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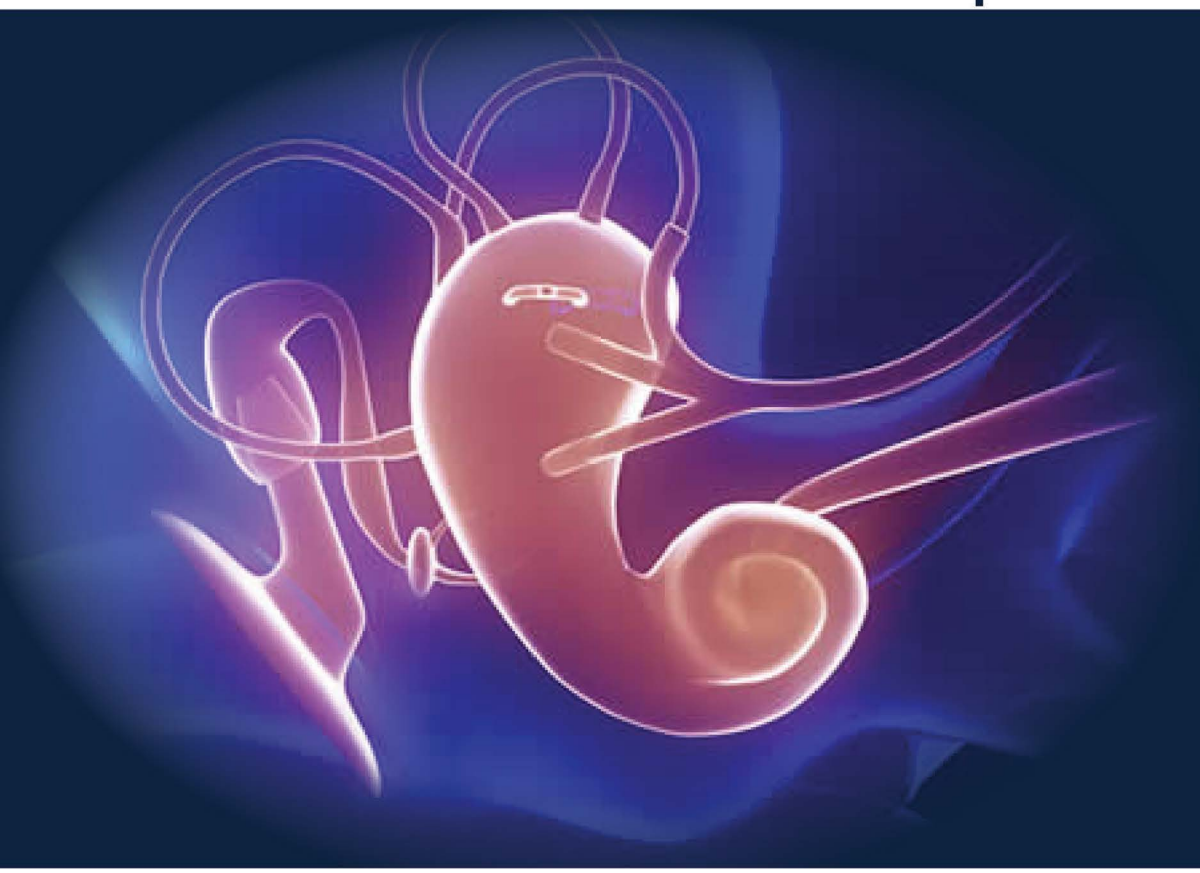
Understanding Benign Paroxysmal Positional VERTIGO

Editors

Alessandro De Stefano • Francesco Dispenza



Foreword

Adolfo M Bronstein

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Chapter

2

Pathophysiology of Benign Paroxysmal Positional Vertigo

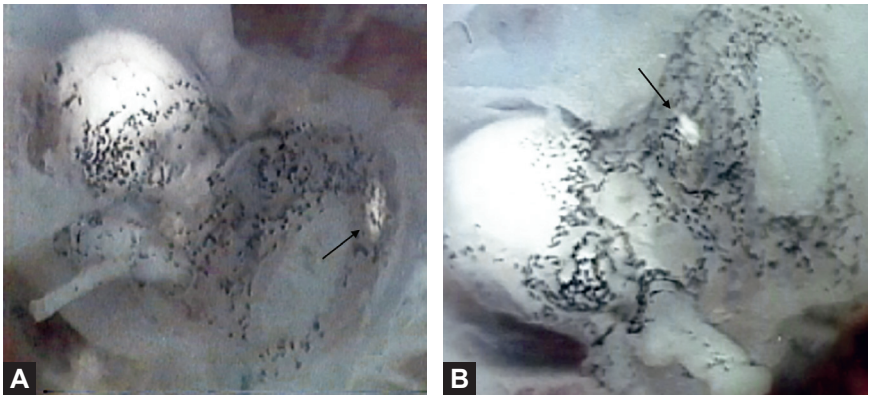
Koji Otsuka, Mamoru Suzuki

PHYSIOLOGICAL EFFECTS OF CUPULOLITHIASIS AND CANALOLITHIASIS IN IN VIVO MODELS

Benign paroxysmal positional vertigo (BPPV) has been classified into two major types based on the nature of nystagmus induced. The most common type is posterior canal canalolithiasis.[1] Nystagmus induced by posterior canal canalolithiasis is characterized by its short duration and latency. Lateral canal BPPV has been further classified into two types according to the direction of nystagmus induced. The underlying mechanism of geotropic nystagmus is thought to be canalolithiasis of the lateral canal while that of apogeotropic nystagmus is considered to be cupulolithiasis of the lateral canal.

We previously used the bullfrog labyrinth in a BPPV model study.[2] The whole membranous labyrinth was used to replicate the human vestibule. The otoconia were dislodged from the utricle by the application of mechanical vibrations to the otic capsule and were driven into the posterior canal (PC) (canalolithiasis model) (Fig. 2.1A). The cupulolithiasis model was created by placing the otoconial mass on the cupula (Fig. 2.1B). The compound action potential (CAP) of the PC nerve was recorded after placing the PC in various positions. The PC was initially placed with the cupula-to-crista axis in the horizontal plane with the utricular side upward. This was designated as the canal-down position. When the PC was placed upside down (i.e. with the canal side up), it was designated as the canal-up position.

In the canalolithiasis model, the canal-down position induced ampullofugal movement of the otoconia, resulting in an excitatory response, while the canal-up position resulted in an inhibitory response (Fig. 2.2). In the excitatory response, the average and standard deviation of the decremental time constant was 6.8 ± 1.0 seconds and the average rise time was 3.5 ± 0.4 seconds. When the otoconia moved inside the canal, neural discharges changed based on the direction of the otoconial movement. A latent period was observed before the discharge change emerged. The otoconia slowly started to move and accelerated with a positional change.



Figs. 2.1A and B: (A) An otoconial mass was detected within the posterior canal. (B) An otoconial mass was on the cupula surface.

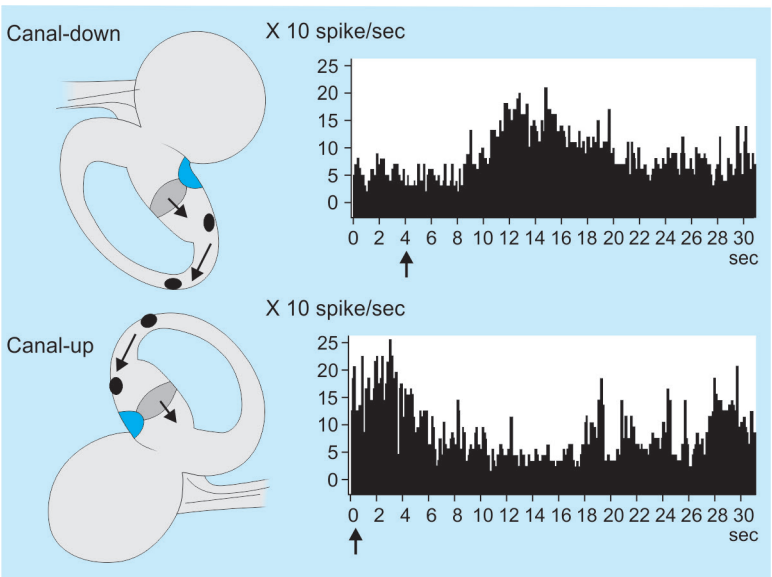


Fig. 2.2: Spike density histograms of compound action potentials in posterior semicircular canal canalolithiasis. The canal-down position induced excitatory discharges, whereas the canal-up position inhibited excitatory discharges. The arrows indicate the onset of positional changes.

The friction force between the otoconia and canal wall was responsible for this slow start of otoconial movement and latency.

On the other hand, in the cupulolithiasis model, the canal-down position induced a depression of the cupula toward the ampullofugal direction, resulting in an excitatory response, while the canal-up position similarly induced depression, resulting in an inhibitory response

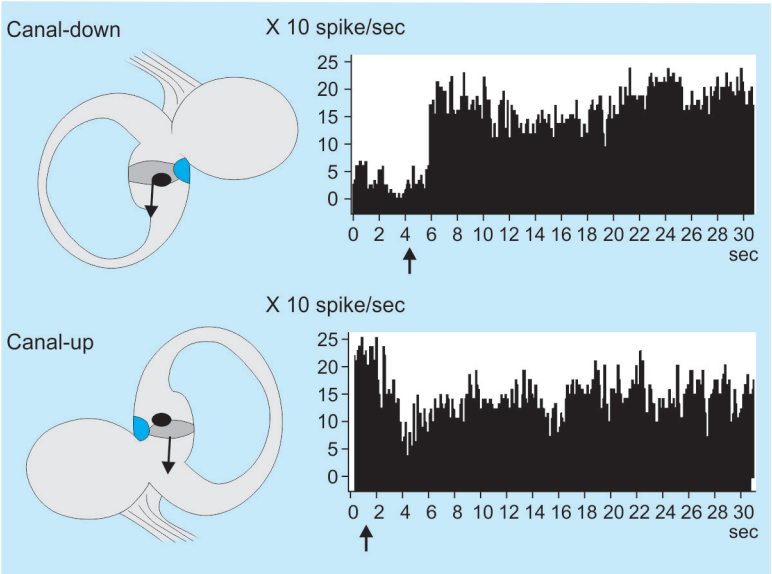


Fig. 2.3: Spike density histograms of compound action potentials in posterior semicircular canal cupulolithiasis. The canal-down position induced sustained excitatory discharges, whereas the canal-up position inhibited excitatory discharges. The arrows indicate the onset of positional changes.

(Fig. 2.3). The average time constant was >43 seconds, which was longer than that of canalolithiasis, and the average rise time was 1.8 ± 0.2 seconds, which was shorter than that of canalolithiasis.

These experiments demonstrated that both cupulolithiasis and canalolithiasis effectively stimulated the cupula and, thus, are potential mechanisms responsible for BPPV. The magnitude of CAP was previously shown to be affected by several physical factors, such as friction between the canal wall and settled otoconia as well as the weight, volume, and acceleration of the moving otoconia.[3] The findings of these model experiments are consistent with the clinical pictures of BPPV. In canalolithiasis, nystagmus has a long latency and short duration, but has a short latency and long duration in cupulolithiasis.

PHYSIOLOGICAL EFFECTS OF OTOCONIA BY MATHEMATICAL MODELS

A mathematical model has been used to analyze the mechanisms responsible for canalolithiasis and cupulolithiasis. House and Honrubia[4] investigated the biophysical fundamental properties of canalolithiasis and cupulolithiasis and reported that the pressure induced by particles moving within the semicircular canal (SC) endolymph was 0.035 dyn/cm^2. Moreover, if an individual particle had a diameter of ~10 μm, the

number of particles required to develop BPPV in the cupulolithiasis model was predicted to be 490, while that required to develop BPPV in the canalolithiasis model was predicted to be 62. However, Squires et al.[5] reported that ~20 otoconia, or 0.1 μg of otoconia, was required to account for the emergence of peak nystagmus with BPPV. A mathematical model of the SCs indicated that endolymphatic pressure changed in the anterior and posterior canals when otoconia moved within the common crus in which both canals joined. The nystagmus induced may become the sum of both canals, resulting in a purely torsional effect.

PHYSIOLOGICAL EFFECTS OF CANALOLITHIASIS AND CUPULOLITHIASIS ON SC ACTIVITY

The effects of BPPV on SC function need to be determined in more detail. In BPPV, the incidence of canal paresis (CP) has been reported to be 20–50%. Canal paresis is assumed to be caused by the direct effects of the otoconia, either on the cupula or in the canal in some BPPV cases. Measurements of SC function in BPPV may enable the size of the otoconia to be evaluated and the side affected by BPPV to be identified. We previously attempted to investigate the effects of the otoconia on SC function in model experiments of BPPV.[6]

Caloric Test

Some BPPV patients have a reduced caloric response. Baloh et al.[7] reported the clinical and oculographic features of 240 cases of posterior canal BPPV (PC-BPPV). A total of 94 patients (39.2%) had CP. In 71 out of the 94 patients with CP, paresis was on the side affected by BPPV. Pagnini et al.[8] showed that 5 out of 11 patients (45.5%) with lateral canal BPPV (LC-BPPV) had caloric CP, all of which occurred on the side affected by BPPV. Baloh et al.[9] also demonstrated that 4 out of 13 patients (30.8%) with LC-BPPV had CP on the side affected by BPPV. Korres et al.[10] reported the electronystagmographic (ENG) findings of 168 BPPV patients and found that 22.1% with PC-BPPV and 21.4% with LC-BPPV had CP.

According to a histological study,[11] loose otoconia were commonly found in the lumens of all SCs. Although these loose otoconia were more commonly detected in the PC, they are also found in the LC and anterior semicircular canal (AC). The presence of the otoconia is typically asymptomatic, and this may be because a small quantity of the otoconia cannot stimulate the SC. Therefore, the presence of the otoconia in the endolymph of the LC may exert a gravitational load on the fluid and, thus, affect caloric responses in a significant number of patients with PC-BPPV.

Caloric responses in BPPV depend on the BPPV condition. Katsarkas[12] divided PC-BPPV patients into two groups according to their ENG

findings; the first group was examined during the active phase of BPPV and the second group during the inactive phase. Caloric test results were frequently found to be abnormal in the first group, but within normal limits in most of the second group. Strupp et al.[13] described a patient with active LC-BPPV canalolithiasis in whom caloric weakness resolved within a few days following particle-repositioning therapy. This reversible caloric CP was attributed to functional plugging of the LC, which eliminated convective flow in the endolymph.

Rotation Test

Rotation Test for PC-BPPV

Iida et al.[14] performed a pendular rotation test with an oscillation of 360° at a frequency of 0.1 Hz in a head-tilted position (60° backward and rotated 45° to either the right or left) in PC-BPPV patients. The vestibulo-ocular reflex (VOR) response of the PC in the affected ear was smaller than that of the AC. However, the VOR improved once vertigo and nystagmus disappeared. Sekine et al.[15a] reported that VOR gains in the PC did not change in PC-BPPV canalolithiasis at any frequencies from 0.1 to 1.0 Hz, and suggested that the volume of free-floating otoconial debris associated with canalolithiasis was too small relative to the endolymph volume to change the SC dynamics.

Rotation Test for LC-BPPV

Sekine et al.[15b] performed a rotation test on LC-BPPV patients, who were rotated around the vertical axis sinusoidally at frequencies of 0.1–1.0 Hz. In canalolithiasis, no significant difference was observed in VOR gain between rotation to the affected and unaffected sides at any frequencies. Although no significant difference was noted in the VOR gain at frequencies of 0.3–1.0 Hz in cupulolithiasis, the VOR gain at 0.1 Hz was significantly smaller during rotation to the affected side than to the unaffected side. This finding suggested that the temporary impairment of LC function was induced by the mechanical restriction of cupular movements.

Head-Shaking Test

Passive high-frequency rotation of the head is commonly performed in the horizontal and sagittal planes. Lopez-Escamez et al.[16] reported the dynamics of canal responses to the horizontal and vertical head-shaking test in BPPV. Head-shaking nystagmus (HSN) was considered to be present when six consecutive beats of nystagmus with a slow-phase velocity (SPV) of at least 2° per second were detected. The main

parameters were the presence of horizontal and vertical HSN, maximum SPV, and time constant of HSN. The maximum SPV of vertical HSN was significantly greater in PC-BPPV patients than in controls. Moreover, the time constant of vertical HSN was smaller for BPPV patients than for controls. These findings suggested that vestibular lithiasis had a limited contribution to the mechanism underlying the generation of HSN.

Head Impulse Test

The head impulse test(HIT) was first introduced by Halmagyi and Curthoys[17] as a clinical test of the VOR to detect lateral semicircular CP. Heidenreich et al.[18] described a patient with active LC-BPPV canalolithiasis who demonstrated robust refixation saccades on HIT, and these were most likely due to rapid mobilization of the otoconial debris. The treatment of LC-BPPV canalolithiasis immediately resolved his vertigo and the HIT results normalized in follow-up testing.

Our Experiments on SC Function in BPPV

We prepared models of canalolithiasis and cupulolithiasis using the isolated PCs of bullfrogs.[6] A glass dish filled with Ringer solution was magnetically fixed on a turntable. The specimen was placed 3 cm from the rotation center with the ampulla toward the inside and the nerve toward the outside. The crista-cupula top axis was on the line facing the rotation center. We first recorded the CAPs of normal specimens and then recorded the CAPs of the canalolithiasis and cupulolithiasis models. The turn table was sinusoidally rotated by a computer-controlled motor. Two rotation cycles, 0.1 and 0.2 Hz, were applied. The rotation cycle was initially set at 0.1 Hz, the rotation angle at 270°, and the maximum angular velocity at 84° per second (Fig. 2.4). The rotation cycle was then set at 0.2 Hz, the rotation angle at 180°, and the maximum angular velocity at 112° per second. The differences between the maximum and minimum spikes in three consecutive recordings were averaged. We designated the spike difference between the spontaneous spikes and maximum spikes as the response intensity (RI). The RIs of canalolithiasis and cupulolithiasis were expressed as a percentage, with 100% representing normal RI. We classified our experiments according to the size of the otoconial mass. An otoconial mass with a span longer than a half of the cupular height (*large otoconia*) was used. In the 0.1 Hz rotation, the RI was 54.6% in canalolithiasis and 17.7% in cupulolithiasis. Cupulolithiasis showed a larger decrease in RI than canalolithiasis. In the 0.2 Hz rotation, the RI was 72.5% in canalolithiasis and 61.8% in cupulolithiasis. The reduction in RI at 0.2 Hz was less than that in RI at 0.1 Hz in cupulolithiasis (Fig. 2.5). An otoconial mass with a span shorter than half of the cupular height

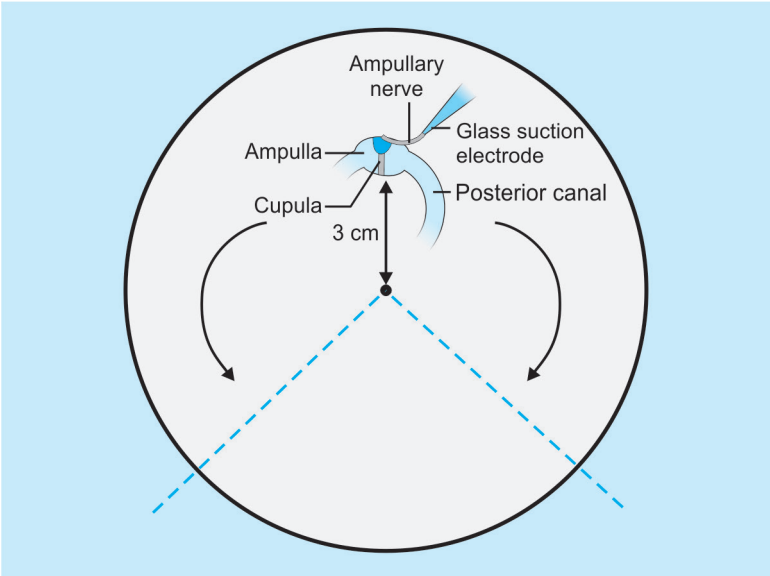


Fig. 2.4: Experimental setting on semicircular canal function in benign paroxysmal positional vertigo. The turn table was sinusoidally rotated by a computer-controlled motor. Two rotation cycles, 0.1 and 0.2 Hz, were applied.

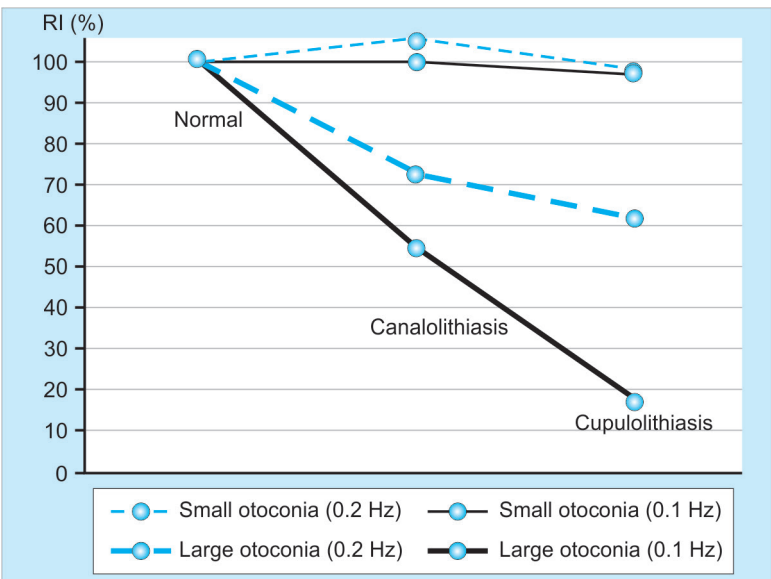


Fig. 2.5: Response intensities (RIs) in canalolithiasis and cupulolithiasis. In the case of small otoconia, the RI was not decreased in either canalolithiasis or cupulolithiasis, or at either 0.1 or 0.2 Hz. In the case of large otoconia, the RI was decreased in both canalolithiasis and cupulolithiasis models. Cupulolithiasis showed a larger decrease in RI than canalolithiasis. The RI at 0.2 Hz did not decrease as much as at 0.1 Hz in cupulolithiasis.

(*small otoconia*) was used. The RI was not decreased in canalolithiasis or cupulolithiasis at either 0.1 or 0.2 Hz (*see* Fig. 2.5).

Large Otoconia versus Small Otoconia

The CAPs markedly decreased and even disappeared in some cases of canalolithiasis involving large otoconia. The otoconia may have formed an embolus, thereby blocking endolymphatic flow. The possibility of otoconial debris forming an embolus was termed a "canalith jam" by Epley.[19] However, CAPs did not change in cases of canalolithiasis involving small otoconia. Some cases of BPPV had a decreased caloric response and VOR gain, whereas others did not. We assumed that this varied according to the size of the otoconial mass.

Canalolithiasis versus Cupulolithiasis

The RI of cupulolithiasis showed a larger decrease than that of canalolithiasis. The direct inhibition of the cupular shift was greater than the inhibition of endolymphatic flow. This suppression of CAPs may have been due to the otoconial weight, which can occur by simply blocking cupular displacement. This finding supported clinical results in which VOR gain was suppressed more in cupulolithiasis than in canalolithiasis.[15b] Our experiments suggested that reduced vestibular responses did not necessarily indicate sensory cell function damage, but rather the inhibition of cupular dynamics. This was supported by the clinical observation in which BPPV vertigo rapidly resolved with adequate physical therapy. The imbalance of cupular mobility between normal and cupulolithiasis ears may be another cause of the dizziness reported in BPPV patients.

0.1 Hz versus 0.2 Hz

The reduction in RI at 0.2 Hz was less than that in RI at 0.1 Hz in cupulolithiasis. A cupula with a large otoconial mass could be moved if the frequency was sufficiently high. This finding supported the clinical result in which VOR gain improved as angular acceleration increased in LC- BPPV.[15b]

FUNCTIONAL EVALUATION OF THE OTOLITHIC ORGAN IN BPPV PATIENTS

Utricular dysfunction has been reported in patients with BPPV. Markham et al.[20] used ocular counter-rolling as a test to reflect the utriculo-ocular reflex. In their study, abnormal ocular counter-rolling was detected in 88.9% of BPPV patients. Sugita et al.[21] examined otolith function in 23

controls and 24 BPPV patients by comparing the VOR gain of off-vertical axis rotation (OVAR) with that of earth-vertical axis rotation (EVAR). Their findings showed that VOR gain during OVAR at 0.8 Hz in BPPV patients was significantly less than that during EVAR, whereas no significant differences were observed in VOR gain between EVAR and OVAR in the controls. They analyzed their findings and suggested the existence of otolith organ dysfunction in BPPV patients.

Vestibular-evoked myogenic potential (VEMP) in cervical muscles (cVEMP) has been widely used as a clinical test of the vesibulocollic reflex, predominantly the sacculocollic reflex. Myogenic potentials around the eyes [ocular VEMP (oVEMP)] have also recently been recorded. Ocular VEMP predominantly reflects utricular functions. Nakahara et al.[22] measured oVEMP and cVEMP in 12 BPPV patients and 12 controls. More BPPV patients exhibited abnormal responses in oVEMP by stimulation on their affected side than the controls, whereas no significant differences were observed in cVEMP between BPPV patients and controls. These findings indicated that oVEMP reflected a specific abnormal condition in BPPV, i.e. utricular function was highly damaged in BPPV patients.

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