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Personal Experiences with Vestibular Evoked Myogenic Potentials as a Modern Method of Diagnosing Vestibular Organ Lesion and Monitoring Treatment

Alina Morawiec-Bajda

Audiology and Phoniatrics Department, Medical University, Łodź, Poland

Abstract: Vestibular evoked myogenic potentials (VEMPs) appear to represent a new and promising technique for the assessment of vestibulospinal reflex function. The primary aims of the study described in this article were (1) to record VEMPs in normal volunteers using available equipment and to establish a range of norms of VEMP parameter values (latency, amplitude); (2) to confirm the saccular origin of VEMPs; (3) to assess the diagnostic significance of VEMPs; and (4) to evaluate the usefulness of VEMPs in monitoring therapeutic results. The study population consisted of 252 patients representing various diagnoses of hearing loss and vestibular organ lesion. Twenty-three patients were treated with an antihomotoxic remedy, and some received placebo. The results of this study demonstrated that VEMPs are helpful in evaluating the physiological and pathological equilibrium system and in monitoring reflex reactions after treatment.

Key Words: sensorineural hearing loss; total deafness; unilateral canal paresis; vestibular evoked myogenic potentials; vestibular neuritis; vestibular schwannoma

Examination of the vestibular system is difficult because of its complicated anatomical structure. The system contains symmetrical vestibular organs and many connections among them. Until now, lack of an unquestionable method of assessing vestibular function has limited study of this area. Assessment of the otolithic organ function is more complicated owing to the impossibility of estimating it without the influence of gravitational force and the canal system. Brainstem neurogenic potentials after stimulation of the horizontal canal are small and require a special apparatus capable of applying high acceleration rapidly and repeatedly to the head. The use of corticovestibular evoked potentials often produces unpredictable results. Vestibular evoked myogenic potentials (VEMPs) seem to represent a new and promising technique for the assessment of vestibulospinal reflex function. Using this technique, proposed by Colebatch and Halmagyi [1,2], with our personal modification [3–5], we undertook a study at the Audiology and Phoniatrics Department of the Medical University in Łódź to determine the clinical significance of VEMPs. Many authors [2,6,7] who described this reliable and noninvasive method either did not assess the role of VEMPs testing exactly in patients with vertigo of various etiologies or presented their results on the basis of testing a small group of patients. The aims of this article are (1) to record VEMPs in normal volunteers using available equipment and to establish a range of norms of VEMP parameter values (latency, amplitude); (2) to confirm the vestibular (and especially) the saccular origin of VEMPs; (3) to assess the diagnostic significance of VEMPs in patients with inner-ear diseases or vestibular nerve lesion; and (4) to assess the usefulness of VEMPs in monitoring therapeutic results.

Reprint requests: Dr. Alina Morawiec-Bajda, 91-487 Łódź, ul. Beczkowa 42 m 12. Poland. E-mail: morawiec@csk.am.lodz.pl

PATIENTS AND METHODS

Patient Selection and Clinical Evaluation

The study population consisted of 252 people ranging in age from 15 to 78 years (average, 46 years). This

group was made up of 165 females (15–78 years) and 87 males (18–69 years). The total number of assessed ears was 504, including 130 healthy ears. The patient population represented diagnoses of unilateral total deafness, sensorineural hearing loss (SNHL), vestibular neuritis, Ménière's disease, unilateral canal paresis, and vestibular schwannoma.

To classify the patients into suitable testing groups according to etiology, after conducting and recording a medical history of all patients, we administered pure-tone audiometry, a click-evoked auditory brainstem test, and computerized electronystagmography. Vestibular examination included the recording of spontaneous nystagmus, positional nystagmus, and optokinetic nystagmus, a smooth-pursuit test, a sinusoidal rotary chair test, and a bithermal caloric test. We used a computer to calculate directional preponderance and canal paresis on the basis of maximum slow-phase eye velocity. In justified cases, we made functional radiological pictures of the cervical spine, USG-Doppler of cerebral arteries, and computed tomographic or magnetic resonance imaging of the head.

Some of the patients (a total of 20 women and 3 men) were treated with an antihomotoxic remedy (cerebrum compositum; Biologische Heilmittel Heel GmbH, Baden-Baden, Germany). The etiology of the diseases was as follows: eight patients had diagnosed Ménière's disease; five suffered from vestibular neuritis; five complained of vertigo caused by vertebrobasilar arterial insufficiency; three exhibited diagnosed vertigo after head trauma; one suffered from benign paroxysmal positional vertigo; and in one, cause of disease was unknown. Seven persons were administered placebo.

Cerebrum compositum is prepared according to Dr. Reckeweg's formula and seems to stimulate the central nervous system and activate control regions in the spinal cord. Cerebrum compositum is composed of active substances of vegetable and animal origin and presumably dilates brain blood vessels. Thanks to such actions, the remedy is employed in the treatment of vertigo (especially chronic vertigo) and buzzing in the ears and also is used to improve memory and mental concentration, as was described recently [3,8].

Vestibular Evoked Myogenic Potentials

VEMP recordings were performed with a MEDELEC–Sapphire 2 ME (MEDELEC, England) with a two-channel averaging capacity. Our own modification of the examination dealt with the position of an examined person, the placements of the fastened electrodes, and the parameters of the stimuli. Patients lay supine and held the head rotated to one side but also bent to the chest. This position allowed the activation of neck flexors on one side. For example, if the record of muscular activity was from the right sternomastoid muscle, the head was turned to the left, and acoustic stimuli were delivered first to the right and then to the left ear. We monitored muscle activation during the test using surface electrodes. An active electrode was placed over the upper half of each sternocleidomastoid muscle (SCMM). A reference electrode was located in the middle of the anterior edge of the clavicle, and a ground electrode was fastened over the upper part of the sternum. The electromyographic (EMG) signal from each side was amplified and band-pass-filtered (20–2,000 Hz). Rarefaction clicks (0.1 msec, 110 dB NHL) were presented through a headphone. The stimulus rate was 5 Hz, and analysis time was 50 msec. We averaged the responses to 512 stimuli twice. Sensitivity ranged from 20 μV to 50 μV.

VEMPs were recorded as a biphasic, positive-negative wave that was described by lowercase letters (p = positive, n = negative). Accounted parameters were as follows: Latencies of the first positive (p) and negative (n) peaks and amplitudes were measured peak to peak (p–n) in ipsilateral and contralateral recordings. The first positive peak was p13 wave and the first negative peak was n21 wave of VEMPs.

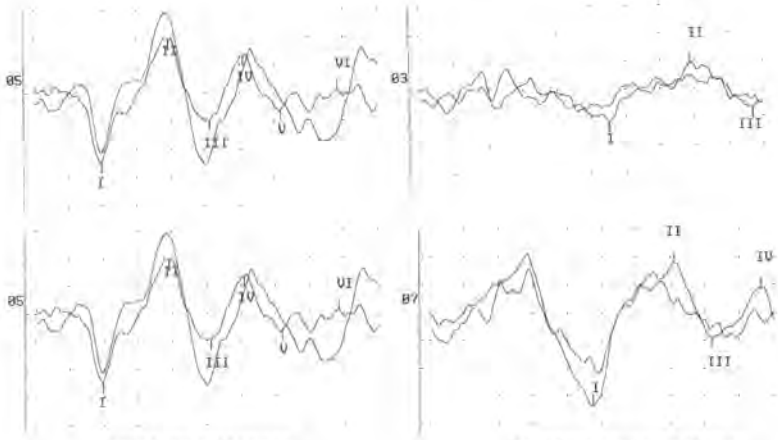
Statistics

In this study, we made a statistical assessment of results using the nonparametric Mann-Whitney test to compare the distribution compatibility of studied variables in control and patient groups and between healthy ears and ill ears in some groups. Comparison tests were considered to be statistically significant ($p < .05\%$). Mean values were given plus or minus one standard deviation (SD), and amplitude was measured peak to peak [9].

RESULTS

Normal Parameters of VEMPs in Control Group

Both ipsilateral and contralateral recordings of VEMPs were recorded in 121 patients and 130 healthy volunteers who formed the control group. VEMPs were absent in response to 110-dB clicks in nine older patients (>50 years). The mean latency (±SD) of the first positive peak (p13) in ipsilateral recordings was 12.69 ± 2.53 msec. If the mean ± 1 SD was defined as the limit of normal range, the normal range of ipsilateral p13 was 10.16–15.22 msec. The mean latency of the first negative peak (n21) in ipsilateral recordings was 20.55 ± 3.22 msec, so the normal range of n21 was 17.33–23.77 msec. The mean latency of p13 in contralateral



Ipsilateral recordings Contralateral recordings

Figure 1. Normal ipsilateral and contralateral recordings.

recordings was 19.08 ± 4.26; thus, the normal range was 14.82–23.34 msec. The mean latency of n21 in contralateral recordings was 26.30 ± 4.72; thus, the normal range was 21.58–31.02 msec.

The mean amplitude (±SD) of ipsilateral p13 was 4.44 ± 4.43 μV; of ipsilateral n21 as 3.54 ± 2.94 μV; of contralateral p13 was 2.37 ± 1.57 μV; and of contralateral n21 was 2.31 ± 1.59 μV. Normal recordings are presented in Figure 1.

Patients with Total Deafness

In patients after stimulation of deaf ears (57 ears), the parameters of mean latency and mean amplitude in both recordings were similar to findings of the control group. We observed normal caloric test results or hypoexcitability of the lateral canal. Comparative statistical analysis of reaction after stimulation of deaf ears and healthy ears disclosed no significant difference (Table 1).

Patients with SNHL

We detected no significant difference in the latencies and amplitudes of VEMPs between intact ears of the control group and affected ears of patients with SNHL (102 ears; Table 2).

Patients with Unilateral Deficiency of Vestibular Excitability in the Caloric Test

We observed no statistically significant difference in the latencies and amplitudes of VEMP recordings after

Table 1. Comparison of Parameters of Vestibular Evoked Myogenic Potentials After Stimulation of Deaf Ears and Intact Ears (Control Group)

Wave	Variable	Statistical Parameters	Control Group	Deaf Ears	Mann-Whitney Test
p14	Ipsilateral	×	12.69	13.66	0.198
		SD	2.53	2.49	
	Ipsilateral-amp	×	4.44	2.59	0.288
		SD	4.33	1.62	
	Contralateral	×	19.08	19.35	0.850
		SD	4.26	5.01	
	Contralateral-amp	×	2.37	2.10	0.836
		SD	1.57	1.10	
n21	Ipsilateral	×	20.55	20.18	0.666
		SD	3.22	2.49	
	Ipsilateral-amp	×	3.54	2.18	0.210
		SD	2.94	0.99	
	Contralateral	×	26.30	25.75	0.726
		SD	4.72	5.95	
	Contralateral-amp	×	2.31	2.08	0.986
		SD	1.59	1.02	

amp = amplitude; SD = standard deviation; × = arithmetical average.
Note: Deaf ears, n = 51; intact ears (control group), n = 121.

Table 2. Comparison of Parameters of Vestibular Evoked Myogenic Potentials After Stimulation of Ears with Sensorineural Hearing Loss and Intact Ears (Control Group)

Wave	Variable	Statistical Parameters	Control Group	SNHL Ears	Mann-Whitney Test
p13	Ipsilateral	×	12.69	12.72	0.5714
		SD	2.53	1.83	
	Ipsilateral-amp	×	4.44	6.43	0.7654
		SD	4.33	8.69	
	Contralateral	×	19.08	17.49	0.2065
		SD	4.26	5.08	
	Contralateral-amp	×	2.37	1.99	0.3504
		SD	1.57	1.23	
n21	Ipsilateral	×	20.55	20.04	0.4498
		SD	3.22	2.70	
	Ipsilateral-amp	×	3.54	5.76	0.6364
		SD	2.94	7.47	
	Contralateral	×	26.30	26.14	0.9013
		SD	4.72	4.15	
	Contralateral-amp	×	2.31	2.66	0.5585
		SD	1.59	1.96	

amp = amplitude; SD = standard deviation; SNHL = sensorineural hearing loss; × = arithmetical average.
Note: Sensorineural hearing loss, n = 102; intact ears (control group), n = 121.

stimulation of ears with weakness of excitability (58 ears) and ears of the control group. In those ears (n = 24) where we found the absence of excitability, we noted either lack of responses or lower amplitudes of VEMPs.

Patients with Vestibular Neuritis

VEMPs were not recorded in some patients with vestibular neuritis (n = 56). We found that VEMPs were normal in four patients (7.1%). A lack of response was detected in 10 patients (17.9%). Responses on the affected side were reduced in 41 patients (73.2%) such that below the lower limit of normal (i.e., mean amplitude) of VEMPs was less than one-third of the amplitude on the normal side. Latencies of ipsilateral p14, ipsilateral n21, contralateral p14, and contralateral n21 of ears afflicted with vestibular neuritis did not differ from latencies of the control group. Statistical analysis confirmed differences of amplitudes of all waves of VEMPs between ill and healthy ears (Table 3).

Patients with Ménière's Disease

The initial biphasic p13–n21 evoked potential was absent from the ipsilateral SCMM in 17.9% of patients with Ménière's disease (n = 67; 12 ill). In the remain-

Table 3. Comparison of Parameters of Vestibular Evoked Myogenic Potentials of Vestibular Neuritis Group and Control Group

Wave	Variable	Statistical Parameters	Control Group	Vestibular Neuritis	Mann-Whitney Test
p13	Ipsilateral	×	12.69	14.55	0.1194
		SD	2.53	3.49	
	Ipsilateral-amp	×	4.44	0.85	0.0005*
		SD	4.33	0.47	
	Contralateral	×	19.08	20.13	0.6059
		SD	4.26	3.51	
	Contralateral-amp	×	2.37	0.77	0.0004*
		SD	1.57	0.71	
n21	Ipsilateral	×	20.55	21.15	0.7163
		SD	3.22	4.89	
	Ipsilateral-amp	×	3.54	0.63	<.00005*
		SD	2.94	0.34	
	Contralateral	×	26.30	27.91	0.4814
		SD	4.72	4.41	
	Contralateral-amp	×	2.31	0.6	0.0003*
		SD	1.59	0.64	

amp = amplitude; SD = standard deviation; × = arithmetical average; * indicates a significant difference.
Note: Vestibular neuritis group, n = 37; control group, n = 121.

Table 4. Comparison of Parameters of Vestibular Evoked Myogenic Potentials in Ménière's Disease Patients and Control Group

Wave	Variable	Statistical Parameters	Control Group	Ill ears	Mann-Whitney Test
p13	Ipsilateral	×	12.69	11.74	0.0435*
		SD	2.53	2.19	
	Ipsilateral-amp	×	4.44	4.00	0.4024
		SD	4.33	4.52	
	Contralateral	×	19.08	16.24	0.0035*
		SD	4.26	4.88	
	Contralateral-amp	×	2.37	1.75	0.0088*
		SD	1.57	1.45	
n21	Ipsilateral	×	20.55	19.40	0.0438*
		SD	3.22	3.50	
	Ipsilateral-amp	×	3.54	3.47	0.1940
		SD	2.94	3.89	
	Contralateral	×	26.30	23.63	0.0211*
		SD	4.72	5.71	
	Contralateral-amp	×	2.31	1.86	0.0374*
		SD	1.59	1.56	

amp = amplitude; SD = standard deviation; × = arithmetical average; * indicates a significant difference.
Note: Ménière's disease, n = 55; control group, n = 121.

ing 82.5% of patients (55 ill), comparative statistical analysis of VEMPs parameters revealed a significant difference of the mean latency of p13–n21 in both ipsilateral and contralateral recordings after stimulation of affected ears of patients with Ménière's disease and the ears of the control group. The latency of p13–n21 waves was distinctly shorter than that in healthy ears if affected ears were stimulated. We found a similar mean amplitude of ipsilateral VEMPs on the affected and intact sides both in the patients and in subjects in the control group. The mean amplitude of contralateral VEMPs was significantly smaller after stimulation of affected ears. Comparison of VEMP parameters detected after stimulation of affected and intact ears is demonstrated in Table 4.

Patients with Vestibular Schwannoma

We observed abnormal VEMPs in all our patients (n = 7) with vestibular schwannoma. We found delayed or decreased VEMP responses after stimulation of affected ears, but most VEMPs were absent after stimulation of ears on the affected side. Findings depended on the size of the tumor. Only small, intracanal vestibular schwannoma that did no damage to all afferents of the vestibular division of the auditory nerve caused elongated latencies of p13–n21 and lower amplitudes. An example of audiometric findings, magnetic resonance imaging, and VEMPs is presented in Figure 2.

Treatment Monitoring

After treatment with cerebrum compositum, we observed changed values of latency and amplitude in all patients treated intramuscularly or orally. Mean latency of VEMPs after stimulation of affected ears before treatment equaled 14.9 ± 1.3 msec for p13 and 21.8 ± 1.7 msec for n21; after treatment, it equaled 12.7 ± 2.3 msec and 19.5 ± 3.3 msec, respectively. Mean amplitude of VEMPs after stimulation of affected ears before treatment equaled 9.31 ± 2.7 μV for p13 and 5.75 ± 3.4 μV for n21; after treatment, it equaled 10.9 ± 4.2 μV and 8.04 ± 2.3 μV, respectively. Similar shortened latencies and higher amplitudes of VEMPs after stopping treatment were noted after stimulation of intact ears. We did not observe these changes of VEMP parameters in those in the placebo group (n = 7). Figure 3 presents findings before and after treatment with certain drugs and with placebo.

DISCUSSION

This study presented VEMPs by use of repeated loud clicks that were recorded on SCMMs. VEMP tracings were registered after stimulation of almost all healthy ears (n = 121) with the exception of nine older patients. Muscle responses were composed of two successive positive and negative waves, labeled by their latencies in milliseconds preceded by a lowercase letter (p or n) according to their polarity. Lowercase letters are also used to distinguish them from neurally generated evoked potentials, which are marked by capital letters (P and N) [10]. Other authors marked these waves as p13–n23 [1,2,6] or p14–n21 [7].

VEMPs were characterized as short-latency's neck reflexes. The earliest muscle response appeared in 7.5 msec (latency of p13 wave), whereas the earliest that other authors had recorded it was 8.2 msec [2]. Values of the mean amplitude of VEMP tracings were lower in this

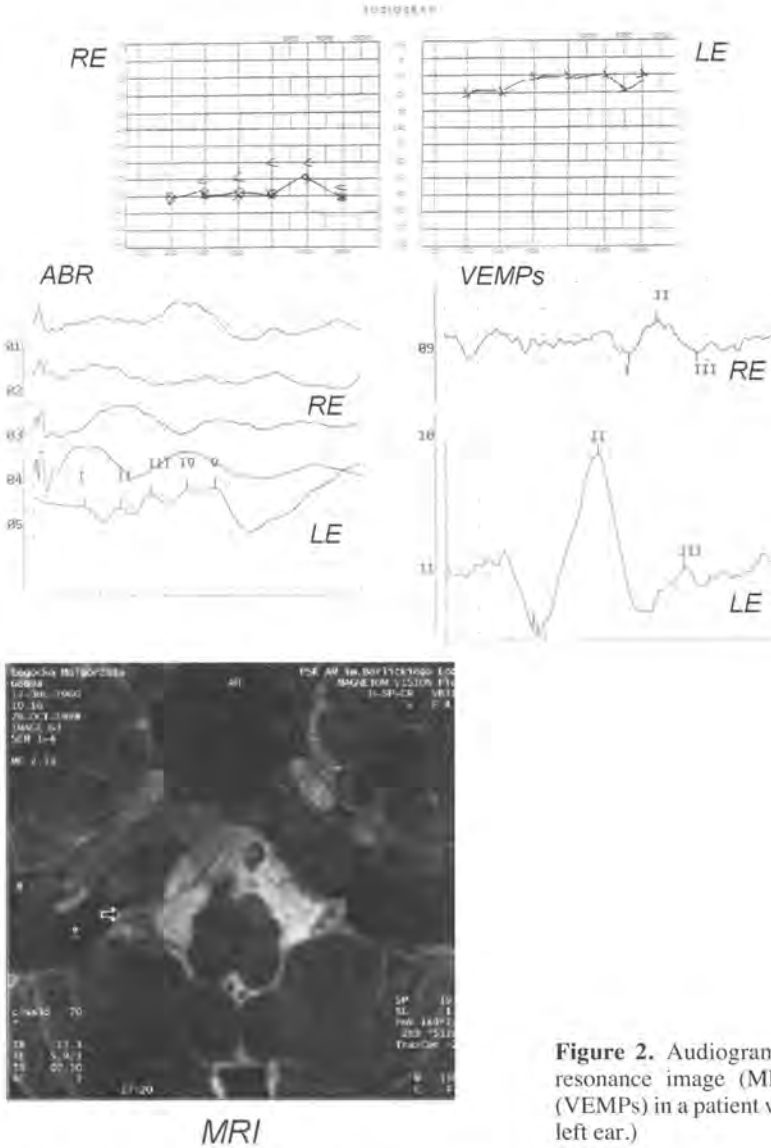


Figure 2. Audiogram, auditory brainstem response (ABR), magnetic resonance image (MRI), and vestibular evoked myogenic potentials (VEMPs) in a patient with vestibular schwannoma. (*RE* = right ear; *LE* = left ear.)

study than those described by some authors [2,7] and similar to those of VEMPs recorded on SCMMs by others [6]. Differences of amplitude values probably resulted from the other technique of examination, especially greater tonic activity of muscles.

VEMPs were received in both ipsilateral and contralateral recordings, but mean latency of p13–n21 was longer in contralateral than in ipsilateral recordings (significant difference). Mean amplitude was higher in ipsilateral recordings but without significant difference. Other authors either did not receive contralateral recordings [2] or observed symmetrical responses in ipsilateral and contralateral recordings [6], or higher amplitudes were placed in contralateral recordings [7]. Later responses on the opposite side to stimulation with similar amplitude may be explained by fewer fibers and more synapses of contralateral tracts [11,12].

VEMPs recordings were received from all patients after stimulation of totally deaf ears, and their mean latency and amplitude did not differ from parameters of VEMPs recorded after stimulation of ears in subjects in the control group. The fact that VEMP recordings might be noted after stimulation of ears that did not respond to clicks in the brainstem evoked auditory response test confirmed their vestibular origin [2]. Similar results in deaf patients were noted by others [2,7].

Normal parameters (latency, amplitude) of VEMPs were also observed after stimulation of ears in which SNHL had been diagnosed and that responded correctly or weakly to caloric stimuli. That finding confirms that response in VEMPs is not mediated by cochlear afferents as well. Other attempts [7] in patients with SNHL did not produce correlation between the degree of hearing loss and the amplitude of VEMPs.

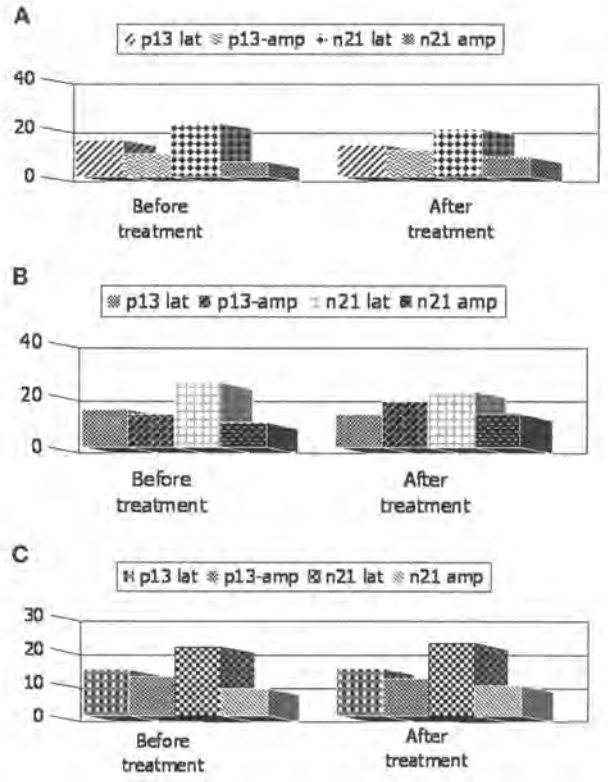


Figure 3. Results of treatment with cerebrum compositum and placebo. (A) Cerebrum compositum treatment of affected ears. (B) Cerebrum compositum treatment of intact ears. (C) Placebo.

Our own results revealed that findings of the caloric test and VEMPs were different. In most of the patients with weakness of excitability (58 ears), we found no statistically significant difference in the latencies and amplitudes of VEMP recordings after stimulation of affected ears and of intact ears of those in the control group. The absence of VEMPs or decreased amplitude of p13–n21 waves was detected in patients in whom excitability was absent (24 ears). It may result from damage to both otolithic and canal parts of the labyrinth or from vestibular division of cranial nerve VIII. Other authors [7] showed no correlation between findings of VEMPs and canal paresis.

Muscle responses on acoustic stimulation on the affected side were detected in 73.2% of patients suffering from vestibular neuritis. In those findings, we observed statistically significant lower amplitudes of VEMPs. Still lower amplitudes might be noted, for instance, in a disease process in which fewer saccular afferents are connected to the superior vestibular nerve. Some 17.9% of patients with vestibular neuritis had no reaction after stimulation of ears on their affected side. The lack of change of EMG activity after sound stimulation might reveal harm to the inferior vestibular nerve, while simultaneous abolition of vestibular excitability on caloric stimulus might demonstrate damage of both superior and inferior divisions of the vestibular nerve. Other authors [13] showed that VEMPs were absent in one-third of patients with vestibular neuritis and explained the abolition of VEMPs as the aftermath of inferior vestibular nerve lesion.

The initial biphasic p13–n21 evoked potential was absent in 12 patients with Ménière's disease. VEMPs were noted in 55 patients, but their parameters revealed a significant difference of mean latency of p13–n21 between findings received from affected ears and intact ears. A mean latency of p13–n21 waves was shorter after stimulation of ill inner ears than that in the ears of control group subjects. Differences of amplitude values (lower after stimulation of affected ears) were noted only in contralateral recordings.

This earlier appearance of changes of EMG activity after sound stimulation of an ill inner ear in Ménière's disease may be explained as a vestibular recruitment phenomenon that meets in inner-ear lesions [14]. Some authors [15] detected the absence of biphasic p13–n21 evoked potential in 54% of patients with Ménière's disease. They explained that endolymphatic hydrops may affect the response of the saccule to a click.

We observed abnormal VEMPs in all our patients with vestibular schwannoma. Delayed or decreased VEMP responses were found after stimulation of the affected ear. Most VEMPs were absent (in 8 of 10 patients). Other authors [16] showed that 80% of patients had abnormal VEMPs with surgically confirmed acoustic neuroma. Sometimes, VEMPs and a caloric test could classify acoustic neuromas according to the involved nerves—inferior or superior vestibular nerve [16]. Detecting VEMPs seems to be especially beneficial when auditory brainstem response is absent and caloric reaction is decreased. If VEMPs are absent in such cases, magnetic resonance imaging absolutely should be obtained. However, the presence of normal VEMPs in this case may show that vestibular nerve division is complete.

In the group treated with cerebrum compositum, we noted shortened latencies and higher amplitudes of VEMPs after stopping treatment (in most cases). These observations applied to both ill and healthy ears. In the patient group that received a placebo, the VEMP test produced ambiguous results. Sometimes, we observed elongated latencies and higher or lower amplitudes of VEMPs after placebo. Mean latency and mean amplitude did not differ before and after the use of placebo. Changes of VEMP parameters obtained after treatment with cerebrum compositum may be evidence that the agent improves the action of vestibulospinal reflex [3]. In this antihomotoxic remedy, the most important component is cocculus. This ingredient, known as *picrotoxin*, stimulates a broad section of the central nervous

system, including the brainstem, because it is a typical antagonist of the inhibitory neurotransmitter GABA. Picrotoxin blocks a chlorine ion channel opened by GABA, so restraining gabaminergic loops reduces a certain part of the inhibition of the brainstem, and the brainstem activity is increased [17,18]. Another author considers GABA as a neurotransmitter in vestibular hair cells [19]. Still others [20], after estimating the influence of betahistine on the vestibular evoked potentials (VEPs), observed that the most relevant VEP parameter is the latency of the waves. During the betahistine trial, these authors noted that after ear stimulation of patients with peripheral vestibular disorders, latencies became progressively shorter and approached the normal levels in recordings. In healthy ear cases and in the group receiving placebo, latencies became progressively longer at each recording session, which authors explained as a phenomenon of progressive fatigue. Other authors [21] investigated the possibility of blockade of afferent nervous fibers in the chicken by tetrodotoxin (TTX) and the use of VEPs as a method of following the influence of chemical remedies on the vestibular organ. TTX cancelled VEPs for 12–24 hours and, after 12 hours, elongated latencies of VEPs as a reaction to pulsate linear acceleration were observed. After 24 hours, VEPs returned to normal values [21].

CONCLUSIONS

Based on the finding of normal VEMP recordings in patients with total deafness or with SNHL, higher-amplitude VEMPs appear to be a result of vestibular organ stimulation. VEMPs seem to be an aftermath of saccule receptor activation because of unchanged VEMP parameters in canal paresis cases. VEMPs are a simple, highly objective, and supplemental technique for assessing the vestibulospinal connections simultaneously with inner-ear or vestibular nerve lesions, because of changes of VEMP parameters or absence of responses in patients with vestibular neuritis, Ménière's disease, or vestibular schwannoma. The use of VEMPs allows the tracing of reflex reactions after treatment.

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