# Assessment the effect of noise on the sacculocolic pathway using vestibular evoked myogenic potential

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**Received** 23 October 2016
**Accepted** 15 January 2017

**The Egyptian Journal of Otolaryngology**
2017, 33:523–527

**Objective**
The aim of this study was to detect the effect of noise on the sacculocollic pathway using cervical vestibular evoked myogenic potential (c-VEMP) and to check the correlation between the hearing thresholds and the c-VEMP responses in noise-induced hearing loss (NIHL) patients.
**Patients and methods**
The study included 40 (80 ears) participants. Their ages ranged between 30 and 45 years. They were divided into two groups: 20 (40 ears) participants with NIHL presenting with bilateral notched audiogram at 4 kHz, and 20 (40 ears) participants as a control group. All participants in the study were subjected to basic audiological evaluation and VEMP tests.
**Results**
As the average pure-tone hearing threshold increased, the c-VEMP latencies prolonged and peak-to-peak amplitude reduced in NIHL patients. Of the 40 ears, c-VEMP was absent in 12 (30%) ears. The latencies prolonged and the peak-to-peak amplitude reduced in 16 (40%) ears. VEMP results were normal in 12 (30%) ears.
**Conclusion**
Noise can cause more damage to the vestibular system, especially the sacculocollic pathway.

**Keywords:**
noise-induced hearing loss, sacculocollic reflex, vestibular system

Egypt J Otolaryngol 33:523–527

1012-5574

## Introduction

Noise is considered one of the most important social health problems. Exposure to high levels of noise can affect the cochlea, causing damage to it, and results in hearing impairment. Many studies on noise exposure have explained the clinical picture of noise-induced hearing loss (NIHL), which is a well-defined entity [1].

Unlike the situation with hearing, noise is not known as a common cause of vestibular disturbances. This is probably a result from the difference in 'tuning' between the cochlear hair cells and the vestibular labyrinth. The hair cells of the cochlea are 'tuned' to respond to frequencies in the range between 20 and 20 000 Hz, whereas vestibular hair cells are 'tuned' to respond to inputs in the range between 0 and 10 Hz [2].

Only few researchers have examined vestibular function in patients suffering from NIHL and examined the possible correlation between vestibular test results and hearing threshold. Golz and colleagues [3–5] had reported that symmetrical or asymmetrical hearing loss may be associated with abnormal vestibular functions that resulted from noise. Exposure to industrial solvents and noise can cause damage to the cochlea and the vestibular system in humans [6]. Moreover, subclinical disturbances of the vestibular system may explain the occurrence of disequilibrium in patients with NIHL [1].

Conversely, Sohmer *et al.* [7] reported that the higher intensity levels (113 dB sound pressure level) cause a clear effect on the cochlea, but no effect on the vestibular end organs. Although loud noise may cause temporary and permanent hearing loss, vestibular changes are less likely understood, possibly because the vestibulo-occular reflex is less sensitive to be affected by the noise even at maximum intensities except if the labyrinth is opened (e.g. superior semicircular canal dehiscence) [8,9].

The neurophysiological and clinical data show that the cervical vestibular evoked myogenic potentials (c-VEMPs) are mediated by a certain pathway that starts when sound stimulates the saccular macula, crosses the inferior vestibular nerve, to reach the lateral vestibular nucleus in the brain stem,



 DOI: 10.4103/1012-5574.206022

and then impulses are sent to the motor neurons of the ipsilateral sternocleidomastoid muscle through the lateral vestibulospinal tract [10]. VEMP is considered a consistent clinical test for the assessment of saccular or inferior vestibular nerve function [11]. Wang and Young [12] reported abnormal caloric and VEMP results in 45 and 50% of individuals exposed to chronic noise, respectively. The vestibular functioning, particularly sacculocollic reflexes, in the noise-exposed individuals are not well understood. Hence, the present study was performed with a purpose of assessing the VEMP in NIHL individuals.

## Aim

This work was designed to detect the effect of noise on the sacculocollic pathway using VEMP and to assess the correlation between the hearing thresholds and the VEMP responses in NIHL patients.

## Patients and methods

This study included a total number of 40 participants (80 ears) who were selected randomly at Zagazig Hospital University.

The 40 participants were divided into two groups of 20 patients each: the study group and the control group. Both the control and case groups were age and sex matched.

All participants provided written consent before participation in the study. The institutional review board approval for this work was obtained on 15 February 2016.

The control group (normal individuals) consisted of 20 participants. Their ages ranged between 30 and 45 years with the following criteria:

(1) All ears had hearing sensitivity within 15 dB hearing level (HL) for frequencies from 250 to 8000 Hz, having 'A' type tympanogram with preserved acoustic reflexes at 500, 1000, 2000, and 4000 Hz.
(2) None of the participants had a recent history or presence of any otological problem (such as ear discharge, earache, etc.), systemic disease, or any neurological symptoms.

The study group (NIHL participants) consisted of 20 participants. Their ages ranged between 30 and 45 years with the following criteria:

(1) All participants were suffering from sensory neural hearing loss ranging from mild to moderately severe degree at 4 kHz (pure-tone ranging from 25 to 70 dB at 4 kHz).
(2) Participants were exposed to intermittent noise greater than 90 dBA daily for 8 h.
(3) Participants working in an industrial setup for 5 years or more.
(4) None of the participants had a recent history or presence of any otological problem (such as ear discharge, earache, etc.), systemic disease, or any neurological symptoms.

All participants in the current study were subjected to the following:

(1) Full history taking including the following:
    (a) Personal history (age, name, sex, and occupation).
    (b) History of hearing loss, tinnitus, discharge, earache, headache, and vertigo.
    (c) Past history of systemic disease, physical trauma, ototoxic drug, and operations.
    (d) History of occupational noise exposure (how many hours per day, days per week, or for how long).
(2) Otological examination.
(3) Basic audiological evaluation: (a) pure-tone audiometry using orbiter 922, Gn Otometrix, Denmark, which included air conduction and bone conduction; (b) speech audiometry; (c) immittancemetry using amplaid 724, Amplifon, Italy, including tympanometry and acoustic reflex threshold measurement.
(4) c-VEMP: using intelligent hearing system evoked potential (Smart EP, Miami, Florida, USA) the stimuli were broad band click with rarefaction polarity, 100 μs duration, and 125 sweeps with a repetition rate of 5 click/s. The recorded potentials were filtered through a bandpass filter of 30–1500 Hz, with analysis time 50 ms and at 95 dBnHL intensity. Electrode montage: surface electrodes were placed on midpoints of each sternocleidomastoid muscle, one for the right side and one for the left side, with the reference electrode on ipsilateral upper sternum. A ground electrode was placed on the forehead.

At least two consecutive averages were recorded from each side to verify reproducibility. The average of two runs was taken for the amplitude and latencies. For latency, the peak latencies of P1 and N1 were measured. P1 is the first positive peak of VEMP and N1 is the first negative peak following P1. The latency was defined as the time from the onset of the

stimulus to the first positive peak. For evaluation of the amplitude, the positive–negative peak P1–N1 was measured. The peak-to-peak amplitude was measured from the maximum positive deflection to the following maximum deflection.

### Statistical analysis

Data from the right and left ears of all participants were collected and tabulated in raw data tables. They were statistically analyzed using statistical package for the social sciences software (statistical computer package version 20; SPSS Inc., Chicago, Illinois, USA). Simple descriptive analysis was performed to calculate the mean±SD of the test variable. The latency and amplitude of VEMPs waves in the case and control groups were compared using the independent *t*-test to calculate the *t* value and its *P*. Pearson's correlation coefficient test was used to test the presence of correlation between effect of exposure to noise and the amplitude and latency of VEMPs waves. The significance level was set at $P$=0.05.

## Results

The study included 40 participants (80 ears). Their ages ranged between 30 and 45 years. They were divided into two groups: 20 (40 ears) participants with NIHL presenting as bilateral notched audiogram at 4 kHz, and 20 (40 ears) participants as a control group. The mean and SD of P1, N1 latencies, and peak-to-peak amplitude obtained at the 95 dBnHL are shown in Tables 1–3.

As demonstrated in Tables 1 and 2 and Figs 1 and 2, there was a significant difference in VEMP responses between the control group and the study group with respect to latencies P1 and N1. The peak-to-peak amplitude was also significantly different between the control group and the NIHL group.

**Table 1 Mean and SD of vestibular evoked myogenic potentials wave latencies (ms) in the control and the study group**

| Variables | Control group (mean±SD) | Study group (mean±SD) | *t* | *P* |
|---|---|---|---|---|
| P13 | 13.3±1.2 | 16.7±2.3 | 7.85 | <0.001** |
| N23 | 22.9±1 | 24.4±0.8 | 6.88 | <0.001** |

This table showed delayed latency in the study group when compared with the control group. **Significant difference when $P$ = 0.05 to >0.01.

**Table 2 Mean and SD of vestibular evoked myogenic potentials wave amplitude (μV) in the control and the study group**

| Variables | Control group (mean±SD) | Study group (mean±SD) | *t* | *P* |
|---|---|---|---|---|
| Amplitude | 29.5±11.7 | 11.6±4.7 | 7.5 | <0.001** |

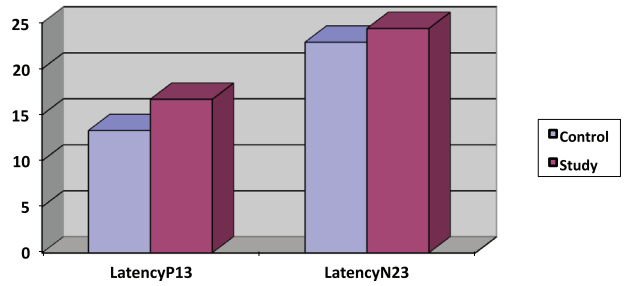
This table showed reduced peak-to-peak amplitude in the study group when compared with the control group. **Significant difference when $P$ = 0.05 to >0.01.

**Table 3 Mean and SD of pure-tone thresholds in dB hearing level of the study group**

| Frequencies (Hz) | Mean±SD |
|---|---|
| 250 | 16.5±5.7 |
| 500 | 16.1±4.9 |
| 1000 | 16±5.3 |
| 2000 | 15.3±5.2 |
| 4000 | 42.2±14.6 |
| 8000 | 24.5±9.2 |

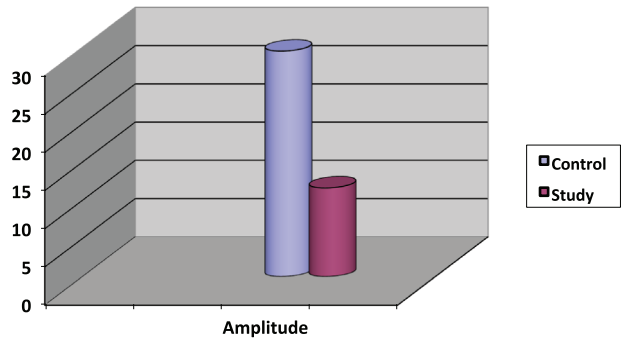
As shown in this table, there were notched hearing loss at 4 kHz.

**Figure 1**




Latency of vestibular evoked myogenic potential in the control and the study group.

**Figure 2**




Amplitude of vestibular evoked myogenic potential in the control and the study group.

**Table 4 Correlation between pure-tone audiometry average at 4 kHz with amplitude and latency of vestibular evoked myogenic potential waves in study group**

| | PTA | |
|---|---|---|
| | *r* | *P* value |
| Amplitude | –0.68 | ? 0.0001**** |
| Latency P1 | 0.75 | ? 0.0001*** |
| Latency N1 | 0.76 | **? 0.0001**** |

This table showed that there was a strong positive correlation between amplitude and PTA average, whereas there was a strong negative correlation between the PTA average and latency of wave (P1 and N1). PTA, pure-tone audiometry. ***Highly significant difference when $P$ = 0.01 to >0.001. ****Very highly significant difference when $P \leq 0.001$.

The correlation was negative for P1 and N1 latencies and positive for peak-to-peak amplitude in comparison with pure-tone average (Table 4).

## Discussion

There is a clear evidence that overexposure to noise causes damage to the cochlear hair cell and to the other pathways of the central nervous system. However, the pathophysiologic aspect of vestibular affection is not well known. Many studies have presented clinical and experimental evidence that high levels of sound can stimulate the vestibular system.

In our study, the patients with NIHL showed bilateral notched audiogram at 4 kHz. NIHL patients demonstrated a significant prolongation of latency and reduction in peak-to-peak amplitude when compared with controls (Tables 1–3). The correlation was negative for P1 and N1 latencies and positive for peak-to-peak amplitude in comparison with pure-tone average (Table 4).

Similarly, Giorgianni *et al.* [13] examined 60 workers with NIHL and 30 office employees. They reported that c-VEMPs were absent in 28.3% (34 ears). In 36.6% (44 ears), the latency was increased and peak-to-peak amplitude was reduced. The c-VEMP results were normal in 35.1% (42 ears). Therefore, the c-VEMP results were abnormal or absent in 64.9% of the workers. All control participants showed c-VEMPs with normal latency and amplitude.

Kumar *et al.* [2] also reported abnormal VEMP (abnormal or absent) in 67% of NIHL patients. Hence, it can be concluded that there is a possibility of vestibular dysfunction, especially the saccular pathway.

Accordingly, the vestibular damage associated with NIHL could be attributed to the mechanical injury or even metabolic damage to the organ of Corti. This damage may comprise ischemia, generation of toxic free radicals, reactive oxygen species, metabolic exhaustion, and ionic imbalance in the inner ear fluid [14]. Although the labyrinthine artery supplies the peripheral vestibular system, the labyrinthine artery mostly arises from the anterior–inferior cerebellar artery, but sometimes it is a direct branch of the basilar artery. In the inner ear, the labyrinthine artery divides into the common cochlear artery that is considered the main blood supply for the cochlea and the anterior vestibular artery that supplies the saccule. Therefore, decreased blood flow may cause permanent hearing threshold shifts and abnormal VEMP responses [14].

In addition to this, as shown in Table 3, there was NIHL at 4 kHz, and it was also observed that, as the pure tone level increased at 4 kHz, the latency was prolonged and amplitude was reduced in NIHL patients (Table 4). Moreover, Wang *et al.* [15] reported abnormal VEMPs in participants with bilateral 4 kHz notched audiogram with a hearing threshold of more than 40 dB at 4 kHz. This may indicate an increasingly damaged saccule.

To explain why a 4 kHz hearing threshold relates to VEMP response, an animal study was conducted in which guinea pigs underwent 136–150 dB sound pressure level for 20 min. This study demonstrated that the most affected structures are the saccule and cochlea (pars inferior), whereas the utricle and the semicircular canals (pars superior) persist free of structural changes [16]. This difference indicates a functional difference between pars superior and inferior, possibly attributable to the presence of the membrana limitans, which is considered as a barrier between two partitions, causing differential sensitivity of cochlear and vestibular sensory cells in the presence of noxious substances [17].

Accordingly, the present findings in our study suggest the affection of the vestibular system with NIHL. This can be identified by VEMP responses indicating the saccular pathway abnormality. Subsequently, there is a correlation between NIHL and VEMPs finding.

## Conclusion

The present study concluded that the VEMP abnormalities in NIHL patients reflect the effect of noise on the sacculocollic pathway. Moreover, with an increase in the pure-tone hearing threshold in noise-exposed individuals, a VEMP abnormality also increases latencies and amplitude (with exclusion of other pathologies that affect pure tone).

### Recommendation

VEMP, a noninvasive and user-friendly procedure, can be used in noise-exposed individuals to assess the sacculocollic pathway.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

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