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Review Article

Association between Simultaneous Occurrence of Occupational Noise-Induced Hearing Loss and Noise-Induced Vestibular Dysfunction: A Systematic Review

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Abstract

Background: Background: Because of functional and structural similarities between the cochlea and vestibular sensory receptors, vestibular dysfunction could be accompanied by noise-induced hearing loss (NIHL) due to occupational noise exposure. We aimed to evaluate the occurrence of vestibular dysfunction (VD) in individuals with NIHL and occupational noise exposure.

Methods: A systematic literature research was carried out within the databases of PubMed, Scopus, Science Direct, and Web of Science for published articles between 1980 and Jan 5, 2023 using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. The methodological quality of the included systematic reviews was assessed with the Joanna Briggs Institute (JBI) checklist. Vestibular system dysfunction parameters were considered as primary outcomes in subjects with NIHL.

Results: We reviewed the evidence (from 19 eligible articles) for VD from noise-induced damage to peripheral vestibular structures. VD can occur after occupational noise exposure or concomitantly with NIHL. Furthermore, this study showed that the saccular organ has a higher susceptibility to noise damage than the vestibular organs of the utricle and semicircular canals (SCCs).

Conclusion: Our results support the role of occupational noise exposure and NIHL as risk factors for developing VD. Further research is needed to investigate the association between the occurrence of VD due to occupational noise exposure or concomitantly with NIHL.

Keywords: Noise-induced hearing loss; Noise-induced vestibular dysfunction; Occupational noise exposure

Introduction

Occupational noise exposure is the second most common risk factor in the industry, which contributes to 22% of workplace-related health issues including auditory and non-auditory health

effects: hearing loss (HL), annoyance, cardiovascular disease, cognitive performance and sleep disturbance (1, 2). According to WHO report, the prevalence of disabling HL (> 40 dB) was 6.12% (466 million people in the world) in 2018 and its number could increase to 8.27% (630 million) by 2030 and 9.6% (900 million) in 2050, respectively (3). A recent WHO report estimated that the global costs of HL (moderate or higher degrees) would be ranged of $750-790 billion annually (4).
NIHL occurs due to occupational noise exposure with the maximal degree in the 3-4 kHz range. Pathological characteristics of NIHL include a bilateral loss of the sensory hair cells and degeneration of neural auditory nerve. Because of regenerative incapability of the human auditory system, NIHL is irreversible (5, 6).
A causal relationship between noise exposures and cognitive deficits including spatial learning and memory ability (7, 8) and spatial memory reduction (9) and proliferating cells in the hippocampal formation (10) has long been recognized. Damage to synapses between the inner hair cells and the primary auditory neuron is the reason for the reduction of cochlea outputs to the brain due to noise exposure without permanent threshold shift (PTS) (11, 12). In addition, NIHL can result in cognitive function deficit (spatial learning/memory) and hippocampal neurogenesis (13).
Such a relationship between peripheral HL and cognitive function has been attributed to a strong anatomical and functional connection between brain regions (hippocampus) and auditory system. Hence, reduction of auditory input results in hippocampal degeneration and impaired memory function (13-16). In addition, hypotheses presented for the causal mechanisms underlying the relationship between HL and cognitive decline include cognitive load hypothesis, information-degradation hypothesis, sensory deprivation hypothesis, and common-cause hypothesis (17, 18).
Because of functional and structural similarities between the cochlea and vestibular sensory receptors in three SCCs and two otolith organs (the utriculus and sacculus), VD could be accompanied by NIHL (19-21). Animal experiments showed the peripheral vestibular system damage due to noise exposure (22-24). The vestibular system is crucial in maintaining balance and gaze stabilization (25, 26). Most common VD symptoms include dizziness, unsteadiness, and vertigo (27). Dizziness as a medical complaint can result in a significant impact on work (28) and cognitive impairments (29).
On the other hand, vestibular signals play an important role in cognition functions such as spatial memory (30, 31), navigation (32), visuospatial ability, attention, memory and executive function (27), and spatial working memory (33). Therefore, a decrease in performance, spatial navigation ability (34-36) and slowing the horizontal head movements (37) during driving have been reported in the literature due to peripheral vestibular disorders.
Numerous studies have been carried out to identify dysfunctions of various organs of the vestibular system (including SCCs and otolith organs) using different methods in subjects who work in various workplaces, including industrial and military. Due to the postulated connections between occupational noise exposure and the possible simultaneous occurrence of NIHL and VD, it is important to research existing knowledge in order to design future research effectively. To the knowledge of the authors, there is no systematic overview of the relationship between NIHL and vestibular dysfunction in subjects exposed to occupational noise.
We aimed to examine and summarize the published literature on the occurrence of dysfunction of the vestibular system in individuals with NIHL and occupational noise exposure.

Methods

Search strategy and selection criteria

Based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (38), this systematic review was carried out in the databases of PubMed, Scopus, Science Direct, and Web of Science for published articles between 1980 and Jan 5, 2023. A search strategy with low specificity and high sensitivity ("Vestibular" and "Noise exposure" OR "Vestibular" and "noise induced hearing loss") was developed to find relevant articles. Due to accessibility, we limited our search strategy only for English-language papers, article types (research articles), and species (human). The search strategy in four databases PubMed, Scopus, Science Direct and Web of Science, resulted in finding 507 articles (Fig. 1). In addition, references of eligible articles were also thoroughly reviewed to find additional relevant articles.

This review study included occupational human studies performed in different workplaces on the relationships between NIHL and VD. Only studies were included in this review that examined the vestibular function by at least one of these test; Electronystagmography (ENG), Videonystagmography (VNG), cervical Vestibular Evoked Myogenic Potentials (cVEMPs), ocular Vestibular Evoked Myogenic Potentials (oVEMPs), and video Head Impulse Test (vHIT). We had excluded studies performed among general population, congress abstracts, and animal models studies.

Screening process

The titles and abstracts of all references were retrieved after removing duplicates (using EndNote X8). They were screened by the inclusion/exclusion criteria. Articles that did not meet the inclusion criteria were excluded from further analyses. Figure 1 shows a PRISMA flow diagram of the study for the screening process. Articles retained were then further filtered out after reviewing full contents and removed any repetition.

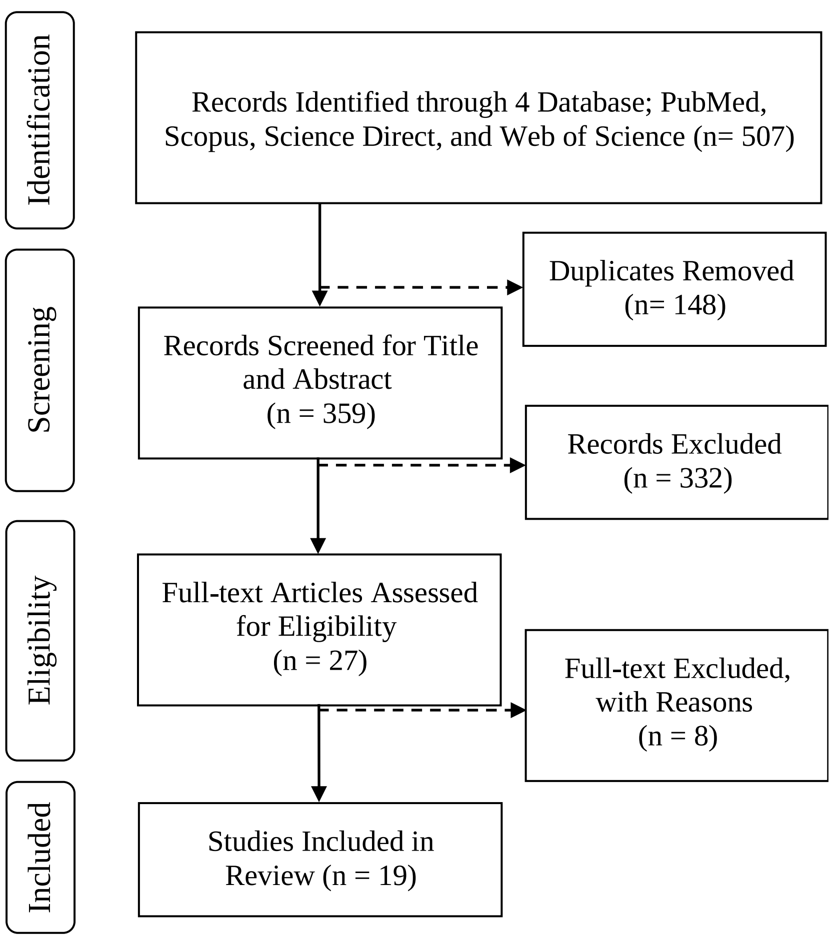

Fig. 1: A PRISMA flow diagram for the screening process of the present review study

Quality assessment of the systematic reviews and data extraction

The methodological quality of the finally included studies in this systematic reviews was assessed with the JBI checklist (39). This checklist consists of 8 questions with Yes, No, Unclear and Not applicable answers for critical appraisal of studies. Therefore, based on the number of positive responses to the questionnaire, each study was allocated a score of 0 to 8. Since our goal was to identify any related study regardless of its quality, we did not set minimum quality threshold criteria.

Results

Description of search

According with the selection criteria we have included 19 studies. Full articles were obtained for the 19 included studies (Fig. 1). Of these studies, 14 articles were carried out in industrial workplaces (with occupational exposure to continuous noise) and 5 articles in military workplaces (with occupational exposure to impulse/impact noise). The main characteristics, findings and quality assessment of the included studies are detailed in Table 1. All included studies were published in English. The quality results of the studies showed that they had an average quality of 6.1 ± 1. Compared to the maximum quality value of 8, these studies were of medium to high quality. The results of these studies are presented in the following three sub-sections.

Vestibular disfunction due to NIHL and occupational exposure to continuous noise

All vestibular organs function including three SCCs and two otolith organs (the utriculus and sacculus) were evaluated among subjects with NIHL and exposed to occupational continuous noise with various tests (Table 1). The evaluation of the function of all SCCs among industrial workers using vHIT were conducted only by two studies, which reported vHIT abnormalities in (lateral canal of) 55% (49) and 47% (53) of workers with NIHL (with predominant abnormality at horizontal SCCs). On the other hand, the function of the horizontal SCCs was evaluated in the various studies with the caloric test (from ENG and VNG tests) (40, 42, 43, 45, 50, 51, 58), and in several of these studies a significant dysfunction of the horizontal SCCs was reported in 47.1% (42), 45% (43), 33% (45), 17.5% and 16.9 % (50) of workers with NIHL. Although there was no significant dysfunction of horizontal SCCs in several of these studies (40, 41, 51, 52). The function of the saccule organ of otolith organs was evaluated in several studies with the cVEMP test (40, 41, 43-48, 51, 52), as several of these studies reported a significant association between the dysfunction of the saccule organ and NIHL (47, 51). In addition, other studies reported significant dysfunction of the saccule organ in 50% (43), 34.6% (44), 70% (45), 36.6% (46), and 30% (47) of the workers with NIHL. In other hand, the evaluation of the function of utricle organ of otolith organs was only carried out using oVEMP test in the study of Tseng and Young (45), which showed that 57% of workers have dysfunction in this organ.

Vestibular disfunction due to NIHL and occupational exposure to impulsive noise

Shupak et al. assessed the function of the horizontal SCCs (using the caloric and rotary chair tests), which showed significant dysfunction in military personnel with NIHL than in controls (20) (Table 2). In addition, Golz et al. demonstrated significant horizontal SCCs dysfunction (using caloric testing) in 46.8% of military personnel with asymmetrical NIHL than with symmetrical NIHL (54). No dysfunction of the horizontal SCCs (with the rotary chair test) was found in military personnel with asymmetrical NIHL than in the control group (56). In addition, the cVEMP test has been used in several studies to assess saccular organ function, and in these studies significant dysfunction was reported in 75% (55), 33% (56), and 63% (57) of military personnel with NIHL compared to the control group. In other hand, in majority of studies in this part, there are no significant correlations found between the vestibular pathology and symptomatology (vertigo or dizziness) (20, 54, 55, 57).

Vestibular disfunction sequence

Several studies included studies in this review reported more vestibular dysfunction in the saccular organ than in other vestibular organs. Investigation of the sequence of vestibular deficits in subjects with NIHL, using cVEMP, oVEMP, and Caloric tests by Tseng and Young (45), showed a decreasing order of abnormal finding from the saccule (70%), followed by utricle (57%), and horizontal SCCs (33%). In addition, Akin et al. demonstrated normal function of the horizontal SCCs compared to a significant abnormal finding of the saccule in all NIHL participants (56). The susceptibility of the saccule organ in the pars inferior (saccule and cochlea) of inner ear than pars superior portion (utricle and SCCs) due to noise exposure has been shown by Wang et al. (59)

Table 1: A summary of articles on the relationship between NILH with VD in subjects exposed to continuous noise (n = 14)

Ref. No.	*Study Aim*	*Study/Control Group*	*Audiological/ Vestibular Examinations*	*The results showed:*	*QAS**
(40)	Investigation of ENG evidence in patients with acoustic trauma.	286 subjects with slight and severe NIHL/40 healthy men	PTA*/ENG	A significant relation between NIHL and vertigo. Increased ENG abnormal results with increased NIHL and presence of vertigo.	4/8
(41)	Investigation of VD in subject with NIHL.	29 airline technicians with mild to profound NIHL (at 4 kHz)	PTA/ENG	No relation between severity of NIHL and VD.	5/8
(42)	Investigation of the VD in subjects with NIHL.	36 subjects with NIHL (at 4 kHz); 20 subjects with vertigo (60.4±17.1 dB HL) and 16 subjects without vertigo (56.7±12.2 dB HL)	PTA/ENG	A significantly reduction in Caloric test response in 47.1% of the subjects with vertigo.	5/8
(43)	Investigation of VEMPs in worker with NIHL.	18 male and 2 female workers from nine industry with NIHL (26-75 dB HL at 4 kHz).	PTA/cVEMP and ENG	Abnormal Caloric (45%) and cVEMP (50%) responses in workers A significant relationship between hearing thresholds and VEMP responses.	6/8
(44)	Investigation of VEMPs in worker with NIHL.	30 workers with NIHL (25-70 dB HL)/30 healthy subjects (age- and sex-matched).	PTA/cVEMP	A significant association between the abnormal cVEMP (34.6%) and hearing threshold.	6/8

(45)	Investigation of the sequence of vestibular deficits in subjects with NIHL.	30 subjects with NIHL (26-62 dB at 4kHz)/30 healthy subject (age- and sex-matched).	PTA/oVEMP, cVEMP, and Caloric (ENG)	Abnormal cVEMP (70%), oVEMP (57%) and caloric (33%) responses in subject with NIHL.	6/8
(46)	Evaluation of VEMPs in worker with NIHL.	60 workers with NIHL (40-70 dB HL at 4 kHz)/30 aged-match healthy office employees.	PTA/cVEMP	A significant association between the abnormal cVEMP (36.6%) and hearing threshold.	4/8
(47)	Investigation of cVEMP in patients with NIHL.	20 workers with NIHL (42.2±14.6 dB HL at 4 kHz)/20 aged-matched normal subjects.	PTA/cVEMP	A significant association between the abnormal cVEMP (30%) and hearing thresholds.	7/8
(48)	Investigation of cVEMP in workers with occupational noise exposure and HL.	40 textile factory male workers with chronic noise exposure (with normal to moderate hearing loss at 4 kHz)/20 age-matched healthy male subjects without noise exposure.	PTA/cVEMP	A significant association between the severity hearing loss and the abnormal cVEMP responses. A significant association between the abnormal cVEMP responses and sense of imbalance.	7/8
(49)	Evaluation of the SCCs function in industrial workers with NIHL.	36 metal industry workers with NIHL (> 40 dB HL at 4 kHz)/30 healthy men.	PTA/vHIT	A significant SCCs deficit in workers with NIHL (55.5%) versus control group (6.6%).	7/8
(50)	Evaluation of the vestibular function in workers with occupational noise exposure.	60 noise-exposed laundry workers with mild NIHL/20 aged-matched healthy subjects.	PTA/VNG	Normal oculomotor tests in both groups. A significantly higher directional preponderance (17.5% versus 3.9%) and unilateral weakness (16.9% versus 3.8%) among the study group than controls.	6/8
(51)	Evaluation of the saccular function in subjects with NIHL and without vestibular symptoms.	25 male subjects with NIHL/25 matched controls with sensorineural hearing loss and without noise exposure.	PTA/cVEMP and Caloric (VNG)	Significant abnormal cVEMPs in subjects with NIHL. Only a significant difference in cVEMPs amplitude (not latency) between the two groups.	6/8
(52)	Assessment of the vestibular function in subjects with chronic noise exposure.	121 police personnel and automobile drivers with at least 5 years noise exposure /121 healthy volunteers without noise exposure.	VNG and cVEMP	Normal vestibular function in terms of VNG and cVEMP tests in both groups.	6/8
(53)	Evaluation of the effect of chronic noise exposure on the vestibular system	17 textile factory workers with mild to moderately severe NIHL/17 healthy subjects.	PTA/vHIT	Significant abnormal vHIT (lateral canal) in 47% of workers with NIHL. Negative correlation between lateral canal gain and hearing thresholds.	7/8

*Pure Tone Audiometry (PTA), Quality Assessment Score (QAS)

Table 2: A summary of articles on the relationship between NILH with VD in subjects exposed to impulsive noise (n=5)

Ref. No.	Study Aim	Population Covered	Audiological/ Vestibular Examinations	The results showed:	QAS
(20)	Examination of the possible changes in the vestibular system function in subjects with NIHL.	22 navy crews with NIHL(≥25 dB in at least one ear)/21 controls with normal hearing.	PTA/ ENG	A significant reduction in VOR gain and caloric responses in the NIHL group. No significant differences in vertigo symptoms, oculomotor tests, directional preponderance between groups.	7/8
(54)	Evaluation of the vestibular system function in subjects with symmetrical and asymmetrical NIHL and exposed to impulse and impact noise.	258 military personnel with NIHL; in 2 groups (134 with symmetrical and 124 with asymmetrical NIHL) and 2 subgroups (presence or absence of vestibular complaints)/32 healthy men (aged-matched without exposure)	PTA/ENG	A significant abnormal vestibular finding in subjects with asymmetrical (46.8%) than symmetrical (5.2%) NIHL. A strong correlation between the vestibular symptoms (dizziness or vertigo) and abnormal ENG findings in subjects with asymmetrical NIHL. A significant correlation between the subjects' symptoms (80.7%) in the group with asymmetrical NIHL and the ENG abnormal findings.	7/8
(55)	Investigation of the effect of impulsive noise exposure on cochlear and saccular function	12 police officers/12 age-matched healthy men.	PTA/cVEMP	A significant hearing deterioration in the police officers (only at 4 and 6 kHz in the left ear) over the 10-year study period compared with healthy controls. A significant abnormal cVEMP responses (75%) in police officers. No symptoms of vertigo or imbalance in police officers.	7/8
(56)	Investigation of the effects of noise exposure on the cVEMP in individuals with asymmetric NIHL	43 subjects with military noise exposure and asymmetric NIHL/14 age-matched controls with normal hearing.	PTA/cVEMP	A significant abnormal cVEMPs (33%) in NIHL subjects. Dizziness is compliant in 49% of NIHL subjects. A significant relationship between the abnormal cVEMPs and the degree of NIHL.	7/8
(57)	Analysis of the cVEMP results of patients with NIHL.	20 military personnel with symmetrical NIHL (at 4, 6 and 8 kHz); in two groups; group 1; NIHL > 68.3 dB and group 2; NIHL<68.3 dB).	PTA/cVEMP	A significant abnormal cVEMP in-group 2 than group 1 (63% versus 28%). No significant difference in the prevalence of vertigo and dizziness symptoms between the groups. A significant higher abnormal cVEMP in subjects with vertigo/dizziness and lower NIHL.	6/8

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Discussion

This review shows the co-occurrence of vestibular dysfunction and NIHL in the working population exposed to continuous or impulsive occupational noise in almost all included studies. In addition to auditory system, occupational exposure to noise can also be a risk factor for the vestibular system. The simultaneous damage to the auditory and vestibular systems from occupational noise exposure could be explained by the functional and structural similarities of these two systems, including the structural similarity of their receptors, the anatomical proximity of the sound energy transmission system to the vestibular system, and their common blood supply. (Via anterior inferior cerebellar artery) (19, 21, 60).

Cochlear pathologies or hearing loss mechanisms due to noise exposure can be classified into mechanical damage and metabolic damage (61). Mechanical damage occurs at high noise levels and can damage cochlear structures (e.g. damage to stereocilia tips, cochlear vessels, tip links, and supporting cells) by transmitting excessive vibration to these structures (62). Due to the proximity and anatomical connections between the auditory and vestibular organs, the mechanical vibrations generated by noise can therefore be transmitted to the vestibular organs and damage their structures. Animal experiments have reported the loss of sensory stereocilia in the otolith organs and SCCs (24), and the degeneration of epithelial cells and the separation of their layers in the saccular maculae (22) due to mechanical damage at high noise levels (116 and 120 dB sound pressure level). On the other hand, an abnormal VEMP response and its association with histological damage in the saccular organ has also been reported after exposure to 115 dB (63). Therefore, the damage to the end organs of the vestibular system due to mechanical damage from loud noise reported in the studies included in this review (20, 54-57) can be attributed to the proximity and structural and functional similarities of the two organs of auditory and vestibular function.

On the other hand, metabolic changes are the main mechanism of NIHL because of chronic noise exposure. Therefore, metabolic changes including reactive oxygen species (ROS), ischemia, free radicals, reactive nitrogen species (RNS), Lipid peroxidation induced by ROS, and metabolic overloads in the organ of Corti are the main mechanism of cochlear damage due to noise exposure (or continuous noise) (61, 64-66). Chronic exposure to noise leads to the generation of free radicals, such as ROS and RNS, from the mitochondria to the cytoplasm of the hair cells and the production of pre-apoptotic factors and eventual apoptosis of hair cells (62, 65, 66). The free radicals generated can persist for days after exposure has ended, leading to progressive damage to the cochlea (66). Due to the same blood supply pathway of the auditory and vestibular systems, it is therefore to be expected that metabolic changes could induce similar damage to the vestibular sensory receptors (67). In addition, the ototoxicity and vestibulotoxicity effects of drugs such as aminoglycoside (68), and carboplatin (69) have recently been demonstrated. The mechanism of ototoxicity of these drugs has been explained by their entry into the cochlea and hair cells through the bloodstream and cell damage and apoptosis due to ROS formation (70). Due to the longer clearance times of the ototoxic drugs from the inner-ear fluids than from the blood, they can remain in the cochlea for several months (with a half-life of 5 to 6 months) after stopping treatments (71, 72). Due to the anatomical proximity and the structural and functional similarity of the two vestibular and auditory organs, the factors that damage the auditory organs can also damage the vestibular organs (19, 21-24). Therefore, the dysfunction of the vestibular system reported in the studies included in this review could be attributed to the mechanism of metabolic damage due to noise exposure (or continuous exposure to noise) (40, 42-47, 49-51).

On the other hand, the absence of symptoms (vertigo or dizziness) in individuals with vestibular dysfunction is likely due to the compensatory mechanism. Thus, the cerebellum seems to play an important role in initiating this mechanism. Although vestibular compensation plays an important role in maintaining balance, it should not be mistaken with full recovery (67). This is because peripheral vestibular abnormalities can result in decreased performance, particularly in unusual situations such as reduced visual input, use of rapid head movements, and the need for spatial navigation skills (34-37).

The inner ear consists of two portions separated by a thin membrane called the membrana limitans including the pars superior (utricle and SCCs) and the pars inferior (saccule and cochlea) (73). The membrane limitans as a partial barrier protecting the utricle and SCCs from acoustic trauma. In other words, the susceptibility of the saccule to noise-induced damage is due to the anatomical proximity of the saccule to the stapes footplate (sound energy transmission system) (22, 24, 74). Moreover, the pars inferior is more susceptible to damage than the pars superior due to high-intensity noise exposure (63). Therefore, due to differences in the susceptibility of the vestibular portions, one part can precede other parts and this can result in discrepancies between evaluation findings of different portions of the vestibular system.

Limitation

The main limitation of this review is the heterogeneous data that precludes meta-analysis. Heterogeneity includes different examination methods, different diagnostic criteria, variability in industry/workplace population, different age groups and variability in sample size.

Conclusion

Almost all studies in this overview have shown a significant association between NIHL and VD among the workplace population exposed to occupational noise. Because of the crucial role of the function of the vestibular system in maintaining balance and gaze stabilization, hearing protection legislation should therefore be extended to cover the vestibular system of individuals who are at risk of damaging it. In view of the importance of medical competence and fitness for work, especially in professions with high demands on balance and gaze stabilization, it is recommended to supplement the obligatory medical examinations with an assessment of the balance function. However, conclusions from the present study needs to be treated with caution. Further research is needed to investigate the association between the occurrence of VD due to occupational noise exposure or concomitantly with NIHL.

Journalism Ethics considerations

Ethical issues (Including plagiarism, informed consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc.) have been completely observed by the authors.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

References

1. Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, Stansfeld S (2014). Auditory and non-auditory effects of noise on health. *The lancet,* 383:1325-1332. doi: 10.1016/S0140-6736(13)61613-X.
2. Nelson DI, Nelson RY, Concha-Barrientos M, Fingerhut M (2005). The global burden of occupational noise-induced hearing loss. *Am J Ind Med,* 48:446-458. doi: 10.1002/ajim.20223.
3. World Health Organization (2018) Addressing the rising prevalence of hearing loss. World Health Organization. Geneva. Available at https://apps.who.int/iris/handle/10665/260336.
4. World Health Organization (WHO) (2017). *Global costs of unaddressed hearing loss and cost-effectiveness of interventions. World Health Organization. Geneva.* Available at https://apps.who.int/iris/handle/10665/254659. ed. World Health Organization.
5. May JJ (2000). Occupational hearing loss. *Am J Ind Med,* 37:112-120. doi: 10.1002/(SICI)1097-0274(200001)37:1<112::AID-AJIM9>3.0.CO;2-%23.
6. Mirza R, Kirchner DB, Dobie RA, et al (2018). Occupational noise-induced hearing loss. *J*

Occup Environ Med, 60:e498-e501. doi: 10.1097/JOM.0000000000001423.
7. Cui B, Wu M, She X (2009). Effects of chronic noise exposure on spatial learning and memory of rats in relation to neurotransmitters and NMDAR2B alteration in the hippocampus. *J Occup Health,* 51:152-158. doi: 10.1539/joh.L8084.
8. Cheng L, Wang S-H, Chen Q-C, Liao X-M (2011). Moderate noise induced cognition impairment of mice and its underlying mechanisms. *Physiol Behav,* 104:981-988.
9. Manikandan S, Padma MK, Srikumar R, Parthasarathy NJ, Muthuvel A, Devi RS (2006). Effects of chronic noise stress on spatial memory of rats in relation to neuronal dendritic alteration and free radical-imbalance in hippocampus and medial prefrontal cortex. *Neurosci lett,* 399:17-22. doi: 10.1016/j.neulet.2006.01.037.
10. Jáuregui-Huerta F, García-Estrada J, Ruvalcaba-Delgadillo Y, Trujillo X, Huerta M, Feria-Velasco A, Gonzalez-Perez O, Luquín S (2011). Chronic exposure of juvenile rats to environmental noise impairs hippocampal cell proliferation in adulthood. *Noise Health,* 13:286-91.
11. Shi L, Liu L, He T, et al (2013). Ribbon synapse plasticity in the cochleae of Guinea pigs after noise-induced silent damage. *PloS One,* 8:e81566. .
12. Liu L, Wang H, Shi L, et al (2012). Silent damage of noise on cochlear afferent innervation in guinea pigs and the impact on temporal processing. *PLoS One,* 7:e49550.
13. Liu L, Shen P, He T, et al (2016). Noise induced hearing loss impairs spatial learning/memory and hippocampal neurogenesis in mice. *Sci Rep,* 6:20374. doi: 10.1038/srep20374.
14. Moxon KA, Gerhardt GA, Bickford PC, et al (1999). Multiple single units and population responses during inhibitory gating of hippocampal auditory response in freely-moving rats. *Brain Res,* 825:75-85.
15. O'Mara S (2005). The subiculum: what it does, what it might do, and what neuroanatomy has yet to tell us. *J Anat,* 207:271-282.
16. Yu Y-F, Zhai F, Dai C-F, Hu J-J (2011). The relationship between age-related hearing loss and synaptic changes in the hippocampus of C57BL/6J mice. *Exp Gerontol,* 46:716-722.
17. Wayne RV, Johnsrude IS (2015). A review of causal mechanisms underlying the link between age-related hearing loss and cognitive decline. *Ageing Res Rev,* 23:154-166. doi: 10.1016/j.arr.2015.06.002.
18. Uchida Y, Sugiura S, Nishita Y, Saji N, Sone M, Ueda H (2019). Age-related hearing loss and cognitive decline—The potential mechanisms linking the two. *Auris Nasus Larynx,* 46:1-9.
19. Santos TG, Venosa AR, Sampaio ALL (2015). Association between hearing loss and vestibular disorders: a review of the interference of hearing in the balance.*IJOHNS,* 4:173-179.
20. Shupak A, Bar-El E, Podoshin L, Spitzer O, Gordon C, Ben-David J (1994). Vestibular findings associated with chronic noise induced hearing impairment. *Acta Otolaryngol,* 114:579-585.
21. Stewart CE, Altschuler RA, Cacace AT, et al (2020). Effects of noise exposure on the vestibular system: a systematic review. *Front Neurol,* 11:1327.
22. Akdogan O, Selcuk A, Take G, Erdoğan D, Dere H (2009). Continuous or intermittent noise exposure, does it cause vestibular damage?: An experimental study. *Auris Nasus Larynx,* 36:2-6.
23. Fetoni AR, Ferraresi A, Picciotti P, Gaetani E, Paludetti G, Troiani D (2009). Noise induced hearing loss and vestibular dysfunction in the guinea pig. *Int J Audiol,* 48:804-810.
24. Stewart C, Yu Y, Huang J, et al (2016). Effects of high intensity noise on the vestibular system in rats. *Hear Res,* 335:118-127.
25. Besnard S, Lopez C, Brandt T, Denise P, Smith PF (2015). The vestibular system in cognitive and memory processes in mammalians. *Front Integr Neurosci,* 9:55.
26. Whitney SL, Marchetti GF, Pritcher M, Furman JM (2009). Gaze stabilization and gait performance in vestibular dysfunction. *Gait Posture,* 29:194-198.
27. Bigelow RT, Agrawal Y (2015). Vestibular involvement in cognition: Visuospatial ability, attention, executive function, and memory. *J Vestib Res,* 25:73-89.
28. Van der Zaag-Loonen H, van Leeuwen R (2015). Dizziness causes absence from work. *Acta Neurol Belg,* 115:345-349.

29. Bigelow RT, Semenov YR, du Lac S, Hoffman HJ, Agrawal Y (2016). Vestibular vertigo and comorbid cognitive and psychiatric impairment: the 2008 National Health Interview Survey. *J Neurol, Neurosurg Psychiatry,* 87:367-372. doi: 10.1136/jnnp-2015-310319.
30. Brandt T, Schautzer F, Hamilton DA, et al (2005). Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. *Brain,* 128:2732-2741.
31. Smith P, Zheng Y (2013). From ear to uncertainty: vestibular contributions to cognitive function. *Front Integr Neurosci,* 7:84.
32. Yoder RM, Taube JS (2014). The vestibular contribution to the head direction signal and navigation. *Front Integr Neurosci,* 8:32. doi: 10.3389/fnint.2014.00032.
33. Guidetti G, Guidetti R, Manfredi M, Manfredi M (2020). Vestibular pathology and spatial working memory. *Acta Otorhinolaryngol Ital,* 40:72-78.
34. Clack TD, Milburn WO, Graham MD (1985). Ear-Eye reflexes while riding in a car. *Laryngoscope,* 95:182-185.
35. Cohen HS, Wells J, Kimball KT, Owsley C (2003). Driving disability and dizziness. *J Safety Res,* 34:361-369.
36. Wei EX, Agrawal Y (2017). Vestibular Dysfunction and Difficulty with Driving: Data from the 2001–2004 national health and nutrition examination surveys. *Front Neurol,* 8:557.
37. MacDOUGALL HG, Moore ST (2005). Functional assessment of head–eye coordination during vehicle operation. *Optom Vis Sci,* 82:706-715.
38. Moher D, Liberati A, Tetzlaff J, Altman DG, Group P (2009). Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLoS Med,* 6:e1000097.
39. Moola S, Munn Z, Tufanaru C, et al (2017). Chapter 7: Systematic reviews of etiology and risk. *Joanna Briggs Institute Reviewer's Manual. The Joanna Briggs Institute,* 5.
40. Man A, Segal S, Naggan L (1980). Vestibular involvement in acoustic trauma (An electronystagmographic study). *J Laryngol Otol,* 94:1395-1400.
41. Oosterveld W, Polman A, Schoonheyt J (1982). Vestibular implications of noise-induced hearing loss. *Br J Audiol,* 16:227-232.
42. Manabe Y, Kurokawa T, Saito T, Saito H (1995). Vestibular dysfunction in noise induced hearing loss. *Acta Otolaryngol,* 115:262-264.
43. Wang Y-P, Young Y-H (2007). Vestibular-evoked myogenic potentials in chronic noise-induced hearing loss. *Otolaryngol Head Neck Surg,* 137:607-611.
44. Kumar K, Vivarthini CJ, Bhat JS (2010). Vestibular evoked myogenic potential in noise-induced hearing loss. *Noise Health,* 12:191-194.
45. Tseng C-C, Young Y-H (2013). Sequence of vestibular deficits in patients with noise-induced hearing loss. *Eur Arch Otorhinolaryngol,* 270:2021-2026.
46. Giorgianni C, Spatari G, Tanzariello MG, Gangemi S, Brecciaroli R, Tanzariello A (2015). Cervical vestibular evoked myogenic potential (c-VEMPs) assessment in workers with occupational acoustic trauma. *Health,* 7:456.
47. Elbeltagy R, Galhom D (2017). Assessment the effect of noise on the sacculocolic pathway using vestibular evoked myogenic potential. *EJO,* 33:523-7.
48. Abd El-Salam NM, Ismail EI, El-Sharabasy AE-S (2017). Evaluation of cervical vestibular evoked myogenic potential in subjects with chronic noise exposure. *J Int Adv otol,* 13:358-362.
49. Yilmaz N, Ila K, Soylemez E, Ozdek A (2018). Evaluation of vestibular system with vHIT in industrial workers with noise-induced hearing loss. *Er Arch Otorthinolaryngol,* 275:2659-2665.
50. Ali AA, El-Maraghy AA, Ahmed AM, Mohany HA (2019). Vestibular Assessment in Chronic Noise Exposure Subjects. *EJHM,* 76:4469-4473.
51. Viola P, Scarpa A, Pisani D, et al (2020). Sub-clinical effects of chronic noise exposure on vestibular system. *Transl Med UniSa,* 22:19.
52. Das S, Kalidoss VK, Bakshi SS, Ramesh S (2022). A Cross-Sectional Study on the Effect of Chronic Noise Exposure on the Vestibular Function of Traffic Policemen and Automobile Drivers. *Noise Health,* 24:231.
53. Elewa MS, Nada EH, Elnabtity NM (2022). Video Head Impulse Test findings in Patients with Chronic Noise Exposure. *EJENTAS,* 23:1-7.

54. Golz A, Westerman ST, Westerman LM, Goldenberg D, Netzer A, Wiedmyer T, Fradis M, Joachims HZ (2001). The effects of noise on the vestibular system. *Am J Otolaryngol,* 22:190-196.
55. Wu C-C, Young Y-H (2009). Ten-year longitudinal study of the effect of impulse noise exposure from gunshot on inner ear function. *Int J Audiol,* 48:655-660.
56. Akin FW, Murnane OD, Tampas JW, Clinard C, Byrd S, Kelly JK (2012). The effect of noise exposure on the cervical vestibular evoked myogenic potential. *Ear Hear,* 33:458-465.
57. Dalgıç A, Yılmaz O, Hıdır Y, Satar B, Gerek M (2015). Analysis of Vestibular Evoked Myogenic Potentials and Electrocochleography in Noise Induced Hearing Loss. *J Int Adv Otol,* 11:127-32.
58. Oosterveld W, Polman A, Schoonheyt J (1980). Noise-induced hearing loss and vestibular dysfunction. *Aviat Space Environ Med,* 51:823-826.
59. Wang YP, Hsu WC, Young YH (2006). Vestibular evoked myogenic potentials in acute acoustic trauma. *Otol Neurotol,* 27:956-961.
60. Hain TC, Helminski JO (2014). *Vestibular Rehabilitation*. Chapter 1: Anatomy and physiology of the normal vestibular system. 4th ed. *F. A. Davis Company*.
61. Hawkins JE, Schacht J (2005). Sketches of Otohistory Part 10: Noise-Induced Hearing Loss. *Audiol Neurotol,* 10:305-309.
62. Kurabi A, Keithley EM, Housley GD, Ryan AF, Wong AC-Y (2017). Cellular mechanisms of noise-induced hearing loss. *Hear Res,* 349:129-137.
63. Hsu WC, Wang JD, Lue JH, Day AS, Young YH (2008). Physiological and morphological assessment of the saccule in Guinea pigs after noise exposure. *Arch Otolaryngol Head Neck Surg,* 134:1099-1106.
64. Kopke RD, Coleman JK, Liu J, Campbell KC, Riffenburgh RH (2002). Enhancing intrinsic cochlear stress defenses to reduce Noise-Induced hearing loss. *Laryngoscope,* 112:1515-1532.
65. Yamashita D, Jiang H-Y, Schacht J, Miller JM (2004). Delayed production of free radicals following noise exposure. *Brain Res,* 1019:201-209.
66. Henderson D, Bielefeld EC, Harris KC, Hu BH (2006). The role of oxidative stress in noise-induced hearing loss. *Ear Hear,* 27:1-19.
67. Jacobson GP, Shepard NT, Barin K, Janky K, McCaslin DL (2020). *Balance function assessment and management.* 3rd ed. plural publishing.
68. Abbas L, Rivolta MN (2015). Aminoglycoside ototoxicity and hair cell ablation in the adult gerbil: a simple model to study hair cell loss and regeneration. *Hear Res,* 325:12-26.
69. Dalian D, Haiyan J, Yong F, Salvi R, Someya S, Tanokura M (2012). Ototoxic effects of carboplatin in organotypic cultures in chinchillas and rats. *J Otol,* 7:92-102.
70. Rybak L, Ramkumar V (2007). Ototoxicity. *Kidney Int,* 72:931-935.
71. Dulon D, Hiel H, Aurousseau C, Erre J, Aran J (1993). Pharmacokinetics of gentamicin in the sensory hair cells of the organ of Corti: rapid uptake and long term persistence. *C R Acad Sci III,* 316:682-687.
72. Aran JM, Erre JP, da Costa DL, Debbarh I, Dulon D (1999). Acute and chronic effects of aminoglycosides on cochlear hair cells. *Ann N Y Acad Sci,* 884:60-68.
73. Nomura Y (2013). *Morphological aspects of inner ear disease.* 1st ed. Springer Science & Business Media.
74. Hara M, Kimura RS (1993). Morphology of the membrana limitans. *Ann Otol Rhinol Laryngol,* 102:625-630.