Video Head Impulse Test findings in Patients with Chronic Noise Exposure

Original Article

Manar S. Elewa[1], Ebtessam H. Nada[2], Nadia M. Elnabtity[2]

Audio-Vestibular Medical Unit, ENT Department, [1]AlAhrar Teaching Hospital, [2]Faculty of Medicine, Zagazig University, Sharkia, Egypt.

ABSTRACT

Objective: There has been raising evidence that long-term noise exposure above a certain level has a harmful impact on health. It is known that temporary or permanent threshold shifts in hearing exist after loud noise exposure. The vestibular system can also be activated and therefore disturbed the equilibrium mechanism. Consequently, this study aimed to assess the effect of noise on the vestibular system in patients with chronic noise exposure using a video head impulse test (vHIT).
Patients and Methods: The present study included 34 subjects. They divided into two groups: the study group consisted of 17 patients with chronic noise exposure and the control group included healthy subjects with normal peripheral hearing sensitivity. Basic audiological evaluation and vHIT were conducted on all subjects in the study.
Results: There was a statistically significant difference in vHIT lateral canal gain between the study and control groups. A statistically significant negative correlation between duration of noise exposure and lateral canal gain was also present. In addition, there was a negative correlation between lateral canal gain and pure tone thresholds at 2,3,4,6&8KHz frequencies.
Conclusion: vHIT is quick and easily applicable in evaluating the vestibular function in patients with prolonged noise exposure.

Key Words: Chronic noise exposure, vestibular function, video head impulse test, VOR.

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Corresponding Author: Manar Salah Elewa, MSc, Audio-Vestibular Medicine, ENT Department, AlAhrar Teaching Hospital, Faculty of Medicine, Zagazig University, Zagazig, Egypt, **Tel.:** 01005547402, **E-mail**: hooradham2016@gmail.com

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INTRODUCTION

The vHIT has recently been used to assess the vestibular function by evaluating the vestibulo-ocular reflex (VOR)[1]. It is a more physiologic test of semicircular canals that used to assess the high-frequency angular VOR[2]. vHIT is a diagnostic method that measures the deficit in VOR by recording eye and head velocity response to brief, unpredictable and passive head rotations, which are called head impulses[3].

VOR is the tool that enables normal individuals to maintain a steady visual image in presence of a moving visual target or moving individual but patients with deficient VOR cannot keep up with high-velocity head turns and generate "catch-up" saccades after head impulses toward the damaged side[4]. The evaluation of the result of vHIT depends on the analysis of both VOR gain and refixation saccades[5].

The vestibular system is highly responsive to head rotation, movement and alterations in orientation with respect to gravity. However, since the vestibular end organs have the same fluid system as the auditory end organs, strong acoustic waves also harm them[6]. Acoustic vestibular system activation occurs not only in pathological situations in which fenestration or canal dehiscence affects the bony canal but also in healthy individuals and in laboratory animals with intact labyrinths[7].

As the cochlea and vestibular receptors, the semicircular canals and otolith organs have close anatomical relationships and phylogenetic similarities. Thus, alterations in the hydrostatic pressure induced in any way (e.g. sound) in one part of these fluid channels can spread to the other parts, because the fluids are incompressible[8] Bekesy *et al*.[9] reported that the initiation and cessation of an intense sound produced a stream of fluid not only in the cochlea but also in the canals. Therefore, many patients with hearing loss may develop vestibular symptoms[10].

The advantages of vHIT include that it is easily applicable, fast, practical, and it can individually evaluate all semicircular canals. For these reasons, it can be used for screening of the defect in the vestibular function[11]. Therefore, Yilmaz *et al*.[11] used vHIT to evaluate vestibular function in patients with noise exposure and reported a significant loss of capacity for VOR gain in patients with noise-induced hearing loss.

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AIM OF THE WORK:

The objective of this study was to assess the effect of noise on the vestibular system using vHIT in patients with chronic noise exposure.

PATIENTS AND METHODS:

1.1 Subjects:

This case-control observational study was carried out in the audio-vestibular unit, ENT department, Faculty of Medicine, Zagazig University Hospitals. This study was approved by the Research Ethics Committee. Approval Code is 4997. Consents were obtained from all participants after an explanation of the test procedures. The sample size was calculated to be 34 subjects of both genders. They were divided into two groups (study and control groups).

Study group: consisted of 17 subjects with an age range from (30-55) with a history of chronic industrial noise exposure (workers of a textile factory). They had bilateral symmetrical sensory neural hearing loss ranged from mild to moderately severe, presented as ‘‘notching’’ of the audiogram at 3, 4 or 6 kHz. The duration of noise exposure ranged from 10 to 36 years. The diagnostic criteria of Noise-induced hearing loss (NIHL) were based on the American College of Occupational and Environmental Medicine (ACOEM, 2014) guidelines[12]. Exclusion criteria: a history of ear infections, head injuries, problems with neurology or general health, history of hearing impairment in the family, acute acoustic trauma or blast injury.

Control group: included 17 healthy adults with an age range from (25-50). They had normal peripheral hearing sensitivity and normal middle ear function. They hadn't any history of exposure to noise or any systemic disease affecting balance {e.g. Neurological conditions, diabetes mellitus, hypertension, etc.}. Both control and study groups were age and gender matched.

1.2 Methodology

A-Basic audiological assessment:

All cases were clinically examined (otoscopic examination). Tympanometry was done in all cases using Tympanometer Ampalid 724 (Amplifon, Italy), only patients with normal middle ear pressure were involved in this study. Audiometric assessment was done by standard pure-tone audiometry (PTA) using Audiometer Orbiter 922 (GN Otometrics, Denmark), air and bone conduction for both ears were performed from250 Hz up to 8000 Hz. Speech audiometry included speech reception threshold (SRT) and word discrimination testing (WD %) was also performed.

B-Video head impulse test

The vHIT was performed using an EYE SEECAM vHIT from Interacoustics. Recordings were obtained for each of the six semicircular canals in all patients [horizontal, left anterior right posterior (LARP), right anterior left posterior (RALP)].

To assess the horizontal SCCs, the rotation of the head was with a small angle (10–20) within the horizontal plane to the left and right direction. To assess the LARP and RALP SCCs, the head was positioned 40 degrees relative to the trunk and rotated either downward or upward to impulse. The head impulse in each plane and direction has performed a minimum of five times.

Measured parameters in vHIT:

***Gain:** it reflects the ratio between the velocity of the eye and the velocity of the head. Gain >0.80 for horizontal SCCs and >0.75 for posterior and anterior SCCs are considered normal results for the control group. vHIT was considered abnormal if reduced VOR gain was present in at least one canal in addition to the presence of saccade.

***Catch-Up Saccade:** it is the reposition of the eyes on the target. Two types of saccades may occur which included overt and covert saccades. Overt saccades are the catch-up saccades, which arise after head impulses while; covert saccades are catch-up saccades that occur during head impulses[13].

Statistical Analysis:

The IBM computer used the SPSS (a statistical program for social science) to analyze data as follows: quantitative variables discribtion as mean, standard deviation. Qualitative variablesdescription as number and percentage. In parametric data, the quantitative variable was compared using an independent t-test. Pearson correlation test was used to rank variousvariables in positive or inverse manner [r= (-1.0) - (1.0)].

RESULTS:

The study group included 17 patients (1 female and 16 male). The mean age was 46.7±7.9 years and the mean noise exposure duration was 22.1±8.8 years. The control group included 17 subjects (5 female and 12 male) and their mean age was 45.1±8.1 years. There was no statistically significant difference between both groups as regards age and gender. The commonest clinical symptoms in the study group were tinnitus (76.4%) followed by hearing loss (52.9%) and lastly vertigo (29.4%) (Table 1).

The number and percentage of patients with vHIT abnormalities in the study group were calculated and

revealed that 8 (47%) of 17 patients with reduced gain in the lateral canal (3 patients with right canal, 2 patients with left canal and 3 patients with bilateral canal deficit). One of the patients (5.9%), how had reduced gain in the left lateral canal also had a reduced gain in the left posterior canal (left posterior and left lateral canal deficit in the same patient). No abnormalities were detected in the anterior canal. Consequently, the total canal deficit was detected in 8 (47%) of 17 patients. Concerning saccades, there was 8 patients (47%) with refixation saccade (6 patients with covert saccade and 2 patients with overt saccade) (Table 2). Patients with overt saccade had the lowest gain in comparison to other patients. Moreover, there was a statistically significant difference between the study and control groups in the right and left lateral canal gain with no significant difference in posterior and anterior canal gain (Table 3).

There was a statistically significant negative correlation between duration of noise exposure with lateral canal gain on both sides. But regarding anterior and posterior gain, there was no correlation with the duration of noise exposure (Table 4). As regards the correlation between vHIT lateral canal gain and pure tone thresholds, there was a negative correlation between lateral canal gain on both sides and pure tone thresholds at 2, 3, 4, 6 & 8KHz frequencies (Table 5).

Table 1: Clinical symptoms in patients with chronic noise exposure

Symptoms	Cases (n=17)	%
Tinnitus		
• Present	13	76.4%
• Absent	4	23.6%
Hearing loss		
• Present	9	52.9%
• Absent	8	47.1%
Imbalance		
• Present	5	29.4%
• Absent	12	70.6%

Table 2: vHIT abnormalities in patients with chronic noise exposure

	Unilateral		Bilateral	Total	
	Rt	Lt		Ears	Subjects %
Lateral canal	3	2	3	11	8 (47%)
Anterior canal	0	0	0	0	0 (zero%)
Post canal	0	1	0	1	1 (5.8%)
Covert saccade	2	2	2	8	6 (35%)
Overt saccade	1	0	1	3	2 (11,7%)
Total canal deficit	3	3	3	12	8 (47%)

Table 3: vHIT canal gain in study and control groups:

Vhit	Study mean ± SD Range	Control mean ± SD Range	t- test	*p-value*
Rt lateral gain	0.83±0.17 (0.47-.0.9)	0.98±0.12 (0.81-1.1)	2.9	0.005*
Rt anterior gain	0.97±0.11 (0.76-1.1)	0.99±0.18 (0.76-1.12)	0.39	0.69
Rt posterior gain	0.98±0.19 (0.76-0.99)	1.03±0.12 (0.77-1.06)	0.9	0.3
Lt lateral gain	0.9±0.2 (0.48-1)	1.1±0.1 (0.81-1.18)	3.6	0.008*
Lt anterior gain	1 ±0.19 (0.76-1.1)	1.07±0.17 (0.76-1.17)	1.1	0.2
Lt posterior gain	0.93±0.16 (0.5-0.98)	1.04±0.17 (0.76-1.07)	1.9	0.06

Table 4: Correlation between duration of noise exposure and vHIT canal gainin patients with chronic noise exposure:

Variable	Duration of noise exposure		
	r^	P	SIG
Rt lateral gain	-0.49	0.04*	S
Rt anterior gain	-0.37	>0.05	NS
Rt posterior gain	-0.3	>0.05	NS
Lt lateral gain	-0.5	0.04*	S
Lt anterior gain	-0.09	>0.05	NS
Lt posterior gain	-0.3	>0.05	NS

Table 5: Correlation between vHIT lateral canal gain and pure tone thresholds in patients with chronic noise exposure:

Variable		Rt lateral gain			Lt Lateral gain		
		r^	P	SIG	r^	P	SIG
250 (HZ)	Rt	-0.07	>0.05	NS	-0.08	>0.05	NS
	Lt	-0.04	>0.05	NS	-0.03	>0.05	NS
500 (HZ)	Rt	-0.3	>0.05	NS	-0.2	>0.05	NS
	Lt	-0.1	>0.05	NS	-0.06	>0.05	NS
1000 (HZ)	Rt	-0.06	>0.05	NS	-0.1	>0.05	NS
	Lt	-0.09	>0.05	NS	-0.05	>0.05	NS
2000 (HZ)	Rt	-0.69	0.002*	S	-0.54	0.02*	S
	Lt	-0.66	0.004*	S	-0.5	0.04*	S
3000 (HZ)	Rt	-0.5	0.04*	S	-0.49	0.04*	S
	Lt	-0.7	0.001**	S	-0.69	0.002*	S
4000 (HZ)	Rt	-0.7	0.001*	S	-0.5	0.04*	S
	Lt	-0.66	0.004*	S	-0.49	0.04*	S
6000 (HZ)	Rt	-0.49	0.04*	S	-0.69	0.002*	S
	Lt	-0.5	0.04*	S	-0.7	0.001**	S
8000 (HZ)	Rt	-0.7	0.001**	S	-0.54	0.02*	S
	Lt	-0.7	0.001**	S	-0.5	0.04*	S

DISCUSSION

Noise exposure has negative consequences on hearing and balance mechanisms[14]. Industrial workers, especially those who have NIHL, frequently suffer from balance disorders like dizziness, spontaneous nystagmus[15,16], oscillopsia, postural instability, and/or motion intolerance. NIHL is characterized by a decrease in hearing sensitivity at or near 4 kHz (noise-notch or audiometric notch) and this notch acts as a biomarker for noise-related cochlear damage[17].

Noise causes destruction of the end organs of the labyrinth by different mechanisms which included metabolic changes due to degeneration of the sensory elements and direct mechanical damage of the neuroepithelium[18]. In addition, reactive oxygen species (ROS) and glutamate excitoxicity are increased; the endogenous antioxidant system is decreased and pro-apoptotic factors release that lead to cell death[19].

Clinical symptoms in the study group included tinnitus in 13 subjects (76%), hearing loss in 9 subjects (53%) and imbalance in 5 subjects (29%) (Table 1). This agreed with the study done by Elkindy.[20] who reported that the most frequent complaint among the noise exposure group was tinnitus in 96 (76.2%) of 126 participants using the visual analog scale (VAS). In addition Kim[21] and Puel *et al*.[22] reported that 54.14% of the workers exposed to noise were complaining of tinnitus.

Tinnitus is the first warning symptom of exposure to extremely loud noises and may reflect an increased vulnerability to injury. It reflects a dysfunction arising from abnormal neural activity in the auditory pathways[23]. Tinnitus associated with excessive noise exposure could be explained by hair cell damage[24].

Only 5 subjects complained of vertigo which agrees with some studies that stated that balance disturbances may not be seen if chronic noise exposure causes

gradual vestibulopathy[25]. In vestibular end-organ damage, compensatory strategies can be developed by the central nervous system. So, the vestibular deficits in patients with chronic exposure to noise were subtle and they did not significantly affect the functional capacity of the patient, as these vestibular deficits are balanced via visual and somatosensory inputs[26].

vHIT results revealed that reduced gain in the lateral canalwas detected in 8 (47%) of 17 patients, one of the patients (5.9%), how had reduced left lateral canal gain also had reduced left posterior canal gain. Consequently, the total canal deficit was detected in 8patients (47%) (Table 2). The lateral canal gain in both ears showed a statistical significance difference between the study and control groups (Table 3). These findings were in agreement with Yilmaz *et al*.[11] who reported that canal deficit was detected in 20 (55.5%) of 36 patients in the noise induced hearing loss group and the lateral canal was the most affected one.

In addition, Yilmaz and Ila.[27] studied 138 employees and divided them into 3 groups. Group 1 was exposed to vibration and noise. Group 2 was exposed only to vibration, Group 3 was not exposed to noise or vibration. They reported that canal deficit was observed in 41 (48.80%) of 84 workers in group 1 and 7 (29.16%) of 24 participants in Group 2. The percentage of canal deficit in group 1 was higher than in group 2. It could be attributed to the synergistic effect of both noise and vibration. They reported also that the lateral canal was the most affected canal.

The vestibular dysfunction that was detected in this study byv HIT can be explained by the close link between the damage pattern found in the cochlea and vestibular structure in patients with NIHL. This means that the mechanisms that cause hearing loss following exposure to noise, causes also vestibular end organs destruction[28]. Less known and less understood in the literature about the affection of lateral canal in particular but it could be related to increase the susceptibility of hair cells in the lateral canal to the damage by noise. Further work is needed to better understand the physiological and functional consequences of noise induced vestibular impairment particularly on the lateral canal.

As regards the correlation between duration of noise exposure and vHIT in the study group, a statistically significant negative correlation between duration of noise exposure with lateral canal gain on both sides was present (longer duration is associated with a decrease in the gain of the lateral canal) (Table 4). It could be explained by Juntunen *et al*.[29] and Ylikoski *et al*.[30] who reported that as the duration and intensity of the noise exposure increased, reduced blood flow may have led to permanent hearing threshold shifts and subclinical disturbance of the vestibular system, resulting in vestibular acoustic trauma. In addition, The Cochlea receives its blood supply mainly from the common cochlear artery, anterior and posterior vestibular arteries supplied the saccule; the labyrinthine artery is the origin of all these arteries. Therefore, as the duration and intensity of the noise exposure increased, a decrease in blood flow may lead to permanent threshold shifts in hearing and abnormal VEMP and caloric responses[31]. The same factor could explain vHIT results also.

The Correlation between vHIT and pure tone thresholds was done among the study group and revealed that there was a statistically significant negative correlation between vHIT lateral canal gain on both sides with pure tone thresholds at 2, 3, 4, 6 & 8KHz frequencies (The more hearing loss degree, the less vHIT responsiveness) (Table 5).These correlations between hearing thresholds and vHIT canal gain might indicate a single mechanism for both cochlear and vestibular noise-induced injury.In contrast,Yilmaz *et al*.[11] examined 36 patients with NIHL by chi-squared test for the presence of vHIT abnormalities based on the degree of hearing loss. They revealed no statistically significant difference between patients with normal hearing, mild and moderate SNHL. Interpretation of these data is limited as NIHL was defined according to the degree of hearing loss at 4,000 Hz instead of a characteristic noise notch[17].

CONCLUSION

Lateral canal is the most frequently affected canal in a patient with chronic noise exposure as detected by vHIT. In addition, a longer duration of noise exposure and increasing the degree of hearing loss (decreasing the audibility) are associated with a decrease in the gain of the lateral canal.

RECOMMENDATIONS

It is recommended to use the vHIT as a screening for the vestibular dysfunction in patients with chronic noise exposure.

CONFLICT OF INTEREST

There are no conflicts of interest.

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