular pathway could be due to metabolic changes in the inner ear such as ischemia, generation of reactive oxygen species, toxic free radicals, metabolic exhaustation and ionic imbalance in the inner ear fluid (Lamm, & Arnold, 2000) or could be due to a direct mechanical injury to the inner ear (Lamm & Arnold, 2000).

However, the latency of cVEMP was more in individuals with NIHL compared to the normal hearing, whereas in the oVEMP results there is no significant difference in the latency of oVEMP parameters between the normal hearing individuals compared to the

individuals with NIHL. This could be due to a differential damage to the innervating structures of the saccule than the utricle.

There was no correlation between the latency of oVEMP and duration of noise exposure, however there was a significant correlation between oVEMP amplitude and duration of noise exposure.

Since, the inner ear cavity contains both the cochlear and vestibular systems, it can be hypothesized that with the increase in years of exposure to the noise there could be a more damage to the vestibular system. However, there was a correlation between amplitude of oVEMP and years of noise exposure but there was no correlation between the oVEMP latency and duration of noise exposure. This could be due to the fact that the saccule system might get affected faster compared to the neuronal system of the vestibular system. Hence, there could be a more reduction in the amplitude of the oVEMP compared to the increase in latency of oVEMP.

There was no significant correlation between latency or amplitude parameters of oVEMP and 4 KHz thresholds in experimental group.

The changes observed in the results of the vestibular evoked myogenic potentials are more likely due to the damage of the utricle and its innervating structures than related to the hearing loss as it has been well established that the presence/absence of oVEMP is independent of the cochlear functions (Bickford et al. 1964; Colebatch et al. 1994; Ozeki et al. 1999).

Chapter-V1

SUMMARY AND CONCLUSIONS

Noise Induced Hearing Loss is a term that refers to acquired loss of hearing due to damage of the inner ear, as a result of exposure to excessive noise. It is also referred to as Noise Induced Hearing Impairment. Noise-induced hearing loss can be temporary or permanent. Temporary hearing loss results from short-term exposures to noise, with normal hearing returning after a period of rest. Generally, prolonged exposure to high noise levels over a period of time gradually causes permanent damage.

Present study was conducted with an aim of studying the results of cVEMP and oVEMP in noise induced hearing loss individuals. The objectives of the study were, studying cVEMP in noise induced hearing loss individuals, studying oVEMP in noise induced hearing loss individuals, studying the correlation between the duration of noise exposure with cVEMP & oVEMP findings in noise induced hearing loss individuals, and studying the correlation between puretone thresholds at 4 KHz and cVEMPs & oVEMP test results in noise induced hearing loss individuals.

To achieve the aim, 10 participants with a history of noise exposure in the age range of 25 to 50 years participated with the mean age of 37.5 years. Also, 10 individuals with normal hearing in the age range of 25 to 50 years with the mean age of 37.8years participated in the study. A detailed case history was taken before doing the testing. A series of routine audiological test battery was conducted which included pure tone audiometry, speech audiometry, immitance, oto-acoustic emission, auditory brain stem responses. Later participants were subjected to cervical vestibular evoked myogenic potential and ocular vestibular evoke myogenic potential.

Cervical vestibular evoked myogenic potentials were recorded with 500 Hz tone-burst stimuli presented at 125 dB SPL in a rarefaction polarity. Ocular vestibular evoked myogenic potentials were recorded with 500 Hz tone burst stimuli presented at 125 dB SPL in a rarefaction polarity. Cervical vestibular evoked myogenic potentials were recorded in an ipsilateral mode whereas the ocular vestibular evoked myogenic potentials were recorded in a contralateral mode.

Following statistical analyses were done

a. Descriptive statistics to obtain mean and standard deviation for P13 latency, N23 latency, and amplitude of P13-N23 amplitude complex of cVEMP for normal hearing individuals and individuals exposed to noise.

b. Repeated Measure ANOVA was done to see the overall effect of noise on cVEMP latency and amplitude parameters with group and ear as between subject factors.

c. Multiple analysis of variance (MANOVA) was done to see the group differences for various latency and amplitude parameters of cVEMP.

d. Independent sample‘t’ test was done to compare the cVEMP latency and amplitude parameters between normal hearing individuals with individuals exposed to noise.

e. Pearson correlation test was done to find out a correlation between cVEMP findings with duration of noise exposure, threshold at 4 kHZ in individuals exposed to the noise.

f. Descriptive statistics to obtain mean and standard deviation for n1 latency, p1 latency, n2 latency and amplitude of n1-p1 amplitude complex and p1-n2

amplitude complex oVEMP for normal hearing individuals and individuals exposed to noise.

g. Repeated Measure ANOVA was done to see the overall effect of noise on oVEMP latency and amplitude parameters with group and ear as between subject factors.

h. Multiple analysis of variance (MANOVA) was done to see the group differences for various latency and amplitude parameters of oVEMP.

i. Independent sample't' test was done to compare the oVEMP latency and amplitude parameters between normal hearing individuals with individuals exposed to noise.

j. Pearson correlation test was done to find out a correlation between oVEMP findings with duration of noise exposure, threshold at 4 kHZ in individuals exposed to the noise.

Results of the study revealed the following:

a. There was a significant main difference between the control group and experimental group for P13 and N23 latency for cVEMP.

b. There was a significant main difference between the control group and the experimental group for the amplitude of P13-N23 complex for cVEMP.

c. There was no significant difference between control group and experimental group for n2 latency of oVEMP. However, there was no significant difference between control group and experimental group for latency of n1 peak and p1 peak.

d. There was a significant difference in amplitude of n1-p1 and p1-n2 peak between the control hearing and experimental group, wherein the amplitude of oVEMP parameters was lesser in individuals with NIHL than the normal hearing individuals.

e. There was no correlation between the latency of cVEMP and duration of noise exposure, however there was a significant correlation between cVEMP amplitude and duration of noise exposure.

f. There was no significant correlation between latency or amplitude parameters of cVEMP and 4 KHz thresholds in experimental group.

g. There was no correlation between the latency of oVEMP and duration of noise exposure, however there was a significant correlation between oVEMP amplitude and duration of noise exposure.

h. There was no significant correlation between latency or amplitude parameters of cVEMP and 4 KHz thresholds in experimental group.

Conclusions:

The findings of the present study reveal that there could be a damage to the sacculocollic and utriculo-ocular pathway in individuals who are exposed to noise. Some of the previous studies have reported damage to the saccule and its innervating structure. cVEMP and oVEMP tests assess the sacculocollic pathway and utriculo-ocular pathway which might be sensitive to the noise exposure. The study also reveals that the as the duration of the noise exposure increases there could be more damage to the saccular or the utricular system. Both cVEMP and oVEMP are non invasive techniques that can be

utilized for assessment of saccular and utricular pathway in individuals with noise induced hearing loss.

Implications of the study:

- Results of the present study add information to the literature, which are helpful in providing information about the otolith dysfunction in noise induced hearing loss individuals.
- Combining oVEMPs and cVEMPs result provides complementary information about saccular & utricular function in individuals with NIHL.

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