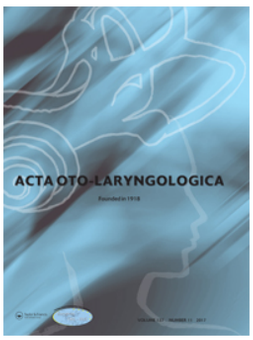

Acta Oto-Laryngologica

ISSN: 0001-6489 (Print) 1651-2251 (Online) Journal homepage: http://www.tandfonline.com/loi/ioto20

Residual dizziness after successful treatment of idiopathic benign paroxysmal positional vertigo originates from persistent utricular dysfunction

Toru Seo, Ko Shiraishi, Takaaki Kobayashi, Kitano Mutsukazu, Takeshi Fujita, Kazuya Saito, Hiroyasu Watanabe & Katsumi Doi

To cite this article: Toru Seo, Ko Shiraishi, Takaaki Kobayashi, Kitano Mutsukazu, Takeshi Fujita, Kazuya Saito, Hiroyasu Watanabe & Katsumi Doi (2017) Residual dizziness after successful treatment of idiopathic benign paroxysmal positional vertigo originates from persistent utricular dysfunction, Acta Oto-Laryngologica, 137:11, 1149-1152, DOI: 10.1080/00016489.2017.1347824

To link to this article: https://doi.org/10.1080/00016489.2017.1347824

ACTA OTO-LARYNGOLOGICA, 2017
VOL. 137, NO. 11, 1149–1152
https://doi.org/10.1080/00016489.2017.1347824

Taylor & Francis
Taylor & Francis Group

ARTICLE

Residual dizziness after successful treatment of idiopathic benign paroxysmal positional vertigo originates from persistent utricular dysfunction

Toru Seo[a], Ko Shiraishi[a], Takaaki Kobayashi[a], Kitano Mutsukazu[a], Takeshi Fujita[a], Kazuya Saito[a], Hiroyasu Watanabe[b] and Katsumi Doi[a]

[a]Department of Otolaryngology, Kindai University Faculty of Medicine, Osakasayama, Japan; [b]Department of Otolaryngology, Osaka Central Hospital, Osaka, Japan

ABSTRACT

Objective: We used ocular vestibular evoked myogenic potentials to investigate the relationship between residual dizziness and utricular function following the canalith repositioning procedure for benign paroxysmal positional vertigo.

Methods: Ocular vestibular evoked myogenic potentials were measured in 44 patients (40 included in analyses, four excluded) with successful results from the canalith repositioning procedure. The patients were examined before treatment and again one week after treatment. We analyzed how various general factors and ocular vestibular evoked myogenic potentials related to residual dizziness.

Results: Residual dizziness was not related to gender, affected side, age, duration of symptoms, recurrence, or the results of the initial ocular vestibular evoked myogenic potential test ($p > .05$). However, residual dizziness was significantly associated with the results of the second ocular vestibular evoked myogenic potential test ($p = .007$).

Conclusions: Residual dizziness after a successful canalith repositioning procedure may be caused by persistent utricular dysfunction.

ARTICLE HISTORY
Received 17 April 2017
Revised 2 June 2017
Accepted 12 June 2017

KEYWORDS
Residual dizziness; utricle; oVEMP; benign paroxysmal positional vertigo; canalith repositioning procedure

Introduction

Benign paroxysmal positional vertigo (BPPV) is one of the most common otological causes of vertigo. BPPV is characterized by rotatory vertigo and torsional nystagmus in response to specific head movements. There is a latency period of several seconds between head movements and the onset of symptoms, and both the vertigo and nystagmus typically lasts less than one minute [1]. The pathophysiological features of BPPV can be explained by free-floating debris within the evoked endolymphatic flow in the posterior semicircular canal (canalolithiasis theory). Histopathological study suggested that the debris originates from dislodged otoconia on the denatured utricular macula [2]. The canalith repositioning procedure (CRP) is based on the canalolithiasis theory and can provide immediate alleviation of positional vertigo and nystagmus [3,4]. Unfortunately, between 38 and 61% of the patients who undergo CRP with successful results still they experience persistent residual dizziness (RD) [5,6].

The origin of RD after successful CRP remains controversial. It occurs more frequently in patients with high scores on the Dizziness Handicap Inventory [5], with higher self-rated anxiety scores [7], and with orthostatic hypotension [8]. Thus, the cause appears to be associated with mental health and autonomic function. Alternatively, RD may be caused by utricular dysfunction that remains after CRP [9]. Studies of ocular vestibular-evoked myogenic potentials (oVEMPs) have revealed that patients with BPPV have abnormal utricular function [10,11], and this dysfunction remains even after a successful CRP [12]. Seo et al. reported that abnormal oVEMP results were observed in three out of the four cases with RD [11]. Sugita et al. studied the vestibulo-ocular reflex gain in off-vertical axis rotation, which reveals otolith function in patients with BPPV. The four cases in this study with dizziness showed significantly lower vestibulo-ocular reflex gain than those without dizziness [13]. Although these clinical studies only include a small number of cases, they suggest that the etiology of RD is persistent utricular dysfunction. The aim of this study was to clarify the relationship between RD and utricular dysfunction using oVEMP testing.

Subjects and methods

We enrolled 61 patients with idiopathic posterior canal BPPV who had undergone successful CRP at Department of Otolaryngology Osaka Central Hospital between July 2011 and June 2012. The inclusion criteria were the cases that did not complain of any otological or traumatic history and the cases that were examined with oVEMP testing both before and after CRP. Exclusion criteria were the cases that had concurrent with or conversion to BPPV of other canal.

CONTACT Toru Seo tseo@med.kindai.ac.jp Department of Otolaryngology, Kindai University Faculty of Medicine, 377-2 Ohno-higashi, Osakasayama 589-8511, Japan

For this purpose, we confirmed the absence of geotropic or apogeotropic nystagmus in supine roll test and atypical nystagmus in Dix-Hallpike test during the course. Based on the criteria, 44 patients were included in this study. The initial oVEMP test was performed several minutes after the Dix-Hallpike test at the first visit. Immediately after this examination, CRP was performed. The effects of the treatment were evaluated by the Dix-Hallpike test one week after CRP, and the second oVEMP test was performed immediately after confirming success of the treatment. When positional nystagmus and vertigo were not present, we defined the treatment as a success. The subjects were then divided into two groups depending on the results of the second Dix-Hallpike test: the non-RD group comprised patients who did not report any dizziness, and the RD group was made up of those who reported persistent and non-positional atypical dizziness. General characteristics and oVEMP results were compared between the two groups. Diagnoses of posterior canal BPPV had been made according to the diagnostic criteria of the American Academy of Otolaryngology-Head and Neck Surgery [1]. Briefly, it is as follows; (a) patient reports repeated episodes of vertigo with changes in head position, (b) they are also fulfilled each of the following criteria; vertigo associated with nystagmus is provoked by the Dix-Hallpike test, (c) there is a latency period between the completion of the Dix-Hallpike test and the onset of vertigo and nystagmus, and (d) the provoked vertigo and nystagmus increase and then resolve within a time period of 60 seconds from onset of nystagmus. CRP was performed as described in a previous report [4]. This retrospective study was approved by the Institutional Review Board.

oVEMP measurements

oVEMPs were measured according to a previously reported method using a Neuropack (Nihon Kohden, Tokyo, Japan) [11]. The active electrodes, the reference electrodes, and the ground electrode were placed just below the eyelid, 2 cm below the active electrodes, and on the forehead, respectively. Subjects lay in a supine position with the eyes in supraduction approximately 30° during recording. A tone-burst sound of 135 dB with a frequency of 700 Hz (rise/fall time, 1 ms; plateau time, 4 ms; repetition rate, 5 Hz) was delivered to the contralateral ear through a headphone. Evoked potentials were recorded and averaged 100 times with the bandpass filter from 5 Hz to 1 kHz.

The initial negative peak and positive peak, which occurred less than 20 ms after the stimulus, were defined as n1 and p1, respectively. Any n1-p1 peak-to-peak amplitude of less than 2 μV was regarded as artifact. As previously described, the peak-to-peak amplitude was evaluated using the impairment ratio of the affected side:

$$\text{Impairment ratio of affected side} = 100 \times (\text{AI} - \text{AA})/(\text{AI} + \text{AA})\ (\%) \quad [11]$$

Where, AI is the amplitude of the intact ear and AA is the amplitude of the affected ear [14]. We considered the oVEMP as reduced when impairment ratio was greater than 31.6% and augmented when impairment ratio was less than −31.6% [11]. Additionally, when the peak latency of n1 and p1 waves were extended beyond the normal range of our institute (8.5–12.2 ms and 14.3–18.6 ms, respectively), we considered the oVEMP as abnormal [11].

Statistical analysis

The Mann–Whitney U test was used to compare the quantitative variables between the two groups, while Fisher's exact test was used to compare the nominal variables. Data were analyzed using EZR 1.33, which is based on the open-source statistical software R [14]. A cutoff of $p < .05$ was used to determine statistical significance. Any cases with no n1-p1 waves in the bilateral ear were excluded from the statistical analysis.

Results

Table 1 shows the general characteristics of the subject group. There were no significant differences between the non-RD group and the RD group with regard to gender, affected side, age, duration of symptoms, or recurrence. For the initial oVEMP test, four of the subjects showed no response in the bilateral ear and were excluded from statistical analysis. The other 40 cases indicated detectable wave at least one ear (Figure 1). Normal results, augmented response, and reduced response were seen in 12, 8, and 10 cases in the non-RD group and in 2, 2, and 6 cases in the RD group, respectively (Figure 2). There were no significant

Table 1. General characteristics of the subject group.

	Non-RD	RD	*p* value
Gender (male: female)	16:18	2:8	.161
Affected side (right: left)	20:14	6:4	1
Age (mean ± SD)	48.8 ± 9.6	46.1 ± 9.3	.424
Days from onset (mean ± SD)	17.6 ± 18.1	11.1 ± 13.6	.139
Recurrence (yes: no)	14:20	3:7	.716

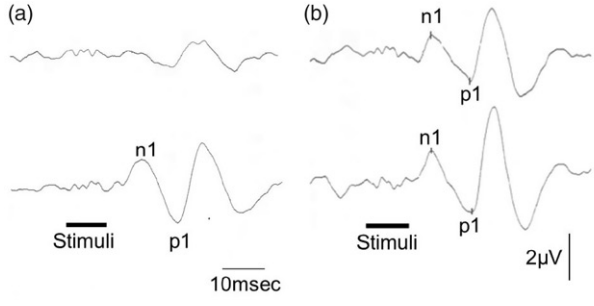

Figure 1. Results of oVEMP before and after treatment in a 51-year-old male patient with BPPV in the left ear. (a) The n1-p1 peak-to-peak amplitude of the initial oVEMP was 4.6 μV on the right ear; however, this was not detected on the left ear (AR:100%). These results show the utricular dysfunction on the left ear. (b) One week after the CRP, no nystagmus or vertigo was observed following the Dix-Hallpike test; however, the patient reported experiencing RD. The amplitudes of the second oVEMPs were 4.7 μV and 3.9 μV for the right ear and the left ear, respectively. AR was 9% (normal). Utricular dysfunction had recovered to normal on the left ear. Upper wave indicates measurement by left ear stimulation and lower wave indicates measurement by right ear stimulation.

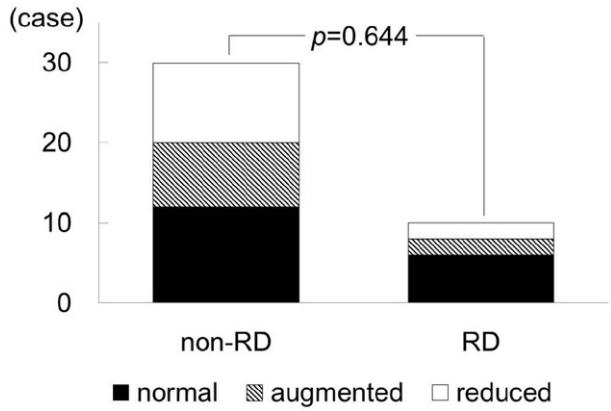

Figure 2. Results of the initial oVEMP tests that were administered prior to the treatment.

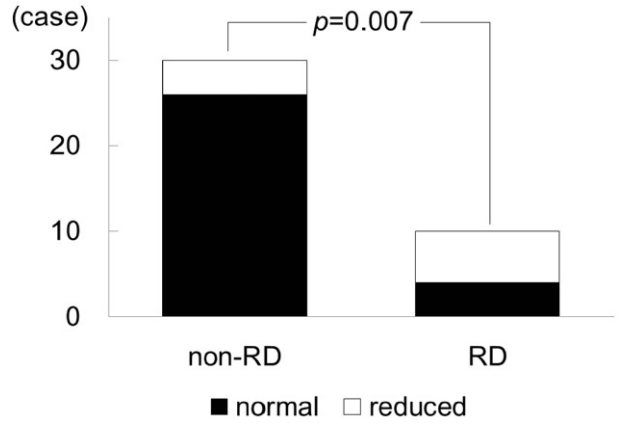

Figure 3. Results of the second oVEMP tests that were administered 1 week after the treatment.

differences between the two groups ($p = .644$). There were no cases of abnormal latency of n1 or p1 waves from the initial oVEMP test results.

For the non-RD group, all eight cases with augmented oVEMP on the initial test showed normal results on the second test. Among the 10 cases with reduced oVEMP on the initial test, five cases recovered to normal results, four cases still showed a reduced response, and one case showed no response in the bilateral ear on the second oVEMP test. There were two cases of augmented oVEMP in the RD group following the initial test. Of these, one case recovered to normal and one case still showed a reduced response after the second test. Among the six cases of reduced oVEMP on the initial test in the RD group, one case recovered to normal results following the second oVEMP test, and five cases still showed a reduced response. In total, four of the 26 cases in the non-RD group showed reduced oVEMP on the second test, and six of the 10 cases in the RD group showed reduced oVEMP on the second test (Figure 3). This represents a significant difference between the two groups ($p = .007$). There were no cases of abnormal latency of n1 or p1 waves in the second oVEMP test.

An additional third oVEMP test was administered in five of the six RD patients for whom symptoms resolved and who had reduced results on the second oVEMP test. This test showed that four out of these five patients showed normal oVEMPs, whereas one still showed a reduced response.

Discussion

Our result demonstrate that the occurrence of RD cannot be predicted by patient background or by the results of an initial oVEMP test. However, we report that RD is related to the results of a second oVEMP test performed one week after treatment. This suggests that the origin of RD is persistent utricular dysfunction, even after a successful CRP. It has been previously shown that the peak-to-peak amplitude of the oVEMP n1-p1 wave in the affected side is larger than that in the unaffected side in certain BPPV cases [11,12]. The cause of augmented response in BPPV was unknown. We could not deny the possibility of false diagnose as augmented in the cases with normal response in the affected ear of BPPV and reduced response in the non-affected ear. However, Shojaku et al. reported that the augmented response in VEMP was recorded during parabolic flight. When otoconia were detached from the otolith membrane under to the microgravity condition, the altered mobility of stereocillia might result the augmented response [15]. Seo et al. suggested that such an augmented result indicates intact hair cells with detached otoconia [11]. Augmented oVEMP was seen in 10 cases in the initial test but was not observed in any cases following the second oVEMP test; thus, augmented oVEMPs were seen only in the early phase of BPPV. Normal results were observed in 18 cases following the initial oVEMP test and in 32 cases following the second oVEMP test. Four out of the five patients in the RD group with a reduced oVEMP following the second test showed normal results in a third oVEMP test. Thus, otolith function appears to recover with time [13]. Our results suggest that delayed recovery of utricular hair cell function may cause RD.

Abnormal results from the second oVEMP test were observed in the absence of symptoms in four patients. Additionally, one case did not report any symptoms but still showed abnormal results in the third oVEMP test. Thus, symptoms were observed to improve earlier than oVEMP. We speculate that this observation is due to central compensation of utricular dysfunction. Compensation for otolith dysfunction can occur in a manner similar to that of semicircular canal dysfunction [16].

In contrast, two patients in the RD group showed normal results on the second oVEMP test. What pathogenesis did cause the symptoms? There was not history of spontaneous vertigo attack, thus the existence of canal dysfunction is unlikely. On the other hand, it was known that some cases of BPPV showed saccular dysfunction, however it was less common than utricular dysfunction [10]. Thus, the existence of saccular dysfunction without utricular dysfunction is unlikely. Previous study indicated that patients with RD often have psychological abnormalities, autonomic nervous system abnormalities [5,7,8]. Although any psychological examinations were performed in this study, the pathogenesis of RD with normal results of second oVEMP may be mental stress rather than vestibular deficit. Brand

suggested the existence of phobic postural vertigo, which involves dizziness and subjective disturbance of balance in patients with obsessive-compulsive type personalities, labile affects, or mild depression following emotional stresses, serious illnesses, or organic vestibular disorders [17]. It has been suggested that patients with the above-mentioned mental stresses develop phobic postural vertigo after the onset of BPPV even if the vertigo was cured with CRP. Our results indicate that the pathology of most patients with RD is residual utricular dysfunction, but that some patients suffer from phobic postural vertigo. For the cases of utricular dysfunction, physical therapy and/or rehabilitation to induce central compensation was required. For the cases of phobic postural vertigo, psychoactive drugs and/or psychotherapy may be required [17]. Our results demonstrate the ability of oVEMP to discriminate between these two causes of RD.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

This study was partly supported by a Grant-in-Aid for Research on Intractable Vestibular Disorder from the Japan Agency of Medical Research and Development.

References

[1] Bhattachayya N, Baugh RF, Orvidas L, et al. Clinical practice guideline: benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg. 2008;139:S47–S81.

[2] Schuknecht HF. Cupulolithiasis. Arch Otolaryngol. 1969;90:113–126.

[3] Epley JM. The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg. 1992;107:399–404.

[4] Seo T, Miyamoto A, Saka N, et al. Immediate efficacy of the canalith repositioning procedure for the treatment of benign paroxysmal positional vertigo. Otol Neurotol. 2007;28:917–919.

[5] Martellucci S, Pagliuca G, de Vincentiis M, et al. Features of residual dizziness after canalith repositioning procedures for benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg. 2016;154:693–701.

[6] Seok JI, Lee HM, Yoo JH, et al. Residual dizziness after successful repositioning treatment in patients with benign paroxysmal posional vertigo. J Clin Neurol. 2008;4:107–110.

[7] Faralli M, Ricci G, Ibba MC, et al. Dizziness in patients with recent episodes of benign paroxysmal positional vertigo: real otolithic dysfunction or mental stress? J Otolaryngol Head Neck Surg. 2009;38:375–380.

[8] Kim H-A, Lee H. Autonomic dysfunction as a possible cause of residual dizziness after successful treatment in benign paroxysmal positional vertigo. Clin Neurophysiol. 2014;125:609–614.

[9] Di Girolamo S, Paludetti G, Briglia G, et al. Postural control in benign paroxysmal positional vertigo before and after recovery. Acta Otolaryngol. 1998;118:289–293.

[10] Nakahara H, Yoshimura E, Tsuda Y, et al. Damaged utricular function clarified by oVEMP in patients with benign paroxysmal positional vertigo. Acta Otolaryngol. 2013;133:144–149.

[11] Seo T, Saka N, Ohta S, et al. Detection of utricular dysfunction using ocular vestibular evoked myogenic potential in patients with benign paroxysmal positional vertigo. Neurosci Lett. 2013;550:12–16.

[12] Kim E-J, Oh S-Y, Kim JS, et al. Persistent otolith dysfunction even after successful repositioning in benign paroxysmal positional vertigo. J Neurological Sci. 2015;558:284–293.

[13] Sugita-Kitajima A, Koizuka I. Recovery of otolith function in patients with benign paroxysmal positional vertigo evaluated by sinusoidal off-vertical axis rotation. Neurosci Lett. 2008;436:124–127.

[14] Kanda Y. Investigation of the freely available easy-to-use software 'EZR' for medical statistics. Bone Marrow Transplant. 2013;48:452–458.

[15] Shojaku H, Watanabe Y, Tsubota M, et al. Evaluation of the vestibular evoked myogenic potential during parabolic flight in humans. Exp Brain Res. 2008;187:477–481.

[16] Takeda N, Igarashi M, Koizuka I, et al. Recovery of the otolith-ocular reflex after unilateral deafferentation of the otolith organs in squirrel monkeys. Acta Otolaryngol. 1990;110:25–30.

[17] Brandt T. Vertigo: its multisensory syndromes. 2nd ed. London (UK): Springer; 2003. Phobic postural vertigo. p. 469–479.