Aud Vest Res (2016);25(3):166-174.

RESEARCH ARTICLE

Vestibular function in patients with vestibular migraine

Younes Lotfi[1], Nasim Mardani[1*], Nima Rezazade[1], Elham Saedi Khamene[2], Enayatallah Bakhshi[3]

[1]- Department of Audiology, University of Social Welfare and Rehabilitation Sciences, Tehran, Iran

[2]- Clinic of Internal Medicine, Amiralam Hospital, Tehran University of Medical Sciences, Tehran, Iran

[3]- Department of Biostatistics, University of Social Welfare and Rehabilitation Sciences, Tehran, Iran

Received: 29 Jul 2016, Revised: 11 Aug 2016, Accepted: 13 Aug 2016, Published: 20 Sep 2016

Abstract

Background and Aim: Vestibular migraine is a type of migraine that causes vertigo, dizziness, and imbalance in addition to typical migraine symptoms. In this study, we aimed to investigate vestibular system in these patients by videonystagmography (VNG), electrocochleography (ECochG), and vestibular evoked myogenic potential (VEMP) testing.

Methods: This empirical study was conducted on 10 patients aged 18-45 years old (mean age: 29.7 years) with vestibular migraine and 10 normal subjects (mean age: 30.9 years). Immittance, audiometry, VNG, ECochG, and VEMP tests were performed in attack-free phase in both groups.

Results: Mean value of spontaneous nystagmus was significantly higher in patients compared to the normal subjects ($p<0.05$). There was no statistically significant difference in oculomotor, positional, caloric, and ECochG test results between the two groups ($p>0.05$). In positional test, pathologic nystagmus was detected in four patients with vestibular migraine, but there was not any significant difference between the two groups in this regard ($p>0.05$). Mean p13-n23 amplitude was significantly lower and mean p13 latency was significantly higher in the vestibular migraine patients than that of the control group.

Conclusion: Vestibular malfunction was more prevalent in patients with migraine than the control group even between attacks. This malfunction can be observed in both peripheral and central systems. Due to heterogeneity of vestibular disorders in patients with vestibular migraine and the variety of pathologic mechanisms that affect its occurrence and progression, conducting one test alone cannot be helpful in diagnosis; thus, test battery approach is crucial.

Keywords: Vestibular migraine; videonystagmography; electrocochleography; vestibular evoked myogenic potential

[*] **Corresponding author:** Department of Audiology, University of Social Welfare and Rehabilitation Sciences, Daneshjoo Blvd., Evin, Tehran, 1985713834, Iran. Tel: 009821-22180100, E-mail: mardani.nasim@yahoo.com

Introduction

Vestibular migraine is a type of migraine that includes symptoms such as vertigo, dizziness, and imbalance. It is diagnosed based on the severity and duration of vertigo (moderate to severe and lasting for 5 minutes to 72 hours), presentation of migraine symptoms, and rejection of other similar disorders. Symptoms of this disorder include spontaneous and positional vertigo lasting for a few seconds to several days, tinnitus, ear fullness, phonophobia, and photophobia [1]. It involves about 1% of the total population, 10% of patients referring to the clinics for balance evaluation, and 9% of patients referring to migraine clinics [2]. The exact cause of this disorder is unknown, but in various studies, different mechanisms have been

reported to explain its causative factors.
Internal auditory artery vasospasm can be one of the causes of this disorder. Another theory about the cause of migraines is the reduction of the emission of cortical electromagnetic waves (cortical spreading depression). Disruption in ion, potassium, and calcium channels can cause the disease symptoms, as well [3]. Various studies were conducted in the field of clinical tests in migraine patients, but none of them has reviewed the balance system performance [4]. Each of these studies reviews a part of the performance of the system. Herein, we aimed to conduct a comprehensive review of the involvement of the balance system in patients with vestibular migraine and determine the effectiveness of videonystagmography (VNG), electrocochleography (ECochG), and vestibular evoked myogenic potential (VEMP) tests in migraine patients.

Methods

In this quasi-experimental study, two groups of vestibular migraine patients and healthy subjects were recruited. There were 10 subjects in patients group including five males and five females (age range: 19-42 years) with a mean age of 30.9 years (SD=7.21). These patients were selected through convenience sampling method based on International Headache Society (IHS) and confirmation of a neurologist. The inclusion criteria were being aged 18 to 45 years, having passed a minimum of two and maximum of five years from the onset of the disease, and not having neck problems such as arthritis, neck pain, and spasms, surgery, or trauma, reduced range of motion and posture problems, hearing threshold of less than 20 dB HL, middle ear dysfunctions, cerumen, uncorrectable visual impairment, and neurological disorders, as well as not consuming medications for neurological diseases. The normal group consisted of 10 healthy individuals (5 males and 5 females) with mean age of 29.7 (SD=7.31) years (age range: 19-44 years) selected through convenience sampling. Data collection was performed during autumn-winter 2015 in Mollasadra Hearing and Balance Clinic. Prior to test, the objectives of the study and the method of performing the study were explained to the participants, and informed consent was obtained from them. Medical case history the patients was taken using a questionnaire; thereafter, otoscopy, immittance evaluation, and pure tone audiometry were conducted within the frequency range of 250-8000 Hz. VNG, ECoG, and VEMP were carried out, if the participant met the inclusion criteria.
In performing the VNG test, the presence of spontaneous nystagmus was first examined, then gaze nystagmus evaluation, smooth pursuit, saccade, and optokinetic tests were performed followed by Dix-Hallpike, positional, and caloric tests, which were carried out with cold and warm air irrigation. In the spontaneous nystagmus test, the presence of pathological nystagmus was investigated. von Berevern et al. studied eye movements in patients with vestibular migraine. Eye movement more than three degrees per second were considered pathological nystagmus [5]. In the gaze test, the stimulus was at visual angle of 0 (opposite), 30 degrees to the right and left, and 30 degrees up and down.
In smooth pursuit testing, slow-phase velocity and gain of nystagmus were evaluated. In saccade test, velocity, latency, and accuracy of nystagmus were investigated. In optokinetic test, slow-phase velocity and nystagmus interest were assessed. In positional tests, nystagmus with the slow-phase velocity of more than 4 degrees per second was considered abnormal. In the caloric test, unilateral weakness, directional preponderance, and visual fixation index were investigated. In the test results, values of unilateral weakness greater than 25%, directional preponderance greater than 30%, and visual fixation index of higher than 70% were considered abnormal [6].
In ECochG test, click stimulus with alternative polarity and rate of 7.1 was used to record the responses. The stimulus was provided unilaterally with ER-3A insert earphone without contralateral masking. Signals were amplified to 75,000 times and 10-1500 Hz bandpass filter was used. The number of sweeps was variable from 1,500 depending on the signal to noise ratio (SNR) and wave amplitude and time

Table 1. Mean (standard deviation) spontaneous nystagmus and caloric test results in migrainous and normal groups

	Normal (n=10)	Migrainous (n=10)	p
Spontaneous nystagmus (^{0}/s)	0.4 (0.69)	2.5 (1.7)	0.007
Unilateral weakness (%)	12.3 (7.13)	17.8 (11.73)	0.31
Directional preponderance (%)	12.5 (4.6)	14.5 (3.34)	0.28
Failure of fixation suppression index	80.1 (7.54)	73 (13.39)	0.24

window was 10 ms. Intensity level of 85 dBnHL was used for acquiring proper waves and three acceptable records with proper repeatability and morphology were gained for each person. Summation potential to action potential ratio (SP/AP) was evaluated on each of the waves. SP/AP values more than 0.42 were considered abnormal [7].

In order to record the responses from the tone burst stimulus, 500 Hz with polarity expansion (rare faction) and 2 ms rise and fall time was applied without plateau. Stimuli were provided unilaterally with repetition rate of 5.1 through inserting earphone without contralateral masking. Electromyography signals were amplified 5000 times and 10-1500 Hz bandpass filter was applied. The number of sweeps was 150 and time window was 100 ms. To monitor muscle contraction in response time recording, acceptable range was at least 50 mV and at most 120 mV and no recording was performed higher and lower than these limits. p13-n23 amplitude and p13 and n23 latency were calculated at 95 dBnHL. In the normal group, all the tests were carried out under similar conditions with identical equipment and testers as in the patient group.

Mean and standard deviation were used to describe the data. To analyze the data, Shapiro-Wilk test was used to check the normality of the data, Levene test for equality of variance, and independent t-test was run to test the equality of two means, using SPSS 22.

Results

Findings of VNG test are provided in Tables 1 and 2. In the spontaneous nystagmus test in healthy subjects, pathological nystagmus was not observed in any of the cases, but in six patients with vestibular migraine remarkable nystagmus was recorded. Mean spontaneous nystagmus in patients with vestibular migraine was significantly higher than in healthy subjects ($p<0.05$). In parameters of oculomotor tests, including gaze nystagmus in right, left, up, and down directions, phase and gain of smooth pursuit latency, velocity and accuracy of saccades, and slow-phase velocity and gain of optokinetic nystagmus, no significant statistical differences were noted between the groups ($p>0.05$).

In the positional test in healthy subjects, no pathological nystagmus was observed in any of the subjects, but in two patients with vestibular migraine, pathological nystagmus was detected, the difference between the two groups was not statistically significant ($p>0.05$).

In Dix-Hallpike test, no pathological nystagmus was observed in any of the two groups and no

Table 2. Frequency of occurrence of positioning and positional nystagmus in migrainous and normal groups

Kind of nystagmus	Normal (n=10)	Migrainous (n=10)	p
Positional	0	2 (20%)	0.07
Positioning	0	0	0.73

Table 3. Mean (standard deviation) summation potential to action potential ratio in migrainous and normal groups

	Normal (n=10)	Migrainous (n=10)	p
Right ear	7.2 (14)	9.35 (16.9)	0.63
Left ear	8.45 (17)	16.71(26.1)	0.43

significant statistical difference was found between the two groups in this regard ($p>0.05$). In the caloric test, no statistically significant difference was observed between the two groups in unilateral weakness, directional preponderance, and visual fixation index ($p>0.05$).

In ECochG test, according to the results presented in Table 3, appropriate repetitive responses were recorded in all the subjects of the healthy group, but in the patients with vestibular migraine, SP/AP ratio was higher than the normal level in two cases. In general, there was no significant difference in the mean SP/AP ratio between the healthy subjects and patients with vestibular migraine ($p>0.05$).

In VEMP test, according to the results reported in Table 4, no statistically significant difference was noted between the two groups in terms of response threshold ($p>0.05$). A statistically significant difference was observed between the two groups regarding the mean response amplitude. In patients with vestibular migraine, the amplitude was significantly lower than in normal subjects ($p<0.05$). The latency of p13 in patients with vestibular migraine was significantly higher than in normal subjects ($p<0.05$). However, no significant difference was observed between latency of n23 ($p>0.05$). The amplitude asymmetry ratio showed no significant difference between the two groups ($p>0.05$).

Discussion

In this study, in the spontaneous nystagmus test, no significant nystagmus was observed in any of the healthy subjects. Nonetheless, in six patients with vestibular migraine, pathological nystagmus was detected with an average of 2.5 degrees per second. These findings suggest that in the assessment of patients in the intervals between attacks abnormal test results are possible. Spontaneous nystagmus test results in this study are consistent with the study conducted by Neugebauer et al. [8]. Similar results were obtained in other studies, as well [9-11]. In a number of studies investigating spontaneous nystagmus in the attack phase of the disease, some degrees of distortion were observed in the test results. In a study performed by patients with vestibular migraine in the acute phase, von Brevern et al. observed spontaneous nystagmus, but in the intervals between attacks, the spontaneous nystagmus was either disappeared or decreased in the same cases [5]. The results of this study are confirmed by other studies [12,13]. In some studies, in the attack phase, pathological spontaneous nystagmus was not observed in any of patients [3,14-16]. The discrepancy between our results and those of other studies might be due to differences in samples, examination time, techniques, equipment, and patholologic limit definition.

In oculomotor tests performed in this study, no abnormal finding was observed. The results of this study are consistent with those of a number of previous ones [14,17]. In a study by Neugebauer et al., the results of gaze, saccade, and optokinetic tests were normal in the initial assessment and follow-up, while in patients with central vestibular migraine, similar disorders in smooth pursuit test were observed in the initial visit and follow-up. Considering the confirmation of the central origin of this disorder, low deficits with slight changes were reported during the study period. It is noteworthy that observing mild abnormalities in the results of tests such as smooth pursuit can be caused by

Table 4. Mean (standard deviation) vestibular evoked myogenic potential parameters in migrainous and normal groups

	Normal (n=10)	Migrainous (n=10)	p
Threshold (dBnHL)	81 (3.37)	83.21 (2.37)	0.35
Amplitude (µv)	129.43 (51.38)	82.34 (40.47)	0.035
p13 Latency (ms)	13.88 (1.32)	15.35 (0.91)	0.009
n23 Latency (ms)	23.28 (1.26)	23.53 (1.14)	0.84
Asymmetry ratio (%)	9.60 (6.09)	8.42 (5.96)	0.96

lack of patient's attention; thus, patient cooperation is of great importance in the results of these tests. In healthy individuals, in case of inappropriate training or cooperation, test results can be distorted. In degenerative diseases of the brainstem and cerebellum, abnormalities can be observed in smooth pursuit test results [8]. Dietrich et al. observed central pathological oculomotor disorders in 65% of patients in the intervals between attacks. They proposed that the cause of this disorder in most migraine cases is ischemia, which leads to infarction in nerve tissue. In the aura phase of the disease, inhibiting the nerve activities causes neurological symptoms in these patients [9]. The development of central oculomotor disorders in patients may suggest vestibular migraine [8].

In the positional test, pathological nystagmus was not observed in any of the healthy subjects, but it was detected in two patients with vestibular migraine; this difference between the groups was not statistically significant. The results of this study are confirmed by other studies [9,11]. Positional nystagmus can occur due to the asymmetry of afferent vestibular neurons in the central or peripheral nervous system. von Brevern et al. examined 10 patients with false benign paroxysmal positional vertigo (BPPV) suffering from migraine headaches. Four of these patients had positional nystagmus associated with vertigo [18]; these results are consistent with ours. Hazzaa and El Mowafy observed positional nystagmus in 60% of patients. They concluded since the oculomotor and caloric tests, are normal positional test seems to be more important for diagnosis of this disorder. However, this test cannot detect whether the lesion is central or peripheral [3]. Polensek and Tusa showed, positional nystagmus was observed in all patients at the time of attack. Although the characteristics of nystagmus in patients with vestibular migraine are different, the results of nystagmus indicate the fact that they are reduced between the intervals of migraine attacks [13]. In several other studies, no pathological nystagmus was recorded in patients [12,17]. This inconsistency in results may be due to differences in the patients participating in the studies, the time elapsed since the onset of the disease, the time of the last attack, and other parameters influencing the test results.

No significant difference was noted between healthy subjects and patients with vestibular migraine in the positioning test results. This result is consistent with some other studies [13,18]. Limited studies are performed investigating the relationship between vestibular migraine and BPPV. Yetiser and Gokmen compared the clinical characteristics of patients with BPPV with and without vestibular migraine and found no significant difference in the rate of treatment through repositioning maneuver. They suggested that in patients with BPPV, migraine may be a secondary complication, but it is not a risk factor for BPPV, and treatment outcomes were similar in both groups [19]. Lempert and Neuhauser stated that the prevalence of BPPV,

Meniere's disease, motion sickness, cerebellar disorders, and psychiatric syndromes in patients with migraine is higher than in the control group [2].

Ishiyama et al. examined, 247 patients with BPPV with torsional nystagmus were examined by Dix-Hallpike test Based on International Headache Society (HIS) criteria, the prevalence of migraine in patients with idiopathic BPPV was three times higher than in patients with BPPV caused by trauma or surgery. In patients with migraine, there exists the probability of recurrent inner ear damages due to vasospasm, which can lead to BPPV [20]. Dash et al. observed torsional nystagmus caused by BPPV in three patients with migraine. After the maneuvers carried out in two of the patients, no headache attacks occurred during the study [4]. A possible cause could be canalithiasis, which can cause headaches similar to migraines. von Brevern et al. demonstrated, in patients with BPPV, those with migraines were 7.5 times the control group [21]. Uneri et al. examined 476 patients with lateral semicircular canal BPPV and concluded that the prevalence of migraine in patients with BPPV was higher compared to healthy individuals [22].

Herein, the parameters under study in the caloric test (unilateral weakness, visual fixation index, and directional preponderance) were within the normal range, which was confirmed by a number of other studies [3,13]. Asymmetry in caloric test results may be due to dysfunction of the vestibular system in semicircular canals (SCC), vestibular nerve, or brainstem. The most common cause of abnormality in caloric test results is disorders of the peripheral vestibular system. In cases of central vestibular system involvement, it is likely for the eighth nerve to be involved, as well. In animal studies, it was found that vestibular nucleus involvement does not lead to poor caloric performance.

In caloric test results, it cannot be stated that the central performance dysfunction does not lead to impairment, but it is less likely to happen in comparing with peripheral factors. The central origin of dysfunction can be confirmed with oculomotor test results. Celebisoy et al. [14] observed unilateral weakness in the caloric test in the intervals between attacks in 20% of patients. In the caloric test at an early stage, Neugebauer et al. found no asymmetry between SCC performance bilaterally, but in the next investigation, unilateral weakness was observed in 25% of patients, which represents progression in vestibular symptoms during disease even in the intervals between attacks [8]. However, in a study by Lempart et al., unilateral function decline in patients with vestibular migraine was found, which has low probability [12].

In the current study, various parameters of VEMP such as response amplitude, wave latency, amplitude asymmetry ratio, and response threshold were measured, and some dysfunctions were noted in test results of a large number of patients. Reduced amplitude and increased latency of p13 were the most common statistically significant findings in this test. cVEMP was bilaterally absent in one of the patients and in one case in the left ear.

Amplitude of p13-n23 showed a significant difference between healthy subjects and patients with vestibular migraine; this result was confirmed by other studies [23-28]. Reduced amplitude and the absence of response could be due to dysfunction of the peripheral vestibular system. Vascular changes in migraine lead to variations in the vestibular branch of internal auditory artery, where these changes may lead to short-term vertigo attacks and irreversible labyrinth damage [29]. Ischemia caused by reduced blood flow of labyrinth structures leads to disrupting the structures of the inner ear such as the saccule and can affect the VEMP amplitude [30].

However, due to the fact that VEMP are pathways are located adjacent to each other in the brainstem, the hypothesis of exclusive involvement of peripheral structures is questionable and the possibility of the involvement of the brainstem due to either abnormal serotonergic regulation or the impact of ischemia caused by reduced blood flow on the vestibular nucleus should be taken into consideration [26]. In a number of studies, there was no significant difference in wave amplitude between patients

with vestibular migraine and normal cases. The discrepancy between these results and our findings may be due to the stimulant used, subjects, time elapsed since the first and last attacks, severity of attacks, response recording method, and response recording criteria.

Healthy subjects and patients with vestibular migraine were significantly different in terms of latency of p13, but the latency of n23 showed no significant difference between the two groups. Ghali and Kolkaila studied patients with migraine with and without dizziness and normal subjects and concluded that vestibular system can affect the delay in peak latencies in patients with vestibular migraine [31], which is confirmed by other studies, as well [17,24,28,32]. Nevertheless, in some studies, the entire complex of p13-n23 is reported to be delayed; in fact, they only investigated the cause of increased latency in p13 and did not mention anything about increased latency in n23 separately [31,32].

Prolonged latency is indicative of retro-labyrinthine lesions, such as vestibular nerve or brainstem lesions [33]. Lack of difference between the two groups of patients and normal subjects in terms of n23 latency can be associated with larger standard deviation of normal values in n23 latencies than p13 [34]. In a study by Vesligaj and Maslovara, in recording the responses to the click stimulus, p13 and n23 latencies were significantly lower in the patient group in comparison with the control group. The difference between stimulation and recording parameters such as the use of click stimulus instead of tone bursts and low sample size in both studies could be the reasons for this difference [35]. In a number of studies, however, no variation in latency of any of the peaks was observed [25-27].

In this study, there was no difference in the average response threshold between the two groups. Boldingh et al. confirmed the results of this study [25]. According to the present results, there was no difference between the two groups in terms of average amplitude ratio. This ratio was within the normal range in all the subjects of both groups, indicating that this condition does not lead to unilateral involvement of vestibular system. The findings of this study are supported by other studies [28,36]. In the study by Vesligaj and Maslovara, the degree of amplitude asymmetry was greater than 35% in more than half of the patients, and in a third of patients, waves were completely eliminated on one side. This disorder can be caused by lesions in the peripheral or central vestibular system, but the exact location of the lesion cannot be determined [35].

In our study, endolymphatic hydrops was not seen in any of the healthy subjects, while in two of the patients with vestibular migraine 49% and 61% increase were noted in SP/AP ratio; however, there was no significant difference between the results of two groups in this regard. Gürkov et al. evaluated 19 patients with migraine and hearing symptoms using magnetic resonance imaging (MRI) test, among whom four patients showed evidence of endolymphatic hydrops in the cochlea and vestibule. Application of MRI for the diagnosis of endolymphatic hydrops in patients with migraine can be useful because some patients with migraine present with symptoms of endolymphatic hydrops. The assessment is beneficial for determining whether the patient has migraine or Meniere's disease that is incorrectly diagnosed as migraine or both disorders simultaneously [37]. Concomitant migraine and Meniere's disease is not a rare complication [38-40] and is seen in a large number of patients.

Many studies have been performed on the relationship between vestibular migraine and Meniere's disease. In a study investigating the prevalence of migraine in Meniere's disease patients, the prevalence of this disorder was reported 56% and 25% in Meniere's disease group and the control group, respectively. They also concluded that the prevalence of migraine in patients with Meniere's disease was higher than in the control group [41]. Probably, it is due to the presence of common factors causing both disorders. Similar genetic factors, dual effects of neurotransmitters (serotonin, norepinephrine, and glutamate), common disorders of ion or neuropeptide channels in the vestibular

and trigeminal neurons might be the causes of concomitant migraine and Meniere's disease.
A hypothesis states that ischemic lesions caused by small vessels in vestibular migraine induce symptoms similar to symptoms of Meniere's disease. Differentiating the two disorders is crucial because their management approaches are different and in case of incorrect diagnosis, the patient may receive wrong treatment and management. In patients not responding to treatment programs and those suffering from dizziness in spite of treatment programs, more detailed studies for correct diagnosis of the disorder and use of appropriate management and treatment practices are of great significance [37,42,43].

Conclusion

Our results indicated that the prevalence of vestibular dysfunction in patients with vestibular migraine was higher compared to the control group even in the intervals between attacks. The main findings of this study include spontaneous nystagmus, increased latency of p13, and reduced response amplitude of VEMP in patients with vestibular migraines, in whom this dysfunction can be seen in both the peripheral and the central parts. Due to the heterogeneity of vestibular dysfunctions in patients with vestibular migraines, similar symptoms with other disorders such as Meniere's disease, BPPV, vestibular neuritis, perilymphatic fistula, and transient ischemic attacks, as well as the existence of different pathophysiologic mechanisms affecting the occurence and development of migraine, application of only one test cannot help with disease diagnosis; thus, application of a battery approach is crucial. In case of correct diagnosis of the disease, appropriate drug treatments and balance rehabilitation (vestibular rehabilitation) are administered, so that unfavorable effects of this disorder on a person's life are diminished. Given the limited number of samples in this study, conducting further studies is recommended to be able to generalize the results to all patients with vestibular migraine.

Acknowledgements

This article is emerged from N. Mardani's MSc thesis in Audiology submitted in University of Social Welfare and Rehabilitation Sciences, Tehran, Iran.

REFERENCES

1. Maslovara S, Soldo SB, Pajić-Penavić I, Alkhamis T, Vešligaj T, Soldo A. Vestibular migraine considering new diagnostic criteria. Neurol Croat. 2014;63(1-2):11-8.
2. Lempert T, Neuhauser H. Epidemiology of vertigo, migraine and vestibular migraine. J Neurol. 2009;256(3):333-8.
3. Hazzaa N, El Mowafy SS. Clinical features of vestibular migraine in Egypt. Egyptian Journal of Ear, Nose, Throat and Allied Sciences. 2016;17(1):17-21.
4. Dash AK, Panda N, Khandelwal G, Lal V, Mann SS. Migraine and audiovestibular dysfunction: is there a correlation? Am J Otolaryngol. 2008;29(5):295-9.
5. von Brevern M, Zeise D, Neuhauser H, Clarke AH, Lempert T. Acute migrainous vertigo: clinical and oculographic findings. Brain. 2005;128(Pt 2):365-74.
6. Baloh RW, Honrubia V, Kerber KA. Baloh and Honrubia's clinical neurophysiology of the vestibular system. 4th ed. New York: Oxford University Press; 2011.
7. Abbas PJ, Brown CJ. Electrocochleography. In: Katz J, Medwetsky L, Burkard R, Hood J, editors. Handbook of clinical audiology. 6th ed. Baltimore: Lippincott Williams & Wilkins; 2009. p. 242-64.
8. Neugebauer H, Adrion C, Glaser M, Strupp M. Long-term changes of central ocular motor signs in patients with vestibular migraine. Eur Neurol. 2013;69(2):102-7.
9. Dieterich M, Brandt T. Episodic vertigo related to migraine (90 cases): vestibular migraine? J Neurol. 1999;246(10):883-92.
10. Kayan A, Hood JD. Neuro-otological manifestations of migraine. Brain. 1984;107 (Pt 4):1123-42.
11. Bir LS, Ardiç FN, Kara CO, Akalin O, Pinar HS, Celiker A. Migraine patients with or without vertigo: comparison of clinical and electronystagmographic findings. J Otolaryngol. 2003;32(4):234-8.
12. Lempert T, Olesen J, Furman J, Waterston J, Seemungal B, Carey J, et al. Vestibular migraine: diagnostic criteria. J Vestib Res. 2012;22(4):167-72.
13. Polensek SH, Tusa RJ. Nystagmus during attacks of vestibular migraine: an aid in diagnosis. Audiol Neurootol. 2010;15(4):241-6.
14. Celebisoy N, Gökçay F, Sirin H, Biçak N. Migrainous vertigo: clinical, oculographic and posturographic findings. Cephalalgia. 2008;28(1):72-7.
15. Vitkovic J, Paine M, Rance G. Neuro-otological findings in patients with migraine- and nonmigraine-related dizziness. Audiol Neurootol. 2008;13(2):113-22.
16. Rzeski M, Stepień A, Kaczorowski Z. Evaluation of the function of the vestibular system in patients with migraine. Neurol Neurochir Pol. 2008;42(6):518-24.
17. Khalil LH, Hazzaa NM, Nour AA. Vestibular migraine: A correlation study between clinical findings and vestibular evoked myogenic potentials (VEMPs). Egyptian Journal of Ear, Nose, Throat and Allied Sciences. 2016;17(1):11-6.
18. Roberts RA, Gans RE. Background, technique, interpretation and usefulness of positional/positioning tes-

ting. In: Jacobson GP, Shepard NT, editors. Balance function assessment and management. 1st ed. San Diego: Plural Publishing Inc; 2008. p. 171-96.
19. Yetiser S, Gokmen MH. Clinical aspects of benign paroxysmal positional vertigo associated with migraine. Int Tinnitus J. 2015;19(2):64-8.
20. Ishiyama A, Jacobson KM, Baloh RW. Migraine and benign positional vertigo. Ann Otol Rhinol Laryngol. 2000;109(4):377-80.
21. von Brevern M, Radtke A, Lezius F, Feldmann M, Ziese T, Lempert T, et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. J Neurol Neurosurg Psychiatry. 2007;78(7):710-5.
22. Uneri A. Migraine and benign paroxysmal positional vertigo: an outcome study of 476 patients. Ear Nose Throat J. 2004;83(12):814-5.
23. Allena M, Magis D, De Pasqua V, Schoenen J, Bisdorff AR. The vestibulo-collic reflex is abnormal in migraine. Cephalalgia. 2007;27(10):1150-5.
24. Wu ZM, Zhang SZ, Zhou N, Liu XJ, Ji F, Chen AT, et al. Neurootological manifestation of migrainous vertigo. Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi. 2006;41(10):726-30. Chinese.
25. Boldingh MI, Ljøstad U, Mygland A, Monstad P. Vestibular sensitivity in vestibular migraine: VEMPs and motion sickness susceptibility. Cephalalgia. 2011;31(11):1211-9.
26. Baier B, Dieterich M. Vestibular-evoked myogenic potentials in "vestibular migraine" and Menière's disease: a sign of an electrophysiological link? Ann N Y Acad Sci. 2009;1164:324-7.
27. Zuniga MG, Janky KL, Schubert MC, Carey JP. Can vestibular-evoked myogenic potentials help differentiate Ménière disease from vestibular migraine? Otolaryngol Head Neck Surg. 2012;146(5):788-96.
28. Moallemi M, Hajiabolhassan F, Fatahi J, Abolfazli R, Jalaie S, Khamseh F. Vestibular evoked myogenic potentials in migraine patients. Audiol. 2011;20(1):16-25. Persian.
29. Baier B, Stieber N, Dieterich M. Vestibular-evoked myogenic potentials in vestibular migraine. J Neurol. 2009;256(9):1447-54.
30. Parker W. Migraine and the vestibular system in adults. Am J Otol. 1991;12(1):25-34.
31. Ghali AA, Kolkaila EA. Migrainous vertigo: clinical and vestibular evoked myogenic potential (VEMP) findings. Egypt J Neurol Psychiat Neurosurg. 2005;42(2):341-50.
32. Liao LJ, Young YH. Vestibular evoked myogenic potentials in basilar artery migraine. Laryngoscope. 2004;114(7):1305-9.
33. Welgampola MS, Colebatch JG. Characteristics and clinical applications of vestibular-evoked myogenic potentials. Neurology. 2005;64(10):1682-8.
34. Murofushi T, Shimizu K, Takegoshi H, Cheng PW. Diagnostic value of prolonged latencies in the vestibular evoked myogenic potential. Arch Otolaryngol Head Neck Surg. 2001;127(9):1069-72.
35. Vešligaj T, Maslovara S. Can a finding of cervical vestibular evoked myogenic potentials contribute to vestibular migraine diagnostics? Med Glas (Zenica) 2016; 13(1):36-43.
36. Taylor RL, Zagami AS, Gibson WP, Black DA, Watson SR, Halmagyi MG, et al. Vestibular evoked myogenic potentials to sound and vibration: characteristics in vestibular migraine that enable separation from Meniere's disease. Cephalalgia. 2012;32(3):213-25.
37. Gürkov R, Kantner C, Strupp M, Flatz W, Krause E, Ertl-Wagner B. Endolymphatic hydrops in patients with vestibular migraine and auditory symptoms. Eur Arch Otorhinolaryngol. 2014;271(10):2661-7.
38. Rassekh CH, Harker LA. The prevalence of migraine in Menière's disease. Laryngoscope. 1992;102(2):135-8.
39. Radtke A, Lempert T, Gresty MA, Brookes GB, Bronstein AM, Neuhauser H. Migraine and Ménière's disease: is there a link? Neurology. 2002;59(11):1700-4.
40. Ibekwe TS, Fasunla JA, Ibekwe PU, Obasikene GC, Onakoya PA, Nwaorgu OG. Migraine and Meniere's disease: two different phenomena with frequently observed concomitant occurrences. J Natl Med Assoc. 2008;100(3):334-8.
41. Neff BA, Staab JP, Eggers SD, Carlson ML, Schmitt WR, Van Abel KM, et al. Auditory and vestibular symptoms and chronic subjective dizziness in patients with Ménière's disease, vestibular migraine, and Ménière's disease with concomitant vestibular migraine. Otol Neurotol. 2012;33(7):1235-44.
42. Shepard NT. Differentiation of Ménière's disease and migraine-associated dizziness: a review. J Am Acad Audiol. 2006;17(1):69-80.
43. Cohen JM, Escasena CA. Headache and dizziness: how to differentiate vestibular migraine from other conditions. Curr Pain Headache Rep. 2015;19(7):31.