The Effect of Noise Exposure on the Vestibular Systems of Dental Technicians

Safa Alqudah
Department of Rehabilitation Sciences, Faculty of Applied Medical Sciences, Jordan University of Science and Technology, Irbid, Jordan

Abstract

Background: Noise exposure is the primary cause of acquired hearing loss in several occupational settings, including dental laboratories and clinics. However, the impact of noise exposure on the vestibular system is not as well researched. Purpose: To investigate the nature of vestibular damage caused by working in dental laboratories and clinics with high levels of noise exposure due to loud dental equipment. Research Design: A descriptive, case study design was used to evaluate the vestibular function of dental technicians. Study Sample: Out of 30 dental technicians, 5 males who had been working for several years in dental settings were selected based on their reports of severe symptoms of imbalance. Data Collection: Audiologic evaluations were conducted in the vestibular unit of the Doctor Tarek Khrais Center in Amman, Jordan, for one year. Each subject underwent several hearing tests, which included otoscopic examination, pure tone audiometry (PTA), impedance measurements, and speech testing. Assessment of vestibular function was then conducted using a diagnostic test battery which included electrocochleography, ocular vestibular evoked myogenic potentials (oVEMP), cervical vestibular evoked myogenic potentials (cVEMP), positional testing using the Thomas Richard-Vitton (TRV) chair, and standing stability testing. Results: All test subjects experienced some form of vestibular impairment, including benign paroxysmal positional vertigo (BPPV), endolymphatic hydrops (Meniere disease), or a combination of both. Three out of five cases displayed little or no hearing loss, indicating that vestibular function is more at risk than hearing acuity to continuous noise exposure in dental settings. Conclusions: Exposure to loud noise in dental laboratories severely impacts the functioning of the vestibular system of the inner ear more than the cochlea. The main clinical implication of this study is that regular vestibular assessments are a necessity for dental technicians.

Keywords: Benign paroxysmal positional vertigo dentistry, endolymphatic hydrops, hearing loss, meniere disease, noise, vestibule

INTRODUCTION

The two most common causes of permanent hearing loss are age-related hearing loss, termed presbycusis, and overexposure to loud noise.1,2 Continuous exposure to excessive noise is a growing and serious health issue.3,4 When the auditory system is exposed to loud sound, permanent damage to the hair cells of the inner ear and the auditory nerve may occur, causing a noise-induced hearing loss (NIHL).5,6 Occupational noise exposure
results in 28% of NIHL cases among workers, which affects their speech comprehension and communication competency and causes substantial health, social and economic challenges.[7-10]

Based on numerous occupational health studies, dental clinics and laboratories are considered high-noise environments.[11,12] Exposure to loud noises in these settings is a significant cause of NIHL.[13] A typical audiologic manifestation of NIHL among dentists and dental auxiliaries during their hearing assessments is a notch in their audiogram within the high frequencies (4000–6000 Hz). This is most often associated with the death of the hair cells of the cochlea and spiral ganglion degeneration of the auditory nerve.[14,15]

Typically, noise levels in the range of 60–99 dBA are generated by dental instruments such as clinical hand-pieces, turbines, and various other pieces of laboratory equipment. Dental technicians are regularly exposed to both scattered and constant reverberating sounds produced by high-speed (88–102 dBA) and low-speed (70–74 dBA) handpieces, high-speed (75 dBA) and slow-speed (77 dBA) suction devices, ultrasonic scalers (107 dBA), and ultrasonic instrument scalers.[16-18]

Acoustic injuries to the ears, as well as anatomical deterioration of the auditory and vestibular sensory organs, are common amongst dental auxiliaries. Messano and Pettit[19] found that dental specialists are more prone to hearing loss than other physicians. These findings agree with the work of Zubick and colleagues[20] from 20 years prior. Furthermore, the use of dental devices, mainly handpieces, causes temporary threshold shifts (TTS) in students working inside dental laboratories which may lead to permanent threshold shifts and hearing loss. Thus, the risk of acquiring NIHL among dentistry professionals increases when noisy equipment is used.[15]

Previously, experiments were used to demonstrate that exposure to intense stimulation of 100 dB (A) leads to more severe damage to the sacculus and Reissner’s membrane of the vestibular system than to the hair cells of the cochlea.[21,22] Additionally, histological samples of the utricle, sacculus, and semicircular canals of the vestibular system revealed significant and abnormal tissue damage following the exposure to loud noise, which suggests that intense noise exposure compromises not only the hearing system but the entire inner ear as well.[23,24] This includes the vestibular structures.

Two previous studies have examined the clinical manifestations of vestibular insult resulting from chronic noise exposure. One of these is a case report by Van der Laan,[25] which revealed that all subjects with a history of noise exposure were later diagnosed with Meniere disease (MD), a progressive vestibular disorder characterized by sudden episodes of vertigo, tinnitus, and fluctuating hearing loss. The other was a cross-sectional study that aimed to assess the integrity of the vestibular system among workers exposed to chronic noise in a printing industry. Among these, 24% reported vertigo attacks, 13% had peripheral insults, and 7% were diagnosed with benign paroxysmal positional vertigo (BPPV).[26] These results imply that vestibular dysfunction may occur in workers following chronic noise exposure.

While many studies have investigated the effects of NIHL, few have focused on dentistry professionals in particular, despite the prevalence and significant impact of noise exposure in this population. The present study is one of only a few which aims to determine whether working with noisy equipment in dental clinics and laboratories negatively impacts vestibular function.

**METHODS**

Five male employees in dental technology laboratories in Amman, Jordan who were regularly exposed to occupational noise were selected for the current study. All participants signed a written informed consent form supported by the Institutional Review Board (IRB) of the Jordan University of Science and Technology. After obtaining consent from each patient, they were asked to complete a questionnaire to assess their workplace concerning the exposure to noise. The questionnaire assessed exposure time, types of noise-generating devices in the environment, precautions used to reduce harmful exposure effects, and vestibular signs and symptoms aggravated by continued work in the same noisy environment.

All subjects underwent a comprehensive hearing and vestibular assessments at Dr. Tarek Kharis’ Clinic in Amman, Jordan. The diagnostic test battery was conducted by qualified audiologists inside soundproof audiology booths and included hearing examinations by otoscopy, tympanometry, and air and bone conduction audiometry as per American Speech-Language-Hearing (ASHA) guidelines. Hearing thresholds at the frequencies most relevant to speech (250, 500, 1000, 2000, 4000, and 8000 Hz) were determined by a pure tone audiometer, and the ultra high frequencies were assessed for the purposes of this study. These signals were delivered via two earphone transducers over the subjects’ ears. Their baseline thresholds were established and recorded on audiograms. Bone conduction thresholds were obtained at 500, 1000, 2000, and 4000 Hz to identify the type of hearing loss.

Hearing loss was diagnosed when the pure tone average (PTA) of thresholds at tested frequencies exceeded 25 dB in one or both ears. The following hearing loss (HL) severity classifications were made [dB (HL)]: mild (26–40), moderate (41–55), moderately severe (56–70), severe (71–90), or profound (>90). Since hearing damage can differentially affect auditory system elements, hearing loss detected in our participants was defined as conductive, sensorineural, or mixed, based on the audiogram findings.
To investigate the health of vestibule, we conducted several vestibular function tests, starting with electrocochleography which assesses the electrical activity that originates from the cochlea and vestibulocochlear nerve to detect MD. Cervical Vestibular Evoked Myogenic Potentials (cVEMP) and Ocular Vestibular Evoked Myogenic Potentials (oVEMP) were next used to assess the integrity of the otolithic organs. Postural steadiness, stability, altered body movements, and sensory inputs (vision, somatosensory, and vestibular) were assessed to identify any balance disorders. We finally checked for positional nystagmus caused by BPPV by applying positional testing using the Thomas Richard-Vitton (TRV) chair.

RESULTS

Patient 1
Age: 23 years
Gender: Male
History: Imbalance and tinnitus started 3 years ago
Noise exposure duration: 4 years
Pure tone audiometry: Normal hearing bilaterally
Tympanometry: Type A bilaterally (normal)
Electrocochleography: No indication of MD
cVEMP and oVEMP: Normal bilaterally
Positional testing using (TRV) chair: Paroxysmal mixed vertical and torsional (upward/rightward) nystagmus while testing the lateral and posterior semicircular canals
Standing Stability: The patient scored 77.63% while standing normally with eyes closed compared to an age-matched average score of 92.20% (sway to the right side)
Diagnosis: BPPV caused by defects in the lateral and posterior semicircular canals
Treatment: Epley Maneuver and Log Roll Maneuver for BPPV

Patient 2
Age: 23
Gender: Male
History: Transient vertigo with tinnitus started one year ago.
Noise exposure duration: 2 years
Pure tone audiometry: Normal hearing bilaterally
Tympanometry: Type A bilaterally
Electrocochleography: No indication of MD
cVEMP and oVEMP: Normal bilaterally
Positional testing using (TRV) chair: Right beating nystagmus while testing the posterior semicircular canals
Standing stability: The patient scored 85.11% while standing normally with eyes closed compared to an age-matched average score of 91.90% (sway to the left side).
Diagnosis: BPPV due to impaired anterior and lateral semicircular canals of the left ear.
Treatment: Log Roll and Head Hanging Maneuvers for treating BPPV.

Patient 3
Age: 47
Gender: Male
History: Vertigo lasting for minutes accompanied by hearing loss, aura symptoms, and a feeling of fullness in the ear. These symptoms started one year ago.
Noise exposure duration: 24 years
Pure tone audiometry: Hearing threshold at 16,000 Hz was 40 dBHL bilaterally
Tympanometry: Type A bilaterally
Electrocochleography: No indication of MD
cVEMP and oVEMP: Normal bilaterally
Positional testing using (TRV) chair: Left beating nystagmus while testing the anterior and lateral semicircular canals
Standing stability: The patient scored 85.11% while standing normally with eyes closed compared to an age-matched average score of 91.90% (sway to the left side).
Diagnosis: BPPV due to impaired anterior and lateral semicircular canals of the left ear.
Treatment: Log Roll and Head Hanging Maneuvers for treating BPPV.

Patient 4
Age: 44
Gender: Male
History: Paroxysmal and positional vertigo dimensioned with vision stabilization, hearing loss, and severe sensation of tinnitus. These symptoms started many years ago.
Noise exposure duration: 21 years
Pure tone audiometry
Right ear: Normal hearing except for a mild drop at 12,000 and 16,000 Hz
Left ear: Slight sensorineural hearing loss with a mild drop at 12,000 and 16,000 Hz
Tympanometry: Type A bilaterally
Electrocochleography: MD in the right ear
cVEMP and oVEMP: Absent oVEMP bilaterally

Standing stability: The patient scored 81.83% while standing normally with eyes closed compared to an age-matched average score of 92.20% (sway to the right side)
Diagnosis: BPPV resulting from damage to the posterior semicircular canal of the right ear.
Treatment: Epley Maneuver for BPPV.
follow-up for endolymphatic hydrops

Epley and Log Roll Maneuvers for BPPV with Treatment:

Diagnosis: BPPV and MD in the right ear

average score of 91.90% (sway to the right side).

Standing stability: The patient scored 89.17% while standing with eyes closed compared to an age-matched average score of 91.90% (sway to the right side).

Diagnosis: BPPV and MD bilaterally and closed.

Treatment: Epley and Log Roll Maneuvers for BPPV with follow-up for endolymphatic hydrops

Patient 5

Age: 32

Gender: Male

History: Severe positional vertigo when moving head and neck and imbalance for many years.

Noise exposure duration: 10 years

Pure tone audiometry: Normal hearing bilaterally except for a moderate drop at 12,000 and 16,000 Hz.

Tympanometry: Type A bilaterally

Electrocochleography: MD bilaterally

cVEMP and oVEMP: Normal and present bilaterally

Positional testing using (TRV) chair: No provoked nystagmus or vertigo when testing any semicircular canals in either ear.

Standing stability: Stability scores matched age-matched average scores when the patient stood with their eyes open and closed.

Diagnosis: MD bilaterally

Treatment: Follow-up for MD

Basic characteristic of the investigated patients are shown in Table 1. Out of 30 dental professionals, five males were chosen based on self-reporting for symptoms of significant imbalance to evaluate their hearing and vestibular functions. Mild hearing insults were diagnosed in some cases (three out of five), especially at high frequencies (40 dBHL at 12,000 and 16,000 Hz). All patients showed normal cVEMP recordings in response to a 500 Hz tone burst presented at the level of 100 dB nHL [Figure 1]. Regarding the oVEMP evaluations of four patients, the initial peaks of oVEMP (P1) occurred at about 9–17 msec followed by negative peaks (N1) occurring at about 12–25 msec, which suggested normal latencies values of oVEMP. However, P1 and N1 were not appeared in collected traces from a one subject, indicating a deviant utricle and superior nerve response [Figure 2]. The ECoChG recordings were from tympanic membrane to click presented in alternating polarity at 100 dB nHL. For three patients, the ratios of the summating potentials (SP) and action potential (AP) amplitudes (<0.4), and areas (<1.5) were normal. However, two cases indicated positive ECoChG findings; one showed abnormal high SP/AP amplitude and area ratios in the left ear (0.9 and 5.1, respectively) while the other was reported to have abnormal SP/AP amplitude (>0.4) and area (>1.5) ratios bilaterally [Figure 3]. The nystagmus was provoked in four out of five participants during positional testing using (TRV) chair. The characteristics of observed nystagmus were varied among cases to include direction-fixed, torsional with up-beating component, horizontal, short lasting, and appeared significantly when testing the lateral and posterior semicircular canals. As performing normal standing paradigm with eyes closed (NS-EC), which is a task of sensory organization test, the same patients scored 77.63%, 81.83 %, 85.11%, and 89.17%, respectively; these results were lower than age-matched average scores and highlighted the presence of abnormal positional sways when the cases were standing on a firm surface with eyes-closed condition.

DISCUSSION

Prior studies have linked noise exposure to the onset of NIHL in many professionals.[127-29] This link is particularly clear in dentistry.[11,12,17,20,30,31,32] However, understanding the potential impact of cumulative noise exposure on the peripheral vestibular system due to occupational activities remains unclear. Thus, the present study assessed the harmful vestibular consequences of exposure to noisy work environments, such as dental clinics. This study involved assessing the vestibular functions of five dental technology workers. It revealed vestibular disorders ranging

Table 1: Gender, exposure, symptoms, and main results of audiologic and vestibular tests of five dental technicians

| No. | Gender | Exposure(years) | Symptoms | PTA | ECoChG | oVEMP | cVEMP | Positional test |
|-----|--------|----------------|----------|-----|--------|-------|-------|----------------|
| 1   | Male   | 4              | Dizz/ tinn | Bil/norm | Bil/neg | Bil/norm | Bil/norm | Nyst/LSCC & PSCC/AD |
| 2   | Male   | 2              | Vert/ tinn | Bil/norm | Bil/neg | Bil/norm | Bil/norm | Nyst/PSCC/AD |
| 3   | Male   | 24             | HL/vert/ aural | Bil/NIHL | Bil/neg | Bil/neg | Bil/neg | Nyst/LSCC & ASCC/AS |
| 4   | Male   | 21             | HL/vert/ tinn | Bil/NIHL | AD/pos AS/neg | Bil/abs | Bil/norm | Nyst/LSCC & PSCC/AD |
| 5   | Male   | 10             | Vert/dizz | Bil/NIHL | Bil/pos | Bil/norm | Bil/norm | Bil/norm |

PTA = pure tone audiometry; ECoChG = electrocochleography; oVEMP = ocular vestibular evoked myogenic potentials; cVEMP = cervical vestibular evoked myogenic potentials; dizz = dizziness; tinn = tinnitus; vert = vertigo; HL = hearing loss; Bil = bilateral; norm = normal; NIHL = noise induced hearing loss; neg = negative; pos = positive; AD = right ear; AS = left ear; abs = absent; nyst = nystagmus; LSCC = lateral semicircular canal; PSCC = posterior semicircular canal; ASCC = anterior semicircular canal.
from BPPV, MD, or a combination of both. Mild hearing insults were reported in some cases (three out of five), suggesting that the vestibular structures of the inner ear are more vulnerable to damage caused by long-term exposure to loud noise created by dental tools than the cochlea.

NIHL and BPPV in dental practitioners

Here, we discuss the possibility that two forms of vestibular diseases in our examinees, BPPV and MD, are caused by a lack of protection of the hearing and balance organs of the inner ear against ongoing exposure to occupational noises in dental clinics and laboratories. We found that BPPV occurred in three out of five cases and was likely formed by acoustic trauma.\(^{[33]}\) BPPV due to noise-related damage occurs when loud sounds cause abnormally strenuous vibrations in the stapes of the middle ear, which is attached to the otolith organs.\(^{[34]}\) With excessive vibration of the stapes, the otoconia may detach from the otolith membrane and migrate to the other balance structures inside the inner ear, most commonly the posterior semicircular canal. Vertigo and imbalance may then occur when the direction of the head changes since this causes the hair cells in the semicircular canals to deflect the otoconia through the action of gravity.\(^{[35]}\) A study by Wang and Young\(^{[36]}\) revealed that patients with NIHL of more than 40 dBHL at 4 kHz might exhibit delayed or absent cVEMP, which reflects abnormal saccule functioning and sacculocolic reflex pathway activity. Kumer (2010) has found that out of 55 pairs of noise-exposed ears, cVEMP was absent in 16 (29.0%).\(^{[37]}\) CVEMP records were normal in twenty (36.4%) pairs; however, the latency of 19 (34.6%) pairs was delayed, and the peak to peak amplitude reduced. Additionally, Wang and Young\(^{[21]}\) studied the caloric and cVEMP responses in 20 patients with chronic NIHL and...
noise-induced hearing loss, indicating as bilateral notched audiogram at 4 kHz. Caloric and cVEMP tests were recorded as abnormal responses in 9 (45%) and 10 (50%) subjects, respectively. Unfortunately, our study’s cVEMP and oVEMP results are inconsistent with the published literature, since the latencies of the positive peak of response (P13) and negative peak (N23) were within normal ranges for all patients except patient 4, whose oVEMP was absent bilaterally. No substantial clinical otolith organ outcomes were noted in our study despite being reported elsewhere in the literature.[21] A disagreement between our findings and others may be attributed to all our participants having normal hearing or hearing impairment that did not exceed 40 dBHL.

**NIHL and MD in dental practitioners**

Several studies have investigated the relationship between NIHL and MD. Pulec[38] indicated that among 120 patients with MD, 3% developed the disease after acoustic or physical injury. Paparella and Mancini[39] reported a further 37 cases of MD caused by acoustic trauma, and Liard and colleagues[40] similarly studied 93 patients with unilateral MD and found that 5% also complained of MD symptoms after exposure to loud sounds. In another study, Ylikoski[41] tracked the history of 18 army soldiers diagnosed with NIHL and found that their risk of experiencing balance symptoms resembling those of MD was 50 to 200 times higher than in a normal population. In a study by Okuno et al.[42] vestibular system functioning was assessed in 475 soldiers, with 1.4% exhibiting MD.

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**Figure 2:** oVEMP recordings from noise exposed dental technicians. Left panel shows oVEMP responses recorded from the right ears of Patient 1 (top) and Patient 4 (bottom) and right panel shows oVEMP responses recorded from the left ears of Patient 1 (top) and Patient 4 (bottom). The stimulus was 500 Hz presented at the level of 100 dB nHL. For Patient 1, the initial peaks of oVEMP (P1) occurring at about 10 msec followed by negative peaks (N1) occurring at about 15 msec, which suggests normal latencies values of oVEMP. However, P1 and N1 are not appeared in collected traces from Patient 4, indicating a deviant utricle and superior nerve response. Recordings from Patient 2, 3, and 5 were normal but not included.
The link between NIHL and vestibular disease is unclear, though some recent work has sought to clarify it. Manabe et al. electrocochleographically and electronystagmographically assessed 36 patients with NIHL and NIHL with vestibular symptoms. They found that vertigo in individuals with NIHL was likely produced by an underlying pathological process parallel to that of MD. Two of our patients also developed MD, likely due to daily noise exposure in the dental laboratory. This conclusion was supported by studies demonstrating the causative links between NIHL and MD. For example, Kimura mentioned that 38% of animals developed MD after acoustic trauma, explaining that the inflammatory reactions coming from noise exposure caused endolymphatic hydrops in the apical ends of the cochlea. Another group of researchers suggested the opposite direction of the cause-and-effect relationship between NIHL and MD: Nakai et al. conducted endolymphatic sac obstruction to generate unilateral MD in guinea pigs. After exposing these animals to PTS stimuli for two hours, they reported histological alternations in the ears with MD compared to the normal contralateral ears.

Despite the links between NIHL and MD, some studies have been less conclusive. One very large study of 17,245 veterans revealed no correlation between the incidence of NIHL or acoustic trauma and MD. In this retrospective study, Segal and Eviator used medical history records of patients to confirm the presence of MD, without the clinical assessments as we did. The limitation of this evaluation method is its dependence on the patient’s subjective symptoms. Researchers have also noted that the prevalence of MD in the general population is relatively low, which may further obscure the relationship between NIHL and MD.

**Figure 3:** ECochG results from noise exposed dental technicians. Left panel shows ECochG responses recorded from the right ears of Patient 3 (top), Patient 4 (middle) and, and Patient 5 (bottom), and right panel shows ECochG responses recorded from the left ears of Patient 3 (top), Patient 4 (middle), and Patient 5 (bottom). The recordings were from tympanic membrane to click presented in alternating polarity at 100 dB nHL. For Patient 1, the ratios of the summating potentials (SP) and action potential (AP) amplitudes (<0.4), and areas (<1.5) were normal. In contrast, Patient 4 showed abnormal high SP/ AP amplitude and area ratios in the left ear (0.9, 5.1, respectively) while the contralateral ear demonstrates normal SP/ AP amplitude and area ratios. Both ears of Patient 5 were reported to have abnormal SP/ AP amplitude (>0.4) and area (>1.5) ratios. All measurements were made with a reference of base line (BL). ECochG traces from Patients 1 and 2 were normal but not included.
definition of their MD symptoms, which sometimes misleads audiologists and ENT physicians to inaccurately diagnose MD, especially without conducting an audiometric examination as well. Thus, the authors most likely missed the causative relationship between NIHL and MD due to this incomprehensive assessment method.

Two of our subjects serve as particular examples of the co-occurrence of MD with BPPV. Some studies have suggested that the floating otoconia of the otolith organs are responsible for the symptoms of BPPV, thus potentially blocking the passage of duct reunions and causing endolymphatic fluid to accumulate gradually, leading to MD.

Therefore, patients with BPPV are at an elevated risk of developing MD.

**Vestibular disorder is more prominent than hearing damage in exposed dental workers**

Studies in animal models have found that degeneration in the vestibular regions of the inner ear occurs more rapidly than in the auditory regions following exposure to loud noise exposure. Furthermore, the saccule and Reissner’s membrane may be damaged immediately following exposure to loud noise; preceding any cochlear injury that occurs. The cellular composition of the utricle, saccule, and semicircular canals may also become disorganized with continuous, prolonged exposure to excessive noise, resulting in irregular auditory and vestibular organ anatomy. Otolith destruction is caused by the obstruction, and subsequent rupturing, of the utricle and saccule walls. The detachment of the ampulla from the underlying connective tissue impacts the semicircular canals, as hair cells are swallowed, stereocilia become deformed or destroyed, and the density of the neural fibers decreases.

**CLINICAL IMPLICATIONS**

As presented in previous studies, our subjects were exposed to a variety of noisy tools and equipment daily, and they experienced the symptoms of balance disorders more than hearing difficulties. While all of these patients were diagnosed with BPPV, MD, or both, their hearing was almost totally intact. This supports the notion that dental technicians should regularly visit audiology clinics, not just for regular auditory assessments, but also to determine whether their vestibular functions are intact. In order to reduce the incidence of vestibular pathologies among dental technicians, an adequate hearing protection must be provided and worn during working hours.

**CONCLUSION**

The present study provides evidence of vestibular and cochlear defects in dental technicians exposed to daily occupational noise. To prevent these disorders, dental technicians should use personal hearing protection at work.

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**Conflicts of interest**

There are no conflicts of interest.

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