Int. Adv. Otol. 2013; 9:(3) 359-367

ORIGINAL ARTICLE

Evaluation of VEMP Findings in Migrainous Vertigo, Migraine and Meniere's Disease

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Objective: Studying the physiopathologic relationship between Meniere's disease and migrainous vertigo, subclinic vestibular exposure in the asymptomatic ear of the Meniere's disease patients and in migraine patients, and the relationship between the hearing of Meniere's disease and vestibular function by applying vestibular evoked myogenic potentials [VEMP] test to migraine, migrainous vertigo and Meniere's disease patients who suffer from headache and/or vertigo.

Study Design: Prospective study.

Setting: University hospital.

Patients: 26 migrainous vertigo, 26 Meniere disease, 22 migrain patients and 27 healthy cases that didn't suffer from headache and vertigo

Intervention(s): Monaural 500 Hz tone-burst stimulant VEMP test was applied on cases. Visual headache stimulation also was applied on migraine cases and the obtained VEMP responses were recorded separately.

Main Outcome Measure(s): VEMP response parameters (treshold, latency, interpeak amplitude, amplitude assimetry ratio).

Results: While a VEMP response was obtained from all the cases in the control group, no response could be obtained from 9 ears in migraine group (20,4 %), 5 ears from migrainous vertigo group (9,6 %), 11 ears which were diagnosed with Meniere's disease (35,5%) and 3 ears in Meniere's disease group which weren't diagnosed with Meniere's disease. When compared to the control group; threshold stimulant intensity was higher in Meniere's disease group, the threshold stimulus intensity was higher, interpeak amplitude value decreased and amplitude asymmetry ratio increased in migrainous vertigo group, the interpeak amplitude value and latency periods were shorter in migraine group. When Meniere's disease is compared to migrainous vertigo group, significant differences were not identified in all parameters. No statistically significant difference was observed between pre and post-headache stimulation VEMP records of migraine cases. No statistically significant difference was detected among healthy and unhealthy ears of Meniere's disease patients and between subgroups of early and late Meniere's disease.

Conclusion: The findings obtained in migrainous vertigo group were considered in favour of peripheral vestibular exposure. The physiopathologic link between migrainous vertigo and Meniere's disease groups was obtained. While subclinical vestibular exposure was identified in the migraine group, lack of change in post-headache stimulation findings gave rise to the thought that the subclinical exposure identified prior to stimulation does not increase with attack. Nonetheless, it was also thought the reason might be that the migraine stimulation mechanism applied is not effective enough to activate the vestibular system. The fact that no difference was identified in comparison of parameters in healthy and unhealthy ears of Meniere's disease was received as finding of asymptomatic saccular exposure in healthy ears of Meniere's disease patients. No relation was detected between hearing thresholds of Meniere's disease patients and VEMP responses and it was shown that hearing function progresses independently from vestibular function. Prognosis of asymptomatic ear and vestibular function can be followed up through VEMP test in patients of Meniere's disease.

Submitted : 04 April 2013 *Accepted : 02 July 2013*

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Introduction

Migraine is an episodic cephalalgia characterized by neurologic, gastrointestinal and autonomic changes. Diagnosis of migraine is substantially established considering the characteristics of cephalalgia and the accompanying symptoms [1].

Understanding the development mechanism of migraine is possible through examination and analysis of the changes occurring during the periods of headache. However, it is not possible to examine patients during the periods of headache mostly. It has been tried to produce experimental headaches in human beings through administration of various substances exogenously [2]. Another experimental headache model is triggering headache by applying visual stimulation. It has been shown in migraine patients with cortical neuronal hyperexcitability that occipital cortical neurons are the most easily stimulated cortical neurons and it is easier to trigger headaches through visual stimulation[3]. Whereas headache could be produced through application of visual stimulation in migraine patients, it could not be produced in control cases [4].

Vertigo is seen in migraine patients 2 or 3 times more frequently than individuals who do not suffer from headache or suffer from tension type headache [5,6]. In addition, it has been proven through various studies that subclinical vestibular affection can occur in migraine patients who do not have complaints of vertigo as well[7-9].

Migrainous vertigo is a clinical picture with an idiopathic character characterized by vertigo accompanying the migraine attack. Migrainous vertigo is the second most frequently seen cause of vertigo following benign paroxysmal positional vertigo in migraine patients [10]. International Classification of Headache Disorders (ICHD-II-2004) does not include the diagnosis criteria of migrainous vertigo [11]. Neuhauser identifies diagnosis criteria of migrainous vertigo as; mild vertigo attack developing at least twice independent of any other cause, accompanied by at least one of the migrainous symptoms in patients diagnosed with migraine according to the ICHD-II-2004 criteria[12].

Meniere's disease is endolymphatic hydrops picture occurring in the forms of attacks characterized by sensorineural hearing loss, vertigo, resonance and aural fullness. Migraine incidence has been found out to be higher in patients with Migraine's disease than it is in normal population through studies conducted [13-15].

Vestibular evoked myogenic potentials [VEMP] testing is based on the principle of recording the inhibitor response produced in the ipsilateral sternocleidomastoid muscle contraction and produced by the vestibulo-colic reflex that the audio stimulus given to ear, saccule, inferior vestibular nerve and central connections create. The response is interpreted with the threshold stimulus intensity, interpeak amplitude value and p13/n23 latency periods of the waves obtained [16].

Materials And Methods

Selection of Cases

This study was started to be conducted after receiving the approval Nr. 2010/05-03 and dated 23.06.2010 of Dokuz Eylül University's Non-interventional Research Ethics Committee. Cases with the diagnosis of Meniere's disease, migraine and migrainous vertigo, aged between 18 and 56 were included in the study. There were 26 cases with the definitive diagnosis of migrainous vertigo according to the Neuhauser's diagnosis criteria of migrainous vertigo in the migrainous vertigo group. 26 patients, five of whom are diagnosed with bilateral Meniere's disease, who bear the characteristics of "certain Meniere's disease" according to the criteria of American Academy of Otolaryngology Head and Neck Surgery Balance and Hearing Committee were included in the study at a period when no complaints and findings of Meniere's disease attack were detected. There were 22 cases diagnosed with migraine according to the ICHD-II-2004 diagnosis criteria of migraine in the group of cases with migraine. Control group consisted of 27 cases similar to the other groups in terms of age and sex with no complaints of headache or vertigo disorder.

An ear-nose-throat examination and pure tone audiometry testing was carried out in every case. Cases with either a history or finding of otitis externa and otitis media, cases with history of otologic surgery or intratympanic intervention, cases in whom conductive

hearing loss was discovered by audiometric tests, cases in whom findings of BPPV examination were discovered, cases with a history of neck surgery", cases taking chronic myorelaxant medication and cases with an overall physical condition disorder were excluded. Patients receiving vestibulo-suppressant or migraine prophylaxis treatment were made to intermit the treatment at least 1 week prior to the testing in all groups. Both patient and control groups were given detailed information on the study and asked to read and sign the consent form.

VEMP Testing and Application of Visual Stimulation of Migraine

VEMP testing was conducted on the cases and control group included in the study. Gold plated disc electrodes were employed in ipsilateral recordings performed through dual channel by monaural stimulation. Active electrodes were connected by a connector and placed on sternum right below the jugular notch; reference electrode was placed on 1/3 center section of the sternocleidomastoid muscle and ground electrode was placed close to the edge of scalp in the midline of forehead.

While lying in recumbent position, the tested individuals were asked to raise their head up to 30^0 flexion as they heard the tone in the examined ear. This move was described to individuals as raising their head and looking at their toes. Tonic activation of SCM muscle was aimed through this.

ICS-CHARTER auditory potential audiometry device was employed in the VEMP application and recording. 500 Hz ton-burst in rarefaction polarity was used as stimulus. Stimulus intensity, starting at 95 dB nHL, was reduced down to threshold and below threshold level. VEMP wave forms that were obtained by taking the average of 150 responses emerging against the stimulus delivered in a filter range of band permeability of 2 Hz-500 Hz , given at 5,1/sec repetition frequency, starting at 95 dB nHL were recorded on computer up until the final intensity of the individuals' threshold. Verification was done by conducting two recordings in order to check responses. Stimulus duration was used as 2-0 cycle/round and 25 msec delay per frequency through Hanning protocol. Interpeak amplitude values of VEMP responses were calculated on waves obtained by the stimulus of 95 dB.

Threshold stimulus intensity of VEMP responses, the initial positive (p13) and the following negative wave (n23) latencies and amplitudes between the peak points of these two waves were analyzed. Amplitude asymmetry ratio was calculated and absolute value of the amplitude asymmetry ratio was used in statistical analysis. Amplitude asymmetry ratio = 100 x (A r – A l) / (A r + A l) (A r: amplitude of right ear; A l: amplitude of left ear).

In the group of patients with migraine, visual stimulation of headache was applied following the VEMP testing. Mechanism of the visual stimulation was prepared in the form of a computer stimulation of a yellow-navy blue dart board, with a color change at 9 Hz frequency, which was set to remain on for 14 seconds and then off for 14 seconds. Patients watched the computer stimulation sitting 30 cm away from the screen for a period of 45 minutes in a dim and quiet environment located in the Hearing-Speaking-Balance Unit. A second VEMP testing was conducted on patients in whom the stimulation produced headache and their responses before and after the stimulation were recorded separately.

Statistical Analysis

SPSS 16.0 (SPSS for Windows 16.0, SPSS Inc. 2007, Microsoft) program was employed for statistical analysis of the study data. All the analyses were carried out in the confidence interval of 95% and $p<0,05$ was regarded as statistically significant. First of all, percentages of cases in which VEMP responses could not be obtained were calculated and comparison of these percentages between the groups was done using the chi-square test. In every analysis in which VEMP response was obtained, threshold stimulus intensity, p13 latency period, n23 latency period, interpeak amplitude value, and mean value and standard deviation value of amplitude asymmetry ratio were calculated. Bonferonni corrected Kruskal-Wallis Variance Analysis was applied for intergroup evaluation; Mann-Whitney U test was applied for the paired comparison of groups and Wilcoxon test was applied for the comparison of dependent groups.

Results

Compared in terms of sex, no statistically significant difference was found out between the groups (p=0,925). A statistically significant difference was found out between the groups in terms of age averages (p<0,05). Whereas a significant difference in terms of age was detected in the paired comparison of the Meniere's disease group, of which age average was calculated to be 42.4, with the control group and migraine group, no significant difference was detected between the groups in other paired comparisons.

VEMP responses could be obtained from all the cases in the control group. Distribution of the ears from which a response could not be obtained by groups is provided in Table 1. When the distribution of ears from which VEMP response could be obtained and could not be obtained in the groups was compared with the control group, a statistically significant difference was discovered (p<0,05).

Bilateral Meniere's disease was present in 5 out of 26 cases in the Meniere's disease group and diagnosis of Meniere's disease was present in 31 out of 52 ears in the group. 11 (35,5 %) of the ears diagnosed with the Meniere's disease and 3 (14,3 %) of the healthy ears did not give VEMP response. A statistically significant difference was not detected in the comparison of distribution of responsive and nonresponsive ears within diseased and healthy ears (p=0,091).

Ears diagnosed with the Meniere's disease were staged by the thresholds of hearing of 0,5, 1, 2 and 3 kHz and subgroups of early and late Meniere's disease were created. When distribution of the nonresponsive ears in subgroups were compared, a statistically significant difference was not detected (p=0,135) (Table 2).

Mean values and standard deviation values of the threshold value, p13 latency period, n23 latency period and interpeak amplitude value of cases from which VEMP responses were obtained were calculated. While ears diagnosed with the disease in the Meniere's disease group were analyzed, both ears were analyzed in the other groups. Amplitude asymmetry ratios were calculated in cases from which bilateral VEMP response was received (Table 3).

In the evaluation carried out by using Bonferonni corrected Kruskal-Wallis Variance Analysis, whereas a statistically significant difference was discovered

Table 1. Distribution of the ears from which a response could not be obtained by groups

Groups	Total	Number of the ears that VEMP could not be obtained from		Ratio of the ears that VEMP could not be obtained from (%)		
		Bilateral	Unilateral	Bilateral	Unilateral	Total
Meniere's Disease	26	5	4	19,2	7,7	26,9
Migrain	22	4	1	18,1	2,3	20,4
Migrainous Vertigo	26	2	1	7,7	1,9	9,6
Control	27	0	0	0	0	0

Table 2. The distribution of the nonresponsive ears in subgroups

Stages	Mean of hearing thresholds	Number of ears	Groups	Number of ears	Number of nonresponsive ears	Ratio of nonresponsive ears
Stage 1	<25 dB	17	Early Meniere's	22	6	% 27,2
Stage 2	26-40 dB	5	disease subgroup			
Stage 3	41-70 dB	7	Late Meniere's	9	5	% 55,5
Stage 4	>70 dB	2	disease subgroup			

between the groups in terms of the threshold stimulus intensity, p13 latency period, n23 latency period and interpeak amplitude value, a significant difference was not detected between the groups in terms of the amplitude asymmetry ratio (Table 4).

Paired comparisons with the control group showed that threshold stimulus intensity increased in Meniere's patients, p13 and n23 latency periods diminished and interpeak amplitude value decreased in migraine cases, threshold stimulus intensity increased and interpeak amplitude value decreased in migrainous vertigo cases.

A significant difference was not detected among all the parameters in the analysis of paired comparison of the Meniere's disease group and migrainous vertigo group (p>0,05).

When migraine and migrainous vertigo cases were compared, it was found out that threshold stimulus intensity, p13 and n23 latency periods increased in migrainous vertigo cases in comparison with migraine cases (p<0,05).

P values obtained in the paired comparison of the groups are provided in Table 5.

Diseased and healthy ears of 15 cases diagnosed with the unilateral Meniere's disease which were bilateral VEMP responsive in the Meniere's disease group were compared. A statistically significant difference was not detected between them in terms of all the VEMP parameters (p>0,005).

When VEMP responsive ears in the early and late Meniere's disease subgroup were compared, a significant difference was not detected between the two subgroups in terms of the VEMP parameters (p>0,05).

VEMP testing was repeated on the cases in the migraine group after producing a headache by means of visual stimulation. A statistically significant difference was not detected in the distribution of the VEMP responsiveness of ears before and after the stimulation and in the VEMP parameters of those that are VEMP responsive (p>0,05).

Discussion

Through an overall assessment of the responses received from patients whose level of affection in the vestibular system was known and on whom VEMP was applied, a general view that latency periods are influenced in pathologies affecting the vestibular nerve, peduncle and vestibulospinal tractus whereas threshold and amplitude values are influenced in pathologies affecting the peripheric vestibular organs has been obtained [17-20].

Table 3. VEMP responds

GROUPS		Threshold Value (dB)	p13 Latency (ms)	n23 Latency (ms)	Interpeak Amplitude Value (µV)	Amplitude Asymmetry Ratio (%)
CONTROL	Number	54	54	54	54	27
	Mean±St. Deviation	82±5,2	16,3±1,7	23,5±2,3	121,4±56	15,6±9,3
MENİERE'S	Number	20	20	20	20	16
DISEASE	Mean±St. Deviation	87±6,9	16,4±2,5	23,4±3	101,2±69,7	17,7±16
MIGRAIN	Number	35	35	35	35	17
	Mean±St. Deviation	82,8±7,3	14,0±1,6	20,8±2,3	102,5±57,8	20,5±15,2
MIGRAINOUS	Number	47	47	47	47	23
VERTIGO	Mean±St. Deviation	86,1±6,6	16,2±2,1	22,6±2,8	89,1±46,1	22,4±12,1

Table 4. Analysis between the groups

	Threshold Value	p13 Latency	n23 Latency	Interpeak Value Amplitude	Amplitude Asymmetry Ratio
p Value	,002	,000	,000	,008	,257

Table 5. The paired comparison of the groups

	Paired groups	Threshold Value	p13 Latency	n23 Latency	Interpeak Amplitude Value	Amplitude Asymmetry Ratio
p Value	Control (n:54)					
	Meniere's disease (n:20)	,004	,884	,966	,080	,940
	Control (n:54)					
	Migrain (n:35)	,656	,000	,000	,036	,392
	Control (n:54)					
	Migrainous Vertigo (n:47)	,001	,586	,110	,001	,030
	Meniere's disease (n:20)					
	Migrain (n:35)	,049	,000	,001	,637	,614
	Meniere's disease (n:20)					
	Migrainous Vertigo (n:47)	,604	,752	,324	,800	,220
	Migrain (n:35)					
	Migrainous Vertigo (n:47)	,038	,000	,005	,404	,613

Incidence vertigo complaint is higher in migraine patients compared to general population [21-25]. In various studies aiming to reveal the subclinical vestibular affection in migraine patients that do not have vertigo complaints, ischemia formed due to vasospasm of labyrinth arteries and temporary vascular disorders in the vestibular nucleus and vestibular tracts was held responsible [7-9]. Whereas a decrease in the interpeak amplitude was detected in comparison to the control group at a study, in which migraine patients were applied the VEMP testing during the painless period, a decrease in latency period was discovered yet a statistically significant difference was not detected [26]. In our study, VEMP response was not received in 20.4 % of the ears in the VEMP testing which was applied to migraine patients during the painless period. A decrease in the interpeak amplitude and shorter p13 and n23 latency periods in comparison to the control group were detected in the VEMP responsive cases. While findings show that vestibulo-colic reflex arch was influenced in migraine patients who do not have vertigo complaints, they also provide information about the presence of subclinical vestibular affection. The fact that the latency periods diminished supports the quality of easy cortical stimulation of migraine patients. It was observed, in the literature, that vestibular tests applied to migraine patients were conducted during asymptomatic periods when the patients have no headache. In our study, on the other hand, experimental migraine attack was produced by means of a visual stimulation and VEMP responses were recorded separately. When VEMP tests conducted before and after the headache were compared, a statistically significant difference was not detected. That no changes in findings occurred during the headache was thought to possibly depend on the fact that the migraine stimulation mechanism was not efficient enough to activate the vestibular system.

Whereas depression wave dispersing cortically influences vestibular nuclei when findings of neuro-otologic tests in migrainous vertigo supported central vestibular affection, ischemia stemming from vasospasm of the internal auditory artery was the cause in migraine when findings supported peripheric vestibular affection. When central and peripheric vestibular disorders were both discovered, it was argued that peptide and ion channel disorders associated with the calcium gene influencing the both systems could be the factor [27-29].

In the VEMP testing applied to cases with migrainous vertigo, it was found out that rates of not receiving any waves and abnormal wave formation were significantly high compared to the control group and there was not any difference in terms of the latency periods [20,30]. In our study, no VEMP wave could be received in 9.6 % of ears in the migrainous vertigo group In responsive ones, on the other hand, threshold stimulus intensity was found

out to be high, interpeak amplitude to be decreased, amplitude asymmetry ratio to be increased compared to the control group while no difference was observed in terms of the latency periods. Our findings are compatible with those in the literature and interpreted to be findings of asymmetric peripheric vestibular affection in cases with migrainous vertigo.

When the migrainous vertigo group was compared with the migraine group, the latency periods were found out to be shorter in migraine cases and the threshold stimulus intensity was found out to be higher in migrainous vertigo cases. While the average of interpeak amplitude value of the migrainous vertigo group was lower, a statistically significant difference was not detected. These findings are interpreted in that the abnormal results of the VEMP testing on individuals with migraine become more evident with the clinically occurring vertigo.

At study evaluating the saccular function in Meniere's patients by means of the VEMP testing, it was found out that VEMP waves are not formed at all or a decrease in the interpeak amplitude was present while the latency periods were not affected [20]. VEMP response could not be received in 35.5 % of the ears diagnosed with the Meniere's disease. When the responsive ears were compared to the control group, it was found out that there was not a difference in terms of the latency periods and the threshold stimulus intensity was higher in ears with the Meniere's disease. Although the interpeak amplitude value was calculated to be lower compared to the control group, a statistical significance could not be obtained. Being compatible with the literature in general, our findings reveal the effect of endolymphatic hydrops on the saccule in Meniere's patients.

In a study, the rate of abnormal VEMP was found out to be 10 % in ears influenced by the Meniere's disease whereas this rate was calculated to be 5,9 % in contra-lateral ears by means of the VEMP testing conducted in order to research the presence of a subclinical vestibular affection in asymptomatic ears of Meniere's patients[31]. In our study, on the other hand, a statistically significant difference was not detected when the distribution of VEMP non-responsive ears and VEMP parameters in responsive ones were compared between the diseased and healthy ears of Meniere's diseases and it was interpreted as findings supporting asymptomatic saccular affection in non-diagnosed ears of Meniere's patients. It is suggested that, that a VEMP response can not be received from non-diagnosed ears, the lowness of the interpeak amplitude in VEMP waves or increase in the threshold can be used for the diagnosis of asymptomatic ear and follow up of its progression.

American Academy of Otolaryngology Head and Neck Surgery Balance and Hearing Committee defined the audiologic staging in Meniere's disease in 1995. In a study analyzing the progressive stage of the Meniere's disease and VEMP responses, it was reported that non-existence of responses and amplitude asymmetry ratios increase with the developing stage [32]. The rates of VEMP non-responsive ears in the sub-groups of early and late Meniere's disease were calculated to be 27,2 % and 55,5 % respectively and a statistically significant difference was not detected in the distribution of VEMP responsive and non-responsive ears between the sub-groups. A statistically significant difference was not identified either in the comparison of the threshold stimulus intensity, interpeak amplitude value and latency periods of sub-groups. While these findings reveal that a significant relationship does not exist between the hearing and saccule functions of Meniere's patients, the findings are assessed in that hearing and vestibular functions can progress independently. Objective digital data to assess the vestibular system besides the pure tone averages, which are objective digital data, in order to draw up a more realistic and consistent staging of the Meniere's disease. Hearing thresholds of patients can not show the vestibular organ damage. VEMP testing is suggested as one of the clinical tests to be employed in staging the vestibular clinic and following the progression. In addition, conduction of the VEMP testing is also recommended for evaluating the vestibular reserve in the stage of destructive surgical intervention.

In our study, a decrease in the interpeak amplitude value and increase in the threshold stimulus intensity existed in the groups of Meniere's disease and migrainous vertigo compared to the control group whereas a significant difference was not discovered in the p13 and n23 latency periods. A significant difference in all parameters was

not discovered in the comparison of the two groups with each other. The results obtained in our study are compatible with the results that Baier et al. obtained by applying the VEMP testing on migrainous vertigo and Meniere's patients [20]. The fact that findings are similar suggests that different pathogeneses influence the similar areas of the peripheric vestibular system or the possibility that migrainous vertigo attacks lead to secondary endolymphatic hydrops. In studies researching the relationship between migraine and endolymphatic hydrops, trigeminal nerve endings were detected in blood vessel walls in the inner ear and it was suggested that the neurogenic inflammation caused by migraine may lead to changes in the inner ear by affecting the blood flow [33]. In addition, transient receptor potential channel vanilloid subfamily-1 (TRPV1), being a migraine-related nociceptor, was detected in the endolymphatic sac [34]. Views suggesting that migraine can lead to secondary endolymphatic hydrops have been put forward due to these findings.

In the differential diagnosis stage of patients with complaint(s) of headache and/or dizziness, it was thought that the discovery of short latency period in the VEMP testing can direct to the diagnosis of migraine; discovery of high threshold and decreased interpeak amplitude can lead to the diagnosis of migrainous vertigo unless audiologic pathological finding exists and to the diagnosis of Meniere's disease if audiologic pathologic finding exists.

References

1. Zarifoglu M. Basagrıları. In: Guldogus F, Gulec S, Uckunkaya N, editors. Bas ve yuz agrilari. Ankara: Gunes Kitapevi; 2007. p. 13-22.

2. Iversen HK. Human Migraine Models. Cephalalgia 2001; 21: 781-785.

3. Young WB, Oshinsky ML, Shechter AL, et al. Consecutive transcranial magnetic stimulation induced phosphene thresholds in migraineurs and controls. Headache 2004; 44: 131-5.

4. Cao Y, Welch KMA, Aurora S, Vikingstad M. Functional MRI-BOLD of visually triggered headache in patients with migraine. Arch Neurol 1999; 56: 548-55.

5. Kayan A, Hood JD. Neuro-otological manifestations of migraine. Brain 1984;107:1123-1142.

6. Vuković V, Plavec D, Galinovic I, Lovrencić HA, Budisic M, Demarin V. Prevalence of vertigo, dizziness, and migrainous Vertigo in patient with migraine. Headache 2007; 47: 1427-35.

7. Harno H, Hirvonen T, Kaunisto MA, et al. Subclinical vestibulocerebellar dysfunction in migraine with and without aura. Neurology 2003; 61: 1748-52.

8. Ishizaki K, Mori N, Takeshima T, Fukuhara Y. Static stabilometry in patients with migraine and tension-type headache during a headache-free period. Psychiatry Clin Neurosci 2002; 56: 85-90.

9. Cutrer FM, Baloh RW. Vestibular pathways involvement in children with migraine: a neuro-otological study migraine-associated dizziness. Headache 2010; 50: 71-6.

10. Neuhauser H, Lempert T. Vertigo and dizziness related to migraine: a diagnostic challenge. Cephalalgia 2004; 24: 83-91.

11. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders. 2nd edition. Cephalalgia 2004; 24: 16-151.

12. Neuhauser H, Lempert T. Vestibular migraine. Neurol Clin 2009; 27: 379-91.

13. Hinchliffe R. Headache and Meniere's disease. Acta Otolaryngol 1963; 63: 383-90.

14. Eklund S. Headache in Meniere's disease. Auris Nasus Larynx 1999; 26: 427-33.

15. Radtke A, Lempert T, Gresty M, Brookes GB, Bronstein AM, Neuhause H. Migraine and Meniere's disease is there a link? Neurology 2000; 59: 1700-4.

16. Murofishi T. Recording and assessing VEMP. In: Murofishi T, Kaga K, editors. Vestibular evoked myogenic potentials, its basic and clinical applications. Tokyo: Springer; 2009. p. 25-34.

17. Cal R, Bahmad F. Vestibular evoked myogenic potentials: an overview. Braz J Otorhinolaryngol 2009; 75: 456-62.

18. 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: 1069-72.

19. Nola G, Guastini L, Crippa B, Deiana M, Mora R, Ralli G. Vestibular evoked myogenic potential in vestibular neuritis. Eur Arch Otorhinolaryngol 2011 Mar 30 [Epub ahead of print] (DOI 10.1007/s00405-011-1592-0)

20. 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.

21. Kayan A, Hood JD. Neuro-otological manifestations of migraine. Brain.1984; 107: 1123-1142.

22. Lempert T, Neuhauser H. Epidemiology of vertigo, migraine and vestibular migraine. J Neurol 2009; 256: 333-8.

23. Vuković V, Plavec D, Galinovic I, Lovrencić HA, Budišic´M, Demarin V. Prevalence of vertigo, dizziness, and migrainous Vertigo in patient with migraine. Headache 2007; 47: 1427-35.

24. Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo, and migrainous vertigo. Neurology 2001; 56: 436-41.

25. Yardley L, Owen N, Nazareth I, Luxon L. Prevalence and presentation of dizziness in a general practice community sample of working age people. Br J Gen Pract 1998; 48: 1131-5.

26. Allena M, Magis D, De Pasqua V, Schoenen J. The vestibulo-collic reflex is abnormal in migraine. Cephalalgia 2007; 27: 1150-5.

27. Dieterich M, Brandt T. Episodic vertigo related to migraine (90 cases): vestibular migraine? J Neurol 1999; 246: 883-92.

28. von Brevern M, Zeise D, Neuhauser H, Clarke AH, Lempert T. Acute migrainous vertigo: clinical and oculographic findings. Brain 2005; 128: 365-74.

29. Celebisoy N, Gökçay F, Şirin H, Biçak N. Migrainous vertigo: clinical, oculographic and posturographic findings. Cephalalgia 2008; 28: 72-7.

30. Hong SM, Kim SK, Park CH, Lee JH. Vestibular-evoked myogenic potentials in migrainous vertigo. Otolaryngol Head Neck Surg 2011; 144: 284-7.

31. Akkuzu G, Akkuzu B, Ozluoglu LN. Vestibular evoked myogenic potentials in benign paroxysmal positional vertigo and Meniere's disease. Eur Arch Otorhinolaryngol 2006; 263: 510-7.

32. Young YH, Huang TW, Cheng PW. Assessing the stage of Ménière's disease using vestibular evoked myogenic potentials. Arch Otolaryngol Head Neck Surg 2003; 129: 815-8.

33. Vass Z, Shore SE, Nuttall AL, Miller JM. Direct evidence of trigeminal innervation of the cochlear blood vessels. Neuroscience.1998; 84 :559-67.

34. Taguchi D, Takeda T, Kakigi A, Takumida M, Nishioka R, Kitano H. Expressions of aquaporin-2, vasopressin type 2 receptor, transient receptor potential channel vanilloid (TRPV)1, and TRPV4 in the human endolymphatic sac. Laryngoscope 2007; 117: 695-8.