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Vestibular evoked myogenic potentials in patients with BPPV

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Summary

Background: The probable cause of Benign Paroxysmal Positional Vertigo (BPPV) is a degeneration of the oto lithic organs (utricle and saccule). The aim of the study is to find possible alterations in Vestibular Evoked Myogenic Potentials (VEMP) recordings in BPPV patients, because the saccule is part of the VEMP pathway.

Material/Methods: 27 BPPV patients (24 unilateral and 3 bilateral) aged 20 to 70 years and 30 healthy age matched controls. BPPV was diagnosed by the upbeating geotropic nystagmus found in the supine position with the head overextended towards one side. The subjects were investigated with pure tone audiometry, bi-thermal caloric test with electronystagmographic (ENG) recording, and VEMP recording.

Results: P1 latency and N1 latency did not present any statistical difference between control ears and affected ears of the BPPV population. The percentage of abnormal VEMP in the BPPV population was statistically higher than in the control ears ($p<0.005$). No significant relationship could be shown between the occurrence of Canal Paresis and abnormal VEMP. No relationship was found between the side (right or left ear) where BPPV appeared clinically and the side where abnormal VEMP was registered.

Conclusions: BPPV is a clinical entity associated with increased occurrence of abnormal VEMP recordings, possibly due to degeneration of the saccular macula, which is part of the neural VEMP pathway.

key words: **Benign Paroxysmal Positional Vertigo • saccule • Vestibular Evoked Myogenic Potentials**

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Background

Although the investigation of inner ear function has recently led to impressive results, there are new discoveries being made daily. Both the auditory and vestibular systems possess properties that are difficult to fully understand. Vestibular evoked myogenic potentials (VEMP) are one of the newest methods used to investigate the vestibular system, based on the vestibulo-spinal reflex. The neural pathway that is tested by VEMP begins at the saccule and leads the stimulus via the inferior vestibular nerve, lateral vestibular nucleus, and medial vestibunospinal tract to the sternocleidomastoid muscle [1]. The stimulus can be either a high intensity sound or a tap.

Von Bekezy [2] was the first who described sound evoked vestibular responses. By using intense sounds of 128 to 134 dB he observed a head movement towards the stimulated ear. In 1992 Colebatch et al. [3] described click evoked potentials that could be averaged out of the electromyographic activity of neck muscles, and Halmagyi [4] claimed that their generation did not involve the lateral semicircular canal. Ever since, VEMP applications have been increasingly used in the audiovestibular test battery. In Meniere's disease, the features of vestibular myogenic potentials [5] are expected to alter, based on the hypothesis that the hydrops affects the membranous labyrinth, part of which is the saccule. Superior Canal Dehiscence Syndrome is a possible diagnosis when enhanced vestibular sensitivity is seen in VEMP recording as amplitude enlargement and threshold lowering [6]. Vestibular neuronitis, a disease affecting the vestibular nerve, has been more commonly investigated by the calorics, which tests the function of the horizontal hemicircular canal, i.e., the superior vestibular nerve. VEMP makes it possible to extend the investigation to the inferior vestibular nerve and check for residual function in cases of deep canal paresis [5]. In the investigation of retrocochlear pathology, i.e., vestibular schwannomas, VEMP can give additional information on Auditory Brainstem Responses (ABR) [7]. Moreover, VEMP are used to monitor labyrinthine function after gentamycin intratympanic injections for chemical labyrinthectiomy [5]. Efficacy of stapes mobilization procedures in otosclerosis patients can be potentially monitored by VEMP [5]. In the investigation of central vestibular pathology, VEMP abnormalities are seen in mid- and lower pontine, as well as medullary lesions, whereas ABR abnormalities are more common in midbrain and pons lesions [8].

Benign paroxysmal positional vertigo is one of the most common peripheral vestibular disorders [9]. It occurs spontaneously in many patients but may also follow head trauma, labyrinthitis, and ischemia in the distribution of the anterior vestibular artery or prolonged bed rest. The first theory to explain the pathophysiology of the disease was proposed by Shuknecht [10] in 1969, who claimed that otoconia, with a specific gravity greater than that of the endolymph, detaches from a degenerating utricular macula and settles on the cupula of the posterior semicircular canal, rendering it gravity sensitive. Hall et al. [11] in 1979 proposed the theory of canalolithiasis, which claims that the otoconia particles are not attached to the cupula, but float freely in the canal lumen. Although recent studies have revealed BPPV affecting the horizontal and the anterior semicircular canal, the posterior semicircular canal is most frequently affected [12,13]. The inferior vestibular nerve synapses with the latter, as well as with the saccule. Although this is a rather common pathological entity, only a few studies with small population samples [14,15] have investigated possible alterations in VEMP in BPPV patients. Akkuzu et al. [14] found a significantly higher frequency of abnormal VEMP in BPPV patients compared with the control group. They had an indication, although not statistically significant, that BPPV patients with abnormal recordings are those with a history of more resistant positional vertigo, leading them to suppose that chronicity of the disease may imply saccular degeneration in addition to the expected utricular degeneration, and state that further studies should include this assumption. They also posed the question of whether the utricle itself could be a part of the reflex arc of VEMP.

The aim of this study is to record VEMP in BPPV patients and to identify any possible relation of alterations in the recordings with the clinical picture.

Material and Methods

The study population consisted of 2 groups.

The first group consisted of 27 patients (54 ears) 14 men and 13 women, ages 20 to 70 years, (median 45 years). Three of them presented bilateral BPPV of the posterior semicircular canal. Of the rest, 11 patients presented BPPV on the right and 13 on the left.

The second group consisted of 30 age-matched healthy controls (60 ears), 17 men and 13 women (median age 47 years).

The patients were diagnosed with BPPV at the start of the investigation. Diagnosis was made with observation of torsional upbeating geotropic nystagmus triggered by the Dix-Hallpike maneuver [16]. The nystagmus should present the peripheral signs, i.e., latency, limited duration, accompanying abrupt and intense subjective vertigo, intensity characterized by crescendo and decrescendo and fatigability on repetitive provocation [17]. A detailed medical history was taken, registering the time of first occurrence of the present vertiginous symptoms, number of recurrences in the past, and number of therapeutic maneuvers (Epley of Semont) they had so far. Patients with diagnosed vestibular neuronitis or any other known vestibular problems or conductive hearing loss were excluded. All patients and controls were evaluated with pure tone audiometry (PTA), bi-thermal caloric test with electronystagmographic (ENG) recording and VEMP recording. ENG recordings were performed with a Life Tech model 3002 (Houston Texas) electronystagmograph, while a Hortmann Airmatic (Neurootometrie) air irrigator was used for the bi-thermal caloric test, and the maximum slow phase velocity was determined with a Life Tech 3100 velocity computer system. The methodology is reported in detail elsewhere [18].

VEMP was registered with the GN Otometrics (Taastrup, Denmark) EP v.5.2 analyzer with a 2-channel averaging capacity. The patient was seated in an upright position, keeping the head turned contralaterally to the stimulated ear. In order to achieve sufficient and constant contraction of the SCM muscle during recordings, we used a blood pressure manometer with an inflatable cuff. The patient was instructed to push

Table1. The table shows the numbers of ears with VEMP abnormalities found in Patient population and in controls.31.5% of patients' ears presented VEMP abnormalities.8.3% of the control ears presented VEMP abnormalities. 30% of the ears affected by BPPV presented VEMP abnormalities, whereas the same percentage in patients' ears non affected by BPPV was 33.3%.

	P1 Delay	N1Delay	P1 &N1 delay	VEMP non recordable	VEMP abnormal	
Patients ears n=54	11	–	1	5	17	(31.5%)
Affected ears n=30	5	–	1	3	9	(30.0%)
Non Affected ears n=24	6	–	–	2	8	(33.3%)
Control n=60 ears	3	1	1	–	5	(8.3%)

Table 2. The table shows the distribution of VEMP abnormalities in relation to the side of ear presenting clinical symptoms of BPPV. The second column shows the number of patients with unilateral BPPV who presented abnormal VEMP in the ipsilateral ear, the contralateral ear or both ears respectively. The third column shows the number of patients with bilateral BPPV who presented abnormal VEMP (there is only one patient who presented VEMP abnormalities in both ears).

Abnormal VEMP	Unilateral BPPV	Bilateral BPPV
Ipsilateral ear	3	
Contralateral ear	4	
Both ears	4	1

their jaw against the hand held inflated cuff so as to generate a specified pressure and maintain it to a constant level, a feedback method proposed by Vanspauwen et al. [19] in order to monitor muscle contraction and reduce VEMP amplitude variability. Two active electrodes were placed over the midpoint of the SCM muscles, with a reference electrode on the upper forehead and a ground electrode in the middle of the forehead. The response of the ipsilateral SCM muscle to monaural stimuli was recorded. The acoustic stimuli (short tone-bursts, 95 dB HL, 500 Hz, rate 5.1/s, ramp=1ms, plateau=0 ms), were delivered monaurally through headphones (TDH-40), with no contralateral masking, and the myogenic potential was recorded ipsilaterally by surface electrodes. The skin was scrubbed and the impedance of the recording electrodes was maintained below 5 KOhms. Electromyographic (EMG) activity of the ipsilateral SCM was recorded and every trial of 150 stimuli was averaged and repeated twice to verify the reproducibility of the waveform and to provide the final VEMP response, which was the average of the 2 recordings. The EMG signal from each side was amplified and bandpass filtered (Hi-pass 2 Hz, Low-pass 500 Hz). The stimulus analysis time for each run was 100 ms. The first positive deflection on the waveforms was marked as P1, and the first negative deflection was marked as N1. The latencies of these waves were calculated and recorded. VEMP response was considered to be absent when there were no recognizable or reproducible biphasic waveforms.

Statistical analysis

SPSS 11.0 was used to perform statistical analysis. Student's T-test was used to compare the means of P1 Latency and N1 Latency between patients and controls, and between affected and non-affected ears in BPPV patients. The association between caloric measurements and VEMP measurements was assessed by Fisher's exact test, and χ^2 method was used to assess the association between the side (right or left ear) of BPPV clinical appearance and the side of abnormal VEMP recording.

Results

All ears (60 ears) of the control population presented VEMP. The mean latency of P1was 16.32ms (SD: 1.59). The mean latency of N1 was 24.62 ms (SD: 2.8). In order to determine the maximum latency considered within normal limits, 2SD was added to each mean latency, thus maximum normal value for P1 latency was 19.50 ms and 30.22 ms for N1 latency. Values higher than those were considered abnormal. The abnormal findings in the control population are reported in Table 1. Mean P1 latency of right ears was 16.34 ms (SD: 1.62) and of left ears 16.31 (SD: 1.59). There was no statistical difference between right and left P1latency ($p>0.05$, paired samples t-test). Mean N1 latency of right ears was 24.78 ms (SD: 3.22) and of left ears 24.45 (SD: 2.35). There was no statistical difference between right and left N1 latency ($p>0.05$, paired samples t-test).

In the patient population (54 ears), 30 ears presented BPPV and 24 ears were unaffected. Abnormal findings were recorded in 31.5% of patients' ears, shown in detail in Table 1.

The percentage of VEMP abnormalities in the non-affected ears of the BPPV population (33.3%) was statistically higher than the percentage of VEMP abnormalities in the control ears (8.3%) Fisher's exact test ($p=0.008$). The proportion of abnormal findings in the BPPV group (31.5%) was significantly higher than the proportion of abnormal findings in the control group (8.3%) ($\chi^2=9.77$, $p=0.002$).

Among the 12 patients who presented VEMP abnormalities, 4 unilateral BPPV patients had abnormal VEMP in both ears and 1 bilateral BPPV patient had bilateral VEMP abnormalities, 3 patients had abnormal findings in the BPPV ear and 4 had abnormal VEMP findings in the contralateral ear (Table 2).

No relationship was found between the side (right or left ear) where BPPV appeared clinically and the side where abnormal VEMP was registered, ie, there was no statistical difference in the proportions of abnormal VEMP occurrence either in the ear presenting BPPV or on the contralateral side, ($\chi^2=0.069$, $p=0.79$).

Table3. The table shows the comparison (independent samples t-test) between mean Latencies of P1 (SD) and N1 of control ears n=60 ears) and mean Latencies of P1 (SD) and N1 respectively of the affected ears (i.e. ears presenting BPPV) with recordable VEMP waveforms (n=27) of BPPV patients. In the Patient group mean latencies are slightly longer than in the controls, but difference is not statistically significant.

	P1 latency (ms) (SD)	N1 latency (ms) (SD)
Control ears	16.32 (1.59) (n=60)	24.62 (2.8) (n=60)
Patient ears with BPPV and recordable VEMP	17.30 (2.68) (n=27)	25.24 (2.87) (n=27)
p value	0.08	0.35

Table 4. The table shows the comparison (paired samples t-test) between the mean P1 latency (SD) of ears presenting BPPV (affected) and mean P1 latency (SD) in the contralateral (non affected) ears of BPPV patients with unilateral BPPV, n=20. (Bilateral BPPV patients i.e. 6 ears and unilateral BPPV patients with non recordable VEMP i.e. 8 ears, were not taken into this account). No statistical difference was found. No statistical difference was found for N1 latency.

	P1 latency (ms) (SD)	N1 latency (ms) (SD)
Affected ears (n=20)	16.80 (1.60)	24.80 (2.30)
Non affected ears (n=20)	17.19 (1.86)	25.14 (1.59)
p value	0.44	0.55

Table 5. The table shows the results of calorics and VEMP in BPPV patients' ears. 31.5% of ears had abnormal VEMP. 24% of ears had Canal Paresis (CP). No statistical relationship was found between the occurrence of CP and VEMP abnormality in BPPV patients' ears. (Fisher's exact test, p=0.617).

	VEMP normal	VEMP abnormal	Total
Calorics normal	28 (51.9%)	13 (24.1%)	41 (76%)
CP	9 (16.6%)	4 (7.4%)	13 (24%)
Total	**37 (68.5%)**	**17 (31.5%)**	**54 (100%)**

Table 3 presents the comparison of means of P1 latency and N1 latency between control ears and affected ears of patients.

P1 latency was compared between affected and non-affected ears of 20 patients who had unilateral BPPV and VEMP was recordable in both ears. The same was done for N1 latency. No statistical difference was found either for P1 latency or for N1 latency (paired samples t-test), shown in Table 4.

In BPPV patients, 31.5% of ears had abnormal VEMP and 24% of patients' ears had abnormal calorics (Table 5); however no significant relationship could be shown between the occurrence of Canal Paresis (CP) and abnormal VEMP in BPPV patients' ears (Fisher's exact test, p=0.617). No significant relationship could be shown between the occurrence of CP and abnormal VEMP in the affected ears of BPPV patients (Fisher's exact test, p=0.441).

Discussion

The degenerative process that leads to the detachment of otoconia in BPPV patients is not yet fully understood. Regarding pathophysiological mechanism, the role of the utricle is widely accepted due to its anatomical proximity with the ampulla of the posterior semicircular canal. However, newer theories [20,21] claim that otoconia-provoking BPPV derives from both maculae, because with increasing age a decrease in the gelatinous layer of the otolithic membrane occurs, which may allow spontaneous dislodgment of the otoconia from the utricle or saccule to occur more easily. Then, otoconia particles, driven by gravity, reach the canal ampulla, where they become trapped. Once they reach a critical mass, they affect the physiological fluid motion and create symptoms when the head takes certain positions. The possibility of saccular involvement in BPPV allows the assumption that degenerated saccular structures may affect the vestibular evoked myogenic potentials, since the saccule is the beginning of their neural pathway [5].

The present study showed significantly more abnormal VEMP recordings in the BPPV patients than in the control population, a finding that supports a previous report by Akkuzu, et al. [14]. Our results support the notion that BPPV is a clinical entity affecting the VEMP pathway in a way that should be further investigated. BPPV is a disease of the labyrinth, ie, a peripheral organ disease, and in this study VEMP recordings in BPPV patients were found to be either normal or absent, or with delayed latencies. This finding contradicts a previous report [22] claiming that in cases of end organ or vestibular nerve damage, VEMP is either normal or absent, whereas in more central neural pathway damage (brainstem disease) VEMP is either present, but with delayed latencies, or is absent. Akkuzu et al. [14] report delayed latencies in some of their BPPV patients, in agreement to our results. Despite the fact that the percentage of abnormal VEMP recordings was statistically higher in BPPV patients than in controls, P1 and N1 means are not statistically different between BPPV patients and controls. This could be explained by the fact that between a normal and an abnormal latency value there is a concrete and defined limit (mean+2SD), meaning that, numerically, a normal and an abnormal value can be very close, and apparently higher

population numbers would be needed in order to show possible statistical differences. Akkuzu et al. [14] had similar results regarding the comparison of latencies.

Another interesting finding was the abnormal VEMP of the contralateral healthy side. Comparing affected to unaffected ears, no statistical difference was found either between P1 latencies or between N1 latencies. In addition, the abnormal findings in BPPV ears and contralateral ears were in almost equal percentages (30% in the BPPV and 33.3% in the contralateral ears of the BPPV population). Therefore, no relationship was found between the side of BPPV and the side where abnormal VEMP was registered. It is very difficult to interpret the fact that VEMP is abnormal in the contralateral ear of a patient with unilateral BPPV. A most probable explanation would be that degeneration of the contralateral saccule might exist, affecting VEMP recording. However, debris mass detached from the utricle (possibly degenerated as well), or the saccule itself, is not sufficient to provoke BPPV symptoms on that side. Another possibility is that there was BPPV in the past on that side, but this was not clearly shown in the patient's history due to insufficient information. A third explanation is a possible down-regulation in the level of vestibular nuclei. This is an assumption based on previous reports trying to interpret a horizontal canal paresis found either on the ipsilateral or the contralateral ear of some BPPV patients [18]. The finding is attributed to an up-regulation of the contralateral medial vestibular nucleus type I neurons, leading to canal paresis in the BPPV ear, or a down-regulation of them, occurring as a compensatory feedback, leading to canal paresis of the contralateral ear. According to this model, one cannot exclude that similar crossed neural pathways could up- or down-regulate the contralateral lateral vestibular neucleus and produce VEMP abnormalities on the contralateral side. This assumption of central interaction between the 2 sides is supported by the fact that the existence of crossed pathways is already proven by galvanic stimulation, eliciting a contralateral VEMP recording at the same time with the ipsilateral [5]. Lastly, one could claim that abnormal VEMP on the contralateral ear of a BPPV patient is a finding completely irrelevant to BPPV, an assumption which is nevertheless contradicted by the high percentage (33.3%) of VEMP abnormalities in the contralateral (i.e. non-affected) ears of our population, which is statistically different from the controls.

Finally, no statistically significant relationship could be shown between the occurrence of VEMP (saccule) and caloric testing (Horizontal canal) abnormalities in our study. The abnormal ENG findings in BPPV patients is an issue already discussed in previous studies and several assumptions attempt to interpret it [18]. Among these, a very interesting hypothesis is that otoconia exists not only in the posterior semicircular canal, but also in minor quantities in the other 2 canals. Although its mass is not able to cause horizontal BPPV symptoms, its presence in the endolymph of the horizontal canal creates a gravitational load in the fluid and may affect the caloric response. Consequently, and in relation with our findings regarding VEMP, an ear with posterior canal BPPV might have ENG abnormalities and normal VEMP, VEMP abnormalities and normal calorics, both tests normal, or both abnormal. The dissociation of VEMP from caloric response findings is also reported in other otological diseases [23] such as Meniere's disease, acoustic neuroma and sudden deafness with vertigo.

Conclusions

The percentage of abnormal VEMP in BPPV patients was statistically different from that in the control population. The conralateral, non-affected side showed an important percentage of abnormal VEMP, similar to that of the affected side. There was no significant relationship between VEMP and caloric testing abnormalities in BPPV patients' ears. The main conclusion of the present study is that in BPPV patients there are subclinical events, taking place in the same or the contralateral ear, which can affect the test battery. Such events could be the degeneration of the saccule in either ear and the presence of subclinical otoconia debris in the horizontal canal. Furthermore, the interaction of the 2 ears through pathways involving the vestibular nuclei might also be an explanation of pathological VEMP.

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