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ORIGINAL ARTICLE

Evaluation of Vestibular System Function Using Video-Nystagmography in Patients with Chronic Noise Exposure

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ABSTRACT

Background: Hearing loss due to noise exposure is considered one of the known harmful effects of noise on human health. Unlike the effect of noise on hearing, it is not usually considered a common etiology of dizziness, vertigo, or other vestibular disorders. With the help of sensory hair cells, the cochlea and the vestibular end organs have a similar evolutionary origin and use the same basic precept of mechano-electric transduction. Therefore, the noise levels that cause damage to the cochlea may also damage the balance system. This study aimed to assess the role of Video-nystagmography (VNG) in the diagnosis of the vestibular dysfunction in individuals with chronic exposure to noise.

Methods: Two groups were included in this study. Control group: 17 healthy subjects with normal peripheral hearing sensitivity and study group: 17 subjects with industrial noise exposure. Basic audiological evaluation and VNG test battery were conducted on all the subjects in this study**. Results:** There was a statistically significant difference in thresholds of pure tone audiogram at 3,4,6,8 KHz frequencies between the study and the control groups. In addition, there was a statistically significant difference in caloric weakness between the two groups. A positive correlation between the noise exposure duration and caloric weakness was also present. **Conclusions:** Long-term noise exposure causes peripheral compensated vestibular dysfunction in addition to its effect on hearing.

Keywords: Chronic noise exposure; Noise-induced hearing loss; vestibular; Video-nystagmography

INTRODUCTION

Noise is defined as any undesirable sound that is considered unpleasant, noisy or disruptive to hearing. Physically, noise and sound are indistinguishable, since both are vibrations transmitted through a medium such as air or water, where the difference arises when the brain receives and perceives favorable and unfavorable sounds [1].

There’s increasing evidence that long-term noise exposure above a specific level can have a negative influence on human health. Elevated workplace or environmental noise can cause hearing disorder, hypertension, ischemic cardiopathy, sleep disturbance, annoyance and changes within the immune system are also attributed to noise [2].

Exposure to noise not only induces hearing loss but also causes deficits in the peripheral vestibular system, in the absence of immediate clinically detectable vestibular signs [3]. There is a remarkable similarity in the injury patterns found in the cochlea and vestibular structures. The effect of noise on the vestibular system involves two mechanisms: direct mechanical destruction, and metabolic changes with associated degeneration of sensory elements. Therefore, it’s likely that individuals who have noise induced hearing loss (NIHL) will have damage to the vestibular end organs in addition to the cochlear lesion [4].

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Being one of the most common vestibular function test used, videonystagmography (VNG) which evaluates both peripheral and central vestibular functions through a battery of tests that measure eye movements assessing vestibular relation to eye movement. These tests record how the eyes react to signals transmitted to the brain by the inner ears. [5]**.** Therefore, this study aimed to assess the role of VNG in the diagnosis of the vestibular dysfunction in individuals with chronic exposure to noise.

SUBJECTS AND METHODS

The Research Ethics Committee at the Faculty of Medicine, Zagazig University Hospitals approved this study. Written informed consent was obtained from all participants after the test procedures had been explained. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Study design and subjects

This case-control observational study was carried out in the audio-vestibular unit, ENT department, Faculty of Medicine, Zagazig University Hospitals. The sample size was calculated to be 34 subjects classified into two groups: Study group involved 17 patients who were exposed to industrial noise. They ranged in age from 30 to 55 years. They had sensory neural hearing loss from mild to moderately severe and presented as audiogram notching at 3, 4 or 6 kHz. NIHL's diagnostic criteria were based on the recommendations of the American College of Occupational and Environmental Medicine (ACOEM, 2014) [6]. *Exclusion criteria:* a history of head injuries, ear infection, a problem with neurology or general health, history of hearing loss in the family and patients with acute acoustic trauma or blast injury. *The control group* involved 17 adults with bilateral normal peripheral hearing sensitivity in the 250-8000 Hz frequency range and normal middle ear function. The participants were both gender and age comparable. Their age ranged from 25 to 50 years. They had no history of noise exposure or other systemic illness affecting equilibrium e.g. diabetes mellitus, hypertension or neurological disorder.

Basic audiological evaluation: All participants were subjected to otoscopic examination, pure tone audiometry, speech audiometry and immittancemetry.

VNG test battery**:** It included the following battery: spontaneous nystagmus, gaze-evoked nystagmus, oculomotor tests (saccade, smooth pursuit and optokinetic tests)**,** Dix-Hallpike, Positional and caloric tests. Recordings were made using version 0.1 of Ulmar video-nystagmography (VNG).

Statistical analysis: We used SPSS version 20 to analyze data. Quantitative variables were presented as mean±standard deviation. A number and percentage were used for the description of qualitative variables. The quantitative variables in parametric data were compared using an independent t-test. In non-parametric data, the independent quantitative variables were compared using the Mann-Whitney-U test. The Pearson correlation test was used to rank various variables [r= (-1.0) - (1.0)] in a positive or negative manner.

RESULTS

Two groups of adults were included in this study. The study group involved 17 patients (16 male and 1 female). Their mean age was 46.7±7.9 years and the mean noise exposure period was 22.1±8.8 years, the control group included 17 subjects (12 male and 5 female) and their mean age was 45.1±8.1 years. No statistically significant difference was present between both groups regarding age and gender. There were statistically significant differences in pure tone audiogram (PTA) thresholds at 3,4,6, and 8 KHz between the study and the control group (Table 1). In addition, there was a positive correlation between the period of noise exposure and PTA threshold at 2,3,4,6&8 kHz in both ears. Otherwise, no statistically significant correlation was present between the period of noise exposure and PTA at lower frequencies. There was no statistically significant difference in Speech Reception Threshold (SRT)but a

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statistically significant difference in Word Discrimination Score (WDS) between study and control groups was present.

Regarding VNG, Spontaneous, oculomotor tests, gaze, positioning, and positional testing showed no significant difference between both study and control groups. Caloric weakness was detected in ten patients (58.8%) three of them (17.6%) showed bilateral weakness while seven others (41.2%) had unilateral weakness. There was a statistically significant difference in the caloric weakness between the study and control groups. While no difference was present in the directional preponderance (DP) between the two groups (Table 2). Moreover, there was a positive correlation between noise exposure duration and the percentage of caloric weakness. A negative correlation between noise exposure duration and cold and worm irrigation values was also present (Table 3). There was a statistically significant positive correlation between caloric weakness and pure tone audiometric threshold at 2,3,4,6 &8 kHz. (Table 4).

Table (1): Comparison of pure tone thresholds at different frequencies between study and control groups

Pure tone threshold (dB)		Study mean ± SD	Control mean ± SD	t- test	p-value
250	Rt	14.4±6.5	12.9±4.7	0.7	0.4
	Lt	16.2±6.9	12.8±4.7	1.6	0.1
500	Rt	13.2±6.3	13.8±5.4	0.3	0.7
	Lt	13.5±4.9	13.8±5.1	0.2	0.8
1000	Rt	15.3±4.7	12.9±3.6	1.1	0.2
	Lt	15.9±3.8	13.8±2.7	0.9	0.3
2000	Rt	20.6±5.4	14.7±3.5	1.7	0.08
	Lt	18.5±5.2	13.8±3.2	1.6	0.1
3000	Rt	32.6±6.9	16.4±4.2	3.8	<0.001**
	Lt	33.8±6.8	17.1±4.3	5.2	<0.001**
4000	Rt	40.8±10.6	15.5±4.6	5.9	<0.001**
	Lt	44.1±13.1	13.8±4.2	8.9	<0.001**
6000	Rt	36.2±5.6	17.4±3.9	4.8	<0.001**
	Lt	36.5±5.2	17.3±4.3	6.1	<0.001**
8000	Rt	31.2±5.7	15.6±5.1	3.8	<0.001**
	Lt	30.3±5.4	16.7±5.3	3.5	<0.001**

Table (2): Comparing caloric weakness and directional preponderance between study and control groups

Variable	Comparison		
Caloric weakness	Mean ± SD	MW (Z-score)	p-value
Study:	21.2±19.7	2.2	0.02*
Control:	7±4.01		
Directional preponderance	Mean ± SD	MW	p-value
Study:	13.6±12.4	1.32	0.18
Control:	6.88±5.2		

Table (3): Correlation between duration of noise exposure, and caloric test in the study group:

	Duration of noise exposure		
Variable	r	P	SIG
Caloric weakness	0.51	0.03*	S
Cold Rt	-0.53	0.02*	S
Cold Lt	-0.56	0.01*	S
Warm Rt	-0.60	0.01*	S
Warm Lt	-0.51	0.03*	S

Table (4): Correlation between caloric weakness and pure tone thresholds at different frequencies in the study group

Variable		Caloric weakness		
		r	P	SIG
250 (HZ)	Rt	0.05	>0.05	NS
	Lt	0.3	>0.05	NS
500 (HZ)	Rt	0.2	>0.05	NS
	Lt	0.1	>0.05	NS
1000 (HZ)	Rt	0.4	>0.05	NS
	Lt	0.4	>0.05	NS
2000 (HZ)	Rt	0.6	0.002*	S
	Lt	0.5	0.02*	S
3000 (HZ)	Rt	0.5	0.001**	HS
	Lt	0.7	0.001**	HS
4000 (HZ)	Rt	0.8	0.001**	HS
	Lt	0.7	0.001**	HS
6000 (HZ)	Rt	0.5	0.001**	HS
	Lt	0.5	0.001**	HS
8000 (HZ)	Rt	0.5	0.02*	S
	Lt	0.5	0.03*	S

DISCUSSION

Noise can elicit several types of responses to the human being in addition to those directly related to hearing. Responses resulting from the activation of vestibular system receptors are one of these nonauditory effects.

Pure tone audiogram thresholds were elevated in the study group especially at 3, 4, 6 and 8 kHz with a distinctive notch at 4 kHz which agreed with **Nada et al.,** [8] and **Mohany et al.,** [9] who stated that there was a significant elevation of pure tone thresholds along with audiometric frequencies 2, 3, 4, 6 and 8 kHz in both sides in studies examining the impact of noise on hearing conducted on workers in textile factories. These findings could be attributed to the outer hair cells injury at the corresponding area of the organ of Corti [10]. In addition, **McGill et al.,** [11] reported that degenerative changes in the organ of Corti are triggered by prolonged noise exposure, most strictly in the 9 to 13 mm part of the cochlea and are responding maximally to the 4 kHz frequency range.

Speech discrimination scores were significantly decreased in the study group. This finding was consistent with elevated hearing thresholds and the assumption that the frequencies affected by noise were the speech frequencies. The level of hearing at 3 kHz is generally accepted to be related to hearing and understanding speech, especially in the existence of noise [12]. Similar findings were reported in studies that were done by **Nada et al.,** [8] and **Emara and Gabr,** [13] who reported that speech discrimination scores were significantly lower for the study group than the control group.

Central vestibular affection was excluded by oculomotor tests which showed no abnormality. Spontaneous, positioning and positional testing showed no significant difference between both study and control groups. This finding was consistent with **Wang and Young,** [14] who stated that VNG couldn't detect either spontaneous or positional nystagmus. In addition, **Ali, et al**., [9] found neither spontaneous nor positional nystagmus in their study group of noise-exposed personnel.

On the contrary, **Oosterveld et al.,** [15] and **Golz et al.,** [16] reported that NIHL patients show a high occurrence of spontaneous, gaze or positional nystagmus using Electronystagmography (ENG) and explained by the vestibular complaint severity which is more common in the patients with asymmetric hearing loss.

These findings may suggest an asymmetrical, centrally compensated decrease in the response of vestibular end-organ associated with a history of exposure to noise. Also, the assumption that: symmetrical affection gives no place for the appearance of signs of decompensation like spontaneous and positional nystagmus [16].

Peripheral vestibular dysfunction was assessed using the caloric test and revealed that 58.8% of the patients showed caloric weakness. Unilateral and bilateral weakness was detected in 41.2% and 17.6% of patients respectively. There was a statistically significant difference in caloric weakness between the study and control groups with no significant difference in the DP (Table 2). These findings indicate peripheral compensated vestibular dysfunction. Similarly, **Wang and Young,** [14] reported abnormal caloric tests in 45% of patients with NIHL including canal paresis in77% of them and caloric areflexia in the remaining 33%.

Moreover, **Ali et al.,** [9] reported that a significant difference was present between the study and control group in unilateral weakness. **Xu et al**. [17] stated that noisy stimuli cause strong functional disability to the end organs of the vestibular system in addition to its effect on the cochlea. Vestibular pathologies in patients with NIHL in their study included unilateral caloric weakness in (20.0%) and bilateral caloric weakness in 26.7 % of the patients.

However, this study disagreed with **Emara and Gabr,** [13] who found out a smaller percentage of caloric weakness in two noise-exposed groups. This might be explained by the fact that not all of the participants in the study group had NIHL as their study group included 40 patients with a history of work-related excessive noise exposure. The group was further classified into two subgroups: twenty patients with NIHL and twenty subjects with normal hearing sensitivity. This may give an impression that there was an association between hearing loss and vestibular lesions.

A positive correlation between the duration of noise exposure and caloric weakness was found (longer duration is associated with an increase in the percentage of caloric weakness). (Table 3). There was also a statistically significant positive correlation between caloric weakness and pure tone threshold at 2,3,4,6 &8 kHz. Regarding low frequencies, there was no statistically significant correlation with caloric weakness (Table 4). These findings were inconsistent with **Ali et al.** [9] who stated a positive association between noise exposure period and auditory and vestibular dysfunction using VNG in their study. Machado et al., [18] also reported that when the duration of noise exposure increased, the severity of dizziness increased and the hearing sensitivity reduced. The compatibility of both vestibular and otological symptoms suggests that, due to prolonged noise exposure, the cochlea and the vestibular system are increasingly deteriorating with each other.

CONCLUSIONS

The present study showed that noise had an obvious effect on both auditory and vestibular systems. Prolonged noise exposure not only causes hearing loss but also leads to essential destruction of the peripheral part of the vestibular system. Some patients with NIHL do not report significant imbalance problems probably due to compensation occurring from the visual, proprioceptive and central vestibular system.

Conflict of Interest: None.

Financial Disclosures: None.

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