Objective assessment of otolith and SCCs functions in individuals with severe to profound hearing loss

Bansal Shalini[1] & Sinha Sujeet Kumar[2]

Abstract

This study was designed to objectively assess the functioning of otolith (saccule and utricle) and three semicircular canals in individual with severe to profound sensorineural hearing loss using cVEMP, oVEMP and vHIT respectively. Twenty adult participants (40 ears) having severe to profound hearing loss ranging in age from 15-40 years in group I. Group-II consisted of 20 adult participants (40 ears) in the age range from 15-40 years with normal hearing sensitivity. All the participants underwent a detailed case history, pure tone audiometry, immitance and reflexometry, cVEMP, oVEMP and vHIT tests. cVEMP was present in 90% and 75% in right and left ear of individual with severe to profound hearing loss respectively. No significant difference between the latencies of both the groups whereas significant difference was found between the p1-n1 amplitude complexes of both the group in which smaller amplitude was found for individual with severe to profound hearing loss. oVEMP was present in 55% and 60% in right and left ear of individual with severe to profound hearing loss respectively. No significant difference between the latencies of n1, p1 and n2 of both the groups whereas significant difference was found for the amplitude complex of p1-n1 and p1-n2 of both the groups. Mean VOR gain values for right and left horizontal canals, right anterior and left posterior canal for individual with hearing impaired is lesser than the individual with normal hearing. There were significant differences between group 1 and group 2 for VOR gain for right horizontal canal and left horizontal canal whereas significant difference was showed in right posterior canal, left anterior canal, right anterior canal and left posterior canal. No association found between cVEMP, oVEMP and different planes of vHIT of right ear. To conclude, vestibular abnormality was seen for both otolith organs (saccule and utricle) and semi circular canals in individual with severe to profound hearing loss. Therefore, vestibular tests should be included along with various audiological tests in the diagnostic protocol for the assessment of individual with severe to profound sensorineural hearing loss.

Key words: *cVEMP,oVEMP, vHIT, VOR gain.*

Introduction

The vestibular system is broadly categorized into both peripheral and central system. The peripheral system is bilaterally composed of three semicircular canals (posterior, superior, lateral) and the otolithic organs (saccule and utricle). The semicircular canals detect rotational head movement while the utricle and saccule respond to linear acceleration and gravity, respectively. These vestibular organs are in a state of symmetrically tonic activity, that when excited stimulate the central vestibular system. This information, along with proprioceptive and ocular input, is processed by the central vestibular pathways (e.g. vestibular nuclei) and maintains our sense of balance and position.

Also, vestibular system is responsible for stabilizing the position of the eyes, head and body in space, and helps to maintain an upright stance. It is composed of two parts, each with different roles: (1) the vestibular-ocular system, responsible for visual stabilization; and (2) the vestibular-spinal system, which maintains the orientation of the body in space and contributes to the postural tone necessary for the acquisition of motor developmental milestones.

Vestibular Evoked Myogenic Potential (VEMP) is a non-invasive test to assess the functioning of otolith organs of inner ear. It is a short latency muscle potential which is elicited by the presentation of loud sound. One of the variant of VEMP is Cervical VEMP (cVEMP) which has been found to be originated from the saccule (Colebatch, Halmagyi, & Skuse, 1994; Todd, Cody, & Banks, 2000). It has been found to be useful in finding out the pathology of the saccule or its end organ pathologies in various vestibular disorders such as: vestibular neuritis (Chihara et al., 2012 ; Manzari, Burgess, & Curthoys, 2012) cerebellopontine angle tumor (Beyea & Zeitouni, 2010; Murofushi & Takehisa, 2001), auditory neuropathy (Sinha, Barman, Singh, Rajeshwari & Sharanya, 2013). Also, the cVEMPs has been found useful in the diagnosis of other vestibular pathologies such as Semicircular canal dehiscence syndrome (Brantberg & Verrecchia, 2012) and multiple sclerosis (Murofushi, Shimizu, Takegoshi, & Cheng, 2001).

Another variant of Vestibular Evoked Myogenic Potential is ocular VEMP (oVEMP), which has been introduced recently and has been suggested to be utricular in origin (Halmagyi Curthoys, Colebatch, 2005; Curthoys, 2010; Welgampola & Carey, 2010; Brandt & Strupp, 2010). It is mediated through vestibulo-ocular reflex pathway. Ocular vestibular evoked myogenic potentials (oVEMPs) also has been utilised in diagnosing inter nuclear ophthalmoplegia (Rosengren & Colebatch, 2011), to differentiate

1. shalinib@gmail.com
2. sujitks@gmail.com

between cerebellar and brainstem lesions (Su&Young.,2011) auditory neuropathy/audiovestibular neuropathy, superior semicircular canal dehiscence syndrome (Rosengren, Aw, Halmagyi, Todd, & Colebatch, 2008) and vestibular neuritis (Murofushi, Nakahara, Yoshimura, & Tsuda, 2011).

Another test which has been utilized recently for the diagnosis is video head impulse test (vHIT). vHIT is quick to administer and noninvasive test. It objectively measures the head velocity and the eye velocity response during brief, abrupt, unpredictable, passive head rotations, and so provides a measure of vestibulo-ocular reflex (VOR) gain and VOR gain asymmetry. It provides an absolute measure of the functional level of every semicircular canal separately. It allows the clinician to diagnose patients with VN acutely while they are ill and assess them again after they have recovered, providing objective evidence of the VOR deficit and the extent of its recovery.

vHIT helps to improve diagnostic accuracy for patients with acute spontaneous vertigo. vHIT is also able to overcome the problems that is being faced while using rotational chair test. As rotational chair test have used big expensive chairs, with low accelerations which put the patient to sleep. vHIT can be performed in a fully lit room and even during acute attacks of vertigo. vHIT help to detect vertical canal dysfunction (MacDougall et al., 2013) . Also, measure the individual SCC's which help to diagnose pheripheral vestibular loss, such as superior and inferior vestibular neuritis.

Anatomical, histological and physiologic similarities between the cochlear and vestibular end organs explain the relation between hearing loss and vestibular disturbs. As both systems are related, in patients with hearing loss it is important to study the complete balance in order to diagnose and prevent a worse vestibular problem. Since vHIT assesses the SCC's, cVEMP assesses the saccule and oVEMP assesses the utricle, the administration of three tests together will complete the picture of the vestibular system in individuals with severe to profound hearing loss.

Nearly 90% of the individual with sensorineural hearing loss is caused by damage to the cochlea or the vestibulocochlear nerve (Agrawal, Platz & Niparko, 2008). The vast majority of those with SNHL have bilateral impairment. Cochlea and the vestibule share the continuous membranous labyrinth of the inner ear through ductus reunions anatomically. So, there are chances that in individuals with sensorineural hearing loss, disturbances of cochlear function could accompany with vestibular impairment. Various studies have reported the prevalence of VEMP abnormality from 53% (Jafari & Asad Malayeri, 2011) to 67% in severe to profound hearing loss individuals (Bansal, Sahani & Sinha, 2013). Affected VEMP is suggestive of affected utricular function being more linked to the cochlea than saccular function in individuals with severe to profound hearing loss. There is dearth of information regarding the function of semicircular canal in individual with severe to profound hearing loss. Also, there is a study reported in the literature regarding the difficulty in balancing among the individuals with sensorineural hearing loss (Voelker & Chole, 2010). Therefore diagnostic evaluation of the vestibular system becomes an essential aspect.

The aim of the present study was to objectively assess the functioning of otoliths (saccule and utricle) and three semicircular canal in individual with severe to profound sensorineural hearing loss using cVEMP, oVEMP and vHIT respectively.

METHOD

Participants:

Twenty adult participants (40 ears) having severe to profound hearing loss ranging in age from 15-40 years in group I. Group-II consisted of 20 adult participants (40 ears) in the age range from 15-40 years with normal hearing sensitivity. All the participants underwent a detailed case history, pure tone audiometry, immitance and reflexometry, cVEMP, oVEMP and vHIT tests. Participants did not have any middle ear pathology, no history of neuromuscular problems in body and neck region, did not have history or presence of neurological problems. Participants did not have history or presence of any ear pain, ear discharge, not have uncomfortable loudness level problems and did not have vestibular sign and symptoms.

Instrumentation:

Calibrated GSI-61 audiometer with TDH-39 headphone encased in MX-41/AR supra-aural cushion was utilized for estimation of air conduction pure tone thresholds.Bone conduction threshold was estimated using Radio ear B-71 bone vibrator. Middle ear status was evaluated by using a calibrated Grason-Stadler Tympstar(GSI) middle ear analyser. Bio-Logic Navigator Pro System was used to record vestibular evoked myogenic potentials (VEMP) and Video head impulse tests were all carried out with prototype ICS impulse video goggles (GN Otometrics, Taastrup, Denmark), with a camera speed of 250frames/s, recording motion of the right eye. All the measurement was carried out in an acoustically treated double room situation. All the testing was carried out in an acoustically and electrically shielded room where the levels was within the permissible limits (ANSI S3.1; 1991).

Test Procedure

Written consent was taken from all the subjects.Pure-tone thresholds was obtained for all the participants

using modified version of Hughson and Westlake sprocedure (Carhart & Jerger, 1959) at octave frequencies between 250 Hz to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction. UCL was obtained in both ears for air conducted speech stimuli using ascending method. Immittance audiometry was carried out in both ears using a probe tone frequency of 226 Hz. Tympanometry was done initially and then ipsilateral and contralateral acoustic reflex threshold was measured for 500, 1000, 2000, and 4000 Hz stimuli.

Cervical Vestibular evoked myogenic potentials (cVEMP) :

cVEMP was recorded from all the participants. Prior to cVEMPs recording the electrode sites was cleaned with abrasive gel (Nuprep). The silver chloride disc type of electrodes was placed on the electrode sites with adequate amount of conduction paste. Surgical tape was used to hold the electrode on the electrode sites. Absolute electrode impedances and inter electrode impedances was maintained below 5000 ohms and 2000 ohms respectively. During the cVEMPs recordings the participants were instructed to sit straight and turn their head to the opposite side of the ear in which stimulus was presented, so as to activate ipsilateral sternocleidomastoid (SCM) muscle, as it gives reliable and greater amplitude. Participants were instructed to maintain the same posture throughout the test run. cVEMPs was recorded using 500 Hz tone burst (2 cycles rise, 1 cycles plateau, and 2 cycles fall, Blackman weighting function) presented at a rate of 5.1/sec using rarefaction polarity. The stimuli were presented to the test ear at single intensity of 125dBSPL using ER - 3A insert ear phones. The responses were recorded for 64msec post stimulus period. The recorded responses were then amplified (X 5000) and band pass filtered between 30 to 1500 Hz. The responses were averaged totally for 150 stimuli. cVEMPs was recorded twice to ensure the replicability of the responses.

For oVEMPs recordings, the electrode sites were cleaned with abrasive gel (Nuprep). The silver chloride disc type of electrodes was placed on the electrode sites with adequate amount of conduction paste. Surgical tape was used to hold the electrode on the electrode sites. Inverting electrode (-) was placed inferior to the lower eyelids of contra lateral eye to the side being stimulated, non-inverting electrode (+) was placed immediately inferior to the inverting electrode and ground electrode was placed on lower forehead. Absolute electrode impedances and inter electrode impedances was maintained below 5000 ohms and 2000 ohms respectively. oVEMPs was recorded for all the participants with upper gaze direction. Participants were instructed to maintain the same upper gaze throughout the test run.

Stimuli used to record oVEMPs were identical to stimuli used to record cVEMPs. 500 Hz tone burst (2 cycles rise, 1 cycles plateau, and 2 cycles fall, Blackman weighting function) presented at a rate of 5.1/sec using rarefaction polarity. The stimuli were presented monaurally at single intensity of 125 dBSPL using ER - 3A insert ear phones. 150 stimuli were used for response averaging. The response was analysed for 64 msec post stimulus period. The recorded electrical responses were amplified (X 30000) and band pass filtered between 1 Hz to 1000 Hz. oVEMPs responses were recorded twice in each ear to ensure replicability of the responses.

Video heed impulse test (vHIT):

Video head impulse test as carried out in well lit room. Target was kept at the eye-level at a distance of 1 m in front of participants. Participants were seated on a height adjustable, rotatable chair was used to maintain ideal height for clinician to deliver horizontal or vertical impulses. vHIT goggles were tightened on the head of each participant to minimize goggles slippage. The target was fixed according to the participant height. Participants were fixated on two projected laser dots separately for calibration of eye position signal. Once calibration was done then participants were instructed to maintain their gaze at the target object, which was located at the eye level beyond the camera at a distance of 1 m straight ahead. A clinician stood behind each participant and rotated the head in horizontal planes in right and left direction. For LARP and RALP positions, the clinician moved the head of the participant upward and downward plane towards right and left side. Each participant underwent a minimum of 20 head impulses in each plane and in each direction. The head was rotated manually and abruptly in each plane at an angle of 10-20 and was randomized. A high speed digital infrared camera which is a part of the instrument was utilized to record the eye movement during and immediately after the head rotation. Mean VOR gain was calculated by taking the average VOR gain of 20 trials in each plane. VOR gain calculation for 20 trials in each plane provides a good response and good test-retest reliability in normal hearing individuals (Bansal & Sinha, 2016).

Response Analysis

Latency of p1, n1 peaks and p1-n1 amplitude complex of cervical VEMP, latency of n1, p1 and n2 peaks, n1-p1 amplitude complex and p1-n2 amplitude complex of oVEMP, latency and amplitude of N3 potentials were analyzed. VOR Gain value responses were analysed for both the groups in vHIT test.

RESULTS

Cervical vestibular evoked myogenic potentials (cVEMP)

Latency of p1, n1 peaks, and amplitude of p1-n1

complex of cVEMP were analyzed for both the groups. In normal hearing group cVEMP potential was present in all 40 ears i.e., in 100% of the ears.

In individuals with severe to profound sensorineural hearing loss cVEMP potentials was present in 18 of the 20 in right ear and 15 of the 20 left ears in the present study. Descriptive statistics was done to calculate the mean and standard deviation for the latency and amplitude of cVEMP parameters for both the ears in normal hearing individuals and individuals with sever to profound sensorineural hearing loss. The values of mean and standard deviation for p1 latency, n1 latency and p1-n1 amplitude complex of both the groups are shown in Table -1

Table 1 : Mean, and standard deviation (SD) for cVEMP potential of individual with normal hearing of right and left ears and individual with severe to profound hearing loss of right and left ears

cVEMP (Normal hearing)	**Mean**	**SD**	**Mean**	**SD**
p1 Latency[msec]	14.30	0.55	14.33	0.35
n1 Latency[msec]	22.19	0.70	22.36	0.50
p1-n1 amplitude [µV]	73.45	66.6	59.29	48.47
cVEMP(Individual with severe to profound hearing loss)	**Mean**	**SD**	**Mean**	**SD**
p1 Latency[msec]	14.40	1.15	14.66	1.06
n1 Latency[msec]	22.18	1.44	21.90	1.03
p1-n1 amplitude [µV]	41.15	43.64	47.56	49.74

It can be seen that mean latencies of p1, n1 of cVEMP potential of individual with severe to profound sensorineural hearing loss is almost similar to normal hearing individuals in both the ears. However, the amplitude of p1-n1 complex in individual with normal hearing is more than individual with severe to profound sensorineural hearing loss.

The obtained data was tested for normality distribution. Shapiro-Wilk test was done for normality check and it showed a non - normal distribution of data ($p<0.05$). Therefore non- parametric statistics was done for the entire data.

To, understand the significant differences, in mean latency and amplitude of different parameters between the two groups of different ears Mann-Whitney U Test was done. The test revealed no significant difference between group 1 and group 2 for latency of p1 of right ear for [$z = 0.19$, $p>0.05$], latency of n1 [$z= 0.47$, $p>0.05$] and amplitude complex of p1-n1 [$z= 1.80$, $p>0.05$]. For left ear of two groups, Mann-Whitney U test revealed no significant difference between group 1 and group 2 for latency of p1 [$z = 0.81$, $p>0.05$] and amplitude complex of p1-n1 [$z= 1.23$, $p>0.05$], however a significant difference was observed between the two groups for latency of n1 [$z= 2.00$, $p<0.05$].

Further to find out the significant differences between the two ears data, Wilcoxson signed rank test was done. Wilcoxson signed rank test did not show any significant difference in cVEMP parameters between the two ears($p>0.05$). Since there were no differences between the data of the two ears for any of the cVEMP parameters, the data of the two ears were combined. Descriptive statistics was done for the overall data to calculate the mean and standard deviation for the latency and amplitude of cVEMP parameters. The values of mean and standard deviation for p1 latency, n1 latency and p1-n1 amplitude complex are shown in Table -2

Table-2 : Mean and standard deviation for cVEMP potential of individual with normal hearing and Severe to Profound sensorineural hearing loss [SNHL]

cVEMP	**Severe to Profound SNHL (Group1) N=33**			**Normal hearing (Group2) N=40**		
	Mean	SD	Median	Mean	SD	Median
p1 Latency[msec]	14.51	1.11	14.32	14.31	0.46	14.31
n1 Latency[msec]	22.06	1.26	21.83	22.26	0.60	22.31
p1-n1 amplitude [µV]	45.51	4.7	26.4	64.79	5.84	50.9

It can be seen from Table-1 that mean latencies of p1, n1 of cVEMP potential of individual with normal hearing is almost similar to individual with severe to profound sensorineural hearing loss. However, the amplitude complex of p1-n1 in individual with normal hearing are larger than individual with severe to profound sensorineural hearing loss.

Further to understand the significant difference in mean latency and amplitude of different parameters of overall data, between the two groups, Mann-Whitney Test was done. Mann-Whitney test revealed no significant difference between group 1 and group 2 for latency of p1 [$z = 0.40$, $p>0.05$]. However, the Mann-Whitney test showed a significant difference for latency of n1 [$z= 2.20$, $p<0.05$] and amplitude complex of p1-n1 [$z= 1.91$, $p<0.05$] between group 1 and 2. To summarize, for the latency of p1 there was no significant difference between the two groups, however latency of n1 and the amplitude of n1-p1was significantly lower for individuals with severe to profound sensorineural hearing loss compared to normal hearing individuals.

Ocular vestibular evoked myogenic potentials (oVEMP)

oVEMP was present in all 40 ears i.e., in 100% of the ears in individual with normal hearing. In individual with severe to profound sensorineural hearing loss oVEMP potentials were present in 11 of the 20 right ears and 12 of the 20 left ears in the present study. Descriptive statistics was done to calculate the mean, standard deviation for the latency and amplitude of oVEMP parameters for both the groups of right and left ears. The values of mean and standard deviation for n1 latency, p1 latency, n2 latency, n1-p1 amplitude complex of normal hearing of both the ears and individual with severe to profound sensorineural hearing loss of both the ears are shown in Table 3

Table 3 : Mean, and standard deviation, for oVEMP potential of individual with normal hearing of right and left ears and individual with hearing loss of right and left ears

oVEMP (Individual with normal hearing)	Mean	SD	Mean	SD
n1 Latency[msec]	10.48	0.42	10.56	0.33
p1 Latency[msec]	15.55	0.47	15.69	0.67
n2 Latency(msec)	20.43	0.64	20.83	0.66
n1-p1amplitude [µV]	3.75	2.93	5.38	7.17
p1-n2amplitude [µV]	3.03	5.17	3.24	1.86
oVEMP (Individual with severe to profound hearing loss)				
n1 Latency[msec]	10.64	1.59	10.84	1.58
p1 Latency[msec]	15.62	1.48	15.67	1.68
N2 Latency(msec)	20.55	0.92	20.73	1.00
N1-p1amplitude [µV]	2.04	1.83	1.49	1.08
p1-n2amplitude [µV]	2.96	4.09	1.36	1.63

It can be seen from Table-3 that mean latencies of n1, p1 and n2 of oVEMP potential of individual with normal hearing is almost similar to individual with severe to profound sensorineural hearing loss. However, the amplitude complex of n1-p1 in individual with normal hearing are larger than individual with severe to profound sensorineural hearing loss.

The obtained data was tested for normality distribution. Shapiro-Wilk test was done for normality check and it showed a non - normal distribution of data ($p<0.05$). Therefore non- parametric statistics was done.

Further, to understand the significant difference in mean latency and amplitude of different parameters between the two groups of right ear, Mann-Whitney U Test was done. Mann-Whitney test revealed no significant difference between group 1 and group 2 for latencies of n1 [$z = 0.62$, p.0.05], p1 [$z= 1.17$, $p>0.05$], latency of n2 [$z= 0.35$, $p>0.05$], amplitude complex of p1-n1 [$z= 1.73$, $p>0.05$] and amplitude complex of n2-p1 [$z= 0.43$, $p>0.05$].

For left ear of two groups, Mann-Whitney U Test revealed no significant difference between group 1 and group 2 for n1 latency [$z = 0.21$, $p>0.05$], p1 latency [$z = 0.89$, $p>0.05$], n2 latency [$z = 0.74$, $p>0.05$] whereas significant difference was observed for amplitude of n1p1complex of [$z = 2.88$, $p<0.05$] and p1n2 [$z= 2.27$, $p<0.05$].

Further to understand the ear differences for different parameters of oVEMP, Wilcoxon signed rank test was done to find out the significant difference between right and left side of oVEMP parameters. The results of Wilcoxson signed rank test are given in Table 4

Table 4 : Wilcoxon signed ranks test in individual with severe to profound hearing loss and individual with normal hearing of oVEMP

oVEMP	Rn1- Ln1	Rp1 - Lp1	Rn2 - Ln2	Rn1p1 - Ln1p1	Rn2p1 - Ln2p1
p value	0.72	0.54	0.60	0.37	0.25
z value	1.16	1.76	0.35	0.61	0.53

Wilcoxson signed rank test revealed no significant differences for any of the parameters of oVEMP for the two groups, hence the data of the two ears for both the groups were combined. Descriptive statistics was done to calculate the mean and standard deviation of overall combined data for latency and amplitude of oVEMP parameters in group 1 and group 2. The values of mean and standard deviation for n1 latency, p1 latency, n2 latency, n1-p1 amplitude complex of normal hearing and individual with severe to profound sensorineural hearing loss are shown in Table -5

Table-5 : Mean and standard deviation for oVEMP potential of individual with normal hearing and Severe to Profound sensorineural hearing loss [SNHL]

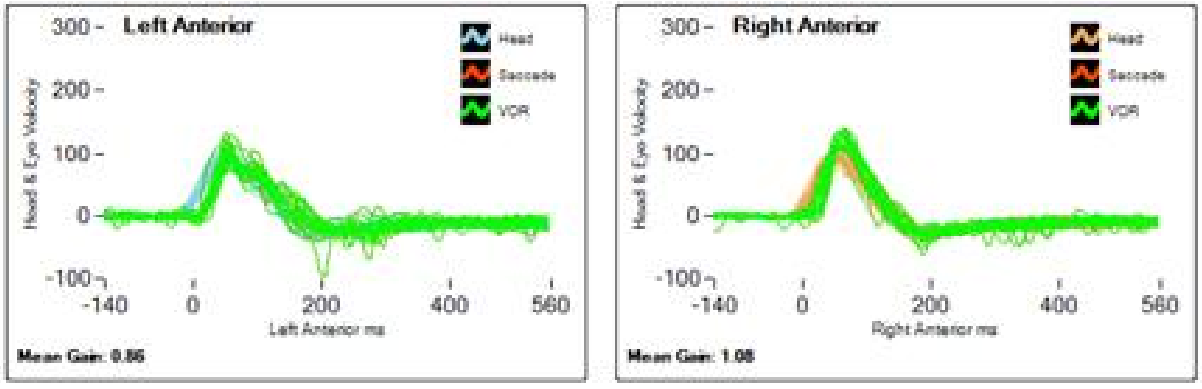
oVEMP	Severe to Profound SNHL (Group1) N=23			Normal hearing (Group2) N=40		
	Mean	SD	Median	Mean	SD	Median
n1 Latency[msec]	10.75	1.55	10.57	10.53	0.37	10.40
p1 Latency[msec]	15.65	1.55	15.83	15.64	0.58	15.45
n2 Latency[msec]	20.68	0.94	20.70	20.63	0.68	20.45
n1-p1 Complex amplitude [µV]	2.18	2.96	1.20	3.47	2.46	2.92
p1-n2 Complex amplitude [µV]	1.69	1.73	1.18	3.73	5.57	1.90

It can be seen from Table-5 that mean latencies of n1, p1 and n2 of oVEMP potential of individual with normal hearing is almost similar to individual with Severe to Profound sensorineural hearing loss. However, the amplitude complex of n1-p1 in individual with normal hearing are larger than individual with severe to profound sensorineural hearing loss.

Further to understand the significant difference in mean latency and amplitude of different parameters for combined data between the two groups, Mann-Whitney Test was done. Mann-Whitney test revealed no significant difference between group 1 and group 2 for latencies of n1 [z = 0.71, p.0.05], p1 [z= 1.31, p>0.05], n2 [z= 0.932, p>0.05]. However, the Mann-Whitney test showed a significant difference for amplitude complex of p1-n1 [z= 3.49, p<0.05] between group 1 and 2. To summarize, for the latency of n1, p1 and n2 there was no significant difference between the two groups, however the amplitude was significantly lower for group 1 compared to group 2.

Video head impulse test (vHIT)

Mean VOR gain was analyzed in vHIT for both the groups. All individual with normal hearing had normal VOR gain for all six SCC's. Mean VOR gain of one individual with normal hearing is shown in fig: 1



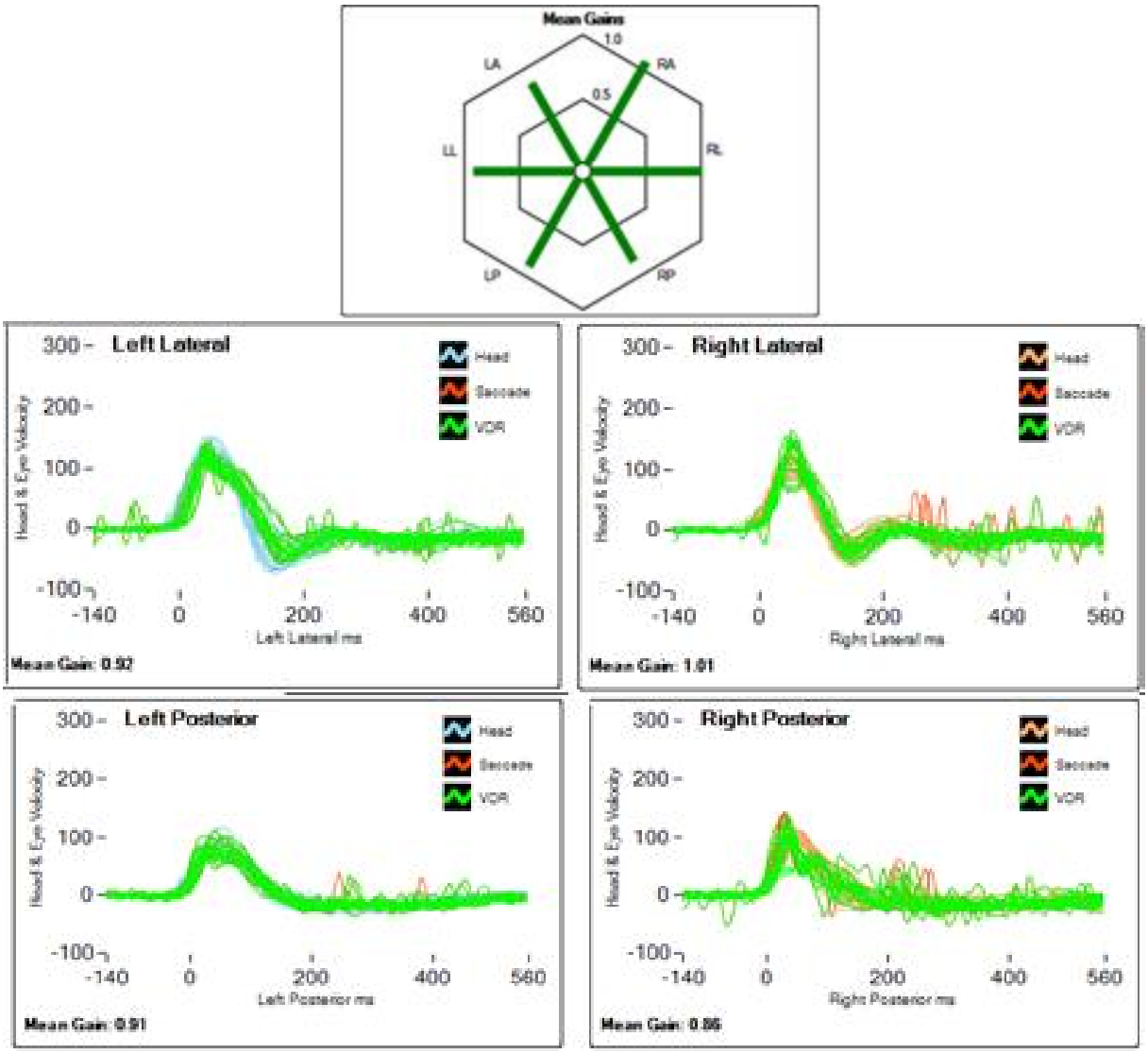


Figure 1 Video head-impulse test results in 3 different planes of a individual with normal hearing participant. The head and eye velocities throughout different head impulses to the right or left side are shown. Also, the VOR gain values are shown in the in the form of Hexaplot.

Individual data was analyzed for individual with hearing impaired and found that 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, 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 left posterior canal was reduced for 5 individuals and increased for 3 individuals. Mean VOR gain of for individual with hearing impaired with normal VOR gain and with reduced VOR gain are shown in fig 2 and 3 respectively.

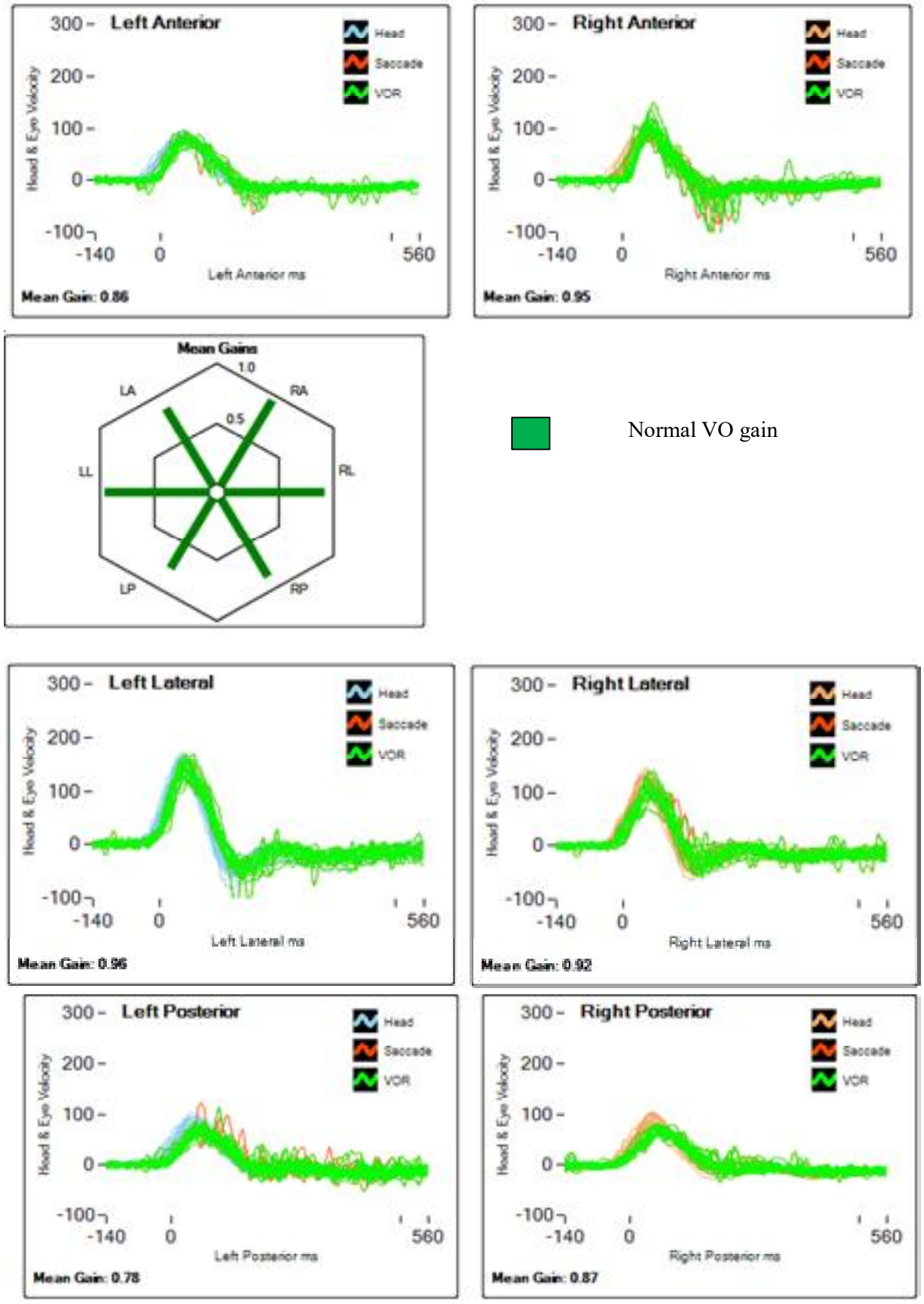
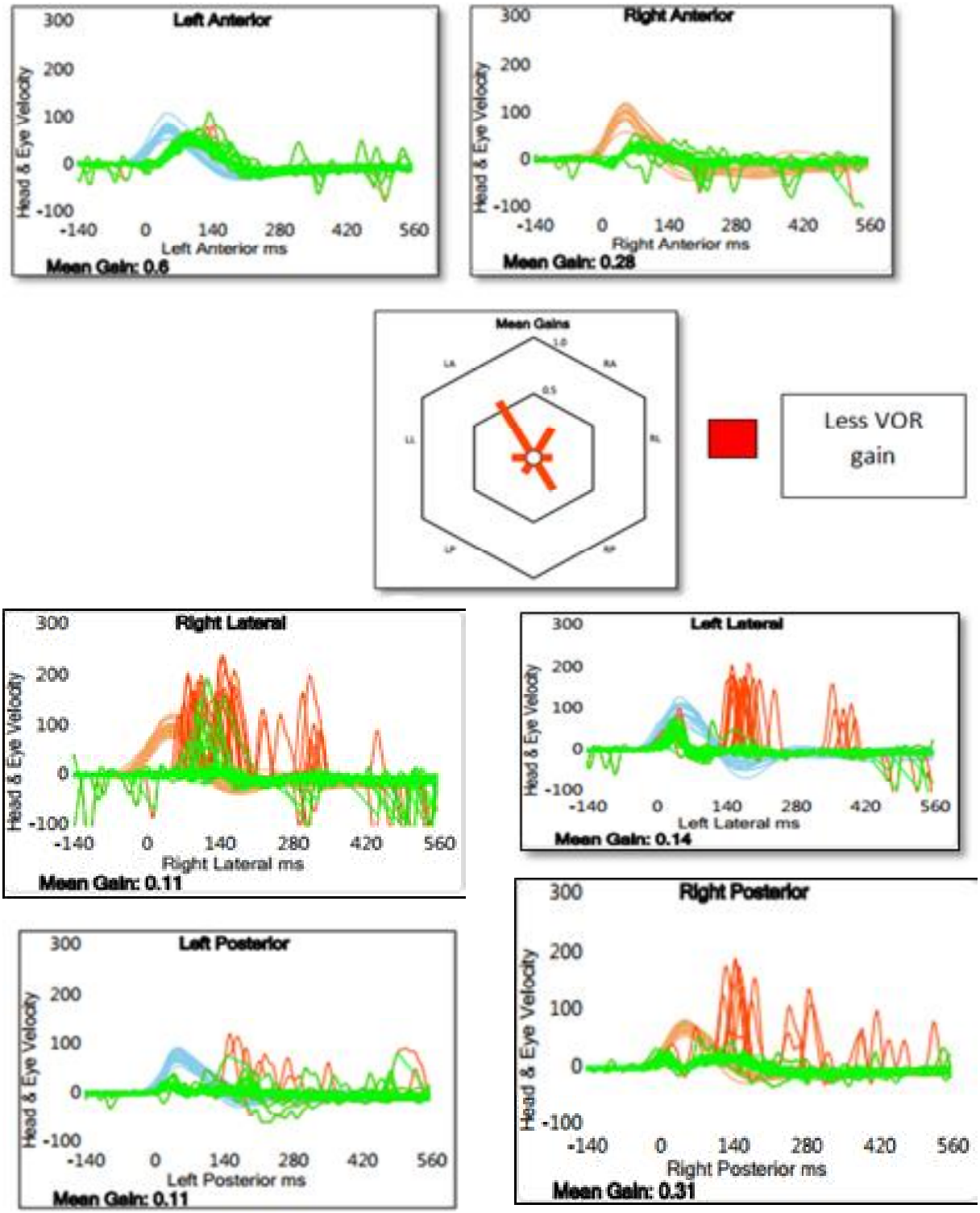


Figure 2 Video head-impulse test results in 3 different planes of a participant with normal VOR gain in individual with severe to profound hearing loss. The head and eye velocities throughout different head impulses to the right or left side are shown. Also, the gain values are shown in the figure in the form of



hexaplot.

Figure 3 Video head-impulse test results in 3 different planes of a participant with abnormal VOR gain in individual with severe to profound hearing loss. The head and eye velocities throughout different head impulses to the right or

left side are shown. Also, the gain values are shown in the figure in the form of hexaplot.

Descriptive analysis was done to calculate mean and gain for both the groups is listed in Table 6

standard deviation of VOR gain in all three planes in both the directions. That is right horizontal (RH), left horizontal (LH), right posterior (RP), left anterior (LA), right anterior (RA), left posterior (LP). Value of VOR

	Group 1 (Individual with severe to profound hearing loss)			**Group 2 (Individual with normal hearing)**		
PLANES	Mean	SD	Median	Mean	SD	Median
Right horizontal	0.82	0.24	0.92	1.02	0.11	1.01
Left horizontal	0.76	0.25	0.84	0.95	0.08	0.96
Right posterior	0.88	0.28	0.88	0.87	0.09	0.86
Left anterior	0.90	0.26	0.93	0.89	0.10	0.86
Right anterior	0.83	0.23	0.84	0.92	0.10	0.89
Left posterior	0.81	0.23	0.84	0.90	0.10	0.88

Table 6 : *Mean and standard deviation was calculated for VOR gain for both the groups*

It can be seen from Table-6 that mean VOR gain values for right and left horizontal canals, right anterior and left posterior canal for individual with hearing impaired (Group-1) is lesser than the individual with normal hearing. Mean VOR gain for right posterior and left anterior canal are similar for both the groups.

The obtained data was tested for normality distribution. Shapiro-Wilk test was done for normality check and there is no significant difference in the VOR gain of different in individual with severe to profound hearing loss which showed a non - normal distribution of data. Therefore non- parametric statistics was done by using SPSS software.

Wilcoxon rank test was done to find the significant difference between different plane of semicircular canals of individual with normal hearing and individual with severe to profound hearing loss and values are shown is table 7

vHIT	**Normal hearing**			**Hearing impaired**		
	LL- RL	LP- RA	LA- RP	LL- RL	LP- RA	LA–RP
z value	1.89	0.14	0.77	2.46	0.78	0.95
P value	0.02	0.43	0.34	0.06	0.89	0.44

Table 7 : Wilcoxon signed ranks test in individual with severe to profound hearing loss and individual with normal hearing

There was significant difference found in left and right lateral plane in individual with normal hearing whereas no significant difference was found between the posterior and anterior semi circular canal of both the ears. Also, there was no significant difference found in all three planes of semicircular canal in both the ears in individual with sever to profound hearing loss.

Further to understand the significant difference in mean values of VOR gain between the two groups, Mann-Whitney U Test was done. Mann-Whitney U Test revealed significant difference between group 1 and group 2 for VOR gain for right horizontal canal [$z = 3.07$, $p < 0.05$] and left horizontal canal [$z = 3.01$, $p < 0.05$] whereas no significant difference was showed in right posterior canal [$z = 0.13$, $p > 0.05$], left anterior canal [$z = 0.10$, $p > 0.05$],right anterior canal[$z = 1.39$, $p > 0.05$] and left posterior canal [$z = 1.02$, $p > 0.05$].

Association between cVEMP, oVEMP and vHIT:

To find the association between the cVEMP, oVEMP, vHIT chi-square test was done and values are shown in table 8.

Test	Right cVEMP			Right oVEMP		
vHIT	Present	Absent	Total	Present	Absent	Total
Right Lateral						
Present	14			7		
Absent	4	1	15	4	8	15
Total	18	1	5	11	1	5
p- Value*	0.74*	2	20	1.68*	9	20
Right Posterior						
Present	10			5		
Absent	8	1	11	6	6	11
Total	18	1	9	11	3	9
p- Value*	0.02*	2	20	0.90*	9	20
Right Anterior						
Present	12			6		
Absent	6	2	14	5	8	14
Total	18	0	6	11	1	6
p- Value*	0.95*	2	20	2.78*	9	20

(*) Chi-Square Test

Table 8 : Association between cVEMP, oVEMP and vHIT of right ear

From the above table, it was observed that there was association between right posterior plane of vHIT and right cVEMP whereas no association found between cVEMP, oVEMP and different planes of vHIT.

Test	**Left cVEMP**			**Left oVEMP**		
vHIT	Present	Absent	Total	Present	Absent	Total
Left Lateral						
Present	9			6		
Absent	6	3	12	6	6	12
Total	15	2	8	12	2	8
p- Value*	0.00*	5	20	1.25*	8	20
Left anterior						
Present	10			8		
Absent	5	3	13	4	5	13
Total	15	2	7	12	3	7
p- Value*	0.73*	5	20	0.03*	8	20
Left posterior						
Present	12			9		
Absent	3	4	16	3	7	16
Total	15	1	4	12	1	4
p- Value*	0.00*	5	20	0.46*	8	20

Table 4.10 : Association between cVEMP , oVEMP and vHIT of left ear

(*) Chi-Square Test

Form above table, it was observed that there are association between left cVEMP and left lateral plane of vHIT ($p<0.05$), left cVEMP and left posterior plane of cVEMP ($p<0.05$) and left oVEMP and left anterior plane of vHIT. However, there was no association was found between other test of left ear.

DISCUSSION

Cervical vestibular evoked myogenic potential

cVEMP was present in 100% in both right and left ear of individual with normal hearing whereas 90% and 75% in right and left ear of individual with severe to profound hearing loss respectively.

The presence of cVEMP in the present study is more compared to the earlier studies. Singh, Gupta, & Kumar, (2012) reported a presence of cVEMP in 87% children of age range 4-12 years with severe to profound hearing loss. Shinjo, Jin, & Kaga, (2007) revealed presence of cVEMP in 75% of the subjects with severe to profound hearing loss. Bansal, Sahni, & Sinha, (2013) reported presence of cVEMP in 98% of individual with sever to profound hearing loss.

Zhou et al., (2009) reported abnormal cVEMP in 21 of 23 children (91%) with sensorineural hearing loss. Zhou et al., (2009) also found significant difference in

amplitudes between children with sensorineural hearing loss and normal hearing. Amplitude was lower in children with sensorineural hearing loss compared to children with normal hearing. Also, Ochi & Ohashi, (2001)showed the prevalence of cVEMP in 66.7% of total ears in individuals with sensorineural hearing loss. Shinjo et al., (2007) reported presence of cVEMP in 50% of individual with hearing loss, asymmetrical responses in 30% of the individuals with sensorineural hearing loss, whereas 20% of individual with severe sensorineural hearing loss had absence of response bilaterally. Similar findings were reported by Tribukait, Brantberg, & Bergenius, (2004), Tribukait et al.(2004) reported normal cVEMP responses in 58% of individual bilaterally, 17% individual with asymmetric response and 25% individual had no VEMP response. Shall & Shall, (2009) reported to have absence VEMP in 22 children of 33 children with profound hearing loss.

The difference in prevalence rate of cVEMP in different studies in sensorineural hearing loss could be due to the difference in population tested. Earlier studies have reported the prevalence in children whereas; the present study bilateral severe to profound sensorineural hearing loss individuals have participated. Also, the etiological factors for the sensorineural hearing loss population tested in these studies were different. In present study, significant difference was found in the amplitude of cVEMP responses.

There was no significant difference found for latencies of right ear whereas significant difference was showed in p1-n1 amplitude complex of right ear of both the groups. However, no significant difference found in the latency of p1 and amplitude complex of p1-n1 of left ear but showed significant difference in latency of n1 of left ear of both the groups. There was no significant difference was found between the two ears in individual with severe to profound hearing loss. However, when the data was combined from two ears, the statistical analysis showed no significant difference between the latencies of p1 peak and n1 peak of cVEMP between the two groups. However, significant difference was found between the p1-n1 amplitude complexes between both the groups in which smaller amplitude was found for individual with severe to profound hearing loss.

Xu et al. (2016) reported that cVEMP was present in 44.4% of individual with profound sensorineural hearing loss and decreased amplitude in cVEMP response in the individuals with sensorineural hearing loss than healthy individuals. Smaller amplitude was found in individual with hearing impaired group compared to normal hearing group. This suggests that there could be abnormality in vestibular function due to similarities in both morphological and physiological between the cochlear and vestibular structures and functions (Singh et al, 2012, Zhou et al., 2009).

Ocular vestibular evoked myogenic potential

In the present study, oVEMP was present in 100% in both right and left ear of individual with normal hearing, whereas 55% and 60% in right and left ear of individual with severe to profound hearing loss respectively. There was no significant difference for latencies of n1, p1, n2 and amplitude complex of p1-n1 and p1-n2 of right ear in group 1 and group 2. However, significant difference was found between the amplitude complex of n1p1 and p1n2 of both the groups and found no significant difference between latencies of n1, p1 and n2 in left ear of group1 and group 2 .Combined data of both the ears were analyzed and found no significant difference between the latencies of n1, p1 and n2 of both the groups whereas significant difference was found for the amplitude complex of p1-n1 and p1-n2 of both the groups.

Similar finding was reported in literature that suggests more utricle dysfunction in individual with severe to profound hearing loss. Previous studies shown that oVEMP response was present in around 60-66% of the individuals with severe to profound sensorineural hearing loss Bansal et al., (2013) . Kaga, Suzuki, Marsh, & Tanaka,(1981) reported hypoactivity of the vestibulo-ocular reflexes in 12 out of 22 children (55%) with severe to profound sensorineural hearing loss based on damped rotation test. Shinjo et al.,(2007) assessed vestibular function using the damped rotation and caloric tests in 20 children with severe sensorineural hearing loss and reported that abnormalities were found in 85% of these children with caloric testing and in 30% with the rotation test. Jacot et al.,(2009) examined 224 children with profound hearing loss, using the caloric and rotation tests. They showed that 50% of the children tested have unilateral or bilateral vestibular dysfunction. Xu et al., (2016) reported to have 38.9% of response rate from oVEMP in individual with PSHL and significantly less amplitude in oVEMP response in individual with profound hearing loss compare to healthy individuals. Niu et al., (2015) reported to have affected oVEMP in 54.8% in individual with sudden sensorineural hearing loss.

Anatomically and physiologically the two parts of the inner ear viz: cochlea and the vestibular organs (semicircular canals and the otolith organs) are closely related to each other (Tribukait et al. 2014). It has also been reported that there are similarities in the vestibular hair cells and cochlear hair cells and the blood supply to both the systems (Starr et al., 2003).The cochlea and the vestibular organs share the same membranous labyrinth of the inner ear and hence the abnormality or the dysfunction of one part may lead to dysfunction of the other part too. In the present study, oVEMP responses are more absent in individual with severe to profound hearing loss than cVEMP that suggest the more utricular dysfunction associated with cochlear

pathology than saccular function in individual with severe to profound hearing loss. Tribukait et al.,(2004) reported that cochlea is more closely linked to the utricle than the any other sensory receptors of the inner ear.

It can be hypothesized that the overt manifestation as well as progression of the auditory deficits would be earlier and greater than that of the vestibular symptoms; this is expected to therefore provide more opportunities for compensation to occur for the vestibular symptoms. This is one of the reasons why most of the individuals of severe to profound sensorineural hearing loss will not report of any kind of vestibular symptoms. Therefore, it may lead to vestibular dysfunction in individual with severe to profound hearing loss.

Video head impulse test (vHIT)

In vHIT, it was found that mean VOR gain values for right and left horizontal canals, right anterior and left posterior canal for individual with hearing impaired is lesser than the individual with normal hearing.

Thus, it can be interpreted from the present study that horizontal canal of both ears are more affected in individual with severe to profound hearing loss than other canals of both the side. Caloric test and ENG was done previously to assess the functioning of horizontal canal. Magliulo et al., (2015) has found abnormal vHIT in individual with Usher syndrome who had established hearing loss and found that 53.3% had significant superior semicircular canal (SSC) deficit, 33.3% individual with ushers syndrome confirmed with horizontal SCC deficits and posterior SCC deficits was presented with 40% of individual with usher syndrome. These results indicated SCC's damage in individual with Ushers syndrome. Lin et al., (2015) reported to have abnormal vHIT that examined horizontal SCC VOR gain in 38.5% of idiopathic sudden hearing loss. Jutila, Aalto and Hirvonen (2013) measured horizontal VOR gain in children with profound hearing loss was 0.77 ± 0.26. In different pathologies had also shown the lesser VOR gain for horizontal canal which shows dysfunction of horizontal SCC. Different studies have been reported in literature to find the function of SCC's in different pathologies.

Martinez-Lopez et al. (2015) reported that vHIT responses are more affected in LARP in the individual with Meniere's disease. The authors concluded that the vHIT can be a useful tool for the diagnosis of semicircular canal dysfunction in individuals with Meniere's disease. Blödow, Pannasch, & Walther (2013) recorded VOR gain of horizontal semicircular canal in 52 individuals with vestibular neuritis using vHIT. Authors found that VOR gain was abnormal in 94.2% of individuals with vestibular neuritis. Chen et al. (2012) reported 7% of individual with beningn paroxymal positional vertigo had abnormal vHIT, 24% individual with beningn paroxymal positional vertigo had abnormal head shaking test whereas caloric test showed abnormality in 71% of individual with beningn paroxymal positional vertigo. Authors concluded that low frequency of semicircular canal frequncy tests are sensitve to find BPPV and vHIT cannot be used to evaluate semicircular function in BPPV.

Association between of cVEMP, oVEMP and vHIT:

There was association between right posterior plane of vHIT and right cVEMP whereas no association found between cVEMP, oVEMP and different planes of vHIT. There are association between left cVEMP and left lateral plane of vHIT, left cVEMP and left posterior plane of cVEMP and left oVEMP and left anterior plane of vHIT. However, there was no association was found between other test of left ear.

The research papers in vHIT have just started to appear in the literature and there are only few stuudies which have tried to correlate the vHIT test results with cVEMP and oVEMP test results in individuals with various vestibular disorders. Walther and Blödow, (2013) tested cVEMP, oVEMP and vHIT and found no association between all these tests in a group of individuals diagnosed with vestibular neuritis. Magliulo et al. (2015) also reported no association between the cVEMP, oVEMP and vHIT test in a group of individuals with vestibular neuritis. Oh et al. (2013) reported no correlation between cVEMP, oVEMP and vHIT test findings in a group of individuals with vestibular neuritis.

Lack of associations between cVEMP, oVEMP and different planes of vHIT due to the fact that cVEMP assess the function of saccule, oVEMP assess the function of utricle and vHIT assesses the function of all 6 SCC's. Also, the stimulus used for vHIT is providing head jerks to stimulate all 6 SCC's of different planes which is more natural way to stimulate the SCC's whereas for cVEMP and oVEMP high intensity acoustic stimulation is used to stimulate saccule and utricle and are more simulated condition.

Conclusions

cVEMP, oVEMP and vHIT provides information of peripheral structure of vestibular system , i.e., otolith organs and all six semicircular canals, hence these tests can be utilised to assess various vestibular pathology. Findings of the present study suggest a high prevalence of and cVEMP and vHIT response compared to the oVEMP in individuals with severe to profound hearing loss, that suggestive of more utricular dysfunction is linked with cochlear loss in individual with severe to profound hearing loss compared to saccule and semi circular canals. Previous studies also reported to have more uticular dysfunction in sensorineural hearing loss than saccule and semicircular canals. There is no association between cVEMP, oVEMP and vHIT response. This suggests that all these tests assess

function of different structure of peripheral vestibular system which is independent to each other. To conclude, abnormality was seen for both otolith organs (saccule and utricle) and semi circular canals in individual with severe to profound hearing loss, and thus, along with other audiological testing, vestibular testing should also be carried out for these individuals with severe to profound sensorineural hearing loss.

REFERENCES

Agrawal, Y., Platz, E. A., & Niparko, J. K. (2008). Prevalence of hearing loss and differences by demographic characteristics among US adults: data from the National Health and Nutrition Examination Survey, 1999-2004. Archives Internal Medicine, 168, (14), 1522- 1530.

Bansal, S., Sahni, S., & Sinha, S. K. (2013). Cervical and ocular vestibular evoked myogenic potentials in individuals with severe to profound hearing loss. Journal of Hearing Science, 3(4), 24.

Bansal, S., & Sinha, S. K. (2016). Assessment of VOR gain function and its test-retest reliability in normal hearing individuals. European Archives of Oto-Rhino-Laryngology?: Official Journal of the European Federation of Oto-Rhino-Laryngological Societies (EUFOS)?: Affiliated with the German Society for Oto-Rhino-Laryngology - Head and Neck Surgery.

Beyea, J. A., & Zeitouni, A. G. (2010). Vestibular evoked myogenic potential latencies in Meniere disease and vestibular schwannoma. Journal of Otolaryngology-Head & Neck Surgery, 39(3), 253-258.

Blödow, A., Pannasch, S., & Walther, L. E. (2013). Detection of isolated covert saccades with the video head impulse test in peripheral vestibular disorders. Auris, Nasus, Larynx, 40(4), 348-51.

Brandt,T.,& Strupp, M. (2010). Clicking the eye muscles? The diagnostic value of sound-evoked vestibular reflexes. Neurology, 75,848-849.

Brantberg K, Verrecchia L. (2012) Effectiveness of different click stimuli in diagnosing superior canal dehiscence using cervical vestibular evoked myogenic potentials. Acta Otolaryngology, 132(10),1077-83.

Carhart, R., & Jerger, J. (1959). Preferred method for clinical determination of pure-tone thresholds. Journal of Speech & Hearing Disorders.

Chen, T., Li, S., Dong, H., Lin, P., Wen, C., Cheng, Y., Ma, Y. (2012). [Analysis of the dysfunction frequency and characteristics of semicircular canal in benign paroxysmal positional vertigo]. Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi = Chinese Journal of Otorhinolaryngology Head and Neck Surgery, 47(10), 793-8.

Chihara, Y., Iwasaki, S., Murofushi, T., Yagi, M., Inoue, A., Fujimoto, C., Yamasoba, T. (2012). Clinical characteristics of inferior vestibular neuritis. Acta Oto-Laryngologica.

Colebatch, J. G., Halmagyi, G. M., & Skuse, N. F. (1994). Myogenic potentials generated by a click-evoked vestibulocollic reflex. Journal of Neurology, Neurosurgery, and Psychiatry, 57, 190-197.

Halmagyi, GM., Curthoys, IS., Colebatch, JG., Aw, ST., (2005) Vestibular responses to sound. Annals of the New York Academy of Sciences, 1039, 54-67.

Jacot, E., Van Den Abbeele, T., Debre, H. R., Wiener-Vacher, S. R., Vibert, D., Hausler, R.,& Saatci, I. (2009). Vestibular impairments pre- and post-cochlear implant in children. International Journal of Pediatric Otorhinolaryngology, 73(2), 209-217.

Jafari, Z., & Asad Malayeri, S. (2011). The effect of saccular function on static balance ability of profound hearing-impaired children. International Journal of Pediatric Otorhinolaryngology, 75, 919-924.

Jutila, T., Aalto, H., & Hirvonen, T. P. (2013). Cochlear implantation rarely alters horizontal vestibulo-ocular reflex in motorized head impulse test. Otology & Neurotology?: Official Publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology, 34(1), 48-52.

Kaga, K., Suzuki, J. I., Marsh, R. R., & Tanaka, Y. (1981). Influence of labyrinthine hypoactivity on gross motor development of infants. Annals of the New York Academy of Sciences, 374, 412-20.

Lin, Y., Gao, L., Han, L., Lu, L., Chen, Y., Zha, D., & Qiu, J. (2015). [Video head impulse test in peripheral vestibular diseases]. Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi = Chinese Journal of Otorhinolaryngology Head and Neck Surgery, 50(9), 724-8.

MacDougall, H. G., Weber, K. P., McGarvie, L. A., Halmagyi, G. M., & Curthoys, I. S. (2009). The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology, 73(14), 1134-41.

Magliulo, G., Iannella, G., Gagliardi, S., & Re, M. (2015). A 1-year follow-up study with C-VEMPs, O-VEMPs and video head impulse testing in vestibular neuritis. European Archives of Oto-Rhino-Laryngology?: Official Journal of the European Federation of Oto-Rhino-Laryngological Societies (EUFOS)?: Affiliated with the German Society for Oto-Rhino-Laryngology - Head and Neck Surgery, 272(11), 3277-81.

Manzari, L., Burgess, A. M., & Curthoys, I. S. (2012). Ocular and cervical vestibular evoked myogenic potentials in response to bone-conducted vibration in patients with probable inferior vestibular neuritis. The Journal of Laryngology & Otology.

Martinez-Lopez, M., Manrique-Huarte, R., & Perez-Fernandez, N. (2015). A Puzzle of Vestibular Physiology in a Meniere's Disease Acute Attack. Case Reports in Otolaryngology, 2015, 460757.

Murofushi, T., Shimizu, K., Takegoshi, H., & Cheng, P. W. (2001). Diagnostic value of prolonged latencies in the vestibular evoked myogenic potential. Archives of Otolaryngology--Head & Neck Surgery, 127, 1069-1072.

Oh, S.-Y., Kim, J.-S., Yang, T.-H., Shin, B.-S., & Jeong, S.-K. (2013). Cervical and ocular vestibular-evoked myogenic potentials in vestibular neuritis: comparison between air- and bone-conducted stimulation. Journal of Neurology, 260(8), 2102-9.

Rosengren, S. M., Aw, S. T., Halmagyi, G. M., Todd, N. P. M., & Colebatch, J. G. (2008). Ocular vestibular evoked myogenic potentials in superior canal dehiscence. Journal of Neurology, Neurosurgery, and Psychiatry, 79, 559-568. http://doi.org/10.1136/jnnp.2007.126730

Rosengren, S. M., & Colebatch, J. G. (2011). Ocular vestibular evoked myogenic potentials are abnormal in internuclear ophthalmoplegia. Clinical Neurophysiology, 122, 1264-1267.

Shall, M. S., & Shall, M. S. (2009). The Importance of Saccular Function to Motor Development in Children with Hearing Impairments. International Journal of Otolaryngology, 2009, 1-5.

Shinjo, Y., Jin, Y., & Kaga, K. (2007). Assessment of vestibular function of infants and children with congenital and acquired deafness using the ice-water caloric test, rotational chair test and vestibular-evoked myogenic potential recording. Acta Oto-Laryngologica, 127(7), 736-747.

Singh, S., Gupta, R. K., & Kumar, P. (2012). Vestibular evoked myogenic potentials in children with sensorineural hearing loss. International Journal of Pediatric Otorhinolaryngology, 76(9), 1308-1311.

Sinha, S.K., Singh, N.K, Barman, A., Rajeshwari, G., & Saranya, R. (2013). Involvement of peripheral vestibular nerve in individuals with auditory neuropathy. European Archives of Otorhinolaryngology. 270, 2207-2214.

Starr, A., Michalewski, H. J., Zeng, F.-G., Fujikawa-Brooks, S., Linthicum, F., Kim, C. S., Keats, B. (2003). Pathology and physiology of auditory neuropathy with a novel mutation in the MPZ gene (Tyr145->Ser). Brain?: A Journal of Neurology, 126(Pt 7), 1604-19.

Su Chiang, Young YE. Differentiating cerebellar and brainstem lesions with ocular vestibular evoked myogenic potential test. European Archeives of Otorhinolaryngology, 268, 923-30.

Todd, N., Cody, F., & Banks, J. (2000). A saccular origin of frequency tuning in myogenic vestibular evoked potentials? Implications for human responses to loud sounds. Hearing Research, 141(1-2), 180-188.

Tribukait, A., Brantberg, K., & Bergenius, J. (2004). Function of semicircular canals, utricles and saccules in deaf children. Acta Oto-Laryngologica, 124(1), 41-8.

Voelker, C. J., & Chole, R. A. (2010). Unilateral Sensorineural Hearing Loss in Adults: Etiology and Management. Seminars in Hearing, 31, 313-325.

Walther, L. E., & Blödow, A. (2013). Ocular vestibular evoked myogenic potential to air conducted sound stimulation and video head impulse test in acute vestibular neuritis. Otology & Neurotology?: Official Publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology, 34(6), 1084-9.

Xu, X.-D., Ding, C.-R., Yu, J., Han, Z., Gu, J., Gao, N.,Chi, F. L. (2016). The hidden dysfunction of otolithic organs in patients with profound sensorineural hearing loss. Hearing Research, 331, 41-46.

Zhu, Y., McPherson, J., Beatty, C., Driscoll, C., Neff, B., Eggers, S., & Shepard, N. T. (2014). Cervical VEMP Threshold Response Curve in the Identification of Meniere's Disease. Journal of the American Academy of Audiology, 25(3), 278-288.