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Persistent otolith dysfunction even after successful repositioning in benign paroxysmal positional vertigo

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ABSTRACT

To evaluate utricular and saccular function during the acute and resolved phases of BPPV, ocular and cervical vestibular evoked myogenic potentials (VEMPs) were studied in 112 patients with BPPV and 50 normal controls in a referral-based University Hospital. Ocular (oVEMPs) and cervical VEMPs (cVEMPs) were induced using air-conducted sound (1000 Hz tone burst, 100 dB normal hearing level) at the time of initial diagnosis and 2 months after successful repositioning in patients with BPPV, and the results were compared with those of the controls. Abnormalities of cVEMPs and oVEMPs in patients with BPPV were prevalent and significantly higher compare to the healthy control group ($p < 0.01$ in each VEMP by chi-square test). In the patient group, difference between the proportions of abnormal responses of cVEMP and oVEMP was not significant in both affected ($p = 0.37$, chi-squared test) and non-affected ($p = 1.00$) ears. The abnormalities were more likely reduced or absent responses rather than delayed ones; reduced or absent responses are 17.6% in cVEMPs ($p = 0.04$, chi-square) and 21.6% in oVEMPs ($p < 0.01$). The non-affected ear in the BPPV group also showed significantly higher abnormalities of cVEMP and oVEMP when compared to the control group. The follow-up VEMPs after repositioning maneuvers were not significantly different compared to the initial values from both stimulated affected and non-affected ears. Although most patients had unilateral BPPV, bilateral otolithic dysfunction was often shown by persistently reduced or absent cervical and ocular VEMPs, suggesting that BPPV may be caused by significant bilateral damage to the otolith organs.

1. Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common cause of peripheral vestibular vertigo [1,2]. In patients with BPPV, the otoconia are dislodged from their usual position within the utricle and migrate into one of the semicircular canals, most often into the posterior semicircular canal probably due to its dependent position [3]. Utricular dysfunction in patients with BPPV has been supported by many post mortem studies that detected damage to the utricular macula on the side affected by BPPV [4]. From these findings, we may expect that patients with idiopathic BPPV have utricular dysfunction more frequently than saccular dysfunction. However, there is increasing evidence that otoconia causing BPPV derive from both maculae [5–9]. Although the pathology that leads to detachment of the otoconia is not yet fully understood, degenerative changes may cause a decrease in the gelatinous layer of the otolithic membrane, which may allow spontaneous dislodgment of the otoconia from the utricular or saccular macula to occur more easily [10,11].

Otolith function can be evaluated with both ocular (oVEMPs) and cervical (cVEMPs) vestibular evoked myogenic potentials. cVEMPs are known to reflect the function of ipsilateral sacculo-collic inhibitory pathways [12,13] while oVEMPs are probably a manifestation of crossed utriculo-ocular reflex pathways [14–16].

The purpose of this study was to characterize utricular or saccular dysfunction in patients with BPPV using cervical and ocular VEMPs in response to air-conducted sound (ACS) and changes of VEMP findings after successful repositioning maneuvers.

2. Methods

2.1. Subjects

Between February 2011 and October 2012, 112 consecutive patients with a diagnosis of BPPV were recruited from the Dizziness Clinic of Chonbuk National University Hospital in Korea (Fig. 1). We only included the patients with the first attack of idiopathic BPPV. Nystagmus was observed without fixation using a video-Frenzel goggle (SLMED, Seoul,

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Korea) system. The diagnosis of BPPV was based on (1) a history of short-lasting positional vertigo, (2) characteristic positional nystagmus; a mixed torsional/up-beating nystagmus in the Dix–Hallpike position or direction-changing horizontal nystagmus beating toward the uppermost (apogeotropic nystagmus) or undermost ear (geotropic nystagmus) in both lateral head turning positions, (3) absence of identifiable central nervous system disorders that could explain the positional vertigo and nystagmus, and (4) absence of any history of neurotological disorder such as Ménière's disease, labyrinthitis, migraine or head trauma.

The affected ear was determined with an assumption that the induced nystagmus is more intense when the head is rotated to the affected side in cases with geotropic HC-BPPV and less intense in cases with apogeotropic HC-BPPV. When the decision was inconclusive due to rather symmetrical head turning nystagmus, the direction of lying-down or head-bending nystagmus was also considered. In recent studies, the direction of lying-down nystagmus was mostly toward the affected ear in apogeotropic HC-BPPV and toward the unaffected ear in geotropic nystagmus while the direction of head-bending nystagmus was largely opposite in each HC-BPPV [10,17]. Cases with PC-BPPV involving bilateral sides of the posterior semicircular canals were excluded in this study analysis.

To exclude patients with positioning nystagmus from central pathologies, all patients also received neurological examinations, including spontaneous nystagmus and gaze-evoked nystagmus (GEN), horizontal and vertical saccades and smooth pursuit, limb ataxia, and balance function.

Age- and sex-matched 50 healthy subjects (aged 43–76 years; mean, 60.1 years; 23 males) without a history dizziness served as control after confirming normal findings with neurological examination and pure tone audiogram (Table 1).

The VEMPs were evaluated 2 separate times in the patients, just after the diagnosis and before the repositioning maneuver, and two months after successful repositioning maneuvers when the positional vertigo and nystagmus were resolved. Patients with persistent dizziness but without positional nystagmus (n = 7) were excluded from the follow-up VEMP study.

2.2. Cervical and ocular VEMP recording

In order to record cVEMPs, subjects were in the supine position on a bed, raised their head approximately 30 from the horizontal, rotated it contralaterally in order to activate the sternocleidomastoid (SCM) muscles, and then the surface EMG activity was measured from an active electrode placed over the belly of the ipsilateral SCM and from a reference electrode on the medial clavicle using self-adhesive Ag/AgCl electrodes. We monitored the background SCM contraction levels in order to match the EMG levels between the sides visually. Although there are many individual variances in the EMG levels, we tried to be equal within 10% of contraction level compare to the other side.

For the recording of oVEMPs, the subject looked up approximately 25 above in the supine position. For each eye, the active recording electrode was placed on the infra-orbital ridge 1 cm below the center of each lower eyelid and the reference electrode was placed approximately 2 cm below that active electrode [14].

Stimuli were generated by customized software (Cadwell Laboratories, Kennewick, WA, USA). We used unilateral 1000 Hz, 5 ms ACS tone bursts as the stimulation, which were provided at an intensity of 100 dB nHL through calibrated headphones. A total of 100 stimuli were delivered at a rate of 5 Hz. The responses were sampled at 10 kHz for 60 ms, from 10 ms before to 50 ms following the stimulus onset. For the responses recorded from the SCM ipsilateral to the stimulated mastoid, the initial biphasic positive and negative peak-to-peak amplitudes were measured. The EMG potentials were amplified, band-pass filtered at 10–3000 Hz, and sampled at 5 kHz, then the data from the stimulus onset to 50 ms was averaged. The amplitude was defined as the value of the peak to peak difference for cervical and ocular VEMPs. The amplitude asymmetry ratio (AR) of oVEMPs and cVEMPs was calculated using the following formula: AR = [(non-affected side − affected side) / (non-affected side + affected side) × 100].

Abnormal VEMPs were defined with any of the three criteria, i.e. absent responses, asymmetric responses (AR > mean + 2 standard deviation [SD]) or delayed latencies (latency > mean + 2 SD). ARs were counted as abnormal for the affected ear if the response on the affected side was smaller and for the unaffected side if the response on the affected side was larger. For both cervical and ocular VEMPs, responses from the neck and eyes are described with the stimulating ear with reference to lesion side i.e., the “affected” or “non-affected” side of the ear in the patient group.

All procedures were in accordance with the Declaration of Helsinki and were approved by the Chonbuk National University Hospital Ethics Committee (IRB No. 2012-05-026). Informed consent was obtained from all participants.

2.3. Statistical analysis

Comparison of the means of p13 and n10 latency, amplitude, and AR between patients and controls was done using Student *t*-tests. To analyze differences between before and after treatment and differences between affected and non-affected ears in BPPV patients, a paired *t*-test were used. Clinical findings between the patients groups and controls were compared using chi-squared and ANOVA. All statistical procedures were performed using SPSS statistical software version 20 (IBM Corp., Armonk, NY, USA).

3. Results

3.1. VEMPs in the healthy control group

cVEMPs were obtained from 94 ears in 50 healthy subjects (94/100 ears, 94%). The mean p13 latency of cVEMPs did not differ between the right and left sides (13.2 ± 1.4 vs. 13.1 ± 1.4 ms, paired t-test, $p > 0.05$). The averaged peak to peak amplitudes of the healthy controls showed a mean of 403.7 ± 254.0 μV on the right and 387.4 ± 261.0 μV on the left (paired t-test, $p > 0.05$). The AR of cVEMPs was 9.8 ± 7.6%. Initial negative responses (n10) of oVEMPs were recorded during ACS stimulation of 90 ears in 50 participants (90/100 ears, 90%). The n10

Table 1
Demographic characteristics of patients with benign paroxysmal positional vertigo (BPPV) and healthy controls.

	Age (years)[a]	Sex (male, %)	Disease duration (days)[a]	Mean number of CRP[a]
PC-BPPV (n = 47)[b]	63.9 ± 12.1	22 (41.5%)	3.8 ± 1.1	2.4 ± 1.1
HC-BPPV (n = 51)[c]	59.9 ± 14.1	25 (49.0%)	3.8 ± 1.3	2.6 ± 1.0
Mixed BPPV (n = 4)[d]	64.6 ± 10.3	1 (12.5%)	4.4 ± 1.4	3.2 ± 0.7
Total (n = 102)	62.8 ± 13.1	48 (42.9%)	3.9 ± 1.2	2.6 ± 1.0
Healthy control (n = 50)	60.1 ± 9.2	23 (46.0%)		
p-Value*	0.17	0.42	0.59	0.22

[a], Mean age ± SD; [b, c, d], BPPV involving the posterior, horizontal, and more than two semicircular canals, respectively.
* Kruskal–Wallis test.

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response had a mean latency of 9.7 ± 1.1 ms, mean amplitude of 6.6 ± 5.5 μV, and a mean AR of 14.4 ± 11.3%.

3.2. Cervical and ocular VEMPs in the BPPV group

In the patients (n = 112), BPPV involved the posterior semicircular canal in 53 patients (53/112, 47.3%), the horizontal semicircular canal in 51 (51/112, 45.5%), and both the horizontal and posterior semicircular canals in 8 (8/112, 7.1%). Of the patients with HC-BPPV, 26 (26/51, 51.0%) showed a geotropic nystagmus, and 25 (25/51, 49.0%) had apogeotropic nystagmus. Ten patients (PC-BPPV in 6, and mixed type BPPV in 4) with bilateral BPPV who revealed upbeating and torsional nystagmus during both sides of Dix-Hallpike maneuver were excluded in the analysis. Therefore, each 102 ears were assigned to the affected ear and to the non-affected ear group (Table 1).

Compared to the healthy controls, the BPPV patients showed longer latency, smaller amplitude, and more asymmetric AR during the affected ear was stimulated when we analyzed only the patients' results who generate VEMPs (Table 2). The proportion of abnormal findings of cVEMPs and oVEMPs in the patient group was prevalent and significantly higher compare to the healthy control group (Fig. 2, Table 3, $p < 0.01$ in each VEMP by chi-square test). A sub-analysis of the HC-BPPV group between geotropic and apogeotropic types showed significantly frequent abnormal responses in each group compared to healthy controls ($p < 0.01$ in apogeotropic type and $p = 0.02$ in geotropic types in each VEMP by Fisher's exact test).

In patients with BPPV, the difference between the proportions of abnormal responses of cVEMP and oVEMP was not significant in each affected ($p = 0.37$, chi-squared test) and non-affected ($p = 1.00$) ear (Table 3). A cVEMP study recorded at the ipsilateral SCM muscle showed abnormal responses in 29 out of 102 affected ears (29/102, 28.4%) including absent (n = 10) or reduced (n = 8) response (18/102, 17.6%) and delayed p13 latency (11/102, 10.8%). In the oVEMP study, among 102 affected ears, 35 ears (35/102, 34.3%) revealed abnormal responses of the n10 component, being absent (18/102, 17.6%), abnormal AR (4/102, 3.9%), delayed latency (11/102, 10.8%), or delayed latency with abnormal AR (2/102, 1.9%) beneath the eye opposite of the affected ear.

To identify if chronicity and treatment resistance of BPPV are associated with VEMP abnormalities, comparison between patients with abnormal and normal VEMPs revealed that disease duration and required number of repeated canalith repositioning maneuvers to resolve vertigo and nystagmus did not show any significant differences (4.1 ± 1.0 vs. 3.8 ± 1.3 days, $p = 0.22$; 2.8 ± 1.0 vs. 2.5 ± 1.0 times, $p = 0.11$; Student t-test). Although there are some difficulties in evaluation of VEMP in acute condition of BPPV, all participants were tested without complications.

3.3. VEMPs at the non-affected ears in patients with BPPV

The proportion of abnormal VEMPs in the non-affected ears of the BPPV population was also significantly higher compared to the healthy control group (Table 3). Abnormal cVEMPs were recorded in 28 of 102 non-affected ears (28/102, 27.5%) of BPPV patients, including no response (n = 4), delayed latency (n = 6), abnormal AR (n = 14), and delayed latency with abnormal AR (n = 4) (Table 3). During oVEMP with stimulation of non-affected ears, 28 out of 102 non-affected ears (28/102, 27.5%) showed abnormal oVEMPs with absent (n = 12), delayed latency (n = 10), asymmetric AR (n = 3), and delayed latency with abnormal AR response (n = 3) (Table 3). Between the affected and non-affected ears, however, there were no statistically significant differences in the results of both cVEMP ($p = 0.88$ by chi-square test) and oVEMP ($p = 0.29$, chi-square test) in each subtype of BPPV. The overall prevalence of abnormal cVEMPs or oVEMPs in patients with BPPV was 42.2% in the affected ear (43/102, 42.2%) and 40.2% (41/102, 40.2%) in the non-affected ear, which were not significant between two sides ($p = 0.78$, chi-square test). Among them, ten patients (10/102, 9.8%) revealed abnormalities on both sides.

3.4. Follow-up VEMPs after successful repositioning maneuvers

Two months after repositioning maneuvers, we evaluated the follow-up VEMPs in 59 of 102 BPPV patients who were successfully managed with repositioning therapies. The follow-up results of p13 latency and AR after repositioning maneuvers were not significantly different compared to the initial values from both stimulated affected and non-affected ears ($p = 0.32$ in the affected ear, $p = 0.65$ in the non-affected ear for p13 latency; $p = 0.43$ for AR; paired t-test; Figs. 3 and 4). In affected ears, a single case who presented normal cVEMP responses before repositioning maneuvers revealed abnormal follow-up results with delayed p13 latency, while 2 cases with initial prolonged responses normalized. In the follow-up oVEMP, the response of n10 revealed mildly increased latency and asymmetric AR compared to the pretreatment values, but these results did not show significant differences ($p = 0.29$, $p = 0.12$ respectively, Figs. 3 and 4). Newly abnormal responses were observed in 10 subjects, including 6 affected ear and 4 non-affected ear stimulations with 7 delayed n10 latency and 3 absent responses beneath each opposite eye.

4. Discussion

In the present study, both cVEMPs and oVEMPs were used to assess saccular and utricular function in patients with BPPV before repositioning maneuvers and 2 months later. Several studies have reported VEMP abnormalities in patients with BPPV [18], but most reports did

Table 2
Comparison of average VEMP parameters between the results of affected ears of patients and of healthy controls.

Subgroups		Latency of initial peak[a]	p-Value*	P-p amplitude[a,b]	p-Value*	AR[a, c]	p-Value*
PC-BPPV[d]	cVEMP	14.1 ± 1.0	<0.01	256.3 ± 170.4	0.04	20.8 ± 15.3	<0.01
	oVEMP	10.8 ± 1.0	<0.01	4.9 ± 5.9	0.02	19.4 ± 16.0	0.04
HC-BPPV[e]	cVEMP	14.4 ± 1.9	<0.01	275.9 ± 168.2	0.04	24.8 ± 20.7	<0.01
	oVEMP	10.4 ± 0.9	<0.01	3.5 ± 2.1	<0.01	19.5 ± 15.7	0.04
Mixed BPPV[f]	cVEMP	14.4 ± 1.0	<0.01**	217.1 ± 25.8	0.05**	10.4 ± 3.20	0.4**
	oVEMP	10.3 ± 0.7	0.12**	4.8 ± 2.4	0.92**	12.9 ± 10.0	0.77**
Patients total	cVEMP (n = 95)	14.3 ± 1.5	<0.01	265.4 ± 165.3	0.04	22.1 ± 17.9	<0.01
	oVEMP (n = 84)	10.6 ± 0.9	<0.01	4.1 ± 4.1	0.04	19.4 ± 15.7	0.02
Healthy	cVEMP (n = 94)	13.2 ± 1.4		395.6 ± 258.9		9.8 ± 7. 6	
	oVEMP (n = 90)	9.7 ± 1.1		6.6 ± 5.5		14.4 ± 11.3	

The absent response was excluded from this analysis of mean parameters which is compared with normal controls by Student t-test (*) and by Mann–Whitney test (**).
[a] Mean value ± SD.
[b] Peak to peak amplitude.
[c] Asymmetry ratio.
[d] PC-BPPV = benign paroxysmal positional vertigo (BPPV) involving the posterior semicircular canal.
[e] HC-BPPV = BPPV involving the horizontal semicircular canal.
[f] Mixed BPPV = BPPV involving the horizontal and posterior semicircular canals.

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Table 3
Cervical and ocular VEMP abnormalities in affected and non-affected ears in patients with BPPV.

Subjects		cVEMPs		oVEMPs	
		Normal	Abnormal	Normal	Abnormal
PC-BPPV[a] (n = 47)	Affected	35	12 (25.5%)	29	18 (38.3%)
	Non-affected	37	10 (21.3%)	33	14 (29.8%)
HC-BPPV[b] (n = 51)	Affected	37	14 (27.5%)	37	14 (27.5%)
	Non-affected	34	17 (33.3%)	39	12 (23.5%)
Mixed BPPV[c] (n = 4)	Affected	1	3 (75%)	1	3 (75%)
	Non-affected	3	1 (25%)	2	2 (50%)
Total (n = 102)	Affected	73	29(28.4%)	67	35(34.3%)
	Non-affected	74	28(27.5%)	74	28(27.5%)

[a,b,c], BPPV involving the posterior, horizontal, and involving the horizontal and posterior semicircular canals, respectively.

not include comparison between the affected and non-affected sides and follow-up studies after successful repositioning. The prevalence of abnormal VEMPs ranged from 10% to 50% in patients with BPPV, and the prevalence was higher in the recurrent BPPV [11,19]. According to our results, although the normal values for VEMPs, especially for the cVEMPs, are relatively small compare to our previous studies [14,20], which can artificially inflate the rate of abnormalities, the BPPV group had significantly higher prevalence of VEMP abnormalities compared to the control group (Fig. 2). It is also needed to be cautious to interpret the results of AC oVEMP studies in cases when there is the high absent rate in normal subjects (10% in current study). Therefore, the actual rate of abnormalities would be lower as false positives are expected. In current study, in affected ear, absence rate was 18/102 (17.6%) and only 7.6% might be caused by BPPV. However, the patients with BPPV appear to have otolithic abnormalities more frequently than normal controls. The frequency and characteristics of abnormal VEMPs did not differ among different types of BPPV (Tables 2 and 3). The overall prevalence of cVEMP abnormality was 28.4% (29/102) in the affected ear and 27.5% (28/102) in the non-affected ear. The oVEMP abnormalities were observed in 34.3% (35/102) of the affected ears and 27.5% (28/102) of the non-affected ears. Thus, the prevalence of abnormal cVEMPs and oVEMPs was similar between the affected and non-affected ears in the patients (Table 3).

Characteristics of abnormal cervical and ocular VEMPs in patients with BPPV were reduced or absent responses rather than delayed responses. Reduced or absent cVEMP responses were 62.1% (18/29 patients with abnormal result) and delayed responses in 37.9% (11/29) ($p = 0.04$, chi-square). Similarly, reduced or absent oVEMP responses were more common than delayed responses (68.6% vs. 31.4%, $p < 0.01$). The VEMP abnormalities might be related to the nature and degree of damage in the corresponding pathways. The reduced or absent VEMPs in the late stage of Meniere's disease was attributed to permanent morphological changes involving the sensory organs. Also, in central vestibulopathy, delayed peak latency might be the result of slowed conduction along the vestibulo-collic or vestibulo-ocular tracts consequent to demyelination [21–23], while the absent or reduced responses were mostly found in brainstem infarct or hemorrhage [24]. VEMPs amplitudes were usually reduced in patients with BPPV [11,18]. However, recently, one study showed a transient increase of oVEMP amplitudes affected ear after successful liberatory maneuvers but no changes in cVEMP amplitudes [25]. They suggested that successful liberatory maneuvers can lead to a repositioning of otoconia to the utricle and cause increase of oVEMP amplitudes. Furthermore, our result revealed that the non-affected ear in the BPPV group also showed significantly higher abnormalities of VEMPs. Therefore, AR of the VEMPs in clinical studies should be interpreted cautiously because it couldn't be assumed that the side with smaller amplitude was abnormal or that the affected side showed small VEMPs. However, our study showed that the amplitudes of the affected sides were reduced and AR of oVEMP as well as cVEMP was not changed between before and after treatment (Figs. 3 and 4). Although BPPV is caused by detached otoconia mostly from the utricular macula due to its anatomical proximity with the ampulla of the posterior semicircular canal, the degenerative processes appear to involve the saccular macula too, resulting in abnormal cVEMPs as well as oVEMPs [5]. Otherwise, the abnormal cVEMPs might have been due to utricular dysfunction. Animal studies showed that

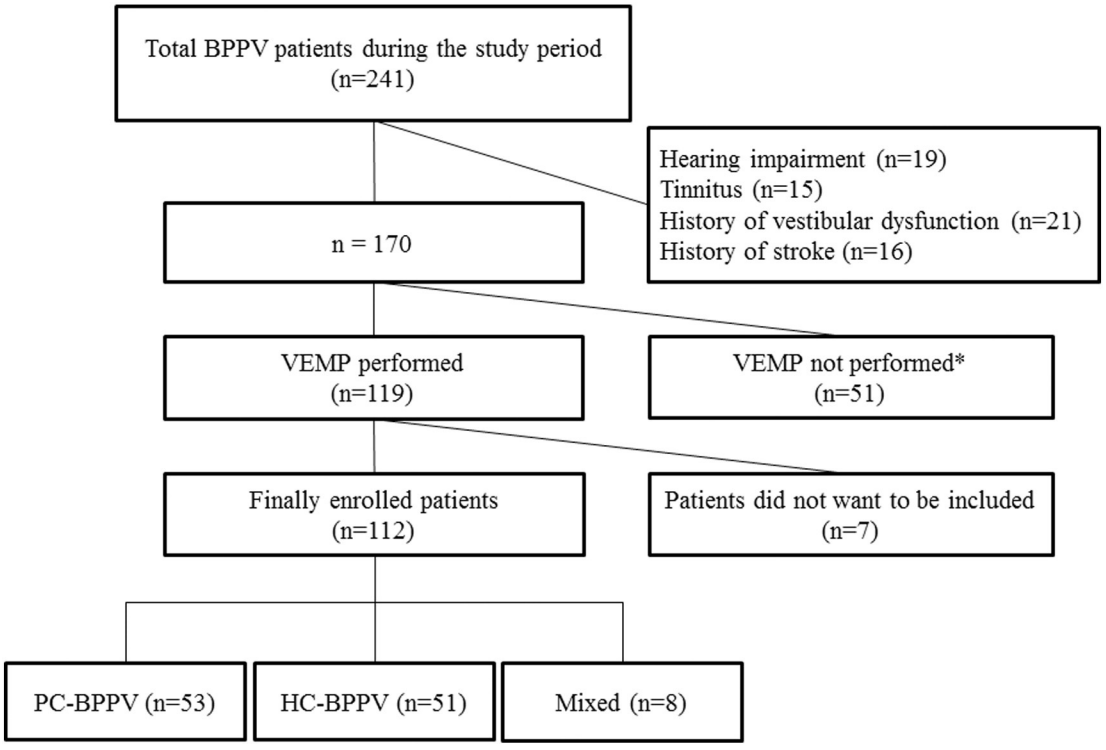

Fig. 1. Flow chart of the study design. *: We excluded the patients who complained persistent dizziness without positional nystagmus in the follow-up VEMP testing (n = 7).

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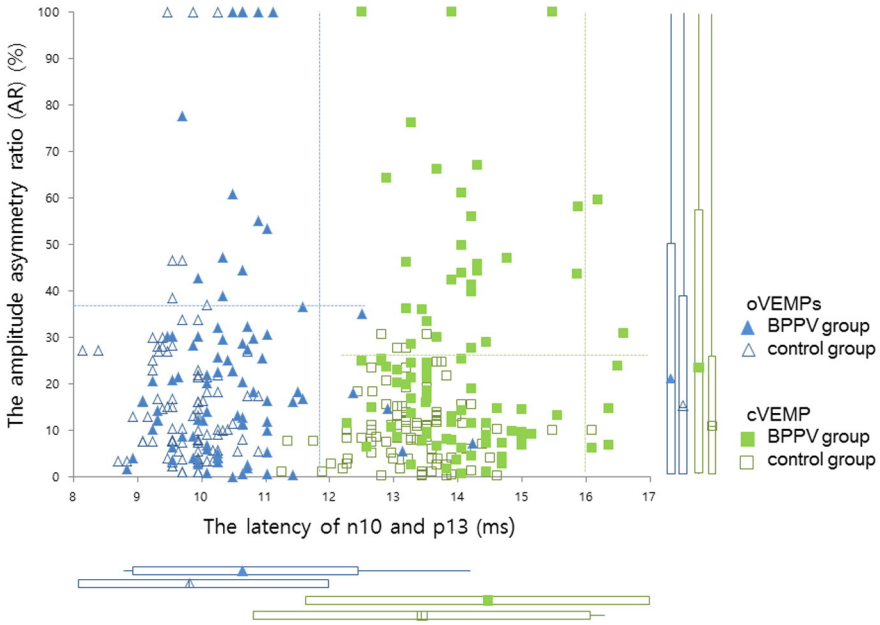

Fig. 2. The VEMP responses for the BPPV patients and normal control groups. The AR and peak latency of the n10 and p13 responses for the patients (filled shapes) and control (open shapes) groups were shown. The blue triangles indicate oVEMPs and the green squares cVEMPs. The means and 95% confidence intervals of AR and initial peak latency are shown to the right and below together with the box plots. The dotted lines signify the normal upper limit of the values of AR (horizontal lines) and latencies (vertical lines). Compared to the healthy controls, the BPPV patients showed longer latency and more asymmetric AR during the affected ear was stimulated ($p < 0.01$ in each VEMP by chi-square test).

utricular nerve stimulation also evoked inhibitory postsynaptic potentials in the ipsilateral SCM motor neurons [26].

Another finding of our study is that the non-affected ear in the BPPV group also showed significantly higher abnormalities of cVEMPs and oVEMPs when compared to the control group (Table 3) [18,27]. Furthermore, the abnormal VEMPs remained even two months after successful repositioning maneuvers in our BPPV patients, which indicate persistent otolithic dysfunction in those patients (Fig. 3). Although we could not check the test-retest reliability in the healthy control group, the normal data of the current study revealed no significant differences compared with the previous control data of our studies [14,20]. The VEMPs may recover after impairment in other types of peripheral vestibular disorders such as vestibular neuritis, such that dynamic function of both saccule and utricle may be regained in several days [28]. It is relatively novel findings that VEMPs are abnormal in the contralateral ear of a patient with unilateral BPPV and persistent abnormalities after successful treatment. Plausible explanation about these results is that a decrease in the gelatinous layer of the otolithic membrane with increasing age may allow spontaneous dislodgment of the otoconia from both sides of otolithic macula to occur more easily even in patients with

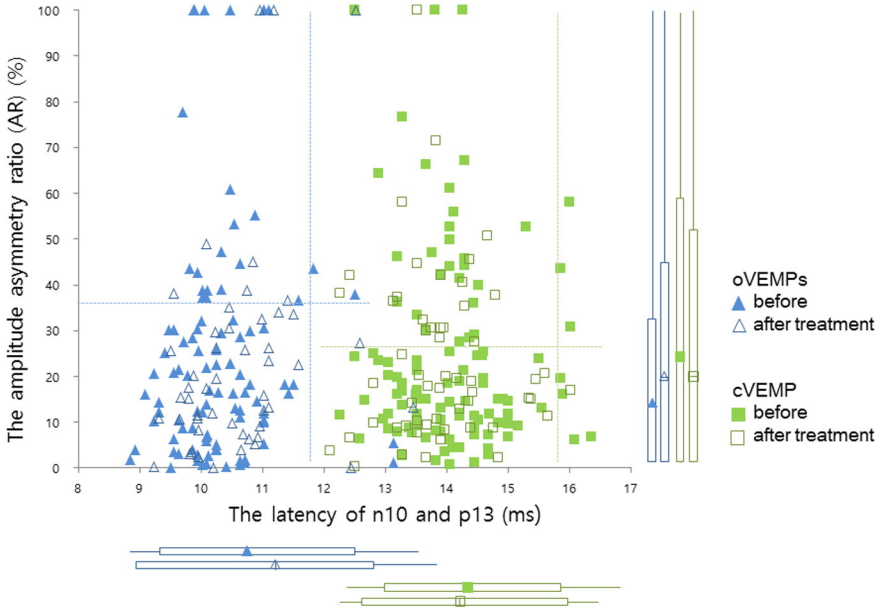

Fig. 3. The VEMP responses for the initial and follow-up tests after successful repositioning maneuvers. The AR and peak latency of the n10 and p13 responses for the initial (before-treatment, filled shapes) and follow-up VEMPs after successful repositioning maneuvers (after-treatment, open shapes). The blue triangles indicate oVEMPs and the green squares cVEMPs. The means and 95% confidence intervals of AR and initial peak latency are shown to the right and below together with the box plots. The dotted lines signify the normal upper limit of the values of AR (horizontal lines) and latencies (vertical lines). There were no differences in initial peak latency and AR of cVEMPs and oVEMPs in the affected ears between pre- and post-treatment repositioning maneuvers (paired *t*-test).

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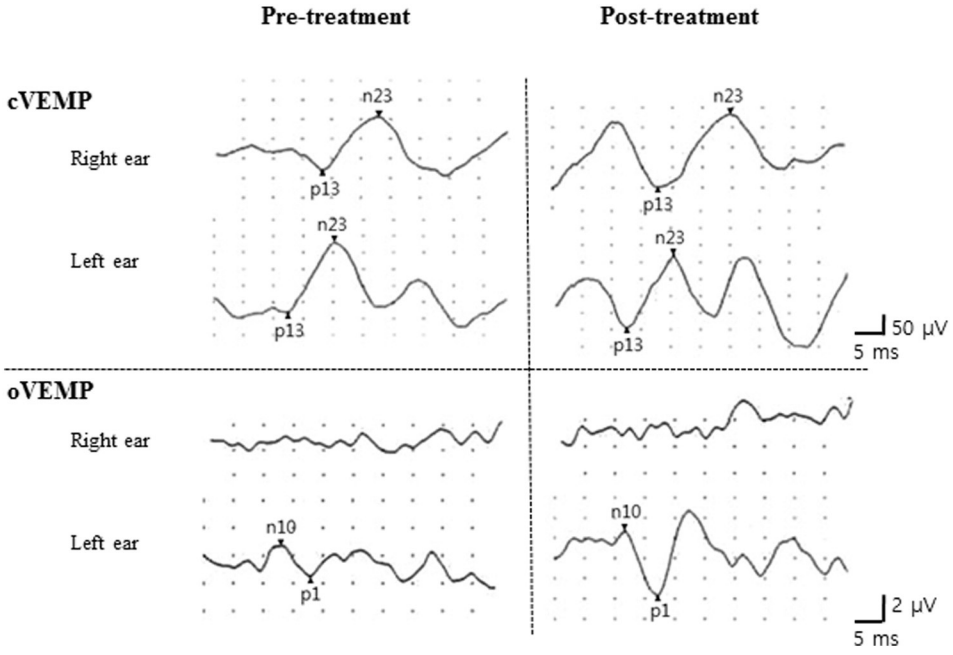

Fig. 4. VEMPs before and after repositioning maneuvers of an index patient with posterior canal type of BPPV on the right ear. This case shows a delayed p13 response on the ipsilateral SCM and an absent n10 response on the contralateral eye to affected ear stimulation, and these findings continued after repositioning maneuvers.

unilateral BPPV [8]. However, otolithic debris detached from the contralateral side is not sufficient to provoke BPPV symptoms on that side but enough to affect the VEMP studies. It has been known that osteopenia, osteoporosis, and vitamin D deficiency may contribute to generation of BPPV via deranged calcium metabolism in the vestibular organs [29]. We can also assume that vestibular degenerative changes with increasing age and abnormal calcium metabolism could contribute to develop bilateral otolithic dysfunction and then their abnormalities were persistent in unilateral symptomatic BPPV.

In conclusion, the overall prevalence of abnormal cVEMPs or oVEMPs in patients with BPPV was over 40% in both affected and non-affected ear, which was significantly higher compared to that observed in the control group. The VEMP abnormalities in BPPV patients were bilateral and persistent, i.e., the abnormalities involved both the affected and non-affected ears and did not recover even long after successful repositioning. Our results suggest that, even in unilateral BPPV patients, there is potential subclinical damage which take place in the ipsilateral or the contralateral ear of both utricle and saccule, and which can affect the VEMPs tests.

Conflict of interest

The authors report no conflicts of interest.

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