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An Exploratory Study of Peripheral Vestibular System in Users of Personal Listening Devices

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Background and Objectives: The widespread use of mobile phones and personal listening devices (PLDs) poses potential health risks, particularly noise-induced hearing loss. Among younger generations, high-volume PLD use is associated with auditory and vestibular system changes. Clinical vestibular testing, including vestibular-evoked myogenic potentials (VEMP) and the video head impulse test (vHIT), may reveal peripheral vestibular impacts from prolonged PLD exposure at volumes over 60%. This study examines VEMP and vHIT results in individuals with normal hearing who have had extended high-volume PLD exposure. **Subjects and Methods**: A cross-sectional comparative study was conducted on individuals aged 15−24 years. All the participants had normal pure tone thresholds with "A" type tympanogram, present acoustic reflexes, and history of PLD usage. Participants were divided into groups according to PLD exposure of <1 year (group A), 1.1−2 years (group B), 2.1−3 years (group C), and 3.1−4 years (group D). The output sound pressure level (dB SPL) near the tympanic membrane was measured. Furthermore, cervical VEMP, ocular VEMP, and vHIT were assessed. **Results**: The VEMP and vHIT findings were statistically analyzed and compared across groups. The peak-to-peak amplitudes of VEMP showed a statistically significant difference between groups A and D. **Conclusions**: Potential subclinical damage to the otolith organs can be associated with increased PLD exposure. No damage to the semi-circular canals was observed as the participants used lower dBA values by the PLDs.

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Keywords: Cervical vestibular evoked myogenic potential; Ocular vestibular evoked myogenic potentials; Vestibular systems; Noise; Head impulse tests.

Introduction

Usage of personal listening devices (PLDs) among the younger generation has been associated with hearing difficulties. Exposure to loud sounds can bring about anatomical and physiological changes in the auditory and vestibular systems. The extent and site of cellular harm are linked to both the intensity and duration of the auditory stimulus and it dictates the severity and duration of non-ischemic impairment to the system.

Peng, et al. [1] revealed changes in hearing thresholds between the 3–8 kHz frequency range, which further expanded with extended exposure to PLDs at elevated intensity levels. Similar to this, another study reported minor preclinical harm to the vestibular end organs and auditory system [2]. Santos, et al, [3] found a close relation between hearing loss and vestibular disorders, highlighting changes in one system can cause significant damage in the other. Noise-induced hair cell or neuronal degeneration can cause lasting loss as the mammalian organ of Corti, auditory sensory epithelium, do not spontaneously regenerate once sensory cells are destroyed [4]. Additionally, Yamamoto and Ishii [5] studied Reissner's

membrane of various lower animals such as guinea pigs, frogs, and fish, as well as in humans. The authors reported human saccular membrane with a tensile strength of 36.33 gf/mm^2 and a breakage pressure of 64 mm Hg, while the Reissner's membrane displays a tensile strength of 49.75 gf/mm^2 and a breakage pressure ranging from 106.7 to 142.2 mm Hg. Thus, making the sacular membrane more vulnerable to damage in any form. There are also reports of a substantial reduction of vestibulo-ocular reflex (VOR) gain in individuals with noise induced hearing loss, suggesting vestibular dysfunction due to extended exposure to PLDs at high volume levels [6].

Vestibular end organs and the organ of Corti within the cochlea exhibit structural resemblance to hair cells, which serve as sensory receptors of the inner ear. Both systems transduce hair bundle displacement into neural activity through the shared vestibulocochlear nerve (CN VIII) [7]. Additionally, it is probable that continuous exposure to loud noise can lead to earlier damage to the saccular membrane compared to the Reissner's membrane. This hypothesis is derived from saccular membrane possessing typically lower tensile strength and a lower breaking point, making it more susceptible to damage [5]. Another study on individuals with hearing loss due to noise exposure and without noise exposure confirmed a subclinical damage to the saccule using cervical vestibular evoked myogenic potential (cVEMP) testing—increased thresholds and decreased p1-n1 amplitude [8]. While previous studies have not directly validated the hypothesis, it remains highly plausible that the saccular membrane is more vulnerable. This suggests that exposure to PLDs may lead to vestibular dysfunction even before cochlear dysfunction occurs, highlighting the importance of including a vestibular test battery in clinical setups.

Numerous studies aim to elucidate the impacts of noise exposure on the cochlear function. However, there is a scarcity of literature addressing the direct impact of noise on the vestibular system, potentially attributed to compensatory mechanisms within the central nervous system that compensate for any impairment in vestibular function. Balance problems have a bigger influence on daily activities than hearing loss and have potential to lead to disability which would lower one's quality of life. Vestibular symptoms such as vertigo, imbalance, abnormalities in gait, etc., differ from hearing loss symptoms. Thus, an earliest feasible start to rehabilitation for these symptoms is recommended. Given that the use of PLDs represents a form of recreational noise exposure, it is important to investigate the potential impact of PLD usage on vestibular function. Therefore, it is crucial to assess the peripheral vestibular system in PLD users. The study aimed to generally profile and study the vestibular test findings with varied durations of PLD exposures at >60% of volume among normal hearing individuals.

Subjects and Methods

Subjects

A cross-sectional study design was used to recruit the participants of the study. A total of 810 participants were screened to fulfil the selection criteria of the study using a questionnaire (Supplementary Material in the online-only Data Supplement). This questionnaire collected information on the type of PLD, duration of PLD exposure, subjective volume levels used to listen to PLDs. Upon obtaining the details from the administered questionnaire, 25 participants were selected using convenient sampling method in each group between 18 to 29 years [9], with PLD exposure of various years.

On the basis of duration of PLD use, participants were divided into 4 groups (Fig. 1): Group A, individuals with <1 year of exposure to PLDs at >60% volume level of its maximum limits; Group B, individuals with 1.1–2 years of exposure to PLDs at >60% volume level of its maximum limits; Group C, individuals with 2.1–3 years of exposure to PLDs at >60% volume level of its maximum limits; and Group D, participants with 3.1–4 years of exposure to PLDs at >60% volume level of its maximum limits.

All the participants signed the written informed consent forms without any cost to participate in the study. Approval was obtained from the Institute Ethical Committee on August 17, 2020, with the approval number BNGRC/T/IEC/05/2020-21.

On average, Group A had 5 months, Group B had 1 year and 6 months, Group C had 2 years and 6 months, and Group D had 3 years and 6 months of PLD exposure, respectively. Selection conditions were taken from earlier research on how PLD affected the auditory system [2,10,11]. Participants with uncomfortable loudness levels of >100 dB HL, tympanogram of "A" type, present acoustic reflexes at 500, 1,000, 2,000, and 4,000 Hz, and normal puretone thresholds within 15 dB HL were recruited.

Participants with conductive hearing loss, neuromuscular disorders, autoimmune diseases, and professionally trained dancers and trainers were eliminated from the study. Individuals were instructed not to take any vestibulotoxic medications or muscle relaxants, or to disclose their usage, at least 48 hours before undergoing the test.

Methods

All assessments were carried out in a room with the American National Standards Institute (ANSI) standards for lighting

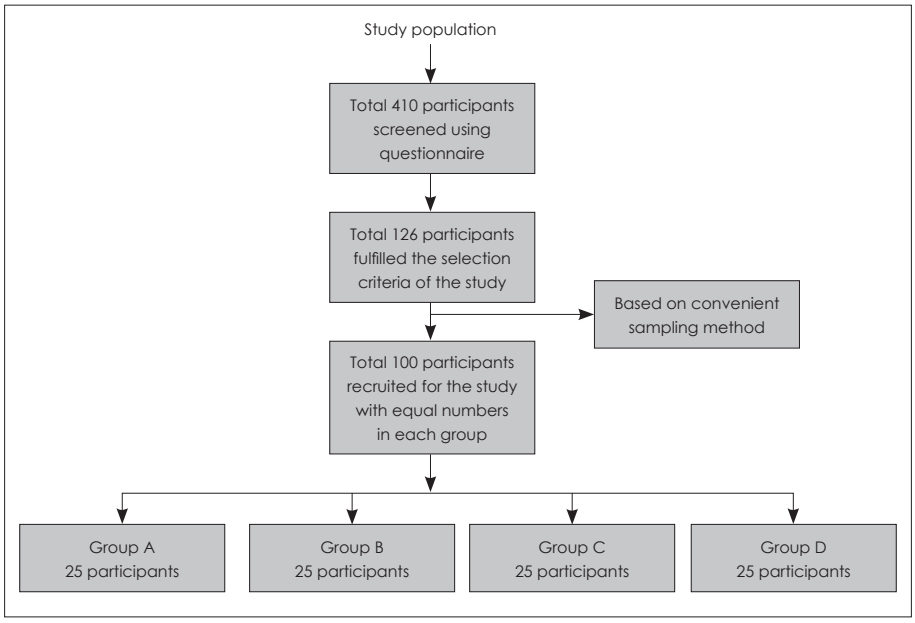

Fig. 1. An algorithm used for recruiting the participants to the study.

and soundproofing [12].

Microphone in the real ear measurement

Objective measurement was used to identify the output sound pressure level (dB SPL) from the PLDs adjacent to the tympanic membrane via a microphone in the real ear (MIRE) measurement [6]. The MIRE procedure ensures the evenness in SPLs through PLDs beyond 60% of volume. The MIRE (objective procedure) supplemented by data obtained from an administered questionnaire (subjective procedure), identified the utilized SPL during the usage of PLDs.

Cervical and ocular vestibular evoked myogenic potential

To obtain vestibular-evoked myogenic potentials (VEMP) from all participants, the Bio-logic Navigator Pro Auditory-Evoked Potential System (Version 7.2.1; Natus Medical Inc., Schaumburg, IL, USA) was employed. Initially, participants were settled on an upright chair with back rest. Subsequently, gold-plated cup electrodes were positioned on the designated recording sites with abrasive Nuprep gel (Weaver and Company, Aurora, CO, USA), to attain absolute electrode impedances (<5 kΩ) and interelectrode impedance (<2 kΩ). Electrodes were carefully affixed with the aid of conductive paste Ten 20 (Weaver and Company) and tape. EMG of sternocleidomastoid muscle contraction was monitored between 100–200 μV and for oVEMP testing, the participant was asked to maintain an upward gaze of 30 degrees. Rectified cVEMP could not be carried out as the software used did not provide rectification for baseline measurements. The protocol for cVEMP [13,14] and ocular vestibular evoked myogenic potential (oVEMP) [15] were adopted from literature.

To evaluate inter-judge reliability and establish accurate peak identification, two experienced audiologists independently analyzed VEMP waveforms. The p1 (positive) and n1 (negative) peaks were identified, and respective peak-to-peak amplitudes, latencies, and interaural difference ratio (IAD), also referred to as the asymmetry ratio (AR), were recorded.

For video head impulse test (vHIT), a target at eye level was fixed at minimum of 1 m in front of them. The participants were told to keep their eyes wide open, relax their neck, and concentrate on the target dot in front of them. Vestibular function of each of the three semicircular canals (SCC) was measured using video goggles (ICS Impulse, Otometrics, Taastrup, Denmark). The vHIT goggles were adjusted to the wearer's head until the least amount of movement was detected at the nasal bridge. To guarantee that head motions would be precisely recorded by the eye, camera location was changed to focus the pupil on video display. Calibration was conducted utilizing a laser fixed at the center of the goggles, under the same lighting conditions as during vHIT assessments. Participants focused a target on the wall while the primary investigator, positioned behind them, administered 20 random head impulses of low-amplitude (10°–20°) for each side and across all three canal planes. Typically, horizontal head velocities ranged from 150°/s to 250°/s, while vertical velocities ranged from 50°/s to 150°/s. Horizontal head turns were initiated from the center for testing the right/left horizontal canal plane.

For right anterior–left posterior (RALP) canal test, participants rotated their head 45° to the left, and impulses were administered by moving the head either backward or forward, with continuous instructions to focus the target. Similarly, for left anterior–right posterior (LARP) canal test, participants turned 45° to the right, and impulses were carried out with head movements either backward or forward [16].

The following parameters were noted for each participant: mean VOR gain of all canals, asymmetry in canals between the ears, and presence of covert saccades.

A standard VOR gain for vHIT is defined as 1.0 [17]. Asymmetry in gain is determined using the formula (gain asymmetry = [left side VOR gain – right side VOR gain]/[left side VOR gain + right side VOR gain]), and the result is expressed as percentage following standardized historical methodologies. Artifacts or invalid impulses resulting from high or low test velocity were also eliminated in accordance with the technique described by Zamaro, et al. [18].

Statistical analysis

Descriptive statistics, Kruskal–Wallis test, and Dunnett C post hoc test were utilized to identify statistically significant differences among groups. Additionally, Spearman's correlation analysis was conducted to determine correlations between PLD exposure duration and parameters of VEMPs across groups.

Table 1. Output sound levels for PLDs across groups

Groups with PLD exposure	Output level (dBA)
Group A	61.43±5.6 (51.67–74.50)
Group B	63.54±1.31 (55.00–76.50)
Group C	68.56±10.18 (55.33–95.84)
Group D	70.01±11.71 (52.50–89.51)

Values are presented as mean±standard deviation (minimum–maximum). PLD, personal listening device; Group A, individuals with <1 year of PLD exposure; Group B, individuals with 1.1 to 2 years of PLD exposure; Group C, individuals with 2.1 to 3 years of PLD exposure; Group D, individuals with 3.1 to 4 years of PLD exposure

Results

The study recorded a 100% response rate. Obtained data was tabulated, and the Shapiro-Wilk test was used to assess normality. Data failed to meet the assumptions of normality (p<0.05). Hence, non-parametric tests were performed to compare the parameters of VEMPs and vHIT across the groups.

To ascertain mean output (dB SPL), the study used MIRE. Descriptive statistics of mean output (dBA) are depicted in Table 1.

Additionally, a statistically significant difference between the groups was found using the Kruskal–Wallis test (χ^2= 10.252, p=0.017). Significant difference was found between Group A and Group D, with a mean difference of 7.13, and between Group A and Group C, with a mean difference of 8.57 (p<0.05). This difference was obtained using Dunnett C post hoc analysis.

Peak-to-peak amplitude of p1-n1, latency of p1 and n1, and AR were tabulated and considered for descriptive statistics and Kruskal–Wallis test to determine the mean, standard deviation, and test statistic of the study parameters. Table 2 displays the findings of parameters across the groups for VEMPs.

Using Dunnett C post hoc analysis, a statistically noteworthy difference was observed for oVEMP peak-to-peak amplitude of n1-p1 between Group A and Group D with a mean difference of 2.03 μV. Similarly, for cVEMP a statistically significant difference was found for peak-to-peak amplitude of p1-n1 between Group A and Group C with mean difference

Table 2. oVEMP and cVEMP findings across the groups

Test parameters	Group A	Group B	Group C	Group D	Test statistic (χ^2)	p value
oVEMP						
n1 latency (ms)	10.27±0.42	10.27±0.52	10.27±0.57	10.27±0.64	0.403	0.966
p1 latency (ms)	14.82±0.63	14.82±0.61	14.82±0.77	14.82±0.71	0.409	0.938
Peak-to-peak amplitude of n1-p1 (μV)	6.38±2.60	5.38±2.71	4.99±2.97	4.34±3.04	19.820	<0.001*
AR (%)	10.31±8.78	10.25±4.35	10.26±15.25	10.30±7.27	0.516	0.915
cVEMP						
P1 latency (ms)	15.04±1.06	15.41±1.40	15.47±0.90	15.15±1.18	5.78	0.122
N1 latency (ms)	23.54±1.21	23.45±1.06	23.79±1.35	23.37±1.68	2.56	0.464
Peak-to-peak amplitude of p1-n1 (μV)	183.98±77.70	176.03±74.7	136.63±76.60	127.17±73.89	23.32	<0.001*
AR (%)	18.68±14.60	19.87±18.38	22.07±17.02	16.95±9.85	1.43	0.697

Values are presented as mean±stadndard deviation. *mean difference is significant (p<0.05). Group A, individuals with <1 year of PLD exposure; Group B, individuals with 1.1 to 2 years of PLD exposure; Group C, individuals with 2.1 to 3 years of PLD exposure; Group D, individuals with 3.1 to 4 years of PLD exposure; cVEMP, cervical vestibular evoked myogenic potential; oVEMP, ocular vestibular evoked myogenic potential; AR, amplitude asymmetric ratio; PLD, personal listening device

Table 3. Mean VOR gain and asymmetry in the semi-circular canals across the groups

Semi-circular canals	VOR gain	Test statistic (χ^2)	*p* value
Left Lateral		6.34	0.09
Group A	0.9±0.06 (0.80−1.03)		
Group B	0.91±0.12 (0.80−1.30)		
Group C	0.93±0.06 (0.84−1.10)		
Group D	0.91±0.05 (0.80−1.02)		
Right Lateral		4.06	0.25
Group A	0.98±0.7 (0.88−1.13)		
Group B	0.96±0.13 (0.78−1.41)		
Group C	0.99±0.73 (0.87−1.13)		
Group D	0.96±0.68 (0.88−1.19)		
Left Anterior		1.449	0.694
Group A	0.9±0.1 (0.80−1.39)		
Group B	0.88±0.09 (0.70−1.13)		
Group C	0.9±0.12 (0.76−1.14)		
Group D	0.91±0.12 (0.80−1.39)		
Right Posterior		1.62	0.65
Group A	0.91±0.12 (0.80−1.30)		
Group B	0.94±0.83 (0.81−1.23)		
Group C	0.90±0.83 (0.81−1.23)		
Group D	0.91±0.12 (0.80−1.30)		
Right Anterior		1.96	0.57
Group A	0.91±0.64 (0.80−1.03)		
Group B	0.88±0.85 (0.78−1.09)		
Group C	0.9±0.7 (0.78−1.08)		
Group D	0.9±0.7 (0.80−1.09)		
Left Posterior		0.64	0.88
Group A	0.91±0.85 (0.78−1.04)		
Group B	0.89±0.72 (0.79−1.03)		
Group C	0.90±0.93 (0.79−1.19)		
Group D	0.91±0.95 (0.76−1.12)		
Lateral asymmetry		0.34	0.95
Group A	6.2±1.51 (3−9)		
Group B	6.12±2.55 (2−11)		
Group C	5.8±2.56 (2−10)		
Group D	6.1±3.17 (1−14)		
Anterior Asymmetry		0.37	0.94
Group A	5.7±2.57 (2−11)		
Group B	5.6±2.5 (2−12)		
Group C	5.92±2.19 (2−11)		
Group D	5.88±3.05 (1−12)		
Posterior Asymmetry		0.95	0.92
Group A	5.56±2.34 (2−10)		
Group B	5.8±2.48 (2−9)		
Group C	5.76±2.27 (2−9)		
Group D	5.44±1.73 (2−11)		

Values are presented as mean±stadndard deviation (range). Group A, individuals with <1 year of PLD exposure; Group B, individuals with 1.1 to 2 years of PLD exposure; Group C, individuals with 2.1 to 3 years of PLD exposure; Group D, individuals with 3.1 to 4 years of PLD exposure. VOR, vestibulo-ocular reflex; PLD, personal listening device

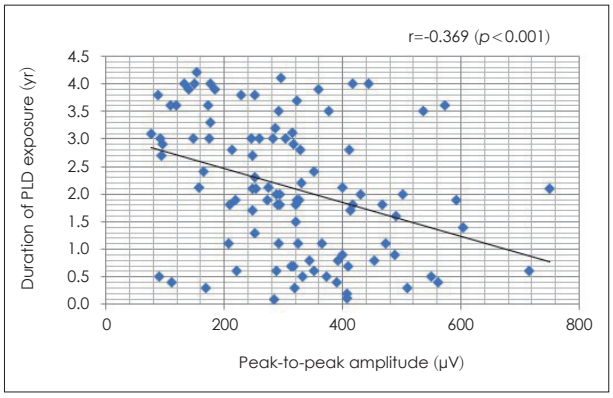

Fig. 2. Correlation between duration of PLD exposure and peak-to-peak amplitude of cVEMP. cVEMP, cervical vestibular evoked myogenic potential; PLD, personal listening device.

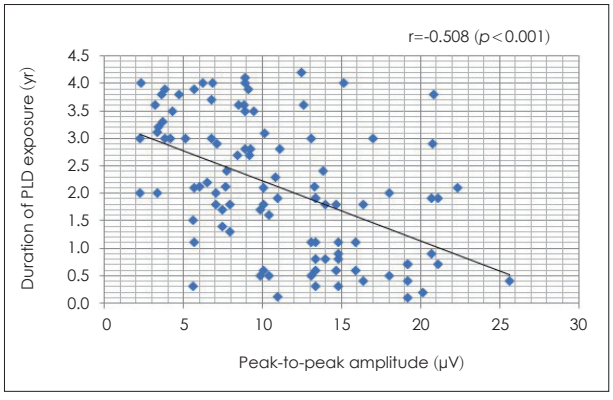

Fig. 3. Correlation between total duration of PLD exposure and peak-to-peak amplitude of oVEMP. oVEMP, ocular vestibular evoked myogenic potential; PLD, personal listening device.

of 47.3 μV, Group A and Group D with mean difference of 56.8 μV, and between Group B and Group D with mean difference of 48.86 μV (p<0.05).

Further, the primary outcome measures assessed in vHIT included the mean VOR gain of each canal and gain asymmetry between the canals of two ears. Table 3 represents the findings of vHIT. A VOR gain cutoff of 0.7 was used and was compared across the four groups [19-21].

In this study, no significant presence of refixation saccades (covert and overt) were observed across the semi-circular testing. Particularly, there was no significant decrease in VOR gain observed in left lateral–right lateral (LLRL), LARP, and RALP over the increasing years of PLD exposure. Likewise, no significant alterations in asymmetry are noted in the anterior asymmetry, posterior asymmetry, or lateral planes across the groups.

As the cVEMP peak-to-peak amplitude and oVEMP peak-to-peak amplitude showcased a notable difference across the groups, we proceeded to analyze the relationship of cumulative PLD usage duration and VEMPs peak-to-peak amplitude through Spearman's correlation analysis. The findings revealed a negative correlation between VEMPs peak-to-peak amplitudes and duration of PLD exposure (Figs. 2 and 3).

Discussion

A response rate of 100% was observed in the study. This finding aligns with an earlier study [22]. However, Singh and Sasidharan [23] indicated that achieving a 100% response rate among PLD users does not essentially specify normal sacculocollic pathway functioning. Instead, it advocates a functional pathway likely owing to exposure to loud sounds bilaterally, as all participants utilized binaural earphones.

Multiple investigations have explored the ill effects of recreational noise on the VEMP pathways. In our study, no identifiable discrepancies in the latencies of VEMP waveforms were detected. Previous studies have suggested delayed latencies in individuals exposed to occupational noise surpassing >90 dBA damage risk criteria (DRC). Similar results are reported for oVEMP findings following noise exposure [24]. These observed delays in latencies of VEMP findings are attributed to variations in the nervous pathway accountable for the waveforms, which include the inferior vestibular nerve and nuclei up to the sternocleidomastoid muscle [25]. Results of our study are consistent with existing literature indicating the absence of neural participation in PLD exposed.

Noteworthy variations in peak-to-peak amplitude were observed in all groups, with a notable trend of decreasing peak-to-peak amplitude of of VEMPs corresponding to increased years of exposure to PLDs (Group D<Group C<Group B< Group A). Research has documented a substantial decline in peak-to-peak amplitude over the course of occupational noise exposure [26,27]. Similarly, Dessai, et al. [28] and Singh and Sasidharan [23] reported similar findings among individuals using PLDs. Observed trend may be ascribed to the stimulation of receptors of macula following acoustic stress to the ears, a phenomenon characteristically lasting from a few hours to days after exposure to any pathology [29].

Reduction in amplitudes of VEMP waveforms across the groups may be linked to subclinical vestibular damage resulting from exposure to recreational noise via PLDs, akin to cochlear damage. Lamm and Arnold [30] explained the reduced amplitude of VEMPs in individuals with NIHL to be due to the vestibular damage associated with NIHL, which, akin to cochlear damage, can be attributed to mechanical injury and metabolic factors such as ischemia, generation of toxic-free radicals, reactive oxygen species, metabolic exhaustion, and ionic imbalance in the inner ear fluid. Furthermore, loud noise damaging the hair cells of cochlea may also impact saccular

macula due to intercommunications of fluid spaces in cochlea and vestibule [31].

Participants in the study used dBA levels, and the results showed a substantial increase in each group (Group D>Group C<Group B<Group A). Nonetheless, used dBA levels by all participants remained below the DRC, this still poses a potential for subclinical vestibular system damage. Prolonged exposure to PLDs is frequently associated with diminished excitability of the saccule and utricle. Resemblance in the structure of hair cells between the cochlea and vestibule, along with the shared blood supply to both cochlear and vestibular end-organs by the same artery, suggests a potential correlation between factors damaging the cochlea and deterioration in the saccule and utricle. Additionally, Tamura, et al. [32] documented peripheral vestibular pathology associated with lengthy exposure to lesser sound intensities. Similarly, these authors reported a reduction in hair cells of vestibular system and a rise in oxidative stress following exposure to sound level of 70 dB SPL for one month. Likewise, McCabe and Lawrence [33] noted damaging effects occurring within minutes of exposure to loud sound levels.

In addition, the cochlear and vestibular receptors originate embryologically in a similar manner and possess a fundamental shared structure: a hair cell that synapses with a primary sensory neuron. However, the susceptibility of the upper part (cochlea and saccule) contrasts with that of the lower part (utricle and SCC), likely due to the presence of a membrane limitans acting as a barrier between the upper and lower parts. This barrier potentially slows the transfer of toxic substances from the lower part to the upper part [34].

Furthermore, AR showed no significant difference between groups. Binge-watching or attending to music represents a form of noise exposure, thus enabling comparison of the findings with older literature on the impact of occupational noise exposure on cVEMP. Literature documents AR (<40%) in around 70% of individuals with noise-induced hearing loss exhibit, although these individuals exhibited asymmetrical hearing loss. Hence, the results of this study are consistent with those reported by Akin, et al. [26]. Absence of noteworthy asymmetry in VEMPs may be credited to the constant practice of listening to PLDs via earbud earphones in both the ears at same intensities, leading to a symmetrical consequence of noise exposure [23]. This theory finds additional support in research related to functioning of cochlear and PLD exposure. These investigations have shown a bilaterally symmetric increase in the threshold and a decrease in the otoacoustic emissions' amplitude [35].

Additionally, the amplitude of VEMPs and the length of noise exposure were negatively correlated. Amplitude decreases as the noise exposure duration increases. This makes sense because the length and intensity of the noise exposure seem to have a significant impact on the amount of noise effect on cochlear blood flow. Anterior and posterior vestibular arteries, which all stem from the labyrinthine artery, supply blood to the otolith while the common cochlear artery supplies the majority of the blood supply to the cochlea. For this reason, decreased blood flow during prolonged and intense noise exposure may have caused abnormal VEMPs.

Further, McGarvie, et al. [36] recommended normative value for VOR gain to be 0.8–1.2 and 0.7–1.2 for lateral and anterior/posterior canals, respectively [37,38]. Similarly, the accepted amount of asymmetry for normal individuals between the two ears must range from 2% to 15% for lateral, anterior, and posterior canals [39]. In our study, VOR gains and amount of asymmetry were within this normative range across all groups. However, no statistically significant difference in mean VOR gains was observed across the groups. Lack of a significant relationship between the usage of PLDs and the mean VOR can be attributed to the decreased SPLs used by the participants on PLD. Loud SPLs were recorded in SCC by Maxwell, et al. [40] in response to sound stimulation, indicating that the SCCs and cochlea receive equivalent amounts of acoustic energy. Hence, low-intensity sounds not exceeding DRC are less likely to cause SCC damage. In our study, the mean output near the tympanic membrane from PLDs was observed at 70.01 (±11.71) dBA in group D, which can less likely cause SCC damage.

Vestibular symptoms were not reported by any of the participants. This might be due to the damage caused bilaterally or the gradual onset of damage, potentially enabling central compensation. Comparison across the groups revealed a reduction in amplitude of VEMP waveforms with increasing exposure to PLD. The brain's compensatory mechanism explains the findings of this study. Subclinical vestibular system damage could therefore not be clinically or functionally apparent in the early stages of injury. Individuals with progressive, persistent exposure-related vestibular illness may not have any vestibular symptoms at all. It may be possible to explain the low occurrence of the clinical symptomatology by considering the ability of the central nervous system to adapt for peripheral vestibular dysfunction. Nystagmus, vertigo, and other autonomic symptoms linked with vestibular insufficiency can be eliminated with complete central vestibular compensation by restoring vestibular nuclei with tonic activity [41]. Moreover, it is possible that the impact of PLDs did not inflict severe damage to the peripheral vestibular system, leading to only diminished responses in VEMPs rather than complete absence.

Limitations of the study

The COVID-19 pandemic necessitated the widespread use of PLDs for various purposes, posing challenges in identifying subjects with consistent exposure durations. Consequently, participants using PLDs with yearly exposure were selected for the study. Furthermore, aligning participants' exposure durations to a specific timeframe proved impractical due to urgent demands during the COVID-19 pandemic and further; making it difficult to meet the set criteria.

Conclusion

The study findings indicate that extended use of PLDs may have a detrimental effect on the utricular and saccular function. Through investigating four groups with differing durations of PLD use, the research highlights the correlation between exposure duration and damage to this pathway. Notably, the effects of prolonged PLD exposure may resemble those seen in cases of occupational noise exposure. Consequently, caution is needed, particularly for younger population. As a precautionary step, it is suggested to restrict daily PLD usage to under one hour, maintaining levels below 60% of the volume, to mitigate possible damage to the utricle and saccule.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.7874/jao.2024.00164.

Conflicts of Interest

The authors have no financial conflicts of interest.

Author Contributions

Conceptualization: all authors. Formal analysis: Teja Deepak Dessai. Methodology: all authors. Supervision: Kaushlendra Kumar, Rashmi J. Bhat. Writing—original draft: all authors. Writing—review & editing: all authors. Approval of final manuscript: all authors.

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None

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