The Journal of International Advanced Otology

J Int Adv Otol 2016; 12(3): 316–20 • DOI: 10.5152/iao.2016.2170

Original Article

Evaluation of Cervical Vestibular-Evoked Myogenic Potential Findings in Benign Paroxysmal Positional Vertigo

Abdullah Karataş, Turgut Yüce, Işıl Taylan Çebi, Gülşah Acar Yüceant, Cemal Hacı, Mehti Salviz

Department of Ear Nose Throat Head, Haseki Training and Research Hospital, İstanbul, Turkey (AK, ITÇ, GAY, CH)
Department of Ear Nose Throat Head, Derince Training and Research Hospital, Kocaeli, Turkey (TY)
Department of Ear Nose Throat Head, Yeni Yüzyıl University School of Medicine, İstanbul, Turkey (MS)

OBJECTIVE: Although there has been a wide consensus on the mechanism of nystagmus and clinical presentation of benign paroxysmal positional vertigo (BPPV), the neuroepithelial pathophysiology of BPPV still remains unclear. In this study, we aimed to clarify the pathophysiology of BPPV by evaluating the cervical vestibular-evoked myogenic potential (cVEMP) findings of patients.

MATERIALS and METHODS: Thirty-six BPPV patients and 20 healthy volunteers were included. Bilateral cVEMP tests were performed on all participants. The participants were divided into the following three groups: those with a BPPV-affected ear, those with a BPPV-unaffected ear, and the healthy control group.

RESULTS: There were no significant differences regarding the latencies of the first positive (p1) and negative (n1) peaks among the three groups. The mean normalized amplitude asymmetry ratio also did not differ between the BPPV and control groups. However, the normalized amplitudes of the BPPV patients (with both affected and unaffected ears) were significantly lower than those of the healthy control group.

CONCLUSION: We detected that the cVEMP data of the affected and unaffected ears of the BPPV patients was similar and that their normalized amplitudes significantly differed from those of the healthy controls. Eventually, we concluded that even if the symptoms of BPPV were unilateral, the findings suggest that the bilateral involvement of the macular neuroepithelium is important in understanding the pathophysiology of BPPV. This finding supports the conclusion that the pathophysiological process starts with neuroepithelial membrane degeneration and continues with otoconia separation.

KEYWORDS: Benign paroxysmal positional vertigo, cervical vestibular-evoked myogenic potential, vertigo

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV), originating from the peripheral vestibular system, is a common type of vertigo. At present, the widely accepted theory about the pathophysiology of the disease is the separation of the otoconia and otoconial debris from the neuroepithelial membrane of the utricular or saccular macula [1]. The otoconia, freely floating in the semicircular canal or sticking to the cupula, provoke short-term nystagmus and vertigo [2]. Due to the topography of the semicircular canals, freely floating otoconia more frequently move into the posterior semicircular canal than into the lateral semicircular canal [3]. The affected canal determines the clinical presentation and direction of nystagmus.

The mechanism resulting in the separation of the otoconia from the neuroepithelial membrane is not yet clear. According to some studies, the separation takes place due to changes in the structure of the otoconia (e.g., osteoporosis, osteopenia, calcium metabolism disorders, or vitamin D deficiency) [4]. Additionally, head trauma and whiplash injuries can cause otoconial fragmentation without neuroepithelial degeneration [5, 6].

However, several studies have proposed that degenerative changes of the neuroepithelial membrane are the main reason behind the separation of the otoconia [7-10]. Aging, diabetes mellitus, hypertension, thyroiditis, hyperlipidemia, stroke, osteopenia, osteoporosis, and vitamin D deficiency are responsible for causing the degenerative changes of the neuroepithelial membrane [11-15]. In particular, over the last 15 years, several studies have investigated the role of vitamin D deficiency and osteoporosis [4, 15-18].

Apart from the most widely accepted theory, there have been other theories about the pathophysiology of BPPV. A temporal bone study conducted by Gacek [19] in 2013 showed focal degenerative changes in the vestibular nerve axons of BPPV patients. Gacek [19] pointed out that some BPPV patients did not benefit from repositioning maneuvers, and in his temporal bone study, no debris

Corresponding Address: Mehti Salviz E-mail: salvizm@yahoo.com
Submitted: 22.01.2016 **Revision received:** 02.04.2016 **Accepted:** 04.04.2016 **Available Online Date:** 01.08.2016

was found in the endolymph of several BPPV patients. Therefore, he suggested that different factors play a role in the etiology of BPPV.

The vestibular-evoked myogenic potential (VEMP) test is an emerging test that allows the specific evaluation of vestibular end organs [20]. VEMPs are recorded by electromyography (EMG) and are derived from the reflex tonic contractions of the sternocleidomastoid (SCM) and ocular muscles evoked by loud auditory stimuli. There are two kinds of VEMPs, ocular VEMP (oVEMP) and cervical VEMP (cVEMP), related to the contracted muscle groups seen by EMG [20, 21]. Murofushi et al. [22] showed that cVEMP is a test of the sacculocollic reflex to sound stimulation, which includes a reflex arc of the saccule, inferior vestibular nerve, and SCM muscle. In contrast, oVEMP recorded from the extraocular muscles is a test of the utriculo-ocular reflex to sound stimuli and has a reflex arc of the utricule, superior vestibular nerve, and extraocular muscles [23, 24].

There has been a wide consensus about the mechanism of nystagmus and clinical presentation of BPPV, but the neuroepithelial pathophysiology of BPPV still remains unclear. In this study, we aimed to clarify the pathophysiology of BPPV by evaluating the cVEMP findings of patients.

MATERIALS and METHODS

Subjects

This study was conducted between July 2014 and October 2014 at Haseki Training and Research Hospital. Thirty-six idiopathic BPPV patients were included (26 females and 10 males), with an average age of 47.2 years (range, 20–63 years). The control group comprised 13 female and 7 male healthy volunteers with an average age of 45.1 years (range, 22–63 years). The patient and control groups were age- and sex-matched, with no statistically significant age or sex differences (p=0.490 and 0.860, respectively) (Table 1).

All participants had undergone a neurotological examination, pure tone audiometry, a bithermal caloric test, and bilateral cVEMP recording.

Exclusion criteria included neurologic and/or otologic disorders, hearing loss documented by pure tone audiometry, history of head and/or neck trauma, chronic systemic diseases, and drug usage affecting the vestibular system. Due to the higher incidence of VEMP abnormalities in elderly people, subjects over the age of 65 years were excluded. Subjects with 20% or higher canal paresis detected by the bithermal caloric test were also excluded.

Benign Paroxysmal Positional Vertigo was diagnosed by the patient history, the Dix–Hallpike test and the head-roll tests. Thirty-four of the 36 patients had posterior canal BBPV, and two had lateral canal BPPV. cVEMPs were recorded before canalith repositioning maneuvers were performed.

Three groups were created for the evaluation of the VEMP test. Groups one and two comprised 36 BBPV patients in total with both affected and unaffected ears, while group three comprised volunteers with bilateral healthy ears (a total of 40 healthy ears).

Our study was reviewed and approved by the Local Ethics Committee (reference number: June-2014/57). In accordance with the Declaration of Helsinki, oral and written informed consent was obtained from all participants.

Table 1. Age and sex distribution of the BPPV and control groups

		BPPV group		Control group		
		Mean± SD/n %	Med (Min-Max)	Mean± SD/n %	Med (Min-Max)	p
Age		47.2±11.4	49 20-63	45.1±11.0	47 22-63	0.490
Sex	Female	26	72%	14	39%	0.860
	Male	10	28%	6	17%	

Free sample t-test/Chi-square test

BPPV: benign paroxysmal positional vertigo; SD: standard deviation

Stimulus Design and Recording Setup

Cervical Vestibular-Evoked Myogenic Potential values were obtained from all participants using bilateral air conduction tone bursts with stimulus frequencies of 500 Hz to test the right and left ears. Calibrated ABR3A insert earphones (maximum intensity level, 100 dB nHL) were used for stimulus transmission. The stimulus profile was adjusted to produce a 2 ms rise, a 2 ms plateau, and a 2 ms fall time with a repetition rate of 5.1 Hz. A frequency of 500 Hz was presented 50–150 times to obtain average responses.

A VEMP evoked potential system (Eclipse EP 25; Interacoustics AS, Assens, Denmark) was used for cVEMP recordings. Disposable silver/silver chloride electrodes (Safelead; Natus Neurology Incorporated, Middleton, WI, U.S.A.) with an impedance of ≤3 kΩ were used. An EMG feedback system (Interacoustics Eclipse; Interacoustics AS, Assens, Denmark) was used for recording muscle responses between 50 and 200 μV. EMG was amplified (60 dB) and bandpass filtered (10–750 Hz). Muscle responses were recorded from 10 ms before the stimulus onset to 60 ms afterwards. To improve reliability and reduce interpatient variability, the test was performed twice, and cVEMP amplitudes were normalized (corrected) by dividing raw amplitudes by the background EMG activity.

cVEMP Recording

Active electrodes were placed over the midpoint of the SCM muscle with a reference electrode placed on the sternum and a ground electrode on the forehead. The measurements were taken while participants were in the sitting position. Participants were asked to contralaterally turn their heads to the stimulated ear and to slightly incline their heads forward to obtain sufficient muscle contraction.

The latencies of the first positive (p1) and negative (n1) peaks and the normalized peak-to-peak (p1-n1) amplitudes were measured for the 500 Hz frequency. Because background muscle activities could interfere with the VEMP amplitudes, the interpeak amplitudes were normalized [2, 25]. The asymmetry ratio was calculated for comparison between the right and left ears, using the formula described by Murofushi et al [22].

Asymmetry Ratio (AR%) = 100(Au − Aa)/(Au + Aa)

Au: p1-n1 (the peak-to-peak amplitude of the unaffected ear)

Aa: p1-n1 (the peak-to-peak amplitude of the affected ear)

Table 2. p1 latency, n1 latency, and interpeak amplitudes in BPPV-affected ears, unaffected ears, and controls

		BPPV-affected ear		BPPV-unaffected ear		Control group		p
p1	Mean±SD		14.2±1.7		14.7±1.8		14.0±1.3	0.271
	Med (Min-Max)	13.7	12.0-19.7	14.3	11.7-18.3	13.7	12.0-18.0	
n1	Mean±SD		21.9±2.0		22.4±2.2		21.8±1.7	0.641
	Med (Min-Max)	22.0	17.7-27.0	22.2	18.7-26.7	22.0	17.8-26.4	
amp	Mean±SD		0.6±0.3		0.7±0.3		1.0±0.4	<.001*
	Med (Min-Max)	0.5	0.2-1.4	0.6	0.2-1.7	1.0	0.3-2.0	

Kruskal-Wallis (Mann-Whitney U test)

*difference of the control group from BPPV affected and unaffected ear

BPPV: benign paroxysmal positional vertigo; SD: standard deviation; amp: interpeak amplitude

Table 3. Interpeak amplitude asymmetry in the BPPV and control groups

	BPPV group		Control group		
	Mean±SD	Med (Min-Max)	Mean±SD	Med (Min-Max)	p
IPAA	16.6±12.8	13 0-52	16.7±13.4	19 0-50	0.738

Mann-Whitney u test
BPPV: benign paroxysmal positional vertigo; SD: standard deviation; IPAA: interpeak amplitude asymmetry

Statistical Analysis

Statistical analyses were performed using statistical software (SPSS 22.0, SPSS Inc.; Chicago, IL, USA). A significant difference was defined as $p<0.05$. The mean, standard deviation, median, minimum value, maximum value, frequency, and ratio were used for the definitive statistics of the data. The Kolmogorov–Smirnov test was used for the distribution of the variables, while the Kruskal–Wallis test, Mann–Whitney U test, and free samples t-test were used for the analysis of quantitative data. The Chi-square test was used for the analysis of qualitative data.

RESULTS

p1 and n1 Latencies

The p1 latencies for the BPPV-affected ears, unaffected ears, and controls were 14.2±1.7 ms, 14.7±1.8 ms, and 14.0±1.3 ms, respectively. The p1 latencies did not differ among these three groups (p=0.271) (Table 2, Figure 1).

The n1 latencies for the BPPV-affected ears, unaffected ears, and controls were 21.9±2.0 ms, 22.4±2.2 ms, and 21.8±1.7 ms, respectively. The n1 latencies did not differ among these three groups (p=0.641) (Table 2, Figure 2).

Amplitudes

The averages of the normalized amplitudes of cVEMP in response to 500 Hz air conducted stimuli (ACS) in the affected ears, unaffected ears, and controls were 0.6±0.3 μV, 0.7±0.3 μV, and 1.0±0.4 μV, respectively. In response to 500 ACS, the amplitudes in the affected ears were not different from those in the unaffected ears (p=0.467), but the amplitude responses in the affected and unaffected BPPV ears were significantly lower than those in the controls (p<0.001) (Table 2, Figure 3).

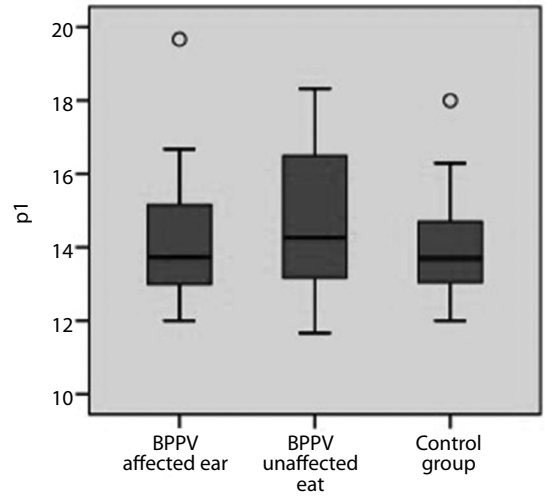

Figure 1. p1 latencies in BPPV-affected ears, unaffected ears, and controls (BPPV: benign paroxysmal positional vertigo)

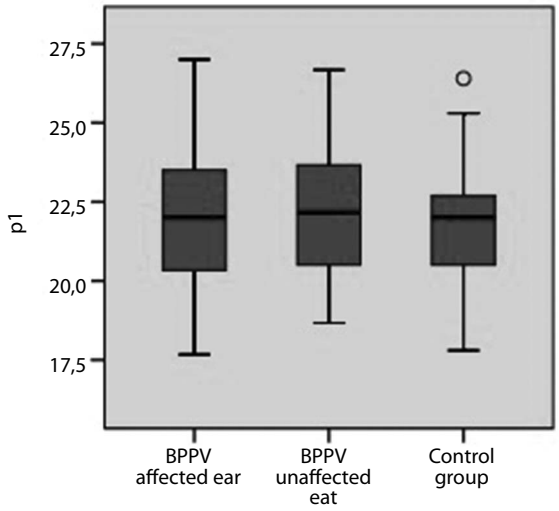

Figure 2. n1 latencies in BPPV-affected ears, unaffected ears, and controls (BPPV: benign paroxysmal positional vertigo)

Amplitude Asymmetry

The amplitude asymmetry rates between the right and left ears were calculated for all participants in the BPPV and control groups. The average amplitude rates for the BPPV and control groups were 16.6±12.8 and 16.7±13.4, respectively. The average amplitude asymmetry rates were not different between these two groups (p=0.738) (Table 3).

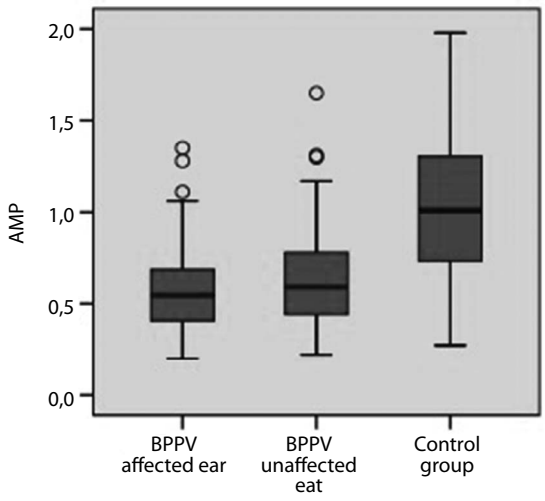

Figure 3. Interpeak amplitudes in BPPV-affected ears, unaffected ears, and controls (amp: interpeak amplitude; BPPV: benign paroxysmal positional vertigo)

When control group data were used to normalize the values, the upper limit for amplitude asymmetry was 43.5 [mean of the control group+(2×standard deviation)]. Only two people in the BPPV group and one person in the control group were at the upper limit.

DISCUSSION

We found no significant difference regarding the p1 and n1 latencies among the three groups; the mean amplitude asymmetry ratio did not differ between the BPPV and control groups. However, the normalized amplitudes of BPPV patients (both affected and unaffected ears) were significantly lower than those in the control group.

There have been several studies discussing cVEMP findings in BPPV patients. Abnormal VEMP findings such as prolonged p1 and/or n1 latencies, decreased interpeak amplitudes, and asymmetric responses in BPPV have been reported in these studies at 10–50% [2, 8, 10]. However, when these studies are thoroughly analyzed, some data regarding the affected and unaffected ears of BPPV patients are controversial.

The first studies on cVEMP abnormalities in BPPV showed prolonged p1 and n1 latencies; however, in recent studies, no alternation in latencies has been reported [2, 10, 26-29]. Similarly, we could not detect any prolongation in the p1 and n1 latencies.

There is a consensus in the literature about the interpeak amplitude changes in the affected ears of BPPV patients. Yetiser et al. [2], Kim et al. [7], Lee et al. [8] and Akkuzu et al. [10] detected lower interpeak amplitudes on the affected sides of BPPV patients. In these studies, the VEMP asymmetry ratios of healthy controls were used as normalized data and compared with those of the BPPV group. On the other hand, there is no information about the comparison of mean amplitudes of BPPV patients (affected and unaffected ears) and controls in these studies. There has also been no standard approach for the evaluation of VEMP asymmetry. Yetiser et al. [2] defined asymmetry over 25% as VEMP asymmetry, although in other studies, data obtained from the control group were regarded as normative data, and VEMP asymmetry was defined as a value above an upper limit calculated by the formula [upper limit=mean of the control group+(2×standary deviation)] [7, 8, 10].

In another study, Kim et al. [7] suggested that when the amplitude of the unaffected ear is lower than that of the affected ear in BPPV, it should be accepted as a VEMP asymmetry in favor of the unaffected ear. However, this notion was not used in other studies. Moreover, in this study, significantly more VEMP abnormalities were detected in both ears of BPPV patients than in the control group.

In our study, the mean interpeak amplitudes of the affected and unaffected ears of BPPV patients did not differ from each other (p=0.467); however, both were significantly lower than the mean value of the control group (p<0.001). The mean VEMP asymmetry values of BPPV patients did not differ from those of the healthy controls, although in 12 BPPV patients, the amplitude values were lower on the unaffected side.

Our findings are valuable because we detected that the cVEMP data from the affected and unaffected ears of BPPV patients are similar. Our research correlates with that in the literature with respect to the fact that interpeak amplitudes are lower in BPPV, and it additionally reveals new information that amplitudes are lower in both ears of BPPV patients. We consider this finding to be particularly important for the assessment of BPPV etiology.

Unilateral involvement is detected by the Dix–Hallpike test or by a head-roll test in a majority of BPPV patients. It is controversial whether the primary pathology is about the structure of the otoconia or the degeneration of the neuroepithelial membrane. Aging, diabetes mellitus, hypertension, thyroiditis, hyperlipidemia, stroke, osteopenia/osteoporosis, and vitamin D deficiency are responsible for causing the degenerative changes of the neuroepithelial membrane, and all these conditions are supposed to cause bilateral involvement [15]. Various studies have reported on the role of osteoporosis and vitamin D deficiency in the development of BPPV [4, 15-18].

Our research suggests neuroepithelial membrane involvement in both ears of unilateral BPPV patients. We think that due to this bilateral involvement, interpeak amplitude values are significantly lower in both ears of unilaterally symptomatic patients. This finding supports the idea that the pathophysiological process starts with neuroepithelial membrane degeneration and continues with otoconia separation. Our research also shows that bilateral otolith dysfunction is probable in unilaterally symptomatic BPPV patients.

The major limitation of our study is that cVEMP is an indirect measurement method for vestibular end organs.

We eventually concluded that even if the symptoms of BPPV are unilateral, findings suggesting bilateral involvement of the macular neuroepithelium are important in understanding the pathophysiology of BPPV. Further research is needed to determine the VEMP characteristics of BPPV patients with comorbidities that can cause neuroepithelial degenerative changes.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Haseki Training and Research Hospital (Reference number: June-2014/57).

Informed Consent: Written informed consent was obtained from patients and patients' parents who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - A.K., M.S.; Design - A.K., M.S.; Supervision - A.K., M.S.; Resources -A.K., T.Y.; Materials - A.K., T.Y., G.A.Y., C.H.; Data Collection and/or Processing - A.K., T.Y., G.A.Y., C.H.; Analysis and/or Interpretation - A.K., I.T.C., M.S.; Literature Search -T.Y., I.T.C., G.A.Y., C.H.; Writing Manuscript - A.K., I.T.C., M.S.; Critical Review - A.K., M.S.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Gacek RR. Pathology of benign paroxysmal positional vertigo revisited. Ann Otol Rhinol Laryngol 2003; 112: 574-82. [CrossRef]
2. Yetiser S, Ince D, Gul M. An analysis of vestibular evoked myogenic potentials in patients with benign paroxysmal positional vertigo. Ann Otol Rhinol Laryngol 2014; 123: 686-95. [CrossRef]
3. Furman JM, Cass SP. Benign paroxysmal positional vertigo. N Engl J Med 1999; 341: 1590-6. [CrossRef]
4. Vibert D, Kompis M, Häusler R. Benign paroxysmal positional vertigo in older women may be related to osteoporosis and osteopenia. Ann Otol Rhinol Laryngol 2003; 112: 885-9. [CrossRef]
5. Pisani V, Mazzone S, Di Mauro R, Giacomini PG, Di Girolamo S. A survey of the nature of trauma of post-traumatic benign paroxysmal positional vertigo. Int J Audiol 2015; 5: 329-33. [CrossRef]
6. Vibert D, Häusler R. Acute peripheral vestibular deficits after whiplash injuries. Ann Otol Rhinol Laryngol 2003; 3: 246-51. [CrossRef]
7. Kim JS, Oh SY, Lee SH, Kang JH, Kim DU, Jeong SH, et al. Randomized clinical trial for geotropic horizontal canal benign paroxysmal positional vertigo. Neurology 2012; 79: 700-7. [CrossRef]
8. Lee JD, Park MK, Lee BD, Lee TK, Sung KB, Park JY. Abnormality of cervical vestibular-evoked myogenic potentials and ocular vestibular-evoked myogenic potentials in patients with recurrent benign paroxysmal postitional vertigo. Acta Otolaryngol 2013; 133: 150-3. [CrossRef]
9. Parnes LS, McClure JA. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. Laryngoscope 1992; 102: 988-92. [CrossRef]
10. 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. [CrossRef]
11. Parham K, Leonard G, Feinn RS, Lafreniere D, Kenny AM. Prospective clinical investigation of the relationship between idiopathic benign paroxysmal positional vertigo and bone turnover: a pilot study. Laryngoscope 2013; 123: 2834-9. [CrossRef]
12. Cohen HS, Kimball KT, Stewart MG. Benign paroxysmal positional vertigo and comorbid conditions. ORL J Otorhinolaryngol Relat Spec 2004; 66: 11-5. [CrossRef]
13. Papi G, Guidetti G, Corsello SM, Di Donato C, Pontecorvi A. The association between benign paroxysmal positional vertigo and autoimmune chronic thyroiditis is not related to thyroid status. Thyroid 2010; 20: 237-8. [CrossRef]
14. 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: 710-5. [CrossRef]
15. Jeong SH, Kim JS, Shin JW, Kim S, Lee H, Lee AY, et al. Decreased serum vitamin D in idiopathic benign paroxysmal positional vertigo. J Neurol 2013; 260: 832-8. [CrossRef]
16. Talaat HS, Abuhadied G, Talaat AS, Abdelaal MS. Low bone mineral density and vitamin D deficiency in patients with benign positional paroxysmal vertigo. Eur Arch Otorhinolaryngol 2015; 272: 2249-53. [CrossRef]
17. Parham K, Kuchel GA. A geriatric perspective on benign paroxysmal positional vertigo. J Am Geriatr Soc 2016; 64: 378-85. [CrossRef]
18. Mikulec AA, Kowalczyk KA, Pfitzinger ME, Harris DA, Jackson LE. Negative association between treated osteoporosis and benign paroxysmal positional vertigo in women. J Laryngol Otol 2010; 124: 374-6. [CrossRef]
19. Gacek RR. A perspective on recurrent vertigo. ORL J Otorhinolaryngol Relat Spec 2013; 75: 91-107. [CrossRef]
20. Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by aclick-evoked vestibulocollic reflex. J Neurol Neurosurg Psychiatry 1994; 57: 190-7. [CrossRef]
21. Zhou G, Cox LC. Vestibular evoked myogenic potentials: history and overview. Am J Audiol 2004; 13: 135-43. [CrossRef]
22. Murofushi T, Iwasaki S, Ushio M. Recovery of vestibular evoked myogenic potentials after a vertigo attack due to vestibular neuritis. Acta Otolaryngol 2006; 126: 364-7. [CrossRef]
23. Shin BS, Oh SY, Kim JS, Kim TW, Seo MW, Lee H, et al. Cervical and ocular vestibular-evoked myogenic potentials in acute vestibular neuritis. Clin Neurophysiol 2012; 123: 369-75. [CrossRef]
24. Iwasaki S, McGarvie LA, Halmagyi GM, Burgess AM, Kim J, Colebatch JG, et al. Head taps evoke a crossed vestibulo-ocular reflex. Neurology 2007; 68: 1227-9. [CrossRef]
25. Salviz M, Yuce T, Karatas A, Balikci HH, Ozkul MH. Diagnostic value of frequency-associated vestibular-evoked myogenic potential responses in Meniere's disease. Audiol Neurootol 2015; 20: 229-36. [CrossRef]
26. Krempaska S, Koval J. The role of vestibular evoked myogenic potentials (VEMPs) in vestibulopathy diagnostics. Bratisl Lek Listy 2012; 113: 301-6. [CrossRef]
27. Kim EJ, Oh SY, Kim JS, Yang TH, Yang SY. Persistent otolith dysfunction even after successful repositioning in benign paroxysmal positional vertigo. J Neurol Sci 2015; 358: 287-93. [CrossRef]
28. Yang WS, Kim SH, Lee JD, Lee WS. Clinical significance of vestibular evoked myogenic potentials in benign paroxysmal positional vertigo. Otol Neurotol 2008; 29: 1162-6. [CrossRef]
29. Hong SM, Yeo SG, Kim SW, Cha CI. The results of vestibular evoked myogenic potentials, with consideration of age-related changes, in vestibular neuritis, benign paroxysmal positional vertigo, and Meniere's disease. Acta Otolaryngol 2008; 128: 861-5. [CrossRef]