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A COMPARATIVE STUDY ON OCULAR VESTIBULAR EVOKED MYOGENIC POTENTIALS IN NORMAL HEARING AND NOISE-INDUCED HEARING LOSS

Speech & Hearing

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ABSTRACT

Background: Noise-induced hearing loss (NIHL) is primarily considered a cochlear pathology; however, emerging evidence suggests associated vestibular involvement, particularly of the utricle and superior vestibular nerve. **Aim:** To evaluate utricular function using oVEMP in individuals with NIHL and compare findings with normal-hearing adults. **Methods:** A total of 200 participants (35–45 years) were divided into Group A (n=100; normal hearing) and Group B (n=100; NIHL). oVEMP responses were recorded using standard electrode montage and stimulation protocols. N1 and P1 absolute latencies were analyzed. Statistical analysis was performed using SPSS v22 with significance set at $p<0.05$. **Results:** Group B showed significantly prolonged N1 and P1 latencies compared to Group A ($p<0.05$) suggesting bilateral utricular and superior vestibular nerve dysfunction in NIHL. **Conclusion:** Chronic noise exposure affects both cochlear and utricular pathways, supporting the concept of NIHL as a cochleo-vestibular disorder.

KEYWORDS

Noise-Induced Hearing Loss (NIHL); Ocular Vestibular Evoked Myogenic Potentials (oVEMP); Utricular Dysfunction; Superior Vestibular Nerve

INTRODUCTION

Noise exposure can lead to both temporary and permanent damage, resulting in associated hearing loss [1]. Prolonged exposure to hazardous noise levels, as well as a single exposure to high-intensity sound, can result in permanent threshold shifts (PTS) [2]. This damage typically results from two primary mechanisms: metabolic changes and direct physical injury. The metabolic changes responsible for cochlear damage include the production of reactive oxygen species (ROS), reactive nitrogen species (RNS), ischemia (reduced blood flow), free radicals, and metabolic stress in the organ of Corti. These factors contribute to the harmful effects of noise exposure on hearing while it is well known that loud sounds can damage the cochlea and cause hearing loss, less attention has been given to how noise affects the vestibular system, which controls balance [3]. However, some individuals with NIHL also experience balance problems and symptoms similar to Meniere's disease. Although there is a clear connection between noise exposure and hearing loss, the impact of noise on balance functions is not as well understood and is often overlooked [4]. This may be due to the difference in the way the hair cells in the cochlea and the vestibular system respond to sound. Cochlear hair cells are sensitive to frequencies between 20 and 20,000 Hz, while vestibular hair cells are responsive to much lower frequencies, typically between 0 and 10 Hz [5].

The vestibular organ consists of three semicircular canals (SCCs) which are anterior, posterior and lateral & two otolithic organs called utricle and saccule. The auditory and vestibular organs share functional and structural similarities, including anatomical proximity, interconnected pathways, and a common blood supply to their receptors [6]. Exposure to loud occupational noise can cause damage to the vestibular system, leading to both asymmetrical and symmetrical hearing loss, often accompanied by abnormal vestibular function [7]. The most common vestibular dysfunction symptoms are vertigo, unsteadiness, and dizziness [8].

NIHL is so common that a majority of the workers believe that it is part of their normal working life course [9]. This is especially disturbing considering that NIHL is nearly always preventable [10]. It has been estimated that 1.1 million people are exposed to excessive noise at work; among these, 0.17 million are predicted to suffer significant ear damage as a direct result of noise [11]. Many workers including those engaged in heavy industry, factories, forge hammering, coal and ore mining, construction, cement plants, gas processing industry and mechanical engineering as well as mill and stationary machine device operators and workers at oil refineries are at risk of developing occupational NIHL [12, 13]. oVEMP assesses excitatory responses of the extraocular muscles, primarily reflecting utricular function and the superior vestibular nerve pathway [14].

Need Of The Study:

Most individuals with NIHL do not report dizziness or balance disturbances, leading to an underestimation of vestibular dysfunction in this population. Therefore, there is a need to systematically investigate vestibular function in individuals with NIHL.

Aim And Objective Of The Study

The aim of the present study was to compare oVEMP findings between normal-hearing subjects and subjects with NIHL.

MATERIAL AND METHODS

The present study included a total of 200 participants with age range of 35–45 years and divided into two groups. Group A consisted of 100 participants with normal hearing, while Group B consisted of 100 participants with Noise Induced Hearing Loss (NIHL). Inclusion Criteria for Group A includes participants with no history of Noise Induced Hearing Loss (NIHL), bilateral normal hearing sensitivity, presence of Type "A" tympanogram. Group B includes participants with a history of Noise Induced Hearing Loss, "A" type tympanogram. Subject who has external or middle ear abnormalities, family history of otological disorders, neurological disease, systemic disease, history of drug intake related to auditory & vestibular, retro cochlear pathology, ear surgery, and previous or ongoing vestibular symptomatology was excluded from the study.

oVEMP are the crossed response, electrode montage includes two reference electrodes which was placed as close as possible underneath the eye in the orbital midline in both sides. The response was recorded from the inferior oblique muscle underneath the contralateral eye. The right electrode was placed under the left eye while the right ear was stimulated. Correct positioning of the electrode on the inferior oblique muscle was essential in obtaining a response. The vertex electrode was placed on the high forehead and the ground or common on the low forehead. Conductive gel was used for placement of electrode.

RESULTS

All statistical analyses were performed using SPSS software version 22. The mean age of Group-A was 38.9 ± 3.01 years, and Group-B was 39.0 ± 2.95 years. Mean and standard deviation values for oVEMP absolute latencies (P1 and N1) values were calculated for both ears in Group-A and Group-B (Table 1). Across all VEMP parameters, Group-B demonstrated prolonged latencies compared to Group-A.

Table 1 presents the mean and standard deviation of absolute latencies for oVEMP in both ears among Group-A and Group-B.

Test	Absolute Latency	Group-A				Group-B			
		Rt Ear		Lt Ear		Rt Ear		Lt Ear	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
oVEMP	N1	12.55	1.79	12.63	1.86	19.19	3.44	19.17	3.48
	P1	15.57	1.30	15.59	1.35	23.70	3.21	23.62	3.21

Within-Group Comparisons

Paired-sample t-tests were used to compare right and left ear oVEMP responses within each group. In Group A, mean N1 latency was 12.55 ms (right) and 12.63 ms (left), showing a very strong positive correlation ($r = 0.96$, $p < 0.05$) with no significant inter-ear difference ($p > 0.05$). Mean P1 latency was 15.57 ms (right) and 15.59 ms (left), also demonstrating a strong positive correlation ($r = 0.89$, $p < 0.05$) without significant asymmetry ($p > 0.05$). These findings indicate symmetrical and preserved utricular function. In Group B, N1 latencies showed a very strong positive correlation between ears ($r = 0.99$, $p < 0.05$), with no significant inter-ear difference, indicating absence of asymmetry.

Between-Group Comparisons (Group-A vs Group-B)

Independent sample t-tests were used to compare Group A and Group B. Extremely high t-values for oVEMP P1 latencies indicate a large and consistent difference between normal and NIHL groups, reflecting significant utricular and superior vestibular nerve dysfunction in NIHL. The magnitude of the effect suggests strong noise-related impact with minimal group overlap. Findings indicate bilateral utricular pathway involvement, and oVEMP appears sensitive in detecting vestibular changes associated with NIHL.

DISCUSSION

The results provide compelling evidence that chronic noise exposure affects not only cochlear mechanisms but also the otolithic organs and semicircular canals, supporting the concept of NIHL as a cochleo-vestibular disorder rather than a purely auditory pathology. The oVEMP results revealed markedly prolonged N1 and P1 latencies in the NIHL group, with extremely high t-values indicating robust statistical and clinical significance. oVEMP responses are known to represent utricular macular function and superior vestibular nerve pathways (Curthoys et al., 2011). The pronounced latency prolongation observed in NIHL participants strongly suggests utricular dysfunction. These findings are consistent with Tseng et al., (2012), who demonstrated abnormal oVEMP responses in individuals exposed to chronic industrial noise, even in the absence of vestibular symptoms. Similarly, Kumar et al., (2010) reported prolonged oVEMP latencies in patients with cochlear hearing loss, suggesting concurrent utricular dysfunction.

Furthermore, the bilateral nature of oVEMP abnormalities in Group-B further supports the notion of diffuse vestibular end-organ involvement, consistent with findings by Stewart et al. (2020), who demonstrated widespread vestibular deficits in chronic NIHL patients using VEMP testing. The bilateral symmetry of oVEMP abnormalities in Group-B again suggests global vestibular system involvement, reinforcing the hypothesis that chronic noise exposure produces widespread vestibular dysfunction rather than isolated end-organ damage.

The findings of the present study demonstrating significant vestibular abnormalities in individuals with NIHL, particularly affecting saccular and utricular pathways. Importantly, these vestibular deficits were not directly proportional to the degree of hearing loss, suggesting that chronic noise exposure itself-rather than cochlear threshold elevation alone-contributes to vestibular receptor damage.

CONCLUSION

Consistent with previous literature, vestibular impairment predominantly affects the saccule, as reflected by elevated VEMP thresholds, prolonged latencies, and reduced P1–N1 amplitudes, even in individuals without overt vestibular symptoms. NIHL is not confined to cochlear pathology alone but extends to the vestibular system, reinforcing the concept of cochleo-vestibular vulnerability to chronic noise exposure.

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