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Clinical Significance of Vestibular Evoked Myogenic Potentials in Benign Paroxysmal Positional Vertigo

*Won Sun Yang, †Sung Huhn Kim, ‡Jong Dae Lee, and *Won-Sang Lee

**Department of Otorhinolaryngology-Head and Neck Surgery, Yonsei University College of Medicine, Seoul, Korea; †Cellular Biophysics Laboratory Department of Anatomy and Physiology, Kansas State University, Manhattan, Kansas, U.S.A.; and ‡Department of Otorhinolaryngology-Head and Neck Surgery, Soonchunhyang University College of Medicine, Bucheon, Korea*

Objective: To investigate the vestibular evoked myogenic potentials (VEMPs) resulting in benign paroxysmal positional vertigo (BPPV) patients and to verify its clinical applications in BPPV.
Study Design: A prospective study.
Setting: Tertiary referral dizziness center.
Patients: Forty-one patients with diagnosis of BPPV and 92 healthy volunteers who underwent VEMP testing.
Intervention: Patients were treated by canalith repositioning maneuvers according to the affected canal, and testing of VEMP was performed at diagnosis and after treatment.
Main Outcome Measures: Testing of VEMP was performed in BPPV patients and in the control group. The number of times the canalith repositioning maneuver was repeated until the patient's report of relief from vertigo and findings of negative positioning test were recorded to find out the relationship between VEMP results and the progress of disease.
Results: Vestibular evoked myogenic potential results of BPPV patients showed prolonged p13 and n23 latencies compared with those of the control group, and we could not find any significant difference in VEMP latencies between patients with posterior and horizontal canal type of BPPV. The number of times that the maneuver was repeated did not correlate with the degree of latency prolongation, but in the "no response" group, the number of times was considerably greater than that in the "response" group.
Conclusion: We found that VEMP latencies are increased in BPPV patients, which may signify neuronal degenerative changes in the macula of the saccule. When an extensive neuronal damage was suspected by VEMP results such as "no response" in VEMP, the disease progress showed a chronic and resistive course. Therefore, we propose that VEMP could be a useful method to determine a clinical prognosis of patients with BPPV. **Key Words:** Benign paroxysmal positional vertigo—Canalith repositioning maneuver—Vestibular evoked myogenic potentials.

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Vestibular evoked myogenic potentials (VEMPs) are electromyographic (EMG) responses to loud auditory stimuli that can be recorded in the sternocleidomastoid (SCM) muscle during tonic contraction. Geisler et al. and Bickford et al. (1) were the first to record these EMG potentials, and Murofushi et al. (2) proved that VEMP are produced by a reflex arc that involves the vestibular saccule, the inferior vestibular nerve, and the SCM muscle.

Benign paroxysmal positional vertigo (BPPV) is the most common disorder of the peripheral vestibular system characterized by episodes of vertigo associated with head movements. In explaining the pathophysiology of BPPV, the concept of a degenerative process that affects the macula of the utricle causing detachment of otoliths has gained popular support (3). However, Akkuzu et al. (4). suggested that this degenerative process might also affect the macula of the saccule. In addition, Gacek (5) reported ganglion cell degeneration of the saccular nerve in a postmortem examination of BPPV patients. We suppose that the saccular degenerative changes can be detected by testing of VEMP, and this type of testing could be valuable for assessing BPPV patients. The aim of this study was to investigate the VEMP results in BPPV patients and to verify its clinical applications.

Address correspondence and reprint requests to Won-Sang Lee, M.D., Ph.D., Department of Otorhinolaryngology-Head and Neck Surgery, Yonsei University College of Medicine, 134 Shinchon-dong, Seodaemun-gu, Seoul, Korea 120-752; E-mail: wsleemd@yumc.yonsei.ac.kr
W. S. Yang and S. H. Kim equally contributed to this work.

MATERIALS AND METHODS

We examined 41 patients diagnosed with BPPV who were primarily referred to our dizziness clinic with complaints of balance problems from September 2005 to December 2006.

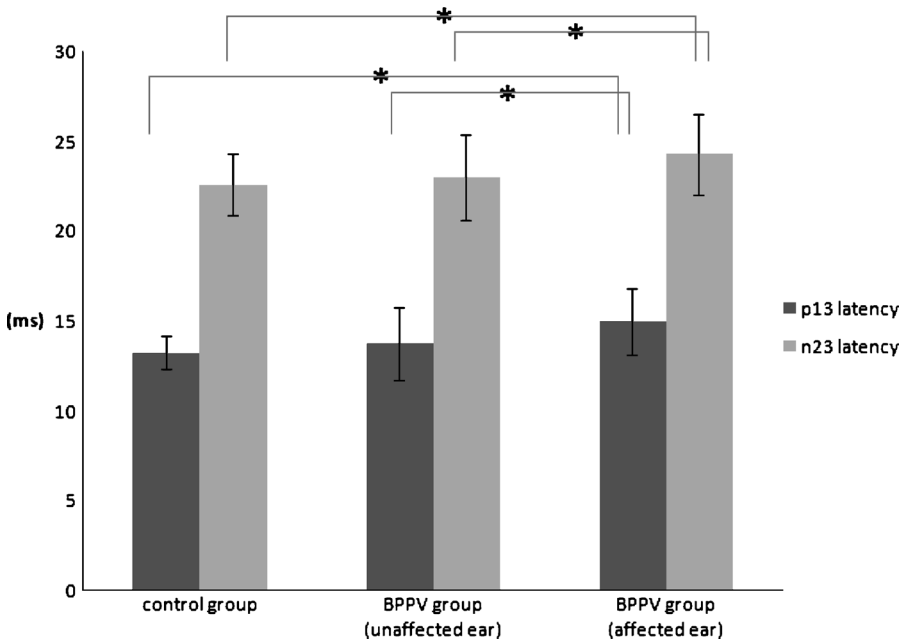

FIG. 1. This chart shows the result of the statistical analysis of VEMP findings in each group. The diagram shows mean ± standard deviation. Asterisk (*) indicates that the differences between the two groups are significant ($p < 0.05$).

Of these patients, 12 (29%) were men and 29 (71%) were women, with a mean age of 59 years. The diagnosis of posterior canal BPPV was based on medical history and findings of characteristic nystagmus, which is upward, and torsional beating towards the "down-side" affected ear produced by the Dix-Hallpike test. The diagnosis of horizontal canal type was made by using a positioning maneuver quickly turning the head 90 degrees toward one side with the patient supine and then rapidly turning the head to the other side. In case of canalolithiasis, this causes a horizontal geotropic nystagmus, which is greater after rotating the head toward the affected side, whereas in case of cupulolithiasis, this causes an ageotropic nystagmus, which is greater after rotating the head toward the unaffected side than the affected side.

There were 34 cases (83%) of posterior canal type and 7 cases (17%) of horizontal canal type of BPPV but no superior canal type. In our study, all the patients with horizontal canal type of BPPV showed a geotropic nystagmus, that is, a canalolithiasis type.

As a control, 92 healthy subjects (mean age, 42 yr; range, 22–74 yr), who had no conductive or sensorineural hearing loss and no known history of vestibular disorders, were selected. Patients and controls with a clinical examination suggesting severe systemic diseases or pathologic conditions of the central nervous system were excluded from the study. All patients and controls gave their informed consent regarding the study and the testing that would be involved.

Vestibular evoked myogenic potential testing was performed for all patients and controls. For the recording of VEMP response, 95-dB clicks were presented through a headphone. Patients were placed in a supine position and asked to raise and turn their head contralaterally to the ear being tested to achieve maximal activation of the SCM ipsilateral to the stimulation. Surface EMG activity was recorded with superficial electrodes placed on the middle third of the SCM, with the reference electrode placed on the upper third of the sternum and the ground electrode on the middle of the forehead. Electromyographic signals were recorded for a series of 95-dB click stimuli delivered at a frequency of 9.1 Hz and were bandpass-filtered (250–1 Hz) using an Audera system machine (GSI, CA, USA). The analysis time window was 90 milliseconds wide (−10 to 80 ms), and responses to 250 stimuli were averaged. The mean peak latencies (in ms) of the two early waves (p13 and n23) and the peak-to-peak (p13–n23) amplitude were measured on the ipsilateral side of the stimulation. p13 was the first positive peak of VEMP, and n23 was the first negative peak following p13. Absence of a meaningful wave form with p13 and n23 was defined as "no response."

The VEMP results of the patient and control groups were analyzed, and the values for latency and amplitude were calculated as mean ± standard deviation.

Benign paroxysmal positional vertigo patients with posterior canal BPPV were treated by Epley maneuvers, and patients with horizontal canal BPPV were treated by Barbecue maneuvers. The canalith repositioning maneuver was repeated everyday until the patient's report of relief from vertigo and findings of negative positioning test. In 18 patients (5 men [28%] and 13 women [72%]; mean age, 60 yr), testing of VEMP was repeated after treatment was completed, and it was compared with that obtained before treatment.

To specify the chronicity of disease and the resistivity to treatment, we recorded the number of times canalith repositioning maneuver was repeated until the patient's report of relief from

TABLE 1. *Vestibular evoked myogenic potential findings according to the type of BPPV*

	PSCC BPPV	HSCC BPPV	*p*
p13 latency, mean ± SD, ms	15.28 ± 1.81	13.51 ± 1.72	0.08
n23 latency, mean ± SD, ms	24.55 ± 2.16	23.13 ± 2.65	0.30
No. of CRM	3.12	4.5	0.64

PSCC indicates posterior canal type; HSCC, horizontal canal type; CRM, canalith repositioning maneuver.

TABLE 2. *Comparisons of VEMP differences before and after treatment*

	Before treatment	After treatment	*p*
p13 latency, mean ± SD, ms	15.35 ± 1.56	14.32 ± 2.20	0.15
n23 latency, mean ± SD, ms	24.98 ± 1.93	24.85 ± 1.84	0.86

vertigo and findings of negative positioning test. Finally, we tried to find out a relationship between the VEMP results and the progress of disease.

For statistical analysis, the values of the patient and the control groups were compared by using Student's *t* test. To analyze differences between values in a BPPV patient, a paired *t* test was used. We also used a correlation analysis to compare VEMP findings with the number of canalith repositioning maneuvers repeated. Statistical significance was set at $p < 0.05$.

RESULTS

Testing of VEMP was performed in each subject, and all volunteers in the control group and 30 patients in BPPV group showed a "response" in VEMP in both ears. However, 11 patients (27%) in BPPV group showed "no response" in VEMP in the affected ears. Both ears of the 30 patients in the BPPV group that showed a "response" in VEMP were tested, and the results were compared with those of the control group by Student's *t* test.

Vestibular Evoked Myogenic Potential Latencies in BPPV Patients

In the control group, the mean latency values for p13 and n23 were 13.25 ± 0.93 and 22.62 ± 1.76 ms, respectively. In the 30 affected ears of patients with BPPV, the mean latencies at p13 and n23 were 14.99 ± 1.89 and 24.31 ± 2.26 ms, respectively, which showed prolongation compared with those of the control group ($p < 0.001$). The affected ears of 13 patients and 6 patients of 41 BPPV patients showed significant prolongation (mean ± two SD of control group) in p13 and n23 latencies, respectively.

In contrast, the values of p13 and n23 latency obtained for the other 30 unaffected ears of BPPV patients were 13.76 ± 2.02 and 23.01 ± 2.41 ms, respectively, which showed no significant difference with those of the control group ($p = 0.27$ and $p = 0.08$, respectively). The amplitude showed no significant difference between the patient and control groups ($p = 0.07$); however, we regarded it as nonspecific value for VEMP because of its large variation and low reproducibility.

Also, we examined intrapersonal differences in BPPV patients, which, as expected, revealed prolonged p13 and n23 latencies in the affected ear compared to those of the unaffected ear ($p = 0.02$ and $p = 0.03$, respectively; Fig. 1).

Vestibular Evoked Myogenic Potential Findings According to the Type of BPPV

This study included 34 cases (83%) of posterior canal type and 7 cases (17%) of horizontal canal type of BPPV but no superior canal type. Horizontal canal type BPPV was exclusively a canalolithiasis type. Nine (26%) of 34 patients in the posterior canal type and 2 (28%) of 7 patients in the horizontal canal type showed "no response" in VEMP in the affected ear. The ratios of the patients with a posterior to horizontal canal type of BPPV in the "no response" group were similar to those in the patient group (4.5 and 4.8, respectively). Twenty-five affected ears in patients with posterior canal type and five affected ears in patients with horizontal canal type which showed a "response" in VEMP were compared by using Student's *t* test, which showed no significant differences in the p13 and n23 latencies ($p = 0.08$ and $p = 0.30$, respectively). The number of times the canalith repositioning maneuver was repeated until the patient's report of relief from vertigo and findings of negative positioning test revealed no significant difference between

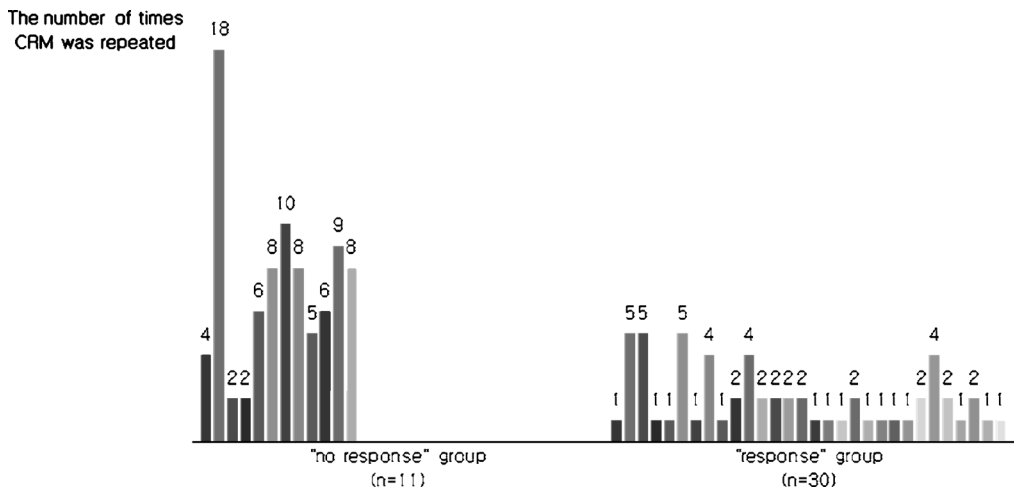

FIG. 2. This chart shows the number of times canalith repositioning maneuver (CRM) was repeated in BPPV patients. In the "no response" group, the number of times the maneuver was repeated was considerably larger than that in the "response" group.

patients with posterior and horizontal canal types of BPPV ($p = 0.64$; Table 1).

Vestibular Evoked Myogenic Potential Findings After Canalith Repositioning Maneuvers

Testing of VEMP was repeated after treatment was completed, and p13 and n23 latencies were not decreased significantly compared to the pretreatment values by a paired t test ($p = 0.25$ and $p = 0.86$, respectively; Table 2.

Vestibular Evoked Myogenic Potential Findings and the Number of Times the Maneuver was Repeated

The number of times canalith repositioning maneuver was repeated until the patient's report of relief from vertigo and findings of negative positioning test was not correlated to the degree of latency prolongation (correlation coefficient, $R = 0.15, 0.23$). However, in the "no response" group, the number of times the maneuver was repeated was considerably greater than that in the "response" group, and the difference was statistically significant ($p < 0.05$). The mean values of each were 7.2 ± 4.3 and 2.0 ± 1.4 times, respectively (Fig. 2).

DISCUSSION

The main objective of this study was to report VEMP findings in BPPV patients and to verify some clinical applications of this test.

We found a significant prolongation of p13 and n23 latencies in BPPV patients when compared with those in the control group, and the interaural differences of p13 and n23 latencies between the affected and the unaffected ears in BPPV patients were obviously noted. We could not find any significant difference in VEMP results between patients with posterior and horizontal canal types of BPPV.

We also tested VEMP amplitude and latencies at p13 and n23, but the values showed a wide range of distribution and low reproducibility. Amplitude means an intensity of a tonic contraction of the relevant muscle, so it could be easily affected by many factors such as basic muscle activity, patient's position, and general conditions. We regarded it as a nonspecific value for VEMP in this study.

As mentioned previously, the degenerative process of various insults that affect the macula of the saccule or the inferior vestibular nerve might be related with the late development of BPPV. In a previous study, Gacek (5) examined temporal bones from five patients with BPPV and reported that the major abnormality in these temporal bones to be a loss of vestibular ganglion cells in the inferior vestibular nerve with sparing of the superior vestibular nerve. They also noted evidence of ganglion cell degeneration in the saccular nerve in two cases and a large number of shrunken neurons with a few normal ganglion cells in the saccular part of the inferior vestibular ganglion with mostly normal posterior canal ganglion cells in one case. Such neural degeneration of saccule could explain the abnormalities in p13 and n23 latencies of VEMP transmitted through the vestibular saccule and the inferior vestibular nerve. The prolongation of p13 and n23 latencies did not decrease significantly after the treatment of patients with canalith repositioning maneuvers, which also implies such irreversible neuronal degenerative changes.

Although canalith repositioning maneuvers have been the standard treatment of BPPV and high success rates have been reported, there is a certain subgroup of patients who do not respond well to this treatment. Previous studies reported that poor prognosis is related to age, cause, and additional vestibular pathologic finding (6–9). We analyzed our test results in BPPV patients to find out if chronicity and resistivity of BPPV cases have any relation with these abnormal results.

In our study, the number of times the canalith repositioning maneuver was repeated in the "no response" group was considerably greater than that in the "response" group. This result suggests that if there are more extensive damages to the saccule, which shows "no response" in VEMP, the disease progress itself reveals as a chronic and resistive course. Patients in the "no response" group had no history of vestibular disorders as the patients in the "response" group; however, one patient in the "no response" group showed 32% of canal paresis in a caloric test in the affected ear. Others in the BPPV group revealed normal caloric test.

In summary, in the pathophysiology of BPPV, the degenerative processes of various insults that affect the macula of the saccule result in abnormal VEMP responses. Especially in the "no response" group, which means that there was more extensive damage that affects the macula of the saccule or the concurrence of other vestibular disorders such as Ménière's disease, vestibular neuronitis, and labyrinthitis that could damage hair cells in the saccule extensively, the disease underwent more chronic and resistive course.

In conclusion, we propose that BPPV is related to vestibular neuronal degeneration of saccule and its prognosis could be predicted by assuming the extent of pathological change in the saccule by testing of VEMP.

CONCLUSION

We found that VEMP latencies are increased in BPPV patients. Abnormal VEMP results may signify neuronal degenerative changes of various insults in the macula of the saccule. When an extensive neuronal damage was suspected by VEMP results such as "no response" in VEMP, the disease progress showed a chronic and resistive course. Therefore, we propose that VEMP could be a useful method to determine the clinical prognosis of patients with BPPV.

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