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

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Abstract

The present study was aimed to evaluate the functioning and susceptibility of the saccule in individuals with Noise Induced Hearing Loss (NIHL) using Vestibular Evoked Myogenic Potentials (VEMPs) 30 individuals (60 ears) with normal hearing sensitivity (control group) and 30 (57 ears) individuals with NIHL (clinical group) in the age range of 25-50 years were taken. All the individuals were tested on a test battery including case history, PTA, Immittance, TEOAEs, ABR & VEMP. 2 questionnaires were administered to obtain information about history of noise exposure and presence/absence of vestibular symptoms. The results showed that VEMP in NIHL group was present in 61.4%. There was statistically significant prolongation of p13 but not for the n23 latency, reduced amplitude for both p13-n23 complex and TEOAE for the clinical group in comparison to the control group. VEMP correlated with the vestibular symptoms in 33 out of 57 ears. VEMP did not correlate with the severity of the hearing loss (HL) for both ears. However, for degree of HL from mild to severe, the frequency of presence of VEMP response decreased. The TEOAE amplitudes are highly correlated with the severity of the HL for both ears. To conclude, the two parameters of VEMP, p13 latency and p13- n23 complex amplitude could be considered to show the effect of noise on saccular system which was obtained significantly different. VEMP is expected to be affected or absent in clients with the dysfunction of the vestibular system, as in the current study, all the individuals with symptoms of "Sensation that you are turning or spinning inside" and "Nausea or vomiting" had absent VEMP responses indicating saccular involvement in NIHL. It is also evident that the cochlea is more susceptible to noise in individuals with NIHL as TEOAE was absent in most of the client with NIHL.

Introduction

Hearing is one of the most important senses in human beings. There are a multitude of factors that can affect the hearing of an individual. The most common factor which can have an adverse effect on our hearing is 'noise'. Since the industrial revolution, an increasing number of ears have been injured by noise via two ways. One is acute acoustic trauma, which is defined as a sudden change in hearing as a result of a single exposure to a sudden burst of sound. Other is the Noise-Induced Hearing Loss (NIHL) which develops slowly over a long period of time as a result of exposure to continuous or intermittent loud noise (ACOEM, 2002). 1.1 million people are estimated to be exposed to excessive noise at work and of these 1 lakh 70 thousand would suffer from significant ear damage as a direct result of noise exposure (South, 2004). Noise has both auditory and non auditory effects. Extreme noise can clearly damage hair cells in the cochlea (Rosler, 1994), the spiral ganglion cells forming the auditory portion of the eighth nerve (Nadol & Xu, 1992) and the central nervous system including the cochlear nuclei, superior olive and inferior colliculus (Kim, Leonard, Smurzynski, & Jung, 1992). Also, negative reactions (Fields, 1994), sleep disturbances

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(Pearsons, Barber, Tabachnick & Fidell, 1995) and detrimental effect on cardiovascular health (Talbott et al., 1996) have been reported resulting from noise exposure. There are battery of audiological tests for evaluating the auditory effects of noise and the early diagnosis of NIHL of which Otoacoustic Emissions (OAEs) provide objectivity and greater accuracy, complementing the behavioral audiogram in the diagnosis and monitoring of the cochlear status following noise exposure (Attias, Abrovitz, Hatib, & Nageris, 2001).

Noise exposure not only damages the cochlea, but threatens the vestibular organs too. (Oosterveld, Polman, & Schoonheyt, 1980). Oosterveld, Polman, & Schoonheyt (1982) reported that individuals with noise exposure could be disabled because of vertigo or balance disorder; an important and perhaps neglected aspect of NIHL. Similar reports of vestibular involvement leading to various vestibular symptoms in individuals exposed to noise have been studied using various test procedures for assessing the vestibular system (Barr, 1886; Chadwick, 1966; Aantaa, Virolainen & Karskela, 1977; Paparella & Mancini, 1983). Vestibular evoked myogenic potentials (VEMPs) which was first described by Bickford, Jacobson & Cody (1964), plays an important role in the vestibular test battery as a non-invasive measure of saccular function (Hall, 2006). VEMPs are mediated by a pathway that includes the saccule, macula, inferior vestibular nerve (IVN), lateral vestibular nucleus (LVN), lateral vestibulospinal tract (LSVT), and motor neurons of the ipsilateral sternocleidomastoid muscle (Halmagyi & Curthoys, 2000).The VEMP waveform consists of two components; of which only the first component (p13- n23) is generated by activation of saccular afferents (Colebatch, Halmagyi & Skuse, 1994).

VEMP has a wide clinical applicability. VEMP has been reported to be useful in the assessment of various peripheral and central vestibular disorders. In a recent study, Wang & Young (2007) reported that patients with bilateral NIHL (bilateral 4 kHz notched audiogram with hearing threshold of 4 kHz > 40 dB) may show abnormal VEMP indicating that vestibular part especially, the sacculocollic reflex pathway has also been damaged. Christina, Kumar & Bhat (2008) also observed abnormal VEMP in 82%, out of which, 36% were having absent VEMP and 46% were having abnormal VEMP, in a total of 6 subjects with noise induced hearing loss.

Need of the study

The vestibular end organs and the cochlea both utilize the same basic principle of mechano-electric transduction with the help of the sensory hair cells (Eisen & Limb, 2007). Also, the bony labyrinth is stimulated in response to high levels of occupational noise. Hence, balance system could also have negative effects secondary to long term noise exposure, along with the hearing sensitivity. The saccule has been reported to be the thinnest membrane (0.015mm) after Reissener's Membrane (0.014mm). Also, saccule can withstand much lesser force (0.57gf/mm) before breakage as against the Reissener's membrane which can withstand a force of 0.84gf/mm (Tetsuo, Nobukazu, & Terufumi, 1990). Furthermore, the distance of the utricle and saccule from the stapes are 0.65mm and 0.4mm respectively which in turn adds to the probability of the balance system getting affected due to noise. It is also reported that saccular maculae among the vestibular structures, are the most sensitive structure to

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

The possible vestibular involvement in patients with NIHL is relatively new and there is dearth of information regarding the same. Individuals exposed to noise either for short or long duration might exhibit vestibular symptoms. VEMP recording might help to unfold the saccular involvement in individuals who are exposed to noise with or without any vestibular symptoms.

Aims of the study

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

Method

Subjects: Two groups of subjects were taken in the age range of 25–50 years. The control group consisted of 30 individuals (60 ears) with normal hearing sensitivity with no history of exposure to noise (mean age= 38.66 years). The clinical group consisted of 30 individuals (57 ears) with NIHL. The clinical group was further subdivided into two groups based on the vestibular symptoms;

Group I: 15 subjects (28 ears) with a mean age of 39.33 years. All the individuals in this group exhibited at least one of the vestibular symptoms that were given in the Dizziness questionnaire. The duration of noise exposure had a mean of 20.93 ears.

Group II: 15 subjects (29 ears) with a mean age of 42.40 years. No individuals in this group exhibited any of the vestibular symptoms. The duration of noise exposure had a mean of 19.47 ears.

Selection criteria

Control group: All the subjects had hearing sensitivity within 15 dBHL at octave and mid octave frequencies from 250 Hz to 8000 Hz with 'A' type tympanogram and normal acoustic reflexes in both the ears. The uncomfortable levels (UCL) for speech for all the subjects were greater than 95 dB HL with good speech identification (SI) scores (≥ 80%).

Clinical group

The subjects were having either normal hearing sensitivity or sensorineural hearing loss with air bone gap not exceeding 10 dB HL with air conduction notch between 3- 6 kHz with any degree of hearing loss. They had noise exposure for duration of 8hrs per day, at least for more than 2 yrs. Immitance measurements showed 'A' type tympanogram with presence/elevated or absence of ipsilateral and contralateral acoustic reflexes in both the ears. TEOAEs showed either normal (in individuals with 3-6 kHz notch), abnormal or absent responses (in individuals having hearing loss indicating cochlear pathology). None of them reported to have hypo/hypertension or spondylitis and did not have any evidence of space occupying lesion (decided based on auditory brainstem response results and /or neurological reports). The uncomfortable levels (UCL) for speech for all the subjects were greater than 95 dB HL with good speech identification scores of 80% or proportionate to the hearing loss.

Instrumentation

A calibrated 2-channel diagnostic MADSEN ITERA audiometer was used to estimate the puretone thresholds (for both air conduction and bone conduction), SI scores and UCL for speech. A calibrated immitance meter GSI- Tympstar was used for both tympanometry and acoustic reflexometry. A calibrated OAE system ILO-V6 was used for the measurement of Transient Evoked Otoacoustic Emission (TEOAE). IHS smart EP version 3.94 US Bez (Intelligent hearing system, Florida, USA) instrument was used to record and analyse VEMP and Auditory Brainstem Response (ABR).

Procedure

1) A detailed case history about history of noise exposure was taken for all the individuals in the clinical group by administering the questionnaire developed by Tharmar (1990). To obtain information about the vestibular symptoms, the II section of dizziness questionnaire developed at Maryland hearing and balance center was used.
2) Puretone thresholds were obtained between 250 Hz to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction at all the octaves and mid octave frequencies, using the Modified Hughson and Westlake procedure (Carhart & Jerger, 1959). PTA2 (average of 1 kHz, 2 kHz and 4 kHz) was also calculated to account for hearing sensitivity at high frequencies. This was considered for the statistical analysis.
3) The SI scores were obtained at 40 dB HL above the speech recognition threshold using monosyllable list developed by Vandana (1998).
4) The UCLs were determined by presenting the running speech through the headphones (TDH-39) at different intensities using ascending method.
5) Immittance audiometry was carried out with a low probe tone frequency of 226 Hz. The ipsilateral and contralateral acoustic reflex thresholds were measured for 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz tones.
6) ABR testing was carried out to rule out any space occupying lesions using the Neuro-diagnostic ABR test protocol. The Subjects who had both the absolute and the inter-peak

latencies within the normal range, with good waveform morphology for both low and high repetition rates were considered as devoid of any space occupying lesions.

7) The OAEs evoked by click trains presented at 84±3 dB pe SPL for the non linear clicks were recorded using an appropriate sized probe tip. The response was acquired using the averaging method. Responses were accepted with a SNR of +6 dB and response reproducibility of ≥ 80%.

8) The VEMP was recorded by instructing the subjects to sit straight and turn their head to the opposite side of the ear in which the stimulus was presented, so as to activate the ipsilateral Sternocleidomastiod (SCM) muscle and were asked to maintain the same throughout the test run. They were also instructed to avoid any extraneous movements of head, neck and jaw to elude muscle artifacts. While recording the VEMP, the tonic EMG level was maintained for each of the subject between 100 to 200 micro volts. A visual feedback which was available in the instrument was provided to each of the subject to monitor tonic EMG level of SCM muscle. The protocol proposed by Wang & Young (2007) was used in the present study to record the VEMP which is given in Table 1.

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

Table 1: Parameters Used to Record VEMP

Results and Discussion

A.VEMP results in the control and the clinical group

Control group: Out of the 60 ears, the VEMP response was present in 51 ears while it was absent in 9 ears. So the response rate for the VEMP was 85%. The overall response rate is consistent with the studies by Townsend & Cody (1971) and Vijayashankar (2008).

The mean, standard deviation (SD) and paired t test results for p13, n23 latency and p13- n23 complex amplitude obtained in individuals with normal hearing were calculated and the results are outlined in Table 2.

Table 2: Mean, Standard Deviation (SD) and t-values with Level of Significance of p13, n23 Latency and p13- n23 Complex Amplitude of VEMP in the Control Group.

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

From the Table 2, it can be inferred that the mean latencies of p13 and n23 was longer for the right ear as compared to the left ear. The variability for the p13 latency measure was higher for the right ear, while for the n23 latency, it was higher for the left ear. Overall, the variability for the n23 latency was greater as compared to the p13 latency. For the p13- n23 complex amplitude, the mean value was larger for the right ear than the left ear while the variability was higher for the left ear. Paired t test results indicated no significant difference between right and left ears for the p13, n23 latency and amplitude of p13- n23 complex. The mean values of p13 and n23 latencies of VEMP response in the present study are almost in agreement with the studies on VEMP by various authors such as Akin, Murnane & Proffitt (2003), Kumar (2006) and Vijayashankar (2008). The amplitude was in accordance with Vijayashankar (2008), he reported mean p13-n23 complex amplitude value around 50 µV and SD of about 25 µV. The amplitude in the control group is slightly greater and the variation is less in the present study as compared to the study by Vijayashankar (2008). The reason for this could be that the EMG level maintained in Vijayashankar (2008) was lower (30-50 micro volts) than the present study (controlled in the range of 100-200 micro volts). It is possible that the EMG level greater than 50 micro volts would have raised the mean amplitude value of p13-n23.

Clinical group: The VEMP response was present in 35 ears and was absent in 22 ears. So, the response rate for the VEMP was 61.4%.

Response patterns of VEMP latency and amplitude: For the p13 latency, 54.29% had normal latency, 40% had prolonged latency and 5.71% had shortened latency. For the n23 latency, 57.14% had normal latency, 34.29% had prolonged latency and 8.57% had shortened latency. The response patterns for amplitude measure showed that 48.57% had normal amplitude while 51.43% had reduced amplitude. The results of the present study are in consonance with Christiana, Kumar and Bhat (2008). They reported that VEMP was abnormal or absent in 67% and normal in 36.4% ears out of 55 NIHL ears evaluated. Out of the 67% ears, VEMP was absent in 45.7% ears. The latency was prolonged and the peak to peak amplitude was reduced in 54.3% ears. They concluded that the possibility of vestibular dysfunction, especially the saccule pathway is high in individuals with NIHL and that VEMP

can be employed in these individuals to assess sacculo-collic reflex. Wang & Young (2007) reported abnormal VEMP responses in 50% of the individuals with NIHL, which included absent VEMPs in 8 and delayed VEMPs in 3 subjects. The absence of VEMP reflects a lesion affecting the sacculocollic reflex pathway, whereas the delayed VEMP latencies are indicative of a retro-labyrinthine or brainstem lesion, especially in the vestibule-spinal tract (Wang & Young, 2006). There are discrepancies seen in the quantitative measures of each of the considered parameter, and this can be attributed to the number of subjects, years of exposure to noise and other recording parameters adopted in different studies.

The mean and the SD for p13, n23 latency and the p13- n23 complex amplitude and the paired t test results obtained in individuals with NIHL was calculated and the results are tabulated in Table 3.

Table 3: Mean, SD and t- values with Level of Significance of p13, n23 Latency and p13-n23 Complex Amplitude in the clinical group.

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

From the Table 3, it can be speculated that the mean latency value and the variability of p13 and p13- n23 complex amplitude was larger for the right ear as compared to the left ear. For the n23 latency, the mean value was smaller for the right ear but the variability was higher for the left ear. Paired t test results indicated that there was statistically no significant difference between right and left ears for the p13, n23 latency and p13- n23 complex amplitude.

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

Comparison of p13 latency: The mean and the SD for p13 latency in ms for both the groups are depicted in Figure 1.

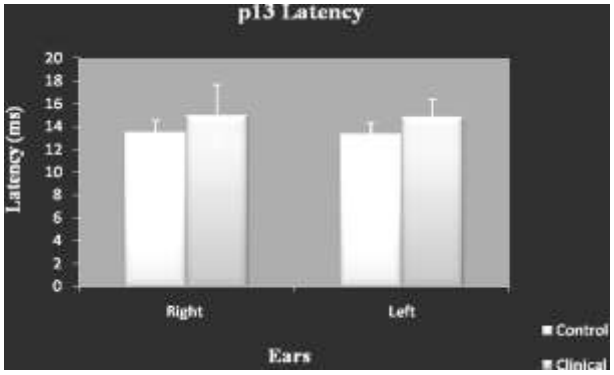

Figure 1: Mean and SD of p13 latency for right and left ears obtained in both the groups.

It can be seen from the Figure 1 that the p13 latency value obtained for the control group is shorter than the clinical group for both right ear and left ear. Mixed ANOVA results revealed that there was a statistically significant difference in p13 latency values obtained between the control and the clinical group [$F (1, 33) = 14.08$, $p < 0.05$]. For within subjects, there was neither ear effect [$F (1, 33) = 0.15$, $p > 0.05$] nor the interaction effect between the group and ear [$F (1, 33) = 0.00$, $p > 0.05$].

Comparison of n23 latency: The mean and the SD for n23 latency for both groups are shown in Figure 2.

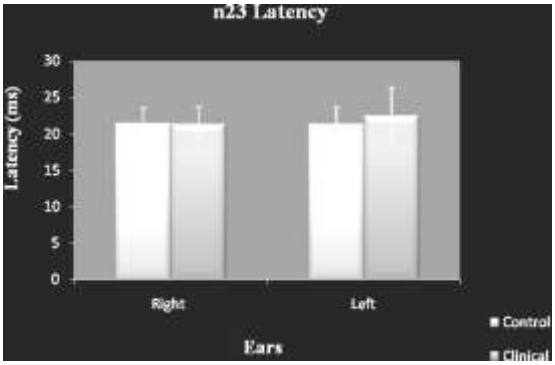

Figure 2: Mean and SD of n23 latency for both the control group and the clinical group.

It can be seen from the Figure 2 that the n23 latency value for the clinical group is longer than the control group for both the right and left ear. Mixed ANOVA results revealed that there was statistically no significant difference for n23 latency values between the control and the clinical group [$F (1, 33) = 2.10$, $p > 0.05$]. For within subjects there was neither ear effect [$F (1, 33) = 0.01$, $p > 0.05$] nor the interaction effect between the group and ear [$F (1, 33) = 0.06$, $p > 0.05$]. The results of the present study are in close agreement with the study by Wang and Young (2007) and Christiana, Kumar & Bhat (2008). Wang and Young (2007) reported specific prolongation of p13 latency, but Christiana, Kumar & Bhat (2008) have reported prolongation for both the peak latencies. Another speculation of the p13 latency being prolonged compared to n23 latency being within normal limits may be reasoned due to the SD value. The SD of n23 was greater than that of p13, resulting in a wider normal range of n23 than p13. Also, the literature on the response consistency of VEMP which was reviewed by Ferber, Dubreuil, & Duclaux (1999) based on the studies done by Townsend & Cody (1971), and others suggest that the consistency is more for p13 and less for n23 of VEMP response.

Comparison of p13- n23 complex amplitude for both the control and the clinical group: The mean and the SD for p13- n23 complex amplitude for both the groups are presented in Figure 3.

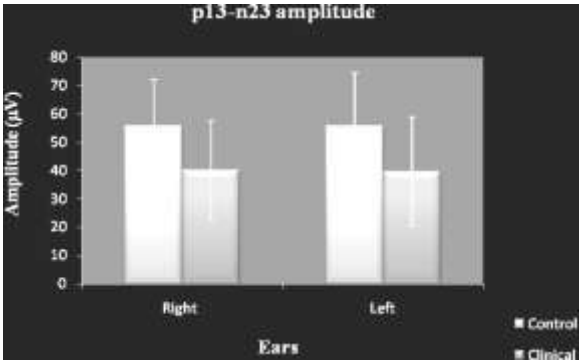

Figure 3: Mean and SD of p13- n23 complex amplitude for both the control group and the clinical group.

It can be seen from the Figure 3 that the p13- n23 complex amplitude for the clinical group in both the right and the left ear is smaller than the control group. Mixed ANOVA results revealed that there was a statistically significant difference in p13- n23 complex amplitude values between the control and the clinical group [$F (1, 33) = 8.60$, $p < 0.05$]. For within subjects, there was neither ear effect [$F (1, 33) = 0.01$, $p > 0.05$] nor the interaction effect between the group and ear [$F (1, 33) = 0.00$, $p > 0.05$]. Christiana, Kumar and Bhat (2008) reported the amplitude being reduced in 19 ears accounting for 34.6% of the abnormal responses. The percentage of the reduced amplitude was higher (51.43%) in the present study which may be because of the difference in the duration as well as the intensity of noise exposure in the study group in the two studies. Also, the variation in the amplitude measure may be due to the mean level of the electromyographic activity (Colebatch, Halmagyi, & Skuse, 1994). It has also been reported in the literature that there are variations in VEMP amplitudes, from a few µV to several 100µV, depending on the muscle tension and the intensity of stimuli (Cheng & Murofushi, 2001a, 2001b; Ochi, Ohashi, & Nishino, 2001). Hence, it could be concluded that although reduced VEMP amplitude does indicate abnormality, it cannot be conclusive as long as the intensity of the signal and more importantly the muscle tension is controlled.

Because of the unequal sample size owing to the absence of response in many of the ears considered in the clinical group, Mann Whitney test was done for the group comparisons of p13, n23 latency and p13- n23 complex amplitude between the two groups. The result is in accordance with the mixed ANOVA results.

C. TEOAE response in the Control and the Clinical group

Control group: The TEOAE being one of the criteria for the selection of subjects in the control group, the response rate for TEOAE was 100%. It was observed that the mean amplitude value for the right ear was larger than the left ear. Also, the variability was higher for the right ear as compared to the left ear. Paired t test indicated that there was no significant difference between right and left ears ($t = 2.73$).

This is in consonance with the literature where prevalence of TEOAE response is reported to be 96%-100% in individuals with normal hearing sensitivity (Probst, Lonsbury, Martin & Coats, 1987). They also reported that right ear OAE's were much greater than the

left ear OAE's. Moulin, Collet, Veuillet and Morgan (1993) reported right ear OAE's being much greater than the left ear OAE's.

Clinical group: The TEOAE was present in 20 ears while it was absent in 37 ears. So, the response rate for the VEMP was 35.09%. It was seen that the mean amplitude value for the right ear was larger than the left ear; whereas the variability for the left ear was higher compared to the right ear. Paired t test results showed no significant difference between right and left ears (t= 1.05). The findings of the study are similar to as reported by Shupak et al. (2007). They reported of reduced TEOAE amplitudes in individuals during the first 2 years of occupational noise exposure. Kowalska and Kotylo (2007) reported that changes in OAE's exactly follow the changes in audiogram related to noise exposure and that patients with NIHL show amplitude reduction and or complete absence of OAE's. They stated that the rationale for using OAE's in patients with NIHL includes the clinical aspect that is confirmation of cochlear lesion.

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

Mixed ANOVA was done to evaluate the group effects, ear effects and interaction between the group and ear effect for TEOAE amplitude. The mean and the SD for TEOAE amplitude for both the control and the clinical group is shown in figure 4.

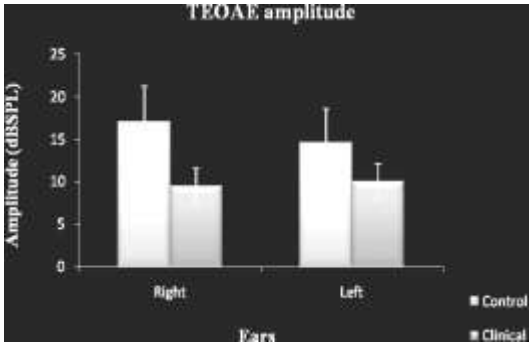

Figure 4: Mean and SD of the TEOAE amplitude for both the control group and the clinical group.

It can be evident from the Figure 4 that the TEOAE amplitude is lesser for the clinical group than the control group for both the right and the left ear. The variability is less for the clinical group than for the control group. Mixed ANOVA results revealed that there was a statistically significant difference in TEOAE amplitude values between the control and the clinical group [$F(1, 35) = 23.22$, $p < 0.05$]. For within subjects, there was neither ear effect [$F(1, 35) = 0.65$, $p > 0.05$] nor the interaction effect between the group and ear [$F(1, 35) = 3.04$, $p > 0.05$]. Mann Whitney t test results are in accordance with the mixed ANOVA results.

E. Comparison of TEAOE and VEMP responses in the Clinical group:

To evaluate the susceptibility of the cochlea versus the saccule, the VEMP responses were compared with the TEOAE responses. This was done using the cross tabulations wherein comparison of the frequency of the presence or the absence of the responses for both

VEMP and TEOAE were made. The frequency of presence and absence of VEMP and TEOAE responses in the clinical group are tabulated in table 4.

Table 4: Frequency of presence and absence of VEMP and TEOAE responses in the clinical group.

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

It can be observed from the Table 4 that the condition in which the TEOAE being absent with VEMP present was more prevalent, followed by both TEOAE and VEMP absent whereas, ears with both TEOAE and VEMP present had intermediate occurrence. It is also evident that only a small percentage has TEOAE present with VEMP being absent.

From the above findings, it can be concluded that it is the cochlea which is more susceptible to noise exposure compared to the saccular part of the vestibular system. This is well supported by the anatomical positioning of the cochlea and saccule wherein the cochlea is at more proximity to the stapes than the saccule. When the ear is exposed to noise, cochlea will be more susceptible. Hence, the outer hair cells of the cochlea would be affected before the macula of the saccule resulting in abnormal TEOAE's prior to abnormal VEMP responses. Ceranic (2007) stated that owing to mechanical force of noise exposure, the most extensive morphological changes are expected to be in the cochlea. Wang & Young (2007) reported abnormal VEMP responses in NIHL subjects and explained that the mechanism of NIHL can be classified either as direct mechanical injury or metabolic damage to the organ of Corti. Talasaka & Schacht (2007) reported that the direct mechanical damage is mostly caused due to chronic noise exposure. The extent of noise effect on cochlear blood flow appears to be heavily influenced by the duration and intensity of the noise exposure (Lamm & Arnold, 2000). Although the cochlea receives its blood supply mainly from the common cochlear artery, the saccule is supplied by anterior and posterior vestibular arteries; all these arteries originate from the labyrinthine artery. Therefore, as the duration and intensity of the noise exposure increases, there is reduction in blood flow which leads to permanent hearing threshold shifts and abnormal VEMP responses.

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

To compare the VEMP responses with the presence or absence of any vestibular symptoms, cross tabulations were done. Here the frequency of the presence or the absence of VEMP was correlated with the presence or absence of vestibular symptoms. The Table 5 depicts the number of individuals exhibiting the vestibular symptoms in correlation with the absence of the VEMP responses. The subjects exhibited either one or more than one

symptom listed below. Two symptoms (Tullio phenomenon and walking in dark) which were not present in the questionnaire are listed in the table as it was reported by the subjects.

Table 5: Vestibular symptoms and the VEMP response in the clinical group.

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

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

Table 6: Frequency of presence or absence of the vestibular symptoms and the VEMP responses in the clinical group.

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

It is evident from the table 6 that the condition in which vestibular symptom was absent with VEMP response being present is most prevalent. Also the percentage of occurrence of the vestibular symptom being present with VEMP absent is higher. So, it can be inferred that out of 57 ears tested in the clinical group VEMP correlated with vestibular symptoms in 33 ears (57.89%).

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

G. Correlation between VEMP responses with the degree of hearing loss in the clinical group: Pearson's correlation analysis was done to evaluate the correlation between the VEMP & the degree of hearing loss for the clinical group. The results of the correlation analysis for the latency and amplitude measures of VEMP for the clinical group are outlined in Table 7.

Table 7: *r* Value and Significance Level for p13 , n23 Latency and p13- n23 Amplitude w.r.t Degree of Hearing Loss for the Clinical Group.

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

It can be seen from the Table 7 that both the latency as well as amplitude measures are not correlated with the severity of the hearing loss for both right and left ear. The VEMP responses across different degrees of hearing loss are tabulated in Table 7.

Table 8: VEMP responses across different degrees of hearing loss.

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

It is evident from the table 8 that in ears with normal hearing with 3-6 kHz notch and those with minimal degree of hearing loss showed equal percentages of presence and absence of VEMP responses. For degree of hearing from mild to severe loss, the frequency of presence of VEMP response decreased and occurrence of absence of response increased and for the severe degree of hearing loss none of the ears showed presence of VEMP. The results of the present study revealed that the degree of hearing loss did not correlate with the VEMP results. Similar findings have been reported by Hsu, et al., (2008) who assessed the saccular functioning in guinea pigs that were exposed to noise and concluded that the saccule can exhibit temporary or permanent functional loss. Wang, Hsu and Young (2006) reported that VEMP test may provide another clue for assessing the hearing outcome. He concluded that VEMPs in patients after acute acoustic trauma showed absent or delayed VEMP responses which indicate poor prognosis with respect to hearing improvement. Young and Cheng (2007) reported more absent VEMP responses with increasing degree of hearing loss in subjects with NIHL. In the present study though there was no correlation between the VEMP responses and degree of hearing loss, the trend of response suggested that as the degree of hearing loss increased the frequency of presence of VEMP response decreased and occurrence of absence of VEMP response increased and for the severe degree of hearing loss none of the ears showed presence of VEMP. Wang and Young (2007) reported that in patients who were exposed to noise with bilateral 4 kHz notched audiogram and hearing threshold of 4 kHz $\geq$ 40 dB showed abnormal (absent or delayed) VEMPs, indicating that the vestibular part, especially the sacculocollic reflex pathway, has also been damaged. Hara and Kimura (1993) attributed the abnormal VEMP findings to the differential sensitivity (possibly because of membrana limitans) of cochlea and saccule from that of other vestibular structures (utricle and saccule). It can be concluded that in general, VEMP does not correlate with degree of hearing loss, but in cases of noise exposure (acoustic trauma and chronic noise exposure) higher degree of hearing loss may affect the VEMP response and thus may be indicative of saccular involvement.

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

Pearson's correlation analysis was done to evaluate the correlation between TEOAE and the degree of hearing loss for the clinical group. The results of the correlation analysis for the TEOAE amplitude measures for the clinical group are outlined in Table 8.

Table 9: r - Value and significance level for TEOAE amplitude for the clinical group.

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

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

It can be seen from the table 9 that the TEOAE amplitude is highly correlated with the severity of the hearing loss for both right and left ear. The TEOAE responses across the different degrees of hearing loss are tabulated in Table 9.

Table 10: TEOAE amplitude responses across different degrees of hearing loss

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

It can be seen from Table 10 that the percentage of presence of TEOAE response reduced as the degree of hearing loss increased. Also, it can be observed that from moderate degree of hearing loss, there was absence of TEOAE response. Findings of the present study are in consonance with literature. Probst, LonsBury, Martin and Coats (1987) demonstrated that noise induced high frequency hearing loss was associated with a reduction in the number of prominent peaks in the spectra of TEOAE's and that TEOAE's were absent for hearing loss above 25-30 dB. Desai, Reed, Cheyne, Richards and Prasher (1999) reported that in 56% of the subjects with NIHL, TEOAEs were absent as compared to controls (0%). They concluded that the reduction in incidence of OAEs in the noise exposed group may be associated with sensory cell damage to localized cochlear regions sub-serving specific frequencies. From the above it can be concluded that noise exposure have severe effect on the OHC's and that TEOAE's are very sensitive to any damage to the OHC's. Hence, the strong correlation between TEOAE and degree of hearing loss is rightly justified.

Conclusion

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

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