

**Assessment of the otolithic and semicircular canal
function in individuals with sensorineural hearing loss**

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ALL INDIA INSTITUTE OF SPEECH AND HEARING

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April, 2018

CERTIFICATE

This is to certify that this dissertation entitled “**Assessment of the otolithic and semicircular canal function in individuals with sensorineural hearing loss**” bonafide work submitted in part fulfillment for the degree of Master of science (Audiology) of the student (Registration No: 16AUD014). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award or any other diploma or degree.

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April, 2018

CERTIFICATE

This is to certify that this dissertation entitled “**Assessment of the otolithic and semicircular canal function in individuals with sensorineural hearing loss**” bonafide work submitted in part fulfillment for the degree of Master of science (Audiology) of the student (Registration No: 16AUD014). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other university for the award or any other diploma or degree. It is also certified that this has not been submitted earlier to any other university for the award or any other Diploma or Degree.

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DECLARATION

I hereby declare that this dissertation entitled “**Assessment of otolithic and semicircular canal function in sensorineural hearing loss**” is the result of my own study under guidance of **Dr. Sujeet Kumar Sinha**, Reader in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Manasagangothri, Mysuru and has not been submitted earlier to any other university for the award or any other Diploma or Degree.

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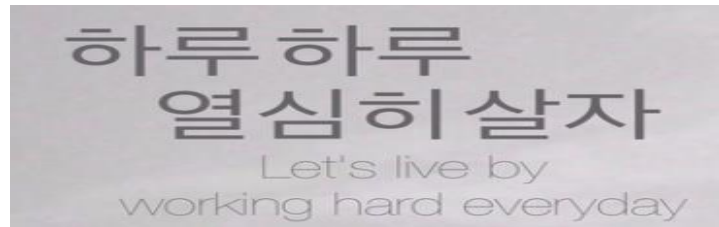
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Abstract

Introduction: There is a great association between vestibular and balance disorders with sensorineural hearing loss as it is anatomically related. Therefore, inner ear diseases can cause damages to both cochlear and vestibular organs.

Aim of the study: The aim of this study was to assess the functioning of otolithic organ and three semicircular canals in individuals with various degree of sensorineural hearing loss.

Methods: two groups of individuals were taken for the study. Group-I consisted of 40 individuals with various degrees of hearing loss within the age range of 18-44 years. Group II consisted of 40 participants with normal hearing within the age range of 18-35 years. All the participants had undergone a detailed case history, administered dizziness questionnaire, pure tone audiometry, Immittance audiometry and acoustic reflex threshold test, cVEMP, oVEMP and vHIT.

Results: Both cVEMP and oVEMP were present in all the individuals with normal hearing sensitivity. However, the cVEMP and oVEMP were absent in most of the individuals with hearing loss. The VOR gain measured with vHIT was normal in individuals with hearing loss, however, there was a presence of saccade in 9 individuals with hearing loss.

Conclusions: Saccular and utricular dysfunction is more prevalent in individuals with hearing loss compared to the canal dysfunction. Hence, the individuals with hearing loss must be assessed for vestibular dysfunction using various test battery.

CHAPTER I

INTRODUCTION

The inner ear contains two sensory organs namely, auditory and vestibular, connected anatomically (receptor cell ultra-structures) and functionally incased within same membranous labyrinth and also shares common labyrinthine artery (Xu et al., 2016; Zhou, Wu, & Wang, 2016).Therefore, damage to the one structure can cause damage to the other also or lesions leading to sensorineural hearing loss could also contribute to dysfunction of the vestibular end organs (Singh, Gupta, & Kumar, 2012).

Studies have reported that the hearing loss is also accompanied by vestibular symptoms like vertigo and nausea sometimes. But most of the time vestibular symptoms are hidden in these individuals. Many studies have found that approximately 20-85% of hearing impaired children has vestibular dysfunction (Kegel et al., 2014). Several authors have reported that hearing impairment is accompanied with vestibular symptoms in various conditions like acoustic neuromas (Dix & Hallpike, 1960), sudden deafness caused by infection and diving (Schuknecht, Kimura, & Naufal, 1973)and hearing loss because of ototoxicity (Moroso & Blair, 1983).

The sensory organ of the vestibular system comprises of the otolithic organs; saccule, utricle and the six semicircular canals (lateral, anterior and posterior). The otolithic organs and semicircular canals sense linear and angular acceleration respectively. The function of otolithic organs can be evaluated by the vestibular evoked myogenic potential (VEMP) test, which was first reported by Colebatch & Halmagyi, (1992). It is now the standard test for otolithic function in people with vestibular impairment. There are two types of VEMP tests: the cervical VEMP (cVEMP) (Colebatch & Halmagyi, 1992) test and the ocular VEMP (oVEMP) test (Rosengren, McAngus Todd, & Colebatch, 2005). These are short latency responses evoked by high

level of acoustic stimuli and are detected with the surface electrodes. VEMPs testing can detect changes occurring in the vestibular organs.

The cervical VEMP (cVEMP) reflects the functioning of the saccule and inferior vestibular nerve, which has strong projections to the sternocleidomastoid muscle and only weak projections to the oculomotor system (Walther & Blödown, 2013). It is characterized by biphasic waves (positive and negative). cVEMP has been utilized for the diagnosis of pathology confined to sacculocollic pathways in various disorders such as Ménière's disease (Young, Huang, & Cheng, 2003), acoustic neuromas (Murofushi et al., 1998), superior canal dehiscence syndrome (Zuniga & Janky, 2013), vestibular neuritis (Manzari, Burgess, McGarvie, & Curthoys, 2012), noise induced hearing loss (Kumar, Vivarthini, & Bhat, 2010), auditory neuropathy (Sinha et al. 2013; Sinha et al. 2014), idiopathic sudden sensorineural hearing loss with vertigo (Iwasaki et al., 2005).

The ocular VEMP (oVEMP) reflects the functioning of the utricle and the superior vestibular nerve, which have strong projections to the inferior oblique muscle of the lower eyelid (Niu et al., 2016). It has been used to diagnose the pathology of the otolith ocular pathways in various vestibular disorders like Ménière's disease (Winters, Campschroer, Grolman, & Klis, 2011; Young et al., 2003), superior canal dehiscence syndrome (Zuniga & Janky, 2013), vestibular neuritis (Manzari, Tedesco, Burgess, & Curthoys, 2010), vestibular schwannoma (Chiarovano, Darlington, Vidal, Lamas, & Waele, 2014), differential diagnosis of brainstem and cerebral lesions (Su & Young, 2011), multiple sclerosis (Gazioglu & Boz, 2012) and auditory neuropathy (Sinha et al. 2014).

The vestibulo-ocular reflex (VOR) maintains stable vision of the target during head movement. Video Head Impulse test (vHIT) is a dynamic vestibular test to assess

all the six semicircular canal which incorporates a new technology that uses a high speed, lightweight video goggle to measure eye velocity and record “catch up” saccades (and other abnormalities) in patients with impaired VOR function. It provides a quick and objective measure of the vestibular ocular reflex (VOR) in response to head movements in the natural range of daily motions.

vHIT detects overt and covert catch-up saccades in patients with various vestibular disorders (MacDougall, Weber, McGarvie, Halmagyi, & Curthoys, 2009). vHIT has been used to detect the pathology in the VOR pathways in various vestibular pathology like Ménière’s disease (Albernaz, 2014) and helps monitoring the effectiveness of intratympanic gentamycin, vestibular neuronitis, central inner ear disorders, metabolic inner ear disorders, vestibular schwannoma (Albernaz, 2014), vestibular neuritis (Bartolomeo et al., 2014), differentiates vertigo from peripheral or central disorders, bilateral vestibular loss (Halmagyi et al., 2017). vHIT also provides effective diagnostic information in the pediatric population (Hamilton, Zhou, & Brodsky, 2015).

NEED OF THE STUDY

1.1. Need of study in sensorineural hearing loss

There is a great association between vestibular and balance disorders with sensorineural hearing loss as it is anatomically related. Therefore, inner ear diseases cause damages to both cochlear and vestibular organs. Xu et al., (2016) reported that, vestibular and cochlear symptoms occurred simultaneously in more than half of the patients. However, most of the patients with sensorineural hearing loss do not exhibit vestibular symptoms (like imbalance, dizziness, spatial disorientation and blurred

vision). Because the vestibular symptoms are hidden and thus easily overlooked in patients with profound sensorineural hearing loss (Zhou, Kenna, Stevens, & Licameli, 2009). The patients with sensorineural hearing loss (SNHL) are likely to have subclinical disorders of the vestibular system, particularly disorders involving the saccule (Sazgar, Dortaj, Akrami, Akrami, & Karimi Yazdi, 2006). Angeli (2003) reported that approximately 70% of children with hearing impairment has vestibular malfunction with 20% - 40% having severe bilateral vestibular loss (Angeli, 2003). Studies have also reported high incidence of vestibular hypofunction in children with SNHL (Rine et al., 2000). Studies reported bilateral vestibular loss in 63% of the children with acquired hearing loss and had unilateral vestibular loss in 37.5% of the children with sensorineural hearing loss (Cushing et al., 2009), 35% of the individuals with sensorineural hearing loss had unilateral vestibular loss and 50% had bilateral vestibular loss (Kaga, 2008). Since the prevalence of vestibular loss is high, there is a need to study the vestibular function in individuals with sensorineural hearing loss.

1.2. Need of study in VEMPs

Cervical VEMP tests the integrity of sacculocollic pathway and ocular VEMP assess integrity of utriculocollic pathway. The function of otolithic organ was evaluated via cVEMP and oVEMP tests and was found that oVEMP responses in 38.9% of ears in patients with PSHL and cVEMP responses in 44.4% of ears in patients with PSHL. When compared to the response rate (100%) in healthy subjects to that of the individuals with PSHL, it indicates that patients with PSHL have a high incidence of damage to the otolithic organs (Xu et al., 2016). Zuniga et al. (2012) reported that there is a strong association between hearing loss and saccular function, suggesting that PSHL patients may exhibit saccular and inferior vestibular nerve dysfunction. Hong et al. (2008) reported that only 26.9% of patients with PSHL exhibited abnormal or absent

cVEMP responses, but it also showed that the more severe the hearing loss, the more likely the saccule was to be involved. Emami & Farahani, (2015) found that there is a correlation between saccular dysfunction and the extent of hearing loss in children with severe sensorineural hearing loss, suggesting that saccular dysfunction may be a concomitant indicator of the severity of hearing disorders. Jin et al. (2010) reported abnormal cVEMP in 25% of the participants with congenital hearing loss. As the various studies showed the saccular involvement in sensorineural hearing loss, there is a need to study the cervical vestibular evoked myogenic potentials in individuals with sensorineural hearing loss.

1.3. Need of study in vHIT

vHIT is a well-established recently developed vestibular function test which helps to assess all six semicircular canals, by measuring the gain of the vestibulo-ocular reflex (VOR). It evaluates the high frequency range of the VOR. It allows the examiner to see corrective saccades with the help of goggles and its sensors. It provides a reliable, repeatable, documented measure of vestibular function. Albernaz (2005) found hypoactive responses of one of the lateral canals in 4 patients and 2 had normal vHIT responses in total of six patients having mild low and high tone hearing losses with normal thresholds for the middle frequencies.

It allows the examiner to rectify the clinical diagnosis and determine whether the entire semicircular canals are affected, or not. For example, in case of vestibular neuritis, this can affect the superior vestibular nerve, damaging the anterior and lateral canal, the inferior vestibular nerve, damaging only the posterior canal, or both. vHIT detects dysfunction of individual lateral and vertical semicircular canals in vestibular dysfunction patients. When combined with the vestibular evoked myogenic potential testing (VEMP) for otolithic organ functioning testing, it gives the function of all

vestibular sense organs. Thus, evidently, abnormality of vestibular function is associated with sensorineural hearing loss and it is most of the time hidden. Therefore, vestibular function testing should be used as an objective procedure to evaluate potentially hidden dysfunction during diagnosis and to choose appropriate treatment for the individuals.

AIM OF THE STUDY

The aim of this study was to assess the functioning of otolithic organ and three semicircular canals in individuals with various degree of sensorineural hearing loss.

OBJECTIVES OF THE STUDY

1. To assess the functioning of saccule in the individuals with sensorineural hearing loss.
2. To assess the functioning of the utricle in the individuals with sensorineural hearing loss.
3. To assess all three semicircular canal in the individuals with sensorineural hearing loss.
4. To find out correlation between the cervical VEMP, ocular VEMP and vHIT with degree or severity of hearing loss.
5. To find out correlation between the cervical VEMP, ocular VEMP and vHIT findings in individuals with sensorineural hearing loss.
6. To find out correlation between the cervical VEMP, ocular VEMP and vHIT findings with the duration of sensorineural hearing loss.

Chapter II

REVIEW OF LITERATURE

The inner ear consists of 2 sensory organs i.e., cochlea for hearing and otolithic and six semicircular canals for vestibular functions and these two organs are connected anatomically. Several studies have reported that the lesion leading to the cochlea also cause lesion to the vestibular organs. Studies have revealed that the hearing loss is accompanied with the vestibular disorders or symptoms but these symptoms are not exhibited, therefore it goes unidentified. Many studies reported that approximately 20-85% of hearing impaired children has vestibular dysfunction (Kegel et al., 2014) and several studies have reported that hearing impairment is accompanied with vestibular symptoms in various conditions like acoustic neuromas (Dix & Hallpike, 1960), sudden deafness caused by infection and diving (Schuknecht, Kimura, & Naufal, 1973) and hearing loss because of ototoxicity (Moroso & Blair, 1983). Zuniga (2012) suggested from result of his study that person with sensorineural hearing loss (PSHL) may exhibits saccular and inferior vestibular nerve dysfunction.

2.1. Vestibular test findings in individuals with different types of sensorineural hearing loss

2.1.1 Sudden sensorineural hearing loss

It is characterized by the sudden loss of hearing of 20dB or more in two consecutive frequencies without any known cause exists within 3 days or less. In sudden sensorineural hearing loss, the results of the various vestibular tests have been reported to be abnormal. Studies have revealed around 40-60% of sudden sensorineural hearing loss exhibited vestibular symptoms along with cochlear.

Park, Jung and Rhee (2001) diagnosed the vestibular deficits in the sudden sensorineural hearing loss with vertigo and correlate with the recovery rate. There were total 125 subjects with sudden sensorineural hearing loss in which 54 subjects had vertigo complaints with 59% of spinning vertigo and 40% reported non-spinning vertigo complaints. The authors carried out an electronystagmography battery (Dix–Hallpike test, caloric test), rotation chair test and computerized dynamic posturography for assessment of vestibular function. The authors reported the vestibular diagnosis as unilateral hypofunction in 30%, BPPV in 25%, directional preponderance in 8%, irritative in 8%, non-specific in 11% and normal in 19%. They also reported that the recovery rates were worse for the individuals with vertigo symptoms as compared to the without vertigo symptoms. The authors concluded that the otolithic and semicircular involvement in the sudden sensorineural hearing loss results in worse recovery rates.

Iwasaki et al. (2005) also tried to evaluate the extent of vestibular lesion in idiopathic sudden hearing loss with vertigo using vestibular evoked myogenic potentials (VEMPs) in with click and galvanic stimuli and caloric test. The vestibular testing was performed within one month after the onset of hearing loss. There were 22 subjects who underwent click evoked VEMPs and caloric test and 8 subjects underwent galvanic VEMPs. Clicks stimulus were delivered through headphone at 95dBnHL and a 3-mA galvanic stimulation was presented to the mastoid (cathode) for recording galvanic VEMP. The authors found absence of click evoked VEMP in 77% in the affected side and normal responses in the unaffected side; reduced caloric responses in 45% in the affected side; normal VEMP and caloric responses in 18% in the affected side. All 8 subjects showed normal responses in galvanic VEMP. The authors

concluded that that most of the participants have lesion in the inner ear and the vestibular nerves.

Rambold et al. (2005) examined differential vestibular dysfunction in 29 subjects with sudden unilateral hearing loss. The authors found vestibular lesion in 45%, of which 53% had a combined impairment of the cochlea and the ipsilateral posterior semicircular canal, possibly reflecting vascular disease in the common cochlear artery.

Sehta (2011) examined vestibular function in 10 individuals with unilateral sudden sensorineural hearing loss using electronystagmography, cervical and ocular vestibular evoked myogenic potentials. The authors reported that absence of cervical evoked myogenic potentials responses in 90% of the affected ears and 10% had normal response; absence of ocular evoked myogenic potentials in 80% and normal in 20%; showed hypoactive responses in 100% in caloric stimulation. There was an association between ocular and cervical VEMP but not with caloric stimulation; no association with degree and duration of hearing loss and the tests results and recommended to use all these tests when diagnosing because all these tests evaluates different systems.

Ogawa et al. (2012) examined vestibular and balance function in 65 patients with sudden sensorineural hearing loss using subjective vertical visual perception (SVV) and vestibular evoked potentials. The authors reported that 23% showed abnormal SVV and 36% showed abnormal VEMP results. They also observed that rate of abnormal SVV and VEMP was greater in person with dizziness symptoms.

Khan, Balraj and Lepcha (2013) tried to investigate the damage to the saccule in 11 subjects with idiopathic sudden sensorineural hearing loss out of which 9 had unilateral loss and 2 had bilateral loss and with or without vertigo. They carried out VEMP using clicks at 95 dBnHL. They reported that in the unilateral unaffected ear,

88% had normal VEMP and 12% had absent VEMP; 53% had normal VEMP and 46% had absent VEMP in affected ears (13 ears). The authors concluded that the saccule damage was seen in these subjects but the extent of damage could not be corresponded with the severity of the hearing loss.

Kim, Na, Park and Shin (2013) investigated vestibular function in 26 patients with sudden sensorineural hearing loss with vertigo. The vestibular function tests implemented were slow phase velocity of spontaneous nystagmus; bithermal caloric tests with the water irrigation for 30 seconds in both ears of 30 and 44 degree C and canal paresis were calculated; subjective vertical visual was carried out in a dark where dim light was displayed in the computer. The line was 3 mm wide and 230 mm long with a dot of 3 mm in diameter which was superimposed along the line p of the rotational axis of the line. The subjects were seated upright on the chair and were asked to adjust the visual rod to the vertical position by manipulating with the remote controller. The subject were instructed that whenever the line appears vertical, click the enter button and it will automatically calculate the deviation in degrees from the true gravitational vertical. The tests were carried on within 10 days of first presentation of the vertigo. The authors reported that spontaneous nystagmus was observed in 58%, abnormal canal paresis in 50% and subjective vertical visual in 10%.

Lee et al. (2014) implemented cervical vestibular evoked myogenic potentials (cVEMP) and caloric test on 92 individuals with idiopathic sudden sensorineural hearing loss and tried to correlate with the severity, pattern and prognosis. The bithermal caloric (44 and 30) was carried out with water caloric irrigator and the cVEMP was done with clicks stimuli. The authors found abnormal caloric responses in 50% and abnormal cVEMP results in 32%. They found no significant differences with respect to the severity and patterns of hearing loss. The authors concluded that the

superior nerve involvement in ISSNHL is more frequent and also results in worse hearing outcome.

Fujimoto et al. (2014) tried to investigate the involvement of vestibular organs in 23 persons with idiopathic sudden sensorineural hearing loss with vertigo using cervical and ocular vestibular evoked myogenic potentials and caloric test. The authors reported abnormal responses of 64% in cervical vestibular evoked myogenic potentials (cVEMPs), and 52% of abnormal responses in caloric test. Ocular vestibular evoked myogenic potentials (oVEMPs) responses were abnormal in 43%. The authors also classified these subjects into cochlear type (26%), cochlear-sacculle type (17%), cochlear- lateral semicircular canal type (4%), cochlear-sacculle-utricle type (4%), cochlear-sacculle-lateral semicircular canal type (9%), cochlear-utricle-lateral semicircular canal type (4%) and cochlear-sacculle-utricle-lateral semicircular canal type (35%). The authors concluded that the vestibular end organs close to the cochlea are affected preferentially.

Nui et al. (2015) assessed 149 individuals with sudden sensorineural hearing loss, with or without vertigo, using cervical vestibular evoked myogenic potentials (cVEMP), ocular vestibular myogenic potentials (oVEMP) and caloric test. The cVEMP was carried out through air conduction of 500Hz toneburst of rise and fall time of 1 ms and plateau of 2 ms at the intensity of 131dB SPL. The authors reported abnormal caloric test results in 53% of the individuals with vertigo and 69% of the individuals without vertigo. cVEMP test results were found to be abnormal in 45% and 43% of the individuals and oVEMP was found to be abnormal in 56% and 70% of the individuals with and without vertigo.

Lui et al. (2017) evaluated 35 subjects with idiopathic sudden sensorineural hearing loss with pure tone audiometry, sensory organization test, cervical and ocular

vestibular evoked myogenic potentials and caloric test and the relationship between severities of hearing loss. The caloric test was carried out with videonystagmography and air caloric irrigator system. The authors found the highest rate of abnormal responses in oVEMP followed by caloric test and cVEMP in subjects with vertigo and without vertigo. SOT results and presence of vertigo symptoms were significantly correlated.

2.1.2 Noise induced sensorineural hearing loss

Oosterveld, Polman and Schoonheydt (1980) assessed 29 individuals with industrial hearing loss of various degrees. The authors observed spontaneous nystagmus in 18, positional nystagmus in 24 persons, cervical nystagmus in 17, nystagmus preponderance in rotational test of 7 individuals was more than 20%. The author did not observe any abnormality in central test and found no correlation with the different degrees of hearing loss. They finally concluded that the noise not only damages the cochlea but also vestibular organs.

Shupak et al. (1994) examined vestibular function 22 individuals with noise induced hearing loss using electronystagmography (ENG) and the smooth harmonic acceleration (SHA) test. The authors reported reduction in vestibular ocular reflex gain and caloric responses. The authors also reported a significant correlation between degree of the hearing loss, reduction in the average VOR gain and ENG caloric lateralization. The authors concluded that there might be a single mechanism for both cochlear and vestibular noise induced injury.

Golz et al. (2001) assessed effects of intense noise exposure on the vestibular organs on 258 individuals categorized into groups of asymmetrical and symmetrical hearing losses as well as on the presence or absence vestibular complaints. The subjects underwent detailed audiological and electronystagmographic evaluation. The authors

found vestibular damages in individuals with asymmetrical hearing loss and also no correlation with the severity and vestibular symptoms.

Wang and Young (2007) did a prospective study to see the consequences of chronic noise exposure in 20 individuals with notched at 4 KHz. All the subjects underwent pure tone audiometry, caloric testing and vestibular evoke myogenic potentials. The authors reported abnormal caloric responses in 45% and vestibular evoked myogenic potentials 50% and reported that the combined abnormal responses were 70%. The authors further concluded that the individuals with history of chronic noise exposure represented as bilateral 4KHz notched and hearing level of more than 40dBHLn might exhibit abnormal responses suggesting sacculocollic reflex pathway dysfunction.

Fakharnia, Sheibanizadeh, Jafari and Hoseini (2009) examined 30 individuals with noise induced hearing loss using cervical vestibular evoked myogenic potential (cVEMP) and caloric test. The authors reported no significant difference in unilateral weakness between two groups and the difference in mean latencies of p13 in the right ear. However, the latency of p13 of left ear was significantly different between the two groups. They also found that, the difference in n23 latency was significantly different only in the right ear and no significant difference between groups in p13-n23 amplitude. They concluded that the pars inferior of the vestibule is more prone to damaged caused by noise exposure.

Manasa (2009) assessed cochlear and vestibular function in 30 individuals with noise induced hearing loss and 30 normal hearing individuals using vestibular evoked myogenic potentials and transient evoked otoacoustic emissions (TEOAEs) to compare which organ is more susceptible to damage due to noise exposure either cochlea or the saccule. The VEMP was recorded using 500Hz toneburst at the intensity of 95dBnHL.

The author found that the response rate for normal was 85% as compared to the response rate for the noise induced hearing loss individuals was 61.4%, reduced p1-n1 amplitude in 51.43% in noise induced hearing loss. The TEOAEs response rate was 100% in normal hearing as compared 35.09% in noise induced hearing loss individuals. The author concluded that the cochlea is more susceptible to noise exposure.

Kumar (2010) recorded cervical vestibular evoked myogenic potentials in 30 individuals with noise induced hearing loss. cVEMP was recorded using 500 Hz tone burst stimuli presented at 99 dBnHL. The authors reported absence of cVEMP responses in 29% of the ears, prolonged cVEMP latency in 34% of the ears and reduced peak to peak amplitude in 36% of the ears. The authors conclude that possibility of the sacculocollic pathway dysfunction is very high in individuals with NIHL.

2.1.3. Congenital sensorineural hearing loss

Kimura (2017) did rotational chair test on 195 children who had profound sensorineural hearing loss and it was further categorized into presence or absence of inner ear malformations. The study showed reduced response in 15.9% in which 5.6% showed a poor response in the rotational chair test 10.3% showed no response.

Saurez (2006) aimed to check the balance sensory integration in children with profound hearing loss and cochlear implants. The children were divided into 2 groups: Group 'A' children had syndromic and non-syndromic hereditary hearing impairment with normal vestibular response. Group B had inner ear malformations, post-meningitis deafness, and one child had non-syndromic hereditary hearing impairment with hypoactive vestibular responses. They concluded that the hearing impaired children are associated with the vestibular hypo-function and hence detailed assessment is necessary.

Shinjo (2006) conducted a study to assess vestibular function in infants and young children with congenital hearing loss and acquired hearing loss. The assessment tool included was ice-water caloric test, rotational chair test and vestibular-evoked myogenic potential (VEMP) recording. The authors reported that only 15% showed normal responses in the caloric test, rotational chair test and VEMP recording bilaterally; 35% showed responses asymmetrically in the caloric test despite normal responses in the rotational chair test and VEMP recording bilaterally; 25% showed hyporeflexia or areflexia in the caloric test bilaterally, but showed normal responses in the rotational chair test and normal reproducible or decreased VEMPs; 25% showed no responses at all in the caloric test, rotational chair test and VEMP recording.

Pajor et al. (2002) examined 126 individuals with unilateral sensorineural hearing loss in which 50% of had vertigo complaints and 30% had dizziness complaints using electronystagmography to investigate the occurrence of dysfunction of vestibular system and to make comparison among the severity and type of the hearing loss. The authors reported abnormal ENG findings in 72% of the individuals.

Zhou et al. (2014) evaluated 12 children with congenital sensorineural hearing loss using cervical vestibular evoked myogenic potential. The test was carried out at different intensities and 90dBnHL being the highest. The analysis of the latency and amplitude was done for 90dBnHL to get the reliable responses. The authors found that the 67% of the children had abnormal responses.

Xu et al. (2016) assessed vestibular function of 43 children with profound sensorineural hearing loss and 20 normal hearing children using cervical and ocular vestibular myogenic potentials. The VEMPs was done for 500Hz tone burst (rise/fall time of 1ms and plateau of 2 ms) at the intensity of 131dB SPL and were increased and decreased depending upon the presence or absence of VEMP responses. The authors

reported that the response rate of cVEMP was 61.9% and oVEMP were 58.1% as compared to the normal hearing children who had 100% response rate.

Cushing et al. (2008) assessed vestibular and balance function in 40 children with severe to profound sensorineural hearing loss and unilateral cochlear implants. Caloric, rotational and vestibular evoked myogenic potentials tests were carried out to assess vestibular function and Bruininks-Oseretsky Motor Proficiency test-II were carried out to assess balance function. The authors found poorer scores in BOT-2 in children with SNHL and cochlear implants users as compared to the normal hearing, abnormal horizontal semicircular function of caloric responses in 50%, abnormal responses in rotational horizontal semicircular canal function in 38% and saccular function was absent bilaterally and unilaterally in 40% .

Kumar et al. (2014) evaluated the vestibular functions in 22 individuals with sensorineural hearing loss of various degrees of severity (mild, moderate and moderately severe). The result revealed that the mild hearing loss had reduced VEMP responses, absent responses in right ear and present responses in left ears (71%) in moderate degree of hearing loss, present VEMP response in 16.6% in moderately severe degree of hearing loss. They further compared across the three degree of hearing loss and no significant relationship was observed.

Cushing et al. (2013) examined vestibular function in 34 children with profound sensorineural hearing loss and 119 had unilateral cochlear implants using caloric, rotational and vestibular evoked myogenic potentials. The authors reported 50% had abnormal caloric responses in which 26% had mild to moderate and 37% had severe abnormal caloric response. Rotational test showed abnormal responses in 47% of these population and reduced VOR in 29% of them. VEMP results showed absent responses 21% and 13% bilaterally and unilaterally respectively. They also reported that the

children who had meningitis and radiologic cochlea-vestibular abnormalities showed horizontal canal dysfunction and the other showed saccular dysfunction. The authors concluded that profound sensorineural hearing loss has some amount of vestibular end organ dysfunction and are sometimes are dependent on the etiology.

Gayle, Roberta and Pohlman (1990) compared the dynamic, static and rotatory balance of the deaf children with normal hearing children. They included 20 children in each category and they concluded that overall balance in deaf children is inferior as when compared to the hearing children. Potter and Silverman (1984) tried to investigate the characteristics of vestibular and balance function in 34 deaf children age ranging from 5-9 yrs. They carried out Post-rotary nystagmus test and standing balance subtests in open and closed eyes conditions. They found significant difference in duration of postrotary nystagmus between hearing and deaf group.

Rashmi (2016) aimed to assess the degree of acquired hearing loss on ocular vestibular myogenic potentials. The oVEMP were implemented on 50 ears with mild to profound hearing loss sub-grouped into mild, moderate to moderately severe and profound degree and 50 ears with normal hearing using 500Hz tone burst at 125 pedBSPL. The author found that oVEMP responses were present in 38 ears out of 50 ears with the response rate of 76% in hearing loss individuals as compared to the normal hearing where response rate were 100%, the latencies were also longer in hearing loss individuals as compared to the normal hearing, the peak-to-peak amplitudes were reduced in hearing loss population as compared to the normal hearing. The authors also reported that the response rates were different compared between different degrees of hearing loss with 90% of response rate in moderate to moderately severe when compared to the 50% response rate of in severe to profound degree of hearing loss individuals. The author also reported that there was no difference in

response rate between mild degree and moderate to moderately severe degree of hearing loss. The authors reported that the latency was increase for the severe to profound degree of hearing loss; peak-to-peak amplitude was large for mild hearing loss and small for profound hearing loss.

2.2 Video Head Impulse Test and findings in various vestibular disorders

2.2.1. Vestibular Neuritis

Vestibular neuritis is a condition where the patient's experiences prolonged severe vertigo. Yoo et al. (2015) assessed 23 vestibular neuritis (VN) subjects during the acute stage and after 1 month follow-up. Gain and asymmetry were analyzed and the authors found that 87% (20/23) subjects had abnormal results in acute stage and 74% (17/23) had in follow up or compensated VN stage.

Blödow, Pannasch and Walther (2013) assessed function of horizontal semicircular canal in 52 vestibular neuritis subjects and compared with 20 healthy subjects. The authors found that 94.2% of VN subjects exhibited abnormal results along with refixation saccades. The isolated covert saccades and isolated overt saccades were observed 13.7% and 34.3% respectively and the combined covert and overt saccades were observed in 52%. The authors concluded that vHIT, using combination of gain and saccades is appropriate tool to diagnosed for diagnosing vestibular disorders.

Bartolomeo et al. (2013) assessed vestibular function in individuals with vestibular neuritis (VN) with video head impulse test in 2 stages, initial stage and follow-up stage (1-3 months). The authors recorded age, gender, caloric deficit and vHIT deficits in both the stages. The authors found that the 51.6% had abnormal results. The authors compared the results of vHIT with the caloric results and concluded that the vHIT lacks sensitivity with moderate vestibular loss.

Redondo-Martínez et al. (2016) carried out vHIT in vestibular neuritis (VN) subjects with vHIT at the initial stage and at two different later stages and tried to compare with the dizziness handicap inventory. The authors found that there was a reduced gain in the affected ears in acute stage of the disease and the gain improved at the later stage. They also reported that the speed of the covert saccades remains same but the speed of the overt saccades reduced in later stages.

2.2.2. Vestibular Migraine

Vestibular migraine is characterized by recurrent attacks of vertigo and history of migraine (Lempert et al., 2012). Blodow et al. (2014) assessed 23 individuals in vestibular migraine (VM) with vHIT. The vHIT was carried out using EyeSeeCam system and horizontal vestibule-ocular reflex was recorded. The subjects were asked to fixate a dot which was at 1.2m away, then the minimum of 10 head impulses were given with low amplitude, high peak velocity and with the duration of 150-200ms. The eye and head velocities were captured at 40, 60 and 80ms after the head impulse and averaging was done. The authors found abnormal results in 9% of VM subject and found reduced VOR gain.

Langhagen et al. (2015) tried to study the clinical findings in 118 children with vertigo associated with vestibular migraine using video head impulse test (vHIT) and performed retrospective chart analysis. They further grouped the patients in 5 different categories using International Classification of Headache Disorders, 3rd edition (beta-version) as: (1) definite vestibular migraine (dVM); (2) probable vestibular migraine (pVM); (3) suspected vestibular migraine (sVM); (4) benign paroxysmal vertigo (BPV); and (5) migraine with/without aura (oM) plus vertigo/dizziness. The authors found that 20% had abnormal hVOR function in which dVM had 8%, pVM had 20%, sVM had 29%, BPV had 50% and no pathologic vHIT in oM.

Kang et al in 2016 tried to find the clinical implication of video head impulse test in 81 VM subjects. The vHIT was performed using ICS Impulse Otometrics at the initial stage, follow-up after 6 months and VOR gain and gain asymmetry were calculated for the diagnosis of deficits in horizontal semicircular canal. The authors found that 11% (9/81) subjects exhibited abnormal results at the initial stage and at the follow up testing 44% (4/9) had abnormal responses and 55.6% (5/9) had catch up saccades.

Albernaz (2014) assessed 200 patients with different cochlear and vestibular disorders and carried out detailed case history, audiological evaluation and vestibular evaluations. The audiological evaluations included the pure tone audiometry, speech discriminations tests, immittance tests. The vestibular tests included spontaneous and semi- spontaneous nystagmus examination, positional nystagmus, pendulum eye tracking, optokinetic nystagmus, pendular rotatory test and video head impulse test (vHIT). For some patients, other tests were also carried such as, auditory brainstem response, caloric test and cervical and ocular vestibular evoked myogenic potentials. The authors reported that in 200 patients there were 15 patients with vestibular migraine in which 12 patients had normal vHIT vestibular ocular reflex (VOR) gain, 1 had hypoactive left lateral canal, 1 had bilateral low VOR gain of the anterior canals and 1 had reduced VOR gain in all the semicircular canals.

2.1.4. Benign positional paroxysmal vertigo (BPPV)

Chen et al. (2012) assessed dysfunction of the semicircular canals in BPPV and the relationship with etopic otoconia using video head impulse test. The study included 214 patients with BPPV in which 107 had posterior semicircular canal canalithiasis, 80 had horizontal semicircular canal canalithiasis, and 27 had horizontal semicircular canal cupulolithiasis with 190 subjects accompanying disease and 24 subjects did not.

The authors found that in 7% of the individuals only the vHIT test results were affected. The authors concluded that the lesion of semicircular canals share similar causal factors with the utricle pathological changes in BPPV and mostly the low frequency range is affected, therefore, vHIT cannot be used for screening the function of semicircular function in BPPV.

Guan et al. (2017) examined 43 patients with BPPV to quantitatively measure VOR gain of 3 pairs of semicircular canal. They found that the gain was reduced in BPPV individuals as compared to the normal and showed higher vertical VOR gain asymmetries compared with the healthy control group.

Perez-Fernandez, Martinez-Lopez & Manrique-Huarte (2014) assessed 12 patients who were diagnosed with BPPV (superior canal). Video head impulse test was performed using GN Otometrics and asked the subjects to keep looking at the target faced at the 1m front of them and turning the head randomly at small angle (10-20 degrees) and 20 impulses were provided for test parameters. The authors found that only one subject had reduced VOR gain for contralesional head impulses and no change in normal limits of VOR gain in any of the canals in other subjects but there was different gain asymmetry for each canal. The authors finally concluded that the VOR gain is normal in individuals with idiopathic BPPV with otoconial debris located in superior semicircular canal.

Fallahnezhad et al. (2017) did a pilot study in 29 individuals with unilateral posterior semicircular canal BPPV in which they aimed to evaluate the VOR gain, gain asymmetry and saccades. They found that the 16/29 (55.17%) had abnormal posterior canal VOR gain in ipsilesional ear, normal horizontal canal VOR gain and reduced VOR gains in the superior canal and saccades were not observed.

2.1.5. Meniere's disease

Blodow et al. (2014) assessed function of semicircular canals in 30 individuals with Meniere's disease in which 25 had definite Meniere's disease and 5 had probable Meniere's disease with affected left ear in 15 patients, right ear affected in 10 subjects and bilateral 5 subjects and 17 subjects were in early stage (5 years) and 13 in their advanced stage (.5 years). The authors reported that the vHIT revealed abnormal gain in 37% of the population with lower mean VOR gain in the affected side than the non-affected side. The authors could not find any correlation between early and advanced stage of MD. Similar findings were found by Park et al in 2015, 55% abnormal gain.

McCaslin, Rivas, Jacobson and Bennett (2014) examined VOR gains in 3 patients diagnosed with definite MD who had moderate degree of flat sensorineural hearing loss. They observed normal vHIT gains and they concluded that there is a differential preservation of high frequency function (evaluated through vHIT).

Rubin et al. (2017) aimed to find the discrepancies in the literature due to different test findings of vHIT in MD. They included 37 patients who had hearing loss of 59dBHL +/- 18 dBHL in which 12 patients had Tumarkin's otolithic crises. They observed normal vHIT results.

2.2. Video head impulse test in sensorineural hearing loss

Sinha and Bansal (2017) aimed to objectively examine the functioning of the 6 semicircular canals in 20 individuals with severe to profound sensorineural hearing loss. They found that the mean VOR gain for left anterior canal was reduced for 5 individuals and increased for 2 individuals; mean VOR gain for right anterior canal was reduced for 6 individuals; left lateral canal was reduced for 6 individuals, right lateral

canal was reduced for 7 individuals, left posterior canal was reduced for 3 individuals and right posterior canal was reduced for 5 individuals and increased for 3 individuals.

Magliulo et al. (2017) examined the VOR gain using video head impulse test in 5 confirmed diagnosis of Usher syndrome type II patients. The author found that 2 subjects showed significant deficit in the superior semicircular canal and horizontal semicircular canal and 4 patients showed significant deficit in the posterior semicircular canal. Bansal and Sinha (2016) examined 20 adults for vestibular function with video head impulse test. The vHIT was performed using ICS Otometrics. The authors found that the mean VOR gain was reduced in the hearing impaired population as compared to the normal hearing individuals. The authors concluded that the vestibular abnormality was found in this population and hence implementation of vestibular test is necessary in daily clinical setting.

To summarize, the review of literature, as the all the test cervical and ocular vestibular evoked myogenic potential (cVEMP and oVEMP) and video head impulse test (VHIT) are affected in the subjects with hearing loss and also assesses different organs of the vestibular system. Therefore, the needs of implementing test are necessary during evaluating the hearing impaired individuals for better rehabilitation process.

Chapter III

METHOD

The present study aimed to assess the functioning of otolithic organ and three semicircular canals in individuals with various degree of sensorineural hearing loss.

PARTICIPANTS:

The participants of this study were divided into two groups Group I and Group II.

a. Group I:

The study included 40 participants (80 ears) within age range of 18-44 years (31 males and 9 females). There were 12 ears with mild, 15 ears with moderate, 15 ears with moderately severe, 21 ears with severe and 17 ears with profound sensorineural hearing loss.

Selection criteria for participants of Group I:

1. All the participants in group I had sensorineural hearing loss ranging from mild degree to severe degree in severity.
2. Bilateral symmetrical/ asymmetrical hearing loss.
3. Participants in group I had negative history of middle ear problems (ear pain, ear discharge) and conductive hearing loss.
4. Participants in group I had no evidence of any retro cochlear pathology.
5. Participants in group I had no definite history of any vestibular disease (eg: Labyrinthitis, vestibular neuritis, Meniere's disease).
6. Participants in group I no complaint of hypertension and diabetes and other neurological problems.
7. Participants in group I had no complaint of neck pain.

Group II:

There were 40 participants (80 ears) within the age range of 18- within age range of 18-35 years (males 19 and females 21) were considered for this study. All the participants in had normal hearing sensitivity.

Selection criteria for participants of Group II:

1. All the participants had normal hearing sensitivity from frequencies 250-8000Hz.
2. Participants had absence of conductive hearing loss and no history of ear pain, ear discharge.
3. Participants had negative history of vestibular symptoms.
4. Participants had no complaint of hypertension and diabetes and other neurological problems.
5. Participants had no compliant of neck pain.

INSTRUMENTATION

1. Calibrated 2-channel Piano Inventis diagnostic audiometer (*Orbiter-922 V-2x, G N Otometrics, Taastrum, Denmark*) with TDH-39 (*Telephonics, 815 Broad Hollow Road, Farmingdale, New York 11735*) and bone vibrator B-71 (*Radioear, KIMMETRICS, 22050 Mohawk Drive, Smithsburg, MD 21783*) was used for obtaining air conduction and bone conduction hearing thresholds and speech thresholds.
2. Calibrated GSI-Tympstar (*GSI VIASYS Healthcare, Wisconsin, USA*) with probe frequency 226Hz was used for tympanometry and reflexometry.
3. IHS (Intelligent Hearing System) Smart EP (3.94 USBez) system (*Intelligent Hearing System, Florida, USA*) with ER-3A Insert ear phones (*Etymotic*

Research, Inc., USA) was used for cervical VEMP and auditory brainstem response testing.

4. Biologic Navigator Pro system (*Natus Medical Incorporated, CA, USA*) with ER-3A inserts ear phones was used for ocular VEMP testing.
5. Otometrics VHIT (*GN Otometrics, North America*) system was used for vHIT testing.

TESTING ENVIROMENT

All the tests were conducted in acoustically treated room with permissible noise level as per ANSI S 3.1 (1991) standards.

PROCEDURE

1. Case history

Detailed case history was taken for each client followed by administration of *dizziness* questionnaire (Maryland Hearing and Balance Centre).

2. Pure-tone audiometry

Hearing thresholds was obtained using modified version of Hughson and Westlake procedure (Carhart, 1959) at octave frequencies between 250 Hz to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction.

3. Immittance audiometry

Tympanograms was obtained with probe frequency 226Hz for both ears followed by acoustic reflex thresholds estimation both ipsilateral and contralateral for 500, 1000, 2000 and 4000Hz.

4. Uncomfortable Loudness level

UCL for speech was done using ascending method for all the subjects.

5. Auditory Brainstem Response

Auditory Brainstem Response was carried out to rule out retro cochlear pathology in subjects with sensorineural hearing loss. Electrode placement site was prepared using a skin preparation gel. Surface disc (AgCl) electrodes were used for recording. Absolute electrode impedances below 5 k Ω and inter electrode impedances below 2 k Ω were maintained. Repeated recordings were done to ensure the reliable responses. The conventional electrode placement was used (non-inverting-upper forehead, inverting-both mastoid and ground-lower forehead). The stimulus used was clicks with rarefaction polarity at the intensity of 90dBnHL and at the rate of 11.1/s and 90.1/s. The filter setting was at 100-3000Hz and the averages taken were 1500 with amplification of 100,000. The analysis time was 10 msrc.

6. Vestibular evoked myogenic potentials

Electrode placement site was prepared using a skin preparation gel. Surface disc (AgCl) electrodes were used for recording. Absolute electrode impedances below 5 k Ω and inter electrode impedances below 2 k Ω were maintained. Repeated recordings were done to ensure the reliable responses.

a. Cervical vestibular evoked myogenic potentials:

Tone bursts of 500Hz (2-0-2) with 95dBnHL were used as stimulus. Rarefaction polarity was used with the repetition rate of 5.1/s. The averages taken were 200 and the analysis time was from -10 to 60ms. The electrode placement used was midpoint of the SCM for non-inverting, the sternal notch for inverting and forehead for ground with the filter setting of 30-1500Hz with amplification of 5000. The recording mode was ipsilateral.

b. Ocular vestibular myogenic evoked potential:

Tone bursts of 500Hz (2-0-2) with 95dBnHL were used as stimulus. Rarefaction polarity was used with the repetition rate of 5.1/s. The averages taken were 200 and the analysis time was from -10 to 60ms. The electrode placement used was Inferior to the lower eyelids for inverting, immediately inferior to the inverting electrode for non-inverting, and high forehead for ground with the filter setting of 1-1000Hz with amplification of 30,000. The recording mode was contralateral.

7. Video Head Impulse Test (vHIT)

The test was carried out in a well lit room. Subjects were seated in a height, adjustable chair and the goggles were placed and secured on the patient. The goggles were tightened to minimized slippage. The examiner instructed the patient to maintain eye focus on a stationary object which was placed at the distance of 1m in front and the clinician delivered a quick, precise head movements in random time and direction. The entire testing was done in three planes-lateral plane, left anterior-right posterior (LARP) and right anterior-left posterior (RALP). To test the anterior and posterior semicircular canals, the head rotations were delivered in the planes of the vertical canals – left anterior-right posterior (LARP) and right anterior-left posterior (RALP). The person's head was positioned about 10-15° turned to the left or right with respect to their body so that the targeted vertical canal plane was approximately aligned with the body sagittal plane, then the head movements were delivered. Total 20 head impulses were given for each of the canal.

ANALYSIS OF THE DATA

- 1) Cervical VEMP:
 - a. Latency of P13 Peak for both the groups (I & II)
 - b. Latency of N23 Peak for both the groups (I & II)
 - c. Amplitude complex of P13-N23 for both the groups (I & II)
1. Ocular VEMP:
 - a. Latency of N1 Peak for both the groups (I & II)
 - b. Latency of P1 Peak for both the groups (I & II)
 - c. Latency of N2 peak for both the groups (I & II)
 - d. Amplitude complex of N1-P1 & P1-N2 for both the groups (I & II)
3. Video Head Impulse test
 - a. Corrective saccades (overt and covert) were analyzed for individuals with sensorineural hearing loss.
 - d. Gain of the VOR for both the groups was calculated using the following formula

$$\text{VOR Gain} = \text{eye velocity} / \text{head}$$

Chapter IV

RESULTS

The aim of this study was to assess the functioning of otolithic organ (sacculle and utricle) and three semicircular canals in individuals with various degree of sensorineural hearing loss and also to find the correlation between the test findings of cervical vestibular evoked myogenic potentials (cVEMP), ocular evoked myogenic potentials (oVEMP) and video head impulse test (vHIT) with degree and duration of hearing loss. 40 individuals with mild to profound degree of sensorineural hearing loss and 40 normal hearing were employed in this study. Shapiro-Wilk test of normality was performed and it indicated that the data was not distributed normally, thus non-parametric test were carried out for statistical analysis. To analyze the data, Statistical Package for the Social Sciences (SPSS) version 20 software was used. The results of the data are presented below:

4.1. Cervical vestibular myogenic potentials findings:

Latency of p1, n1 and peak-to-peak amplitude (p1n1) of cVEMP was analyzed in both the groups. In the normal hearing group, cVEMP was present in all the participants. In the hearing impaired group, out of 12 mild ears cVEMP was present in 3 ears, 4 out of 15 ears with moderate sensorineural hearing loss, 1 out of 15 ears with moderately severe sensorineural hearing loss, 1 out of 21 ears with severe sensorineural hearing loss and 2 out of 17 ears with profound hearing loss. Thus, in the hearing loss individuals out of 80 ears cVEMP was present in 11 ears (13.75%).

cVEMP recorded from one normal hearing individuals and individuals with hearing loss with normal and absent cVEMP are represented in figure below.

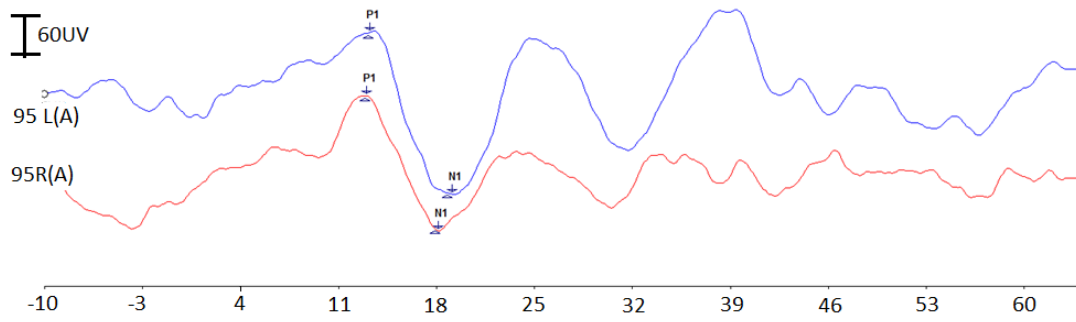


Figure.4.1. cVEMP waveforms in one of the normal hearing participants in both the ears.

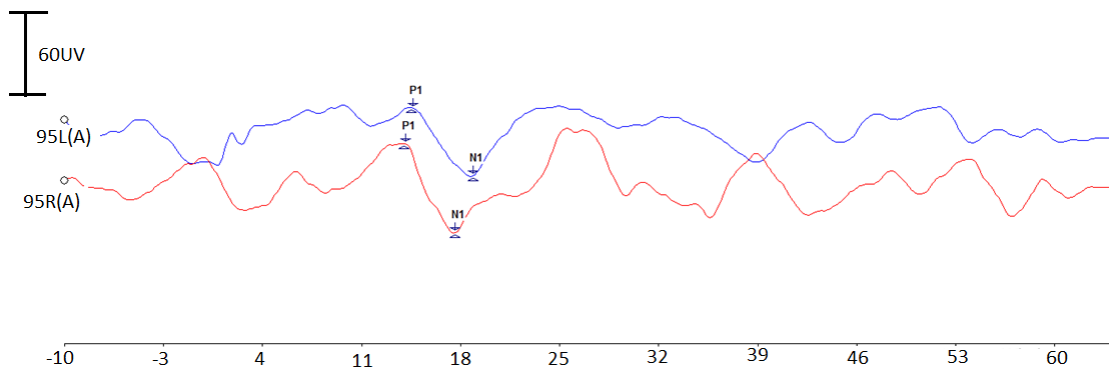


Figure.4.2. cVEMP waveforms in one of the hearing loss participants with presence of cVEMP in both the ears.

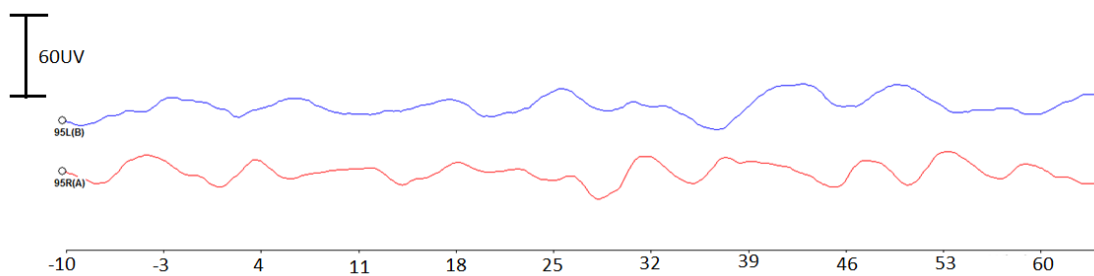


Figure.4.3. cVEMP waveforms in one of the hearing loss participants with absence of cVEMP in both the ears.

Descriptive statistics was done to calculate mean and the standard deviation for the latency and the amplitude of cVEMP for both the ears in normal and hearing loss groups. The mean values and the standard deviation for p1 latency, n1 latency and peak-to-peak

amplitude of p1n1 for individuals with normal hearing sensitivity is shown in Table 4.1 and for individuals with different degree of hearing loss is shown in Table 4.2

Table: 4.1. Mean and standard deviation (SD) for cVEMP in left and right ear in normal hearing.

	Right ear			Left ear		
	n	Mean	SD	N	Mean	SD
p1 Latency (in m seconds)	40	13.89	1.45	40	13.93	1.71
n1 Latency (in m seconds)	40	20.39	1.99	40	19.82	3.70
p1-n1 amplitude (in μV)	40	17.11	5.29	40	16.77	4.36

Table: 4.2. cVEMP responses in individuals with different degree of hearing loss.

		Right ear		Left ear		
		N	Mean	SD	Mean	SD
<i>Mild sensorineural hearing loss</i>	p1 latency	1	17.50	2	17.56	0.96
	n1 latency	1	20.13	2	19.75	6.71
	p1n1 amplitude	1	18.25	2	11.29	13.51
<i>Moderate sensorineural hearing loss</i>	p1 latency	2	16.31	2	17.58	
	n1 latency	2	24.94	2	24.00	

	p1n1	2	7.1	2	6.85	
	amplitude					
<i>Moderately severe</i>	p1 latency	1	13.38	0		
<i>sensorineural hearing loss</i>	n1 latency	1	17.00			
	p1n1	1	4.73			
	amplitude					
<i>Severe sensorineural</i>	p1 latency			1	15.63	
<i>hearing loss</i>	n1 latency			1	19.13	
	p1n1			1	4.67	
	amplitude					
<i>Profound sensorineural</i>	p1 latency			2	17.69	0.97
<i>hearing loss</i>	n1 latency			2	22.75	2.34
	p1n1			2	5.06	5.17
	amplitude					

It can be seen from Table 4.1 and Table 4.2 that, for normal hearing individuals the cVEMP responses were present for all the participants whereas, it was absent for most of the individuals in various degree of hearing loss. It can also be seen that as the severity of hearing loss increased, the number of participants in whom the response was present reduced. As the cVEMP was absent for most of the participants in hearing loss group, the data from all the individuals from hearing impaired group were combined to get mean and standard deviations for cVEMP latency and amplitude. Table 4.3 shows the mean and the standard deviation for the combined data of cVEMP latency and amplitude from the hearing impaired group.

Table.4.3: Mean and standard deviation (SD) for cVEMP in left and right ear in hearing loss.

	Right Ear		Left Ear	
	Mean	SD	Mean	SD
p1 Latency	15.87	2.23	15.29	5.69
n1 Latency	22.06	3.77	20.63	3.99
P1-n1 amplitude(μv)	15.09	8.60	7.29	6.65

The data was tested for normality distribution using Shapiro-Wilk test and it showed that the data were not distributed normally. Therefore non-parametric statistics were performed. Mann-Whitney U test revealed no significant differences between normal hearing and hearing loss group for p1 latency of right ear [$Z = 1.82$; $p = 0.69$], n1 latency for right ear [$Z=1.15$; $p: 0.25$], n1 latency in left ear [$Z=0.48$; $p=0.62$] and p1n1 amplitude complex for the right ear [$Z =3.35$; $p= 0.76$], however, Mann Whitney U test revealed a significant difference between normal hearing and hearing loss groups for p1 latency for left ear [$Z=2.70$; $p=0.00$] and p1n1 amplitude complex for left ear [$Z=3.13$; $p= 0.00$] was observed.

4.1.1. Correlation of cVEMP latency and amplitude with degree and duration:

Latency and amplitude of the cVEMP of the hearing impaired group was correlated with the degree and duration of hearing loss in hearing impaired individuals. Spearman's rank correlation test revealed no correlation between p1 latency for right ear and degree of hearing loss [$r_s= -0.63$; $p=0.36$], n1 latency for the right ear and degree of

hearing loss [$r_s = -0.31$; $p = 0.68$], and no correlation between p1n1 amplitude complex for right ear with degree of hearing loss [$r_s = -0.63$; $p = 0.36$]. Spearman's rank correlation test revealed no correlation between p1 latency for left ear with degree of hearing loss [$r_s = 0.73$; $p = 0.87$], n1 latency in left ear [$r_s = 0.54$; $p = 0.25$], p1n1 amplitude complex for left ear with degree of hearing loss [$r_s = 0.63$; $p = 0.12$].

Latency and amplitude of the cVEMP were also correlated with duration of hearing loss. Spearman's rank correlation test revealed no correlation between p1 latency for the right ear with duration of hearing loss [$r_s = -0.40$; $p = 0.60$], n1 latency for right ear with duration of hearing loss [$r_s = -0.40$; $p = 0.60$] and p1n1 amplitude complex for right ear with duration of hearing loss [$r_s = -0.80$; $p = 0.20$]. Spearman's rank correlation test revealed no correlation between p1 latency for left ear with duration of hearing loss [$r_s = 0.01$; $p = 0.96$], n1 latency for the left ear with duration of hearing loss [$r_s = 0.25$; $p = 0.58$], p1n1 amplitude complex for left ear with duration of hearing loss [$r_s = -0.42$; $p = 0.33$].

4.2. Ocular evoked myogenic potentials (oVEMP) findings:

Latency of n1, p1, n2 and peak-to-peak amplitude of n1p1 and p1n2 were analyzed. In the normal hearing group, oVEMP was present in 100% (80 ears) and in the hearing loss individuals out of 80 ears oVEMP was present in 13 ears (16.25%).

oVEMP recorded from one normal hearing individuals and individuals with various degree of hearing loss are represented in figure.

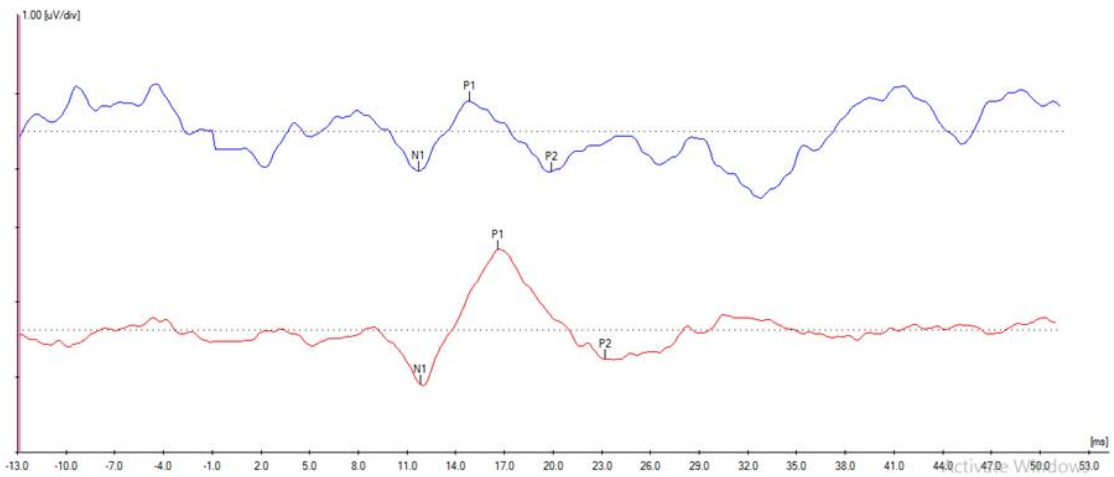


Figure.4.4. oVEMP waveforms in normal hearing individual's right and left ear.

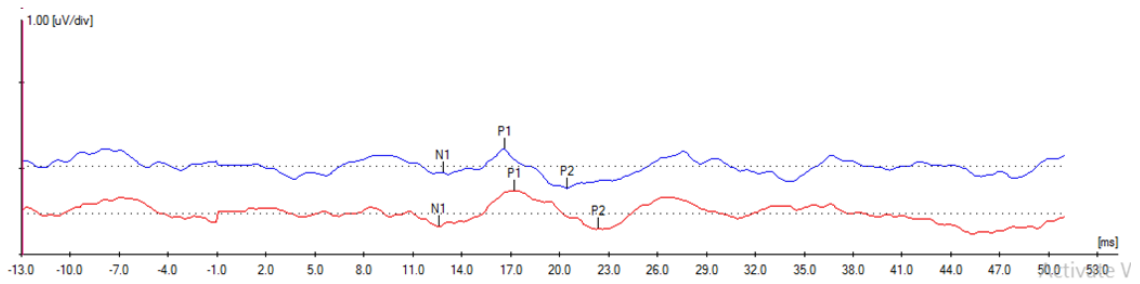


Figure.4.5. oVEMP waveforms of one of the hearing loss individuals with normal oVEMPs in both right and left ear.

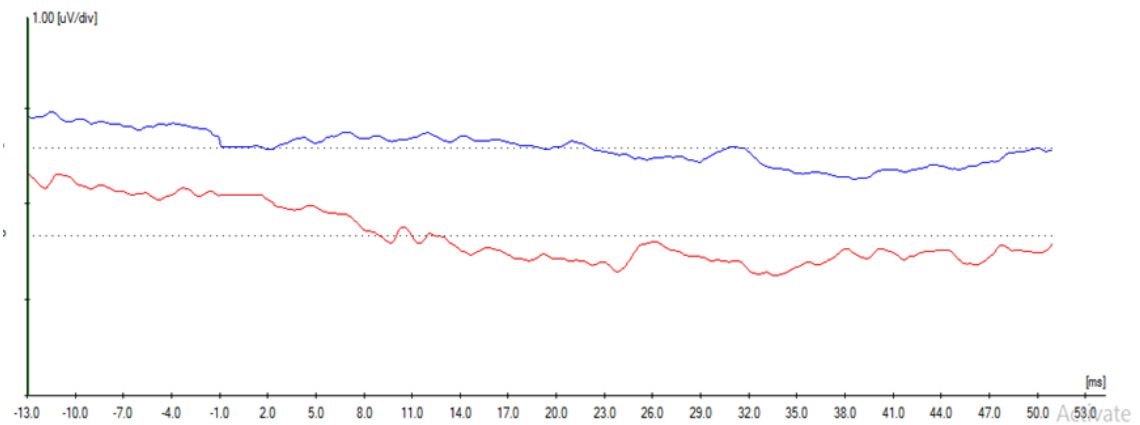


Figure.4.6. Absent oVEMP waveforms in one of the individuals with hearing loss in both the ears.

Descriptive statistics was done to calculate mean and the standard deviation for the latency and the amplitude of oVEMP for both the ears in normal and hearing loss groups.

The mean values and the standard deviation for n1 latency, p1 latency, n2 latency and peak-to-peak amplitude of p1n1 and p1n2 for both the groups is shown in the table 4.4 and for the hearing impaired individuals in Table 4.5

Table.4.4. Mean and standard deviation (SD) for oVEMP in left and right ear in normal hearing individuals.

	Right ear		Left ear	
	Mean	SD	Mean	SD
n1 Latency (msec)	9.59	1.66	9.72	4.03
p1 Latency (msec)	14.04	1.98	13.94	1.97
n2 Latency (msec)	18.90	2.75	19.27	3.43
n1p1 amplitude (μ V)	4.33	2.33	4.00	1.50
p1-n2 amplitude (μ V)	5.92	1.88	4.37	2.74

Table 4.5. Mean oVEMP latency and amplitude of the individual degree of hearing loss in both the ears.

		Right ear			Left ear		
		N	Mean	SD	N	Mean	SD
<i>Mild sensorineural</i>	n1 latency	1	11.85	-	0	-	-
<i>hearing loss</i>	p1 latency	1	15.72	-	0	-	-
	n2 latency	1	19.10	-	0	-	-
	p1n1 amplitude	1	0.40	-	0	-	-
	p1n2 amplitude	1	0.00	-	0	-	-
<i>Moderate</i>	n1 latency	1	10.10	-	2	11.34	1.59
<i>sensorineural</i>	p1 latency	1	13.47	-	2	16.28	1.85
<i>hearing loss</i>	n2 latency	1	18.22	-	2	20.09	3.00
	p1n1 amplitude	1	4.16	-	2	1.00	0.64
	p1n2 amplitude	1	1.67	-	2	1.00	0.51
<i>Moderately</i>	n1 latency	2	11.41	1.14	2	10.35	0.35
<i>severe</i>	p1 latency	2	16.78	1.51	2	16.53	1.50
<i>sensorineural</i>	n2 latency	2	23.66	0.97	2	22.03	2.56
<i>hearing loss</i>	p1n1 amplitude	2	0.56	0.34	2	0.81	0.52
	p1n2 amplitude	2	0.09	0.12	2	0.04	0.05
<i>Severe</i>	n1 latency	3	10.55	1.12	2	10.78	1.50
<i>sensorineural</i>	p1 latency	3	14.51	2.03	2	17.16	2.39
<i>hearing loss</i>	n2 latency	3	19.51	3.21	2	20.35	2.82
	p1n1 amplitude	3	0.78	0.87	2	0.87	0.79
	p1n2 amplitude	3	0.09	0.21	2	0.07	0.03

<i>Profound</i>	n1 latency	1	10.97	-	0	-	-
<i>sensorineural</i>	p1 latency	1	16.10	-	0	-	-
<i>hearing loss</i>	n2 latency	1	21.72	-	0	-	--
	p1n1 amplitude	1	2.84	-	0	-	-
	p1n2 amplitude	1	0.27	-	0	-	--

It can be seen from Table 4.4 and Table 4.5 that the oVEMP was present for all the individuals with normal hearing sensitivity whereas it was absent for most of the individuals with sensorineural hearing loss. As the date of the different hearing loss was less, data from all the groups were combined and was made as a single group of hearing loss and latency and amplitude of the oVEMP parameters were compared with individuals with normal hearing sensitivity. The mean and the standard deviation for the combined data is given in Table 4.6.

Table.4.6. Mean and standard deviation (SD) for cVEMP in left and right ear in hearing loss.

	Right ear		Left ear	
	Mean	SD	Mean	SD
n1 Latency (in m seconds)	10.92	0.94	10.82	1.08
p1 Latency (in m seconds)	15.30	1.64	16.66	1.56
n2 Latency (in m seconds)	20.61	2.75	20.82	2.36
n1p1 amplitude	1.35	1.45	0.89	0.52

(in μV)				
p1-n2 amplitude	0.14	0.65	0.34	0.56
(in μV)				

The data was tested for normality distribution using Shapiro-Wilk test and it showed that the data were not distributed normally. Therefore non-parametric statistics were performed. To understand the significant differences between normal hearing and hearing loss groups in mean latency and amplitude Mann-Whitney U test was done. Mann-Whitney U test revealed no significant difference for p1 latency for right ear [Z=1.92; p= 0.05], n2 latency for the right ear [Z=1.62; p= 0.10], n1 latency for left ear [Z=1.67; p= 0.95], p1 latency for left ear [Z=2.28; p=0.22], n2 latency of left ear [Z=1.13;p= 0.25] and p1n1 amplitude complex for the right ear [Z=3.35; p=0.762]. However significant differences between normal hearing and hearing loss group was noted for n1 latency of right ear [Z=2.60; p=0.00], n1p1 amplitude complex of the right ear [Z=3.38; p: 0.00], p1n2 amplitude complex for the right ear (Z=4.07; p=0.00), n1p1 amplitude complex for the left ear [Z=3.54; p=0.00] and p1n1 amplitude complex for the left ear [Z=3.10; p= 0.002].

4.2.1. Correlation between oVEMP latency and amplitude with degree and duration of hearing loss.

Latency and amplitude of the oVEMP were correlated with the degree and duration of hearing loss. Spearman's Rank-Order Correlation revealed no correlation between n1 latency for the right ear with degree of hearing loss [$r_s=0.34$; p=0.40], p1 latency of the right ear with degree of hearing loss [$r_s=0.05$; p=0.90], n2 latency of the right ear and degree of hearing loss [$r_s=0.14$; p=0.74], p1n1 amplitude complex for right

ear with degree of hearing loss [$r_s=0.17$; $p=0.67$] and p1n2 amplitude complex for right ear with degree of hearing loss [$r_s=0.39$; $p=0.33$]. Spearman's Rank-Order Correlation revealed no correlation between n1 latency for the left ear and degree of hearing loss [$r_s=0.40$; $p=0.43$], p1 latency for left ear with degree of hearing loss [$r_s=0.20$; $p=0.69$], n2 latency in left ear with degree of hearing loss [$r_s=0.06$; $p=0.90$], p1n1 amplitude complex for left ear with degree of hearing loss [$r_s=0.15$; $p=0.77$] and p1n2 amplitude complex for left ear with degree of hearing loss [$r_s=0.58$; $p=0.22$].

Spearman's Rank-Order Correlation revealed no correlation between n1 latency for the right ear with duration of hearing loss [$r_s=0.50$; $p=0.20$], p1 latency for right ear with duration of hearing loss [$r_s=0.16$; $p=0.69$], n2 latency in right ear [$r_s=0.25$; $p=0.54$], p1n1 amplitude complex for right ear with duration of hearing loss [$r_s=0.59$; $p=0.11$] and p1n2 amplitude complex for right ear with duration of hearing loss [$r_s=0.30$; $p=0.45$]. Spearman's rank order correlation revealed between n1 latency for the left ear with duration of hearing loss [$r_s=0.52$; $p=0.28$], p1 latency for left ear with duration of hearing loss [$r_s=0.19$; $p=0.71$], n2 latency in left ear with duration of hearing loss [$r_s=0.08$; $p=0.87$], p1n1 amplitude complex for the left ear with the duration of hearing loss [$r_s=0.00$; $p=1.00$], p1n2 amplitude complex for left ear with duration of hearing loss [$r_s=0.75$; $p=0.08$].

4.3 Video head impulse test (vHIT) test findings

Mean vestibulo-ocular reflex (VOR) gain and correctives saccades were measured for both the groups. In the normal hearing groups VOR gain was normal in all six semicircular canals. However out of 72 ears analyzed, reduced VOR gain was found in 28 ears (38.88%) and correctives saccades were observed in 9 ears (12.5%) with hearing

impaired individuals. Mean VOR gain of one normal hearing individual is shown in figure.

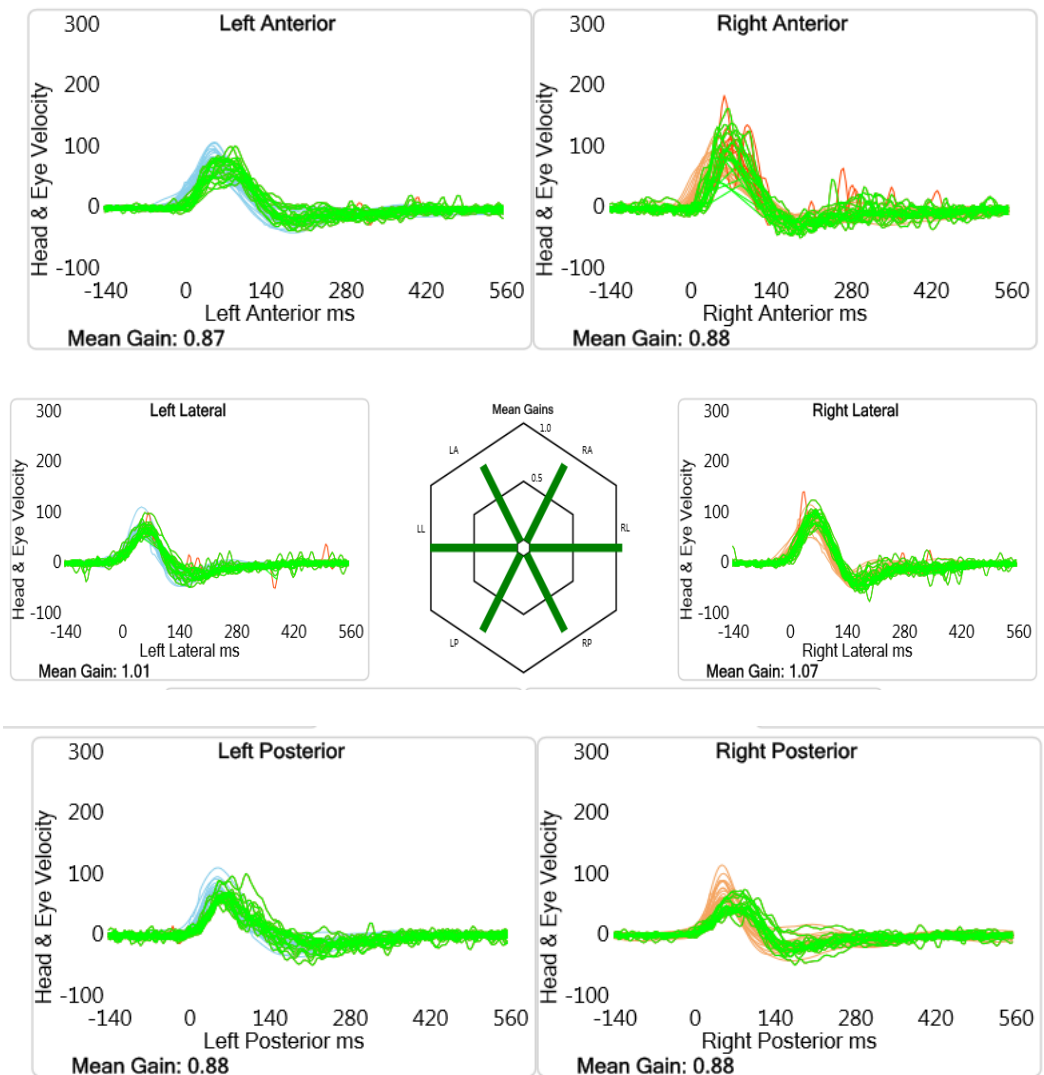


Figure.4.7. Video head impulse test results in 3 different planes of one participant with normal VOR in individual with normal hearing. The head and eye velocities throughout different head impulses are shown and the hexaplot of the gain values are shown.

Individual data was analyzed for the hearing loss individuals and found that the mean VOR gain of right and left lateral canal was reduced for 2 individuals and 5 individuals respectively. Mean VOR gain for right and left anterior canals were reduced for 2 and 7 individuals respectively. Mean VOR gain of right and left posterior canals were reduced for 13 individuals and 2 individuals respectively. Mean VOR gain of individuals with hearing loss with normal VOR gain and reduced VOR gain are shown in figure below 4.8 and 4.9 respectively.

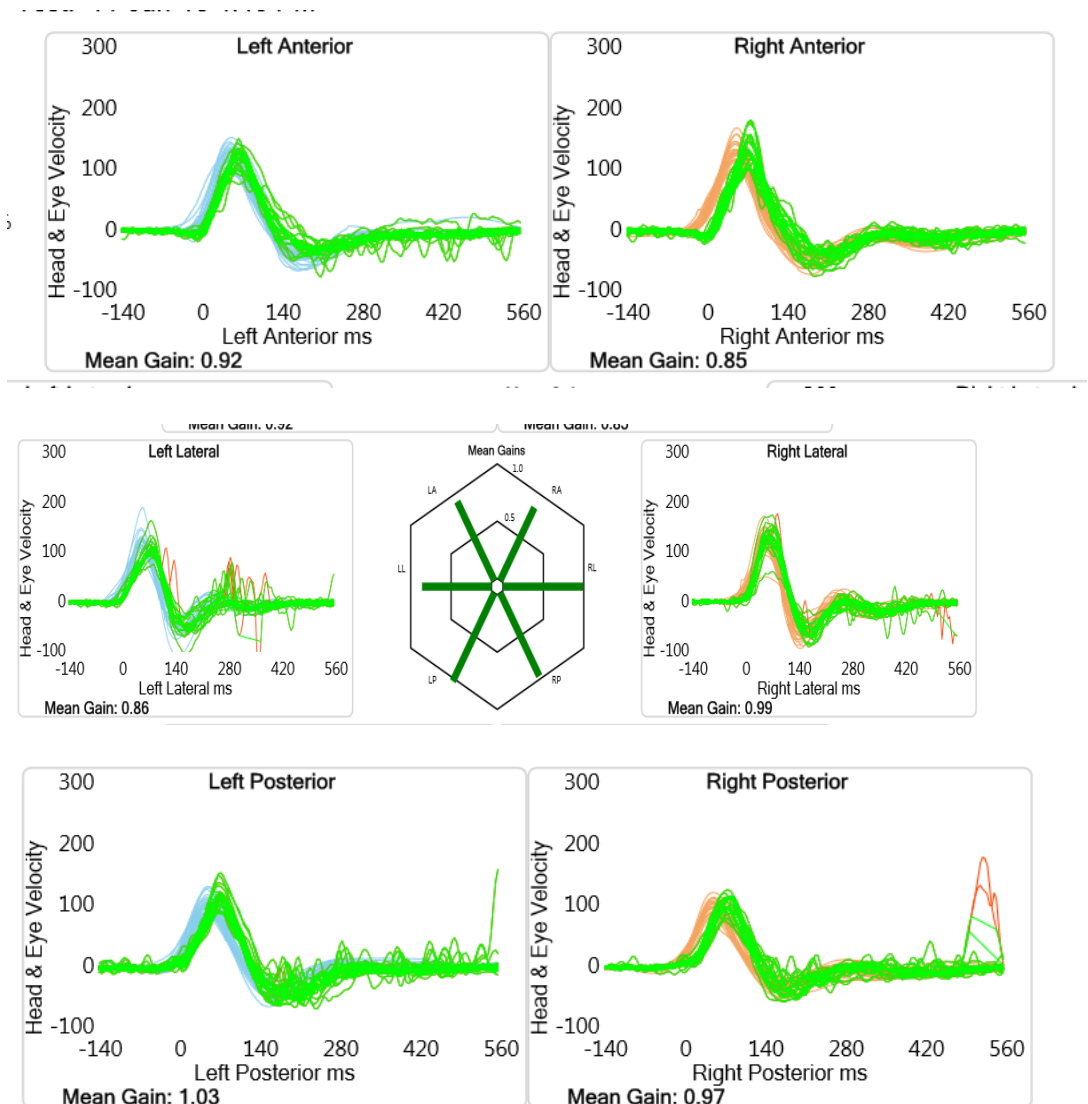


Figure.4.8. Video head impulse test results in 3 different planes of one participant with normal VOR in individual with hearing loss. The head and eye velocities throughout different head impulses are shown and the hexaplot of the gain values are shown.

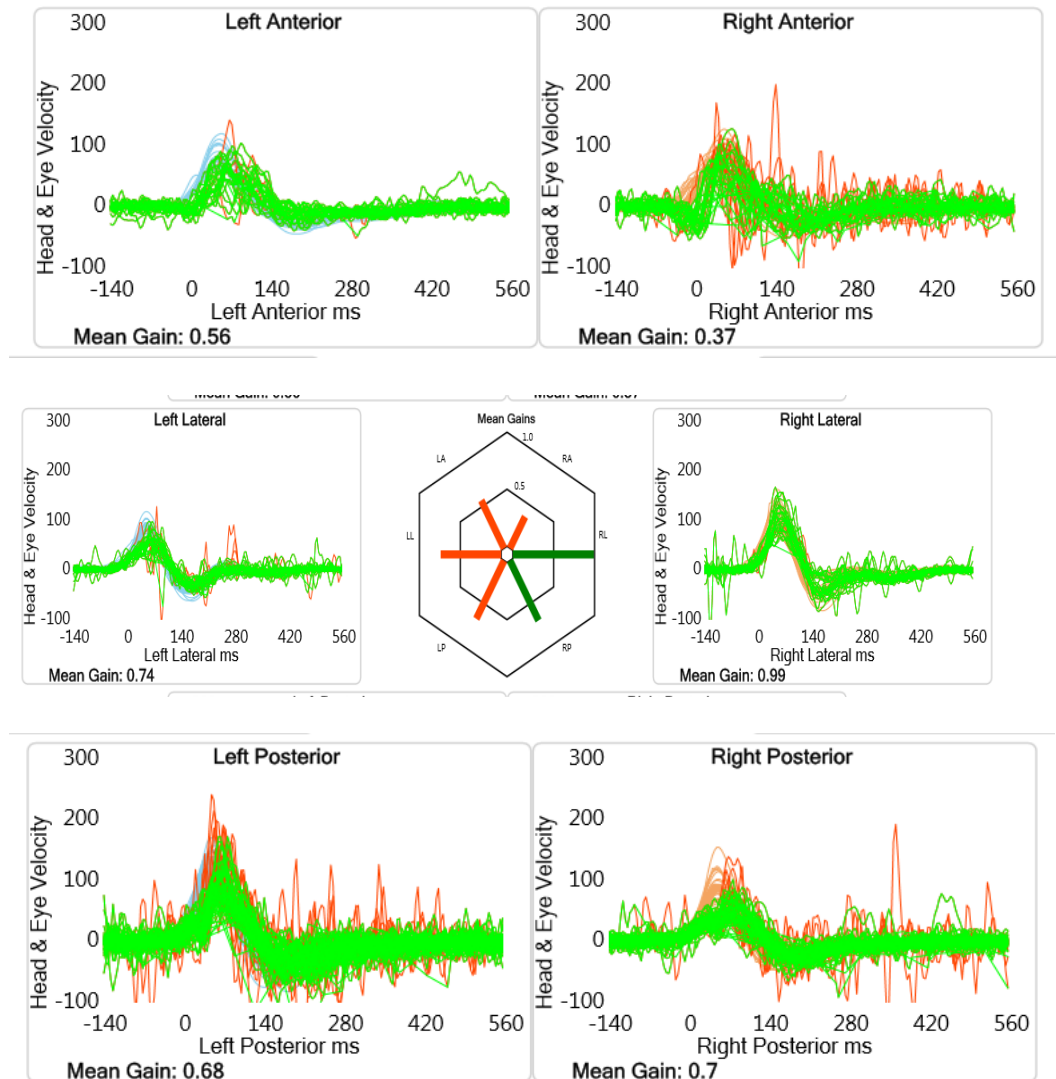


Figure.4.9. Video head impulse test results in 3 different planes of one participant with reduced VOR in individual with hearing loss. The head and eye velocities throughout different head impulses are shown and the hexaplot of the gain values are shown.

Descriptive analysis was done to calculate mean and standard deviation of VOR gain in all 3 planes in both the directions. The right horizontal (RH), left horizontal (LH),

right anterior (RA) and left anterior (LA), right posterior (RP) and left posterior (LP). The mean values for the all six semicircular canals for both the groups are shown in Table 4.6/ Table 4.7 shows the presence of corrective saccades in individuals with hearing loss.

Table.4.7. Mean and standard deviation (SD) for VOR gain in left and right ear in normal hearing and hearing loss individuals.

Planes	N	Normal hearing		N	Hearing loss	
		Mean	SD		Mean	SD
Right Horizontal	40	0.99	0.19	40	0.91	0.14
Left Horizontal	40	0.90	0.15	40	1.00	0.16
Right Anterior	40	0.97	0.17	40	0.80	0.37
Left Anterior	40	0.79	0.36	40	0.99	0.16
Right Posterior	40	0.75	0.19	40	0.89	0.13
Left Posterior	40	0.89	0.13	40	0.75	0.19

Table.4.8. Presence of refixation saccades in left and right ear hearing loss individuals.

Planes	Saccades
Right lateral	2
Left lateral	2
Right anterior	1
Left anterior	0
Right posterior	3
Left posterior	1

The data was tested for normality distribution using Shapiro-Wilk test and it showed that the data were not distributed normally. Therefore non-parametric statistics were performed. To understand the significant differences between normal hearing and hearing loss groups in mean VOR gain, Mann-Whitney U test was done. The test revealed no significant difference for left horizontal canal [$Z=1.77$; $p=0.07$], right horizontal canal [$Z=0.08$; $p=0.93$], left anterior canal [$Z=1.92$; $p=0.05$], right anterior canal [$Z=0.932$; $p=0.34$], left posterior canal [$Z=1.07$; $p=0.28$], however there was significant difference seen for the right posterior canal [$Z=3.28$; $p=0.00$].

4.3.1 Correlation between VOR gain value with degree and duration of hearing loss

Spearman's rank correlation revealed no correlation between VOR gain of right lateral canal with degree of hearing loss [$r_s=0.88$; $p=0.59$], VOR gain of right anterior canal with degree of hearing loss [$r_s=0.02$; $p=0.86$], VOR gain of right posterior canal with degree of hearing loss [$r_s=0.06$; $p=0.71$]. Also, no significant correlation was found between VOR gain of right lateral canal with duration of hearing loss [$r_s=0.25$; $p=0.10$], VOR gain of right anterior canal with duration of hearing loss [$r_s=0.13$; $p=0.42$], VOR gain of right posterior with duration of hearing loss [$r_s=0.09$; $p=0.57$].

Spearman's rank correlation revealed no correlation between VOR gain of left lateral canal with degree of hearing loss [$r_s=0.08$; $p=0.62$], VOR gain of left anterior canal with degree of hearing loss [$r_s=0.05$; $p=0.76$], VOR gain of left posterior canal with degree of hearing loss [$r_s=0.01$; $p=0.93$], VOR gain of left lateral canal with duration of hearing loss [$r_s=0.11$; $p=0.48$], VOR gain of left anterior canal with duration of hearing loss [$r_s=0.17$; $p=0.29$], VOR gain of left posterior canal with duration of hearing loss [$r_s=0.20$; $p=0.22$].

4.4 Association between cVEMP, oVEMP and VHIT test findings

To find the association between the cVEMP, oVEMP, VHIT test findings in hearing impaired group, chi-square test was done. Table 4.7 shows chi-square value for the association between cVEMP and oVEMP test results.

Table 4.9. Association between cVEMP and oVEMP test results in hearing impaired individuals.

		oVEMP Left ear			oVEMP Right Ear		
		Present	Absent	Total	Present	Absent	Total
Left	Present	0	7	7			
cVEMP	Absent	7	26	33			
Total		7	33	40*			
Right	Present				2	4	6
cVEMP	Absent				7	27	34
Total					9	31	40*

*p>0.05 (Chi-Square test)

Table 4.10. Association between cVEMP and VHIT test results in hearing impaired individuals.

		cVEMP Left ear			cVEMP Right Ear		
		Present	Absent	Total	Present	Absent	Total
Left lateral canal	Normal	6	25	31			
	Reduced	0	5	5			
Total		6	30	36			

Left anterior canal	Normal	6	26	32			
	Reduced	0	4	4			
Total		6	30	36			
Left posterior canal	Normal	6	28	34			
	Reduced	0	2	2			
Total		6	30	36			
Right lateral canal	Normal				2	32	34
	Reduced				0	2	2
Total					2	34	36
Right anterior canal	Normal				2	32	34
	Reduced				0	2	2
Total					2	34	36
Right posterior canal	Normal				2	21	23
	Reduced				0	13	13
Total					2	34	36

*p>0.05(Chi-Square test)

Table 4.11. Association between oVEMP and VHIT test results in hearing impaired individuals.

		oVEMP Left ear			oVEMP Right Ear		
		Present	Absent	Total	Present	Absent	Total
Left lateral canal	Normal	5	26	32			
	Reduced	2	3	4			

Total		7	29	36		
Left anterior canal	Normal	6	26	32		
	Reduced	1	3	4		
Total		7	29	36		
Left posterior canal	Normal	6	28	34		
	Reduced	1	1	2		
Total		7	29	36		
Right lateral canal	Normal				4	30
	Reduced				0	2
Total					4	32
Right anterior canal	Normal				4	30
	Reduced				0	2
Total					4	32
Right posterior canal	Normal				2	21
	Reduced				2	11
Total					4	32

* $p > 0.05$ (Chi-Square test)

Thus, no association was found between the results of the cVEMP, oVEMP and vHIT tests in individuals with hearing loss.

To summarize the results of the present study, cVEMP was present in 100% in right and left ear in normal hearing individual whereas cVEMP was present in 13.25% in individuals with hearing loss. There was no significant difference found for latencies and amplitude complex of right ear. However, there was a significant difference for p1 latencies and pln1 amplitude complex in the left ear.

oVEMP was present in 100% in normal hearing individuals whereas it was present in 16.25%. There was no significant difference found between hearing impaired and normal hearing individuals for the latencies of n1 of left ear, p1 of left and right ear, n2 latency of right and left ear and p1n1 amplitude complex of right ear. However significant differences were found between normal hearing and hearing loss group for n1 latency of right ear, n1p1 amplitude complex, p1n2 amplitude complex for the right ear, n1p1 amplitude complex for the left ear and p1n1 amplitude complex for the left ear.

In VHIT, it was found that the mean VOR gain was not different in individuals with hearing loss and normal hearing individuals for right and left horizontal canals, right and left anterior canals and left posterior canals. However there was significant difference was found in right posterior canal when compared between hearing loss and normal hearing individuals.

Chapter V

DISCUSSION

Vestibular evoked myogenic potentials

cVEMP was present in 100% of the individuals with normal hearing, whereas, it was present only in 13.75% in individuals with hearing loss. There was a significant difference between normal hearing and hearing loss groups for p1 latency for left ear and p1n1 amplitude complex for left.

In the present study, oVEMP was present in 100% of the normal hearing individuals whereas oVEMP was present in only 16.25% of hearing impaired individuals. There were significant differences observed between normal hearing and hearing loss group for n1 latency of right ear, n1p1 amplitude complex of the right ear, p1n2 amplitude complex for the right ear, n1p1 amplitude complex for the left ear and p1n1 amplitude complex for the left ear.

Several other studies in the literature have also reported absence of cVEMP in individuals with various degree of sensorineural hearing loss. Iwasaki et al. (2005) reported absence of clicked evoked vestibular evoked myogenic responses in 77% in individuals with idiopathic sudden sensorineural hearing loss. Fujimoto et al. (2014) reported abnormal cervical vestibular evoked myogenic potentials responses in 64% of the individuals with idiopathic sensorineural hearing loss. Zhou et al. (2009) reported abnormal cVEMP in 91% children with severe to profound hearing loss. Cushing et al. reported that the cervical VEMP revealed normal responses in 71% in children with profound sensorineural hearing loss. Bansal, Sahni and Sinha (2016) reported absence of cVEMP in 100% of the subjects with severe to profound hearing loss. Sehta (2011) reported absence of cervical vestibular evoked myogenic potentials in 90% of the subjects with unilateral sudden sensorineural hearing loss. The differences in prevalence rates across the studies could be due to different methodologies or perhaps to the different ages of participants or due to different degree of hearing loss in the participants.

Xin et al. (2015) reported that the response rate of oVEMP in subjects with profound sensorineural hearing loss was 58.1% and also the amplitude of oVEMP was reduced in subjects with sensorineural hearing loss. Fujimoto et al (2014) reported abnormal oVEMP responses in 43% of subjects with idiopathic sudden sensorineural hearing loss. Nui et al (2014) found that the oVEMP responses were abnormal in 56% and 70% in individuals with sudden sensorineural hearing loss with or without vertigo respectively. Nui et al. (2014) also reported that abnormality of oVEMP is higher than any other vestibular test in individuals with sudden sensorineural hearing loss. In the present study, oVEMP responses were also correlated with the degree of hearing loss and no significant correlations were found. However, a contrasting study was reported by Rashmi (2011) where she found significant correlation with degree of hearing loss and oVEMP responses, as with the increase in the degree of hearing loss, the oVEMP responses were reduced. Bansal, Sahni and Sinha (2016) found presence of oVEMP responses in 66% of individuals with severe to profound sensorineural hearing loss and suggested that the utricular function is more linked with cochlear as compared to the saccule.

In the present study cVEMP and oVEMP responses were absent in most of the individuals with hearing loss of various degrees, suggesting saccular and utricular function be more related to the cochlea than semicircular function in individual with hearing loss. Studies have reported high incidence of vestibular abnormality in individuals with hearing loss attributing to the close physical connection between cochlea and otolithic organs sharing the common membranous labyrinth.

In the present study it was found that the p1n1 amplitude complex of the oVEMP were smaller in hearing impaired group than the normal hearing group suggesting there is an abnormality in the utricle in hearing loss individuals. This

abnormality could be due to the great association with vestibular and hearing anatomically related, thus, damage to the inner ear might cause damaged to both of the organs. Studies have reported that there is a strong association between hearing loss and saccular function and those who have been diagnosed with sensorineural hearing loss may already have saccular disorders.

Further, neither cVEMP nor oVEMP results had any correlation between the different degree and duration of hearing loss.

Singh et al. (2013) recorded vestibular evoked myogenic potentials in 15 children with severe to profound sensorineural hearing loss and found that VEMP was either absent or abnormal in all 15 participants. Zhou et al. (2009) recorded vestibular evoked myogenic potentials in 23 children with sudden sensorineural hearing loss and found that the VEMP was abnormal or absent in 21 out of 23 children. The relationship between hearing level and vestibular dysfunction in sensorineural hearing loss patients remains inconclusive. Most researchers considered that there was a more severe hearing loss in sensorineural hearing loss patients with abnormal caloric test (Korres et al. 2011; Xu et al. 2014). As to vestibular evoked myogenic potentials (VEMPs), in some studies, it was found that patients with profound hearing loss showed a higher abnormal rate of cVEMP test (Korres et al. 2011), but the association between abnormal cVEMP or oVEMP and hearing level was not revealed in other studies(Ogawa et al. 2012; Nagai et al. 2014; Fujimoto et al. 2015).

Video Head Impulse test

In the present study it was found that the VOR gain was normal in all the participants with normal hearing whereas in hearing loss groups, out of 72 ears analysed, reduced VOR gain was found in 28 ears (38.88%) and correctives saccades were observed in 9 ears (12.25%) with hearing impaired individuals. There was no significant difference observed in the VOR gain of hearing impaired group and normal hearing group for left horizontal canal, right horizontal, left anterior canal, right

anterior canal, left posterior canal; however there was significant difference seen for the right posterior canal in both the groups.

Sinha and Bansal (2017) reported reduced VOR gain of all six canals in individuals with severe to profound sensorineural hearing loss. Magliulo et al (2017) found significant deficit in the superior semicircular canal and horizontal canal in 2 subjects and posterior canal in 4 subjects with Usher syndrome type II. Bansal and Sinha (2016) reported that the mean VOR gain was reduced in the hearing loss participants as compared to the normal hearing participants. Lin et al (2015) reported abnormal horizontal SCC VOR gain in 38.5% of subjects with individuals with idiopathic sudden hearing loss. Komazec et al (2017) found reduced VOR gain of horizontal semicircular canal in VHIT in children with cochlear implants. Nassif, Balzanelli and Zinis (2016) reported no significant lateral semicircular canal high frequency VOR gain between cochlear implant users and normal hearing peers. The difference in results of the vHIT study could be because of the different population of SNHL studied. In the present study usual cases of sensorineural hearing loss were taken, whereas Sinha and Bansal (2017) had taken participants with congenital sensorineural hearing loss. The reduced VOR gain observed by Sinha and Bansal (2017) could be because of the profound degree of hearing loss taken for the study whereas, in the present study subjects hearing loss ranged between mild to severe hearing loss.

Refixation saccades indicates impaired vestibule ocular reflex and semicircular function which helps us in maintaining gaze in motion (eye velocity and head velocity should be equal and opposite). It occurs when the eye is not able to stabilize gaze and moves with the moving object and when this happens, central system gives information that the target is in fixed, then the compensatory movement of the eye to correct the

gaze leads to saccades or also called as corrective saccades. Studies have reported presences of refixation saccades in various vestibular pathologies as a compensatory mechanism for the impaired VOR gain. In a study it was also reported that the saccades were present in all the severe to profound sensorineural hearing loss.

In this present study, some individuals with hearing loss had refixation saccades might suggests presence of vestibular loss. Presence of refixation saccades in the presence of a normal or reduced VOR gain values is an indication of vestibular pathology (Weber et al. 2008; Magliulo et al. 2015). This is the first study which has measured the presence or absence of refixation saccades in adults with different degree of hearing loss. Earlier studies have measured only VOR gain values in individuals with severe to profound hearing loss. However, few of the earlier studies have reported presence of refixation saccades in 16%-43% of the individuals with various vestibular deficits (Weber et al. 2008; Magliulo et al. 2015).The difference in amount of refixation saccades in different studies could be because of the different population studied.

Association with three different test, cVEMP, oVEMP and VHIT on hearing loss group:

There was no association seen between the three tests, cVEMP, oVEMP and VHIT. This lack of association can be attributed to testing of different organs or functions by these tests as cVEMP assesses saccule, oVEMP assess utricule and vHIT assesses semicircular canals. The cVEMP pathways are longer than the oVEMP pathways and also cVEMP is recorded from sternocleidomastoid muscles which are thicker than the oVEMP recording site, inferior oblique muscle (Sarvanan, 2013).

Chapter-VI

SUMMARY AND CONCLUSION

The peripheral hearing and vestibular system consists of cochlea and otolith organs (sacculle and utricle) and 3 pairs of semicircular canals- lateral, anterior and posterior. The vestibular organs and the cochlea is situated in the inner ear, connected anatomically (receptor cell ultra-structures) and functionally incased within same membranous labyrinth and also shares common labyrinthine artery. Due to anatomical, histological and physiological similarities between these two organs, damage to the one structure can cause damage to the other also or lesions leading to sensorineural hearing loss could also contribute to dysfunction of the vestibular end organs.

Since, peripheral vestibular structures consists of many organs, one particular test cannot assess all the structures. Hence, different tests are employed to assess various peripheral structures. The cVEMP assesses sacculle, oVEMP assesses utricle and vHIT assesses semicircular canal, the three tests will give a complete image of the vestibular organs in hearing loss individuals. Hence, the aim of the present study was to assess the functioning of otolithic organ and three semicircular canals in individuals with various degree of sensorineural hearing loss using cVEMP, oVEMP and VHIT. Therefore, the objectives of the present study are:

1. To assess the functioning of sacculle in the individuals with sensorineural hearing loss.
2. To assess the functioning of the utricle in the individuals with sensorineural hearing loss.
3. To assess all three semicircular canal in the individuals with sensorineural hearing loss.

4. To find out correlation between the cervical VEMP, ocular VEMP and vHIT with degree or severity of hearing loss.
5. To find out correlation between the cervical VEMP, ocular VEMP and vHIT findings in individuals with sensorineural hearing loss.
6. To find out correlation between the cervical VEMP, ocular VEMP and vHIT findings with the duration of sensorineural hearing loss.

To achieve the aim of the present study, two groups were taken for the study. Group-I consisted of 40 individuals with various degrees of hearing loss within the age range of 18-44 years. Group II consisted of 40 participants with normal hearing within the age range of 18-35 years. All the participants had undergone a detailed case history, administered dizziness questionnaire, pure tone audiometry, immittance audiometry and acoustic reflex threshold test, cVEMP, oVEMP and VHIT.

cVEMP and oVEMP was recorded using 500 Hz tone burst stimuli presented at 95 dBnHL. For cVEMP recording the positive electrode was placed on the sternocleido mastoid muscle, negative electrode was placed on sternoclavicular joint and ground electrode was placed on the forehead. For oVEMP recording, the positive electrode was placed 1cm below the eyes, negative electrode was placed 1 cm below the positive electrode and ground electrode was placed on the forehead. Both the responses were analysed in a 70 msec time window including -10 msec pre stimulus time. For recording vHIT, the clients were made to sit one meter away from the target and head thrust were applied in lateral, LARP and RALP plane.

The waveforms of cVEMP, oVEMP responses and VHIT VOR gain were obtained from all the participants from both the groups. Analysis of p1 and n1 latency and p1n1 amplitude complex were done for the cVEMP; n1, p1, n2 latency, p1n1 and

p1n2 amplitude complex analysis was carried out for oVEMP and VOR gain and saccades were analysed for the VHIT.

Mean and standard deviation were calculated for both the groups and the following statistical analysis were done:

- Test of normality and found non-normality distribution of data, therefore non-parametric tests were performed.
- Comparison of group I and group II were done using Mann-Whitney test for all the three tests.
- Chi-square test was done to find out the association between three tests.

The results obtained for the above statistical analysis revealed following:

Cervical vestibular evoked myogenic potentials:

- cVEMP was present in 100% of the participants in normal hearing group and in hearing loss group cVEMP responses were present in 13.75%.
- There was no significant difference found for latencies and amplitude complex of right ear between individuals with normal hearing sensitivity and individuals with hearing loss.
- There was a significant difference for p1 latencies and pln1 amplitude complex in the left ear between individuals with normal hearing sensitivity and individuals with hearing loss.
- There was no correlation between cVEMP tests findings with degree and duration of hearing loss.

Ocular vestibular evoked myogenic potentials:

- oVEMP was present in 100% in normal hearing individuals whereas it was present in 16.25% % in hearing loss individuals.

- There was no significant difference found between hearing impaired and normal hearing individuals for the latencies of n1 of left ear, p1 of left and right ear, n2 latency of right and left ear and p1n1 amplitude complex of right ear between individuals with normal hearing sensitivity and individuals with hearing loss.
- There was significant differences were found between normal hearing and hearing loss group for n1 latency of right ear, n1p1 amplitude complex, p1n2 amplitude complex for the right ear, n1p1 amplitude complex for the left ear and p1n1 amplitude complex for the left ear between individuals with normal hearing sensitivity and individuals with hearing loss.
- There was no correlation between the oVEMP test findings with degree and duration of hearing loss.

Video head impulse test:

- The mean VOR gain was not different in individuals with hearing loss and normal hearing individuals for right and left horizontal canals, right and left anterior canals and left posterior canals between individuals with normal hearing sensitivity and individuals with hearing loss.
- There was significant difference was found in right posterior canal when compared between hearing loss and normal hearing individuals.
- Further there was no association found between cVEMP, oVEMP, and vHIT test results in hearing impaired individuals.

CONCLUSION

cVEMP, oVEMP and VHIT provides information of the peripheral vestibular systems i.e., saccule, utricle and semicircular canals respectively, thus these tests can be utilized to identify and diagnose various vestibular pathology. Findings of the present study suggest high prevalence of vestibular dysfunction mainly in saccular and utricular structures in hearing loss individuals as compared to the normal hearing sensitivity. Previous studies have also reported the saccular and utricular damage in hearing loss individuals. There was no association seen in the cVEMP, oVEMP and VHIT test findings in the hearing impaired population because all the three tests assess three different organs. To conclude, saccular and utricular dysfunction is more prevalent in individuals with hearing loss compared to the canal dysfunction. Hence, the individuals with hearing loss must be assessed for vestibular dysfunction using various test battery.

Implications of the study:

- This study provides information regarding the diagnostic significance of combination of vHIT, cVEMPs, and oVEMPs in individuals with sensorineural hearing loss.
- This study provides basis for selection of the different kinds of vestibular rehabilitation in individuals with sensorineural hearing loss.
- The study will help in identifying the exact site of lesion in individuals with sensorineural hearing loss.

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