



## CERVICAL VESTIBULAR EVOKED MYOGENIC POTENTIALS IN INDIVIDUALS WITH NOISE-INDUCED HEARING LOSS

### Speech & Hearing

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### ABSTRACT

**Background:** NIHL is traditionally regarded as a cochlear pathology; however, emerging evidence indicates that the vestibular system—particularly the saccule—may also be vulnerable to acoustic trauma. **Aim:** To evaluate saccular function and inferior vestibular nerve integrity using cVEMP in individuals with NIHL compared to normal-hearing adults. **Methods:** In this cross-sectional study, 200 participants aged 35–45 years were divided into Group A (n=100; normal hearing) and Group B (n=100; NIHL). cVEMP responses were recorded and analysed. **Results:** The NIHL group demonstrated significantly prolonged bilateral P1 and N1 latencies compared to controls ( $p < 0.05$ ). Responses were largely symmetrical, although mild asymmetry was observed in P1 latency. **Conclusion:** Chronic noise exposure affects the sacculo-collic pathway in addition to cochlear structures, supporting the concept of NIHL as a cochleo-vestibular disorder.

### KEYWORDS

Noise-Induced Hearing Loss (NIHL), Cervical Vestibular Evoked Myogenic Potentials (cVEMP), Vestibular Dysfunction, Saccule

### INTRODUCTION

Noise is an unwanted, unpleasant, undesirable and disturbing sound in nature. Noise can cause severe auditory effect as well as non-auditory effects on human<sup>[1,2]</sup>. Excessive exposure of noise may damage the cochlear system which leads to permanent hearing loss<sup>[3]</sup>. The loss of hair cells in the cochlea, especially the outer hair cells is the major pathogenic feature of noise-induced hearing loss (NIHL)<sup>[4]</sup>. NIHL is an insidious and cumulative problem that worsens over time. It is a consequence of multi-factorial damage to auditory structures following exposure to occupational, environmental, or recreational sources of loud sound<sup>[5]</sup>. The duration and severity of noise-induced hearing loss (NIHL) depend on the extent and location of cellular damage within the auditory system, which are closely related to the intensity and duration of noise exposure<sup>[6]</sup>.

The vestibular system is anatomically and physiologically linked to the cochlea, sharing the membranous labyrinth, similar mechanosensory hair cells, a common vascular supply, and a continuous fluid environment<sup>[7,8]</sup>. The auditory and vestibular organs share functional and structural similarities, including anatomical proximity, interconnected pathways, and a common blood supply to their receptors<sup>[9]</sup>. 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<sup>[10,11]</sup>. The vestibular system is essential for the maintenance of postural balance and gaze stabilization. The most common vestibular dysfunction symptoms are vertigo, unsteadiness, and dizziness<sup>[12]</sup>. It plays an important role in cognitive functions, including spatial memory, navigation, attention, memory, visuospatial ability, executive function, and spatial working memory. Peripheral vestibular disorders can lead to a decline in performance, difficulty with spatial navigation, and slower head movements, especially when driving<sup>[13]</sup>. Body sway also increases in patients with NIHL that suggests sub-clinical disturbances of the vestibular system<sup>[14,15]</sup>.

Vestibular evoked myogenic potentials (VEMP) are short-latency muscle responses triggered by sound, vibration, or electrical stimulation. These responses can be elicited by air or bone conduction, or through vibration or electrical signals<sup>[16]</sup>. VEMPs provide objective evidence of noise-related vestibular dysfunction, with cervical VEMPs reflecting saccular–inferior vestibular nerve pathways. Reliable vestibular activation typically requires high-intensity acoustic stimuli ( $\geq 100$  dB SPL), levels comparable to those known to cause cochlear injury<sup>[17]</sup>. Biomechanical studies further suggest that the saccular membrane may be more susceptible to acoustic stress than cochlear membranes, increasing the likelihood of vestibular involvement during hazardous noise exposure<sup>[18]</sup>.

### Purpose Of The Study:

Noise Induced Hearing Loss (NIHL) has traditionally been regarded as a disorder confined to cochlear pathology. However, emerging evidence suggests that chronic noise exposure may also affect the

vestibular system due to the close anatomical, embryological, and physiological relationship between cochlear and vestibular structures. Therefore, there is a need to systematically investigate vestibular function in individuals with NIHL.

### Aim & Objective Of The Study

To compare cervical vestibular evoked myogenic potentials (cVEMP) responses in normal-hearing and noise-induced hearing loss (NIHL) subjects.

### 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 were bilateral normal hearing sensitivity, presence of Type “A” tympanogram. Group B includes participants with a history of NIHL, bilateral moderate to moderately severe sensorineural hearing loss, “A” type tympanogram. Exclusion criteria were 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.

The subject was asked to lie down at 30 degree on the couch, lift and turn their head in the opposite direction to the stimulus. The surface area of the subject's skin was cleaned using cotton wool with mild abrasive paste to ensure a good contact between the electrodes, the skin and to achieve low impedance. Recording of cVEMP includes four electrode montage which includes two reference electrodes were placed on the upper belly of the SCM muscle on both sides. The vertex electrode was placed on the clavicular joint (or on the high forehead) and the ground or common on the low forehead.

### RESULT

Mean age was comparable between groups (Group A:  $38.9 \pm 3.01$  years; Group B:  $39.0 \pm 2.95$  years). Analyses were performed using SPSS v22 at a 95% confidence level ( $p < 0.05$ ). Descriptive statistics showed prolonged cVEMP P1 and N1 latencies in Group B compared to Group A (Table 1).

Table 1 presents the mean and standard deviation of absolute latencies for cVEMP 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
cVEMP	P1	14.62	0.91	14.80	1.06	18.73	1.83	18.64	1.89
	N1	22.71	1.51	22.87	1.57	28.76	1.89	28.71	2.04

### Within-Group Comparisons

Paired t-tests were used to compare ears. In Group A, although small

but statistically significant inter-ear differences were observed for P1 and N1 ( $p < 0.05$ ), correlations were strong (P1:  $r = 0.76$ ; N1:  $r = 0.91$ ), indicating functional symmetry. In Group B, P1 latencies showed a significant inter-ear difference ( $p = 0.02$ ) with very high correlation ( $r = 0.98$ ), suggesting mild asymmetry, whereas N1 latencies showed no significant difference ( $p > 0.05$ ) despite strong correlation ( $r = 0.97$ ).

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

Independent t-tests (after Levene's test) revealed significant differences in both P1 and N1 latencies bilaterally ( $p < 0.05$ ), with Group B showing prolonged responses. Findings suggest NIHL is associated with altered saccular function, likely due to shared cochlear and saccular hair cell vulnerability to noise exposure.

### 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. In the present study, individuals with NIHL (Group-B) showed significantly prolonged cVEMP P1 and N1 latencies bilaterally when compared to normal hearing individuals (Group-A). Since cVEMP primarily reflects saccular function and inferior vestibular nerve integrity, these findings suggest saccular involvement in NIHL. These results are consistent with the work of Murofushi et al. (1999), who reported that prolonged cVEMP latencies indicate impaired saccular or inferior vestibular nerve conduction. Similarly, Rauch et al. (2004) demonstrated that noise exposure can damage vestibular hair cells due to their anatomical proximity and shared vascular supply with cochlear structures. The bilateral prolong and symmetrical of latencies of cVEMP observed in Group-B aligns with findings by Clinical vestibular studies using VEMPs strongly corroborate this anatomical rationale. Cetinbag-Kuzu et al. (2023) reported significantly elevated cVEMP and oVEMP thresholds and reduced N1–P1 amplitudes in individuals with NIHL. Viola et al. (2020) documented increased VEMP evocation thresholds, while Tseng and Young (2013) observed a graded pattern of vestibular involvement, with the highest abnormality rates in the cochlea and saccule, followed by the utricle and semicircular canals. Giorgianni et al. (2015) found abnormal or absent cVEMP in 64.9% of NIHL subjects, including prolonged latencies and reduced amplitudes.

### CONCLUSION

Chronic acoustic exposure results in vestibular involvement in addition to cochlear damage, supporting the concept of noise-induced hearing loss as a cochleo-vestibular disorder. 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.

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