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Clinical utility of cervical vestibular-evoked myogenic potentials in predicting residual dizziness after benign paroxysmal positional vertigo

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HIGHLIGHTS

- We performed multivariate analysis to derive cVEMP parameters associated with residual dizziness.
- cVEMP testing can be used for the prediction of residual dizziness after the recovery of BPPV.
- Increased cVEMP-modified IAD ratio on the affected side is associated with residual dizziness.

ABSTRACT

Objectives: In the present study, the value of cervical vestibular-evoked myogenic potential (cVEMP) as a predictive factor for residual dizziness after recovery of benign paroxysmal positional vertigo (BPPV) was evaluated.

Methods: The present study included 65 patients who had BPPV and underwent cVEMP testing. Patients were divided into two groups depending on the presence or absence of residual dizziness after recovery of BPPV. Univariate and multivariate analyses were performed to determine the factors associated with residual dizziness using age, gender, affected semicircular canal, affected side, BPPV duration, and cVEMP parameters.

Results: In univariate analysis, cVEMP-modified interaural amplitude difference (IAD) ratio and p13 latency showed a relatively significant association ($p < 0.20$) with residual dizziness. Based on multivariate analysis, increased cVEMP-modified interaural amplitude difference (IAD) ratio at the affected side ($\geq$25%; $p = 0.018$, OR 6.623) remained as an associated factor.

Conclusions: Increased cVEMP-modified IAD ratio at the affected side is associated with residual dizziness. BPPV patients with increased cVEMP-modified IAD ratio at the affected side are more likely to have residual dizziness after recovery of BPPV.

Significance: cVEMP testing could be used for the prediction of residual dizziness. An increased cVEMP-modified IAD ratio at the affected side may be used as a predictor of residual dizziness.

1. Introduction

Benign paroxysmal positional vertigo (BPPV) is one of the most common vestibular disorders with a lifetime prevalence of approximately 2.4% in the general population (von Brevern et al., 2007). Otoconial particles, which have fallen from otolith organs into semicircular canals, induce BPPV (Bhattacharyya et al., 2008). BPPV can be cured through appropriate canalith repositioning maneuvers (Fife et al., 2008; Helminski et al., 2010). However, despite clear pathogenesis and treatment, some patients still experience an imbalance after successful repositioning maneuvers. The imbalance, termed residual dizziness (Seok et al., 2008), includes a sensation of dizziness or lightheadedness in the absence of vertigo or nystagmus and reportedly persists from a few days to several weeks (Singh and Apeksha, 2016). The prevalence of residual dizziness ranges from 31 to 61% (Seok et al., 2008; Jung et al., 2012; Teggi et al., 2013; Kim and Lee, 2014).

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Otoconia are a major cause of BPPV and originate from degenerating otolithic organs (Gacek, 2003; Korres and Gkoritsa, 2011; Hoseinabadi et al., 2016; Singh and Apeksha, 2016; Chang et al., 2017), thus, residual dizziness might be induced by the disorder of otolithic organs. A relationship between residual dizziness and disorder of otolithic organs was reported in several studies (von Brevern et al., 2006; Yetiser et al., 2014; Seo et al., 2017).

Vestibular-evoked myogenic potentials (VEMPs) are electromyographic responses caused by vibration or sound stimulation originating from the vestibule and reflect the function of otolithic organs (Colebatch and Halmagyi, 1992). There are two types of VEMPs, cervical vestibular-evoked myogenic potentials (cVEMPs) and ocular vestibular-evoked myogenic potentials (oVEMPs) (Colebatch and Halmagyi, 1992; Colebatch et al., 1994; Chihara et al., 2007). cVEMP and oVEMP reflect saccular and utricular functions, respectively (Chihara et al., 2007; Govender et al., 2015). Seo et al. reported that oVEMPs are associated with residual dizziness (Seo et al., 2017) and due to the anatomical proximity, oVEMPs are considered more associated with residual dizziness than cVEMPs. However, the reflex pathway of cVEMPs also includes utricular components (Govender et al., 2015). In addition, Yetiser et al. reported the cVEMP results are associated with the duration of symptoms (Yetiser et al., 2014). In our previous study, the prognosis of BPPV was also shown associated with the cVEMP results (Chang et al., 2017). Considering that cVEMP is a more widespread test than oVEMP, a study investigating the association between cVEMP results and residual dizziness is important. In the present study, we evaluated the value of cVEMP as a predictive factor for residual dizziness after BPPV.

2. Materials and methods

2.1. Patients

A retrospective analysis of patients diagnosed with canalithiasis type of BPPV between April 2011 and March 2016 was performed. Patients treated for BPPV before visiting our center or diagnosed with secondary or bilateral BPPV were excluded. For example, patients with persistent dizziness or spontaneous nystagmus who were suspected of having unilateral weakness in canal function were excluded. A cVEMP test was performed within 7 days after diagnosis at our tertiary care center. All cVEMP tests were performed before recovery of BPPV, but some cVEMP tests were performed after repositioning maneuvers. This study was approved by the institutional review board (IRB) of our center (1711-013-16119).

We performed positioning maneuver and measured nystagmus. Based on these results, the diagnosis of BPPV, the affected side, and semicircular canal were determined. The patients were treated with repositioning maneuvers that removed the otoconia from the affected semicircular canal: Epley's maneuver was used for patients with posterior semicircular canal BPPV and barbecue roll maneuver for patients with lateral semicircular canal BPPV. Patients underwent the positioning maneuver at the following outpatient clinic. Whether BPPV was cured was determined by the disappearance of typical nystagmus and positional dizziness. Recovery from BPPV was defined as the absence of typical positional dizziness and nystagmus. If BPPV was not cured, the appropriate repositioning maneuver was performed again.

When the patient experienced persistent and non-positional atypical dizziness which interfered with daily life after recovery of BPPV, residual dizziness was diagnosed. All patients with residual dizziness underwent a bithermal caloric test on the day when recovery of BPPV was confirmed. The patients were divided into two groups depending on the presence or absence of residual dizziness. Group 1 consisted of patients diagnosed with no residual dizziness and group 2 consisted of patients diagnosed with residual dizziness.

We performed statistical analysis using age, gender, affected semicircular canal, affected side, BPPV duration, and cVEMP parameters to determine the predictive factors for residual dizziness.

2.2. Cervical vestibular-evoked myogenic potential

cVEMP was measured using Navigator Pro (Bio-logic Systems, Mundelein, IL, USA) according to the method described in a previous study (Chang et al., 2017). Briefly, surface electromyographic (EMG) activity was recorded in the sitting position with the head in rotation. The extent of sternocleidomastoid muscle (SCM) contraction (30 mmHg) was monitored with a self-monitored feedback method using a blood pressure manometer with an inflatable cuff. Acoustic stimuli (short tone bursts; 500 Hz, 95 dB HL, 5.1 times/second, 1-2-1 ms) were delivered through inserted earphones. The upper third of the SCM, the upper border of the sternum, and the glabella were used as the sites for the active, reference, and ground electrodes, respectively. The EMG signal from each side was amplified and bandpass-filtered between 10 and 1500 Hz. Typically, 200 responses from each ear were averaged. We measured the latencies of each peak (p13 and n23), interpeak latencies (ms), and p13–n23 amplitudes (μV). The modified IAD ratio was used to more intuitively understand the response of the affected side. The modified IAD ratio was calculated as follows: (affected side ear amplitude − unaffected side ear amplitude) ÷ (affected side ear amplitude + unaffected side ear amplitude). A modified IAD ratio of 25% was the criterion for an abnormal modified IAD ratio in our vestibular laboratory obtained by testing normal subjects. We considered the response of the affected side as augmented when the modified IAD ratio was >25% and reduced when the modified IAD ratio was <25%.

2.3. Caloric test

All patients with residual dizziness underwent a bithermal caloric test. The external auditory canal was irrigated alternately with a constant flow of warm (44 °C) and cold (30 °C) water for a constant period (30 seconds). Eye movements were recorded through a video-based system (ICS Chartr 200; GN Otometrics, Taastrup, Denmark). The maximum slow-phase velocity of nystagmus was calculated and a unilateral weakness in the caloric test was determined according to the Jongkees formula. A value >25 % was defined as abnormal.

2.4. Statistical analysis

Statistical analysis was performed in two steps. First, univariate analysis was used to identify factors associated with residual dizziness ($p < 0.20$). Age, gender, affected semicircular canal, affected side, BPPV duration, and cVEMP parameters were included in the univariate analysis. Next, multivariate logistic regression analysis was performed using the associated factors found in the univariate analysis. If the odds ratio (OR) was higher than 1 (or lower than 1), the associated factor was considered more (or less) related to residual dizziness. The difference between the two groups was compared using the Mann-Whitney U test. A p-value less than 0.05 was considered statistically significant. Statistical analyses were performed using the IBM SPSS software version 21.0 (IBM, Armonk, NY, USA).

3. Results

The present study included 65 patients with canalithiasis type of BPPV. The clinical characteristics of patients were summarized in Table 1. Among the 65 patients, 49 (75.4%) reported no dizziness after recovery of BPPV (group 1). Sixteen (24.6%) patients reported persistent and non-positional atypical dizziness after recovery of BPPV (group 2). The clinical characteristics of patients in group 2 were summarized in Table 2. All patients in group 2 showed no abnormality in the caloric test.

To determine the factors related to residual dizziness, we performed univariate and multivariate analyses. Univariate analysis revealed a relatively significant association ($p < 0.20$) between residual dizziness and the modified IAD ratio (Figs. 1 and 2) and p13 latency (Table 3). Age, gender, affected semicircular canal, affected side, BPPV duration, cVEMP n23 latency, interpeak latencies, and p13–n23 amplitude were not associated with residual dizziness. Multivariate analysis was performed using the relatively significantly associated variables, modified IAD ratio and p13 latency. In multivariate analysis, a modified IAD ratio $\geq$25% ($p = 0.018$, OR 6.623; 95% CI 1.385–31.683) remained an independent factor associated with residual dizziness (Table 4).

This study included both lateral and posterior canal BPPV. We further analyzed whether the affected canal affected the modified IAD ratio. Mean modified IAD ratios of lateral and posterior canal BPPV were 0.0% (±43.0%) and 8.9% (±39.0%), respectively. There was no significant difference between two groups ($p = 0.603$, Mann-Whitney U test). Analyzed among those with residual dizziness, mean modified IAD ratios of lateral and posterior canal BPPV were −6.8% (±33.4%) and 7.4% (±51.5%), respectively. There was no significant difference between two groups ($p = 0.913$, Mann-Whitney U test). The affected canal had no effect on the modified IAD ratio.

Table 1
Clinical characteristics of patients.

Characteristic	All patients (n = 65)	Group 1 (n = 49)	Group 2 (n = 16)
Age, years	53.1 (14.0)	52.4 (14.7)	55.1 (11.6)
Gender			
Male	17	14	3
Female	48	35	13
cVEMP			
p13 Latency (ms)	16.5 (2.2)	16.6 (2.0)	15.7 (1.8)
n23 Latency (ms)	24.1 (2.3)	24.3 (2.1)	23.5 (2.5)
Interpeak latency (ms)	7.6 (1.6)	7.8 (1.7)	7.8 (1.6)
p13–n23 Amplitude (μV)	74.3 (56.2)	73.3 (61.7)	53.9 (40.3)
Modified IAD ratio			
−25% < Modified IAD ratio < 25%	32	27	5
Modified IAD ratio ≤ −25%	22	17	5
Modified IAD ratio ≥ 25%	11	5	6
Affected SCC			
Posterior canal	47	36	11
Lateral canal	18	13	5
Affected side			
Right	33	23	10
Left	32	26	6
Duration of BPPV	7.1 (7.9)	6.5 (7.9)	9.0 (8.2)

All values are presented as either mean (standard deviation) or number of patients. Group 1 consisted of patients diagnosed with no residual dizziness. Group 2 consisted of patients diagnosed with residual dizziness. Three patients in group 1 and 2 patients in group 2 showed no cVEMP response. Measurements of p13 latency, n23 latency, interpeak latency, and p13–n23 amplitude were excluded in those patients. Modified IAD ratio was defined as follows: (affected side ear amplitude − unaffected side ear amplitude) ÷ (affected side ear amplitude + unaffected side ear amplitude).
cVEMP = cervical vestibular evoked myogenic potential; IAD = interaural amplitude difference; SCC = semicircular canal.

4. Discussion

BPPV is one of the most common diseases causing dizziness. Because its pathogenesis is relatively well known, BPPV can easily be treated with the canalith repositioning maneuver. However, some patients complain of dizziness referred to as residual dizziness (Seok et al., 2008) even after successful recovery of BPPV. Several studies were performed to determine the related factors and causes of residual dizziness. Consequently, several factors have been suggested to be associated with residual dizziness including duration of vertigo before successful repositioning maneuvers (Seok et al., 2008; Teggi et al., 2013; Faralli et al., 2016), old age (Oghalai et al., 2000; Pritcher et al., 2008; Teggi et al., 2011), anxiety disorders (Brandt, 1996; Huppert et al., 2005; Teggi et al., 2011; Faralli et al., 2016), and otolithic organ disorder (von Brevern et al., 2006; Yetiser et al., 2014; Seo et al., 2017). Among these factors, one of the most studied and evident is otolithic organ disorder. Several studies have reported causal relationships between otolithic organ disorder and residual dizziness. Thus, patients with BPPV may have otolith organ disorders at the same time because otoconia, a major cause of BPPV, originate in the degenerating otolith organ (Welling et al., 1997; Gacek, 2003; Korres and Gkoritsa, 2011; Hoseinabadi et al., 2016; Singh and Apeksha, 2016). BPPV can be treated with repositioning maneuvers, but otolith organ disorders may persist after these maneuvers. Therefore, the residual dizziness may remain if otolith organ disorder persists even after recovery of BPPV.

The relationship between residual dizziness and otolithic organ disorder has been suggested in several studies. von Brevern et al. reported that otolith-ocular reflex was decreased in patients with BPPV and the decrease persisted for one month after recovery of BPPV. They suggested that a decrease in otolith-ocular reflex was responsible for residual dizziness (von Brevern et al., 2006). Yetiser at al. reported that BPPV patients with a longer duration of symptoms had significantly longer p1 latency on cVEMPs than individuals having a shorter duration of symptoms. They suggested the outcomes of cVEMP could be associated with residual dizziness (Yetiser et al., 2014). Seo et al. reported that residual dizziness was significantly associated with the oVEMP results and suggested that residual dizziness is caused by persistent utricular dysfunction (Seo et al., 2017).

In the present study, the prevalence of residual dizziness was 24.6%, which was slightly lower than the results in previous studies (Seok et al., 2008; Jung et al., 2012; Teggi et al., 2013; Kim and Lee, 2014). We showed that if the cVEMP response of the affected side is significantly higher than the unaffected side, residual dizziness will likely remain after successful recovery of BPPV. These results are consistent with previous studies showing an association between otolith organ disorder and residual dizziness. The most important implication of the present study is that we can predict the occurrence of residual dizziness using cVEMP results. cVEMP is a test that can be performed quickly without discomfort, and the results can be reliably obtained. Therefore, prediction of the occurrence of residual dizziness based on the results of cVEMP may be helpful for the management of BPPV patients. In addition to this finding, there are two noteworthy findings in our study. First, the results were obtained using cVEMP testing, not oVEMP. Due to the anatomical proximity, otoconia, which induce BPPV, are likely to originate from the utricle. Therefore, cVEMP, which mainly reflects saccular function rather than utricular function, may be assumed not to be associated with residual dizziness. However, a previous report showed the association between residual dizziness and cVEMP (Yetiser et al., 2014), and in several studies

Table 2
Clinical characteristics of patients with residual dizziness.

Patient No.	Gender	Age	BPPV			UW in the caloric test (%)	Duration of residual dizziness (days)
			Affected side	Affected SCC	Duration (days)		
1	F	56	R	Post.	3	21	43
2	F	27	R	Post.	3	7	33
3	F	35	R	Post.	13	3	20
4	M	67	R	Post.	9	14	12
5	F	65	R	Post.	5	9	14
6	F	64	R	Post.	3	18	31
7	F	51	R	Lat.	3	2	7
8	F	57	R	Lat.	3	11	13
9	F	54	R	Lat.	14	4	24
10	F	55	R	Lat.	21	6	39
11	F	69	L	Post.	3	10	23
12	F	51	L	Post.	30	11	29
13	F	61	L	Post.	11	3	15
14	M	69	L	Post.	17	10	24
15	F	48	L	Post.	3	9	12
16	M	52	L	Lat.	3	3	7

BPPV = benign paroxysmal positional vertigo; SCC = semicircular canal; F = female; M = male; R = right; L = left; Post. = posterior; Lat. = lateral; UW = unilateral weakness.

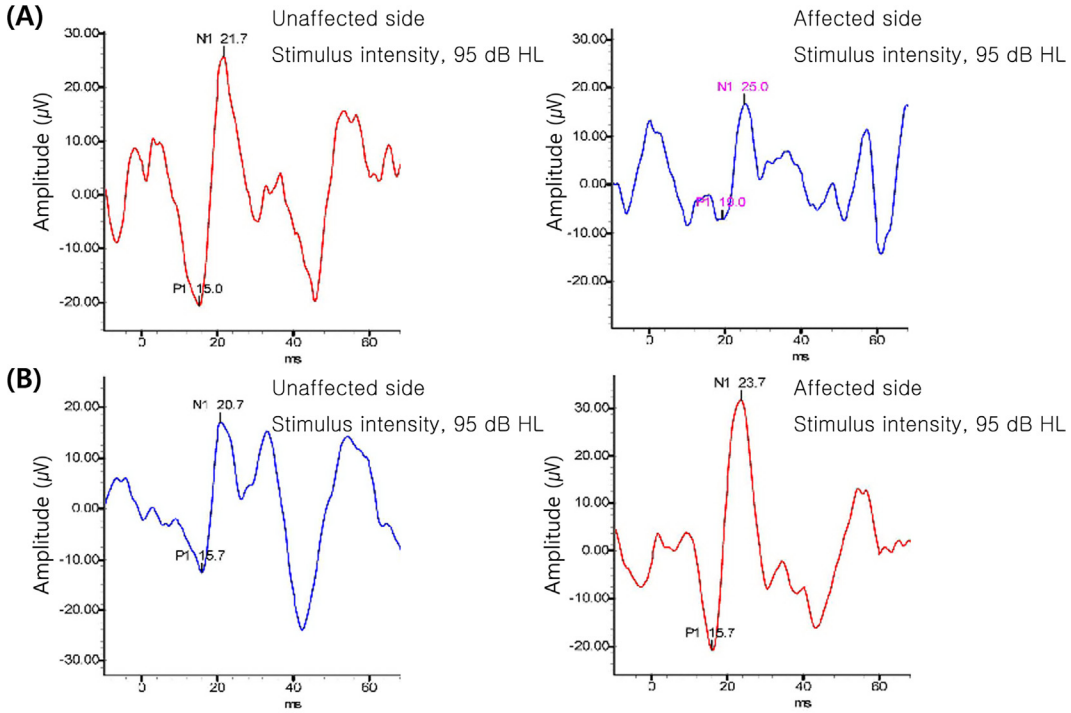

Fig. 1. cVEMP results. (A) A 57-year-old female with left lateral semicircular canal BPPV. cVEMP parameters were as follows: p13–n23 amplitudes at the unaffected side = 46.34 μV; p13–n23 amplitudes at the affected side = 23.98 μV; and modified IAD ratio = −31.8%. She did not experience imbalance after successful recovery of BPPV. (B) A 64-year-old female with right posterior semicircular canal BPPV. cVEMP parameters were as follows: p13–n23 amplitudes at the unaffected side = 29.71 μV; p13–n23 amplitudes at the affected side = 52.27 μV; and modified IAD ratio = 27.5%. She experienced imbalance after successful recovery of BPPV. Modified IAD ratio was defined as follows: (affected side ear amplitude − unaffected side ear amplitude) ÷ (affected side ear amplitude + unaffected side ear amplitude). cVEMP = cervical vestibular-evoked myogenic potential; BPPV = benign paroxysmal positional vertigo; IAD = interaural amplitude difference.

the association of BPPV with cVEMP was reported (Akkuzu et al., 2006; Hong et al., 2008; Korres and Gkoritsa, 2011; Longo et al., 2012; Lee et al., 2013; Sreenivasan et al., 2015; Hoseinabadi et al., 2016). Our previous study also showed the cVEMP results were associated with the prognosis of BPPV (Chang et al., 2017). Therefore, predicting residual dizziness based on cVEMP results has some empirical support. Further studies will be carried out to investigate the association between oVEMP-IAD ratios and residual dizziness. Another important finding in our study was that only the increased cVEMP-modified IAD ratio at the affected side was associated with residual dizziness, and the decreased cVEMP-modified IAD ratio was not associated with residual dizziness. Considering that several studies have reported that otolith function is reduced in BPPV patients (von Brevern et al., 2006; Longo et al., 2012; Lee et al., 2013), the increased cVEMP-modified IAD ratio is presumed to mean otolith organ disorder. However, there is still uncertainty as to the difference in mechanisms that lead to increased and decreased IAD ratios, and further studies are needed.

In some studies, the mechanism of residual dizziness has been attributed to factors other than the otolith organ disorder. Faralli et al. suggested the brain adapts to the presence of BPPV and cannot adapt to the situation in which the BPPV is fully cured, resulting in residual dizziness (Faralli et al., 2016). Inagaki et al. reported

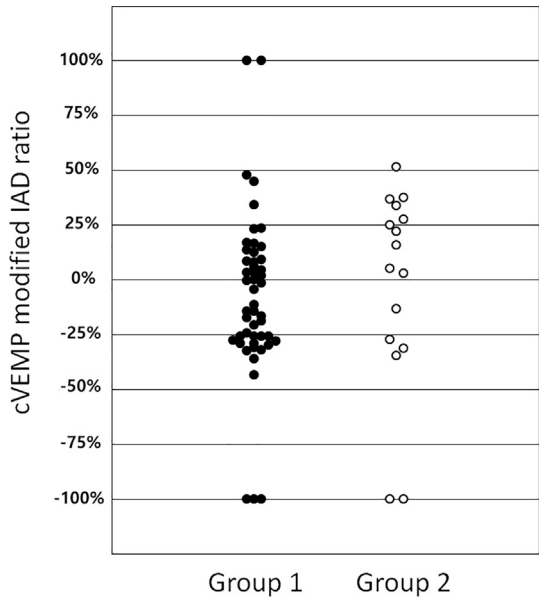

Fig. 2. Distribution of cVEMP-modified IAD ratios in groups 1 and 2. Group 1 consisted of patients diagnosed with no residual dizziness. Group 2 consisted of patients diagnosed with residual dizziness. cVEMP-modified IAD ratio was defined as follows: (affected side ear amplitude − unaffected side ear amplitude) ÷ (affected side ear amplitude + unaffected side ear amplitude). cVEMP = cervical vestibular-evoked myogenic potential, IAD = interaural amplitude difference.

that after the repositioning maneuver, the movement of otoconia entering the utricle causes residual dizziness (Inagaki et al., 2006). Di Girolamo et al. suggested that even after the repositioning maneuver, a small amount of otoconia remained in the semicircular canal causing residual dizziness (Di Girolamo et al., 2000). Based on the results of these studies, residual dizziness may not be caused solely by otolith organ disorder. Residual dizziness appears to be a type of syndrome caused by various factors rather than a disease originating from a single cause. However, otolith organ disorder has been considered the most important factor in the development of residual dizziness and cVEMP may play an important role in predicting residual dizziness.

Table 4
Multivariate analysis of factors potentially associated with residual dizziness after BPPV.

Characteristic	*p*-value	OR	95% CI
cVEMP modified IAD ratio			
−25% < modified IAD ratio < 25%	Reference		
Modified IAD ratio ≤ −25%	0.625	1.506	0.291–7.785
Modified IAD ratio ≥ 25%	0.018	6.623	1.385–31.683
p13 latency (ms)	0.233	0.805	0.564–1.149

Modified IAD ratio was defined as follows: (affected side ear amplitude − unaffected side ear amplitude) ÷ (affected side ear amplitude + unaffected side ear amplitude).
OR = odds ratio; 95% CI = 95% confidence interval; cVEMP = cervical vestibular evoked myogenic potential; IAD = interaural amplitude difference.

5. Conclusion

The major finding from our study is that increased cVEMP-modified IAD ratio at the affected side was associated with residual dizziness. BPPV patients with an increased cVEMP-modified IAD ratio at the affected side were more likely to have residual dizziness after a successful repositioning maneuver. Therefore, we propose that the cVEMP-modified IAD ratio can be used as a predictor of residual dizziness.

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Table 3
Univariate analysis of factors potentially associated with residual dizziness after BPPV.

Characteristic	Group 1	Group 2	*p*-value	OR	95% CI
Age, years	52.4 (14.7)	55.1 (11.6)	0.512	1.014	0.972–1.058
Gender					
Male	82.4%	17.6%	Reference		
Female	72.9%	27.1%	0.441	1.733	0.427–7.029
cVEMP					
p13 Latency (ms)	16.6 (2.0)	15.7 (1.8)	0.172	0.778	0.543–1.115
n23 Latency (ms)	24.3 (2.1)	23.5 (2.5)	0.227	0.832	0.617–1.121
Interpeak latency (ms)	7.8 (1.7)	7.8 (1.6)	0.975	1.006	0.703–1.438
p13–n23 Amplitude (μV)	73.3 (61.7)	53.9 (40.3)	0.240	1.008	0.995–1.021
Modified IAD ratio					
−25% < Modified IAD ratio < 25%	84.4%	15.6%	Reference		
Modified IAD ratio ≤ −25%	77.3%	22.7%	0.801	0.856	0.255–2.869
Modified IAD ratio ≥ 25%	45.5%	54.5%	0.017	5.280	1.340–20.802
Affected SCC					
Posterior canal	76.6%	23.4%	Reference		
Lateral canal	72.2%	27.8%	0.714	1.259	0.367–4.318
Affected side					
Right	69.7%	30.3%	Reference		
Left	81.3%	18.8%	0.283	0.531	0.167–1.688
Duration of BPPV	6.5 (7.9)	9.0 (8.2)	0.273	1.038	0.971–1.109

All values are presented as either mean (standard deviation), percentage of patients or each parameter. Group 1 consisted of patients diagnosed with no residual dizziness. Group 2 consisted of patients diagnosed with residual dizziness. Three patients in group 1 and 2 patients in group 2 showed no cVEMP response. Measurements of p13 latency, n23 latency, interpeak latency, and p13–n23 amplitude were excluded in those patients. Modified IAD ratio was defined as follows: (affected side ear amplitude − unaffected side ear amplitude) ÷ (affected side ear amplitude + unaffected side ear amplitude).
BPPV = benign paroxysmal positional vertigo; cVEMP = cervical vestibular evoked myogenic potential; IAD = interaural amplitude difference; SCC = semicircular canal.

Conflict of interest

The authors have no potential conflicts of interest to disclose.

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