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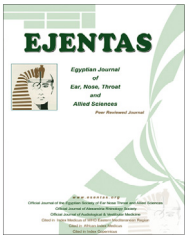

ORIGINAL ARTICLE

Role of cervical vestibular-evoked myogenic potentials testing in vestibular migraine

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KEYWORDS

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Abstract *Background:* Vestibular dysfunction has been long described in patients with migraine; this relation has been addressed as vestibular migraine. The pathophysiology as well as the peripheral or central localization of this deficit is unclear. Cervical vestibular evoked myogenic potential (cVEMP) is a validated method to test saccular function and vestibulocollic pathway.

Objectives: The current work was designed to assess the characteristics of cVEMP response in patients with vestibular migraine and compare them with the results of healthy controls, and to find out if the cVEMP could be useful as a complementary tool for testing vestibular function in vestibular migraine.

Methods: Twenty five patients with definite vestibular migraine were involved as a study group. Twenty healthy volunteers of comparable age and sex were taken as a control group. The amplitude and latency of cVEMP were measured. Electronystagmography (ENG) test battery including caloric testing was done.

Results: Our study demonstrated significant reduction in cVEMP amplitudes, and more frequently absent response in patients with vestibular migraine compared to healthy controls. There was no correlation between cVEMP amplitudes and caloric testing. ENG tests showed peripheral vestibular lesion in 36% of patients, central lesion in 16%, mixed lesion in 4%.

Conclusion: cVEMP is a useful complementary tool for testing vestibular function in vestibular migraine. Reduced cVEMP amplitude or absent response were the most frequent features in vestibular migraine. The saccule and or the sacculo-collic pathway are affected in vestibular migraine, with more tendencies for peripheral vestibular dysfunction in our patient group.

1. Introduction

Migraine has long been recognized to be associated with several vestibular syndromes, and sometimes vertigo is the cardinal symptom. This type of migraine is called vestibular

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migraine (VM).[1,2] Vestibular migraine has gained recognition as a distinct clinical entity in recent years. VM has a 6–10% incidence, and is one of the most common diagnoses in dizziness units, but it still not included in the International Headache Society (IHS) criteria as a subgroup of migraine.[3] Neuhauser et al. have proposed operational diagnostic criteria for vestibular migraine.[4,5] The vestibular symptoms in VM can occur before the migraine headache in a similar pattern as an aura phenomenon, during the headache, or completely independent of the headache. Many also report a chronic subtle feeling of vertigo and disequilibrium all the time, with exacerbations during migraine attacks.[6] The pathophysiology of the VM is unclear, it could be localized anywhere in the vestibular system. Previous studies of VM describe a variety of abnormal vestibular findings, some indicating dysfunction in the central vestibular system, and others in the peripheral vestibular system.[7–9] The sensation of vertigo in VM patients may arise from a decreased or an increased activation of the vestibular system, either peripheral, or central, generating a tonic imbalance between these structures.[6]

Functional abnormalities of the vestibular system in vestibular migraine were tested in different studies; recently it was evaluated by the cervical vestibular evoked myogenic potential (cVEMP).[6,10–12] cVEMP is a short-latency myogenic response which is evoked by sound and recorded from neck muscles. The currently understood cVEMP was first described by Colebatch and Halmagyi (1992), and Colebatch et al. (1994).[13,14] Since then, cVEMP have become a standard clinical test of otolith (predominantly saccular) function. The conventional method for recording the cVEMP involves measuring electromyographic activity from surface electrodes placed over the tonically-activated sternocleidomastoid muscles. The cVEMP is thus a manifestation of the vestibulo-collic reflex. However, recent research has shown that VEMP can also be recorded from the extraocular muscles, using surface electrodes placed near the eyes. These ocular VEMP (oVEMP) are a manifestation of the vestibule-ocular reflex.[15] cVEMP have been applied in cases of peripheral vestibular diseases such as Meniere's disease, vestibular neuritis, vestibular schwannomas, and superior semicircular canal dehiscence.[16–20] However studies on disorders of central vestibular origins are not only rare, they are also generally inconclusive.[21–23]

There is a growing amount of interest in cVEMP studies on migraine in the recent years with different results. Liao and Young (2004), and Boldingh et al. (2011) have reported absent or delayed cVEMP,[12,16] whereas Baier and Dieterich (2009), Allena et al. (2007) and Roceanu et al. (2008) have found cVEMP of normal latency but, reduced amplitude.[11,10,24] Kandemir et al. (2012) recorded normal values of p13, n23 latencies, and amplitudes.[25]

The aim of the present study is to assess characteristics of cVEMP response in patients with vestibular migraine and compare them with the results of healthy controls, and to find out if the cVEMP could be useful as a complementary tool for testing vestibular function in vestibular migraine.

2. Methods

The study was conducted in the audiology clinic, Assiut University, Egypt from 2013 to 2014. The study protocol was approved by the local ethics committee. Informed consent was obtained from all participants. Twenty five patients with VM (14 female and 11 male; mean age 24.6 ± 6.75 with an age range of 19–40 years) were diagnosed to have definite VM according to the criteria of Neuhauser and colleagues[4,5]: (1) Recurrent episodic vestibular symptoms of at least moderate intensity. (2) Current or previous history of migraine according to criteria of the International Headache Society.[26] (3) One of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras. (4) Other causes ruled out by appropriate investigations. Triptans, antiepileptic drugs and vestibular suppressant drugs were stopped 1 week before testing. The mean disease duration was 1.65 years. In all patients, vertigo attacks were characterized by spinning sensation, 72% had positional symptoms, and 16% had unsteadiness. In 9 (36%) patients, vertigo attacks associated with headaches, in six (24%) patients, vertigo occurred after the headache; in four (16%) it preceded the headache. In six (24%) vertigo attacks not related to headaches and sometimes associated with headaches. Headache was hemicranial in 15 patients, holocranial (affecting the whole head) in seven patients and in three patients it may be hemicranial and sometimes holocranial. Nausea, and or vomiting were reported to be present in 19 patients (76%) during the vertigo attacks or headaches. Hearing loss, middle ear pathology and other vestibular disorders were exclusion criteria.

The control group included age and sex matched 20 healthy volunteers (14 female and 6 male, mean ages 23.4 ± 5.0) with no history of vestibular symptoms or migraine. Comparing age and gender between study and control groups revealing no significant difference, $P = 0.086$ for age; $P = 0.258$ for sex). All subjects were submitted to full history taking, otological examination, and neurological examination, which are free in all subjects. Audiological evaluation was performed for both groups to ensure normal hearing sensitivity. Two channel audiometer Madsen model, Orbiter 922 and interacoustics AZ 26 tympanometer were used.

2.1. cVEMP recording

Testing of cVEMP was recorded by interacoustics, EP 15 version 3.03; it was done in the interictal period (absence of migraine headache and vertigo attacks for at least, the last two days). VEMP test was recorded in all subjects while turning their head to the opposite side to activate the sternocleidomastoid (SCM) muscle. The electromyography was recorded with surface electrodes mounted in the middle third of SCM on the tested side. The reference electrode was placed on the ipsilateral mastoid. Tone burst of 500 Hz was presented to the tested ear at intensity of 100 dB nHL with 5.1/s. stimulus rate. Four millisecond (ms) rise/fall time was used with a plateau time of 2.0 ms. The analysis time was 80 ms and the filter setting was 10–750 Hz. The latencies of peaks p13 and n23 and peak to peak amplitude were measured. Since the amplitude of the p13–n23 response is largely determined by the level of tonic SCM contraction,[27] the EMG activities were monitored during recording, and subjects received a visual feedback so as to maintain muscle activities at constant level. To normalize the raw VEMP magnitudes, an EMG scaling was done by dividing the raw amplitude for each recording on the mean rectified EMG activity from the pre-stimulus period. To determine

the relation of the amplitudes of both sides in one patient, an asymmetry ratio (AR) was calculated using the following formula: AR% = $100(A_l - A_s)/(A_l + A_s)$, Where A_l and A_s are the larger and smaller, amplitudes obtained from stimulating each ear.[19]

2.2. ENG testing

A full ENG test battery was done by Micromedical tech. (Meta 4 version 8.1), using monopolar electrode montage, and it included a search for nystagmus both with and without fixation, with horizontal and vertical gaze deviation, and with a post-head shaking test. Also, nystagmus was studied using both the Dix–Hallpike test (positioning test) for the right and left side, and positional tests including the supine, head right and head left, and right lateral and left lateral positions. An assessment of saccades, smooth pursuit, and optokinetic nystagmus was also done. In addition, caloric function was studied with the use of alternate bithermal caloric irrigation with water at 30 °C and 44 °C. A difference of 20% was considered unilateral caloric weakness and the magnitude of a nystagmus of a slow-component velocity of 6 degree/s was considered significant during positional, and post-head shake testing, or nystagmus tests.

Data collected, and analyzed by means of statistical software package for social sciences (SPSS) version 21. Data expressed as mean, Standard deviation, number, and percentage. Mann whitney test was used to determine significance for numeric variable. Chi Square was used to determine significance for categorical variable. Spearman Rank correlation test was used for correlations between groups. A difference was considered to be statistically significant (*) when the probability (P) value was $\leqslant 0.05$.

3. Results

Patients with vestibular migraine showed significant lower p13–23 mean amplitudes on both sides compared to controls ($P < 0.05$ for each side) (Table 1). Sixteen patients with vestibular migraine (64%) had significantly reduced VEMP amplitude; lower than the lower limit of the 95% CI (confidence interval) of the controls. Four of them (16%) had reduced amplitude on the right, four (16%) in the left and eight (32%) had reduced amplitudes bilaterally. VEMP response were present in all normal subjects but six of the 25 patients (24%) with vestibular migraine had absent VEMP response, four of them (16%) had no waveform on the right side, one patient (4%) had no waveform on the left side, and one patient (4%) had no waveform bilaterally. Collectively, 19 (76%) of the 25 patients showed abnormal VEMP response (either reduced amplitude, or absent response, or both).

Studying the relation of amplitudes of both sides, the asymmetry ratio compared between migraine patients, and controls showed a statistically significant difference ($P = 0.005$) (Table 1), which means a reduced amplitude in one side than the other in patients with vestibular migraine (Fig. 1). The asymmetry ratio could not be recorded in six patients because of absent waveform either unilaterally or bilaterally. It was recorded in 19 patients, and in 14 of them (74%), it was more than the upper limit of the 95% CI (0.108) of the controls.

With regard to latencies, a mean p13 of 16.94 ms (SD ± 2.15) and an n23 of 26.5 ms (SD ± 2.89) were calculated for the right side in patients with vestibular migraine. On the left side, they had a mean of 17.69 ms (SD ± 3.0) and 27.26 ms (SD ± 3.95) for n23. In the control group, the latency for p13 on the right side was 16.79 ms (SD ± 2.92) and on the left side 16.23 ms (SD ± 2.29). The latency for n23 on the right was 25.43 ms (SD ± 3.49) and 24.9 ms (SD ± 4.3) on the left. There was neither a group difference of the latencies for the p13 nor the n23 on both sides (p13 right side $P = 0.854$, left side $P = 0.084$, n23 right side $P = 0.301$, left side $P = 0.068$) (Fig. 2). However six patients (24%) had more prolonged latency than the upper limit of 95% CI of the control group.

In patients with vestibular migraine, 16 out of 25 participants (64%) had abnormalities in at least one of the ENG tests (Table 2). Positioning and positional tests revealed significant nystagmus in 52% of patients, which included direction fixed or geotropic direction changing nystagmus and two patients only showed a geotropic direction changing nystagmus. The second abnormality was presence of unilateral caloric weakness (20%) that is followed by abnormal post head-shaking (16%). In addition 12% had abnormal saccade, 12% had abnormal pursuit, and 8% had abnormal OPK. There was no evidence of spontaneous, or gaze nystagmus in the study group. ENG test results in the control group showed no significant nystagmus, symmetrical caloric response, and normal oculomotor test.

To test an association between the cVEMP amplitude, and the caloric function, Spearman Rank correlation test was performed, showing no significant correlation on the right or left side (right side $r = 0.022$; $p = 0.894$, left side $r = 0.219$; $p = 0.157$).

From ENG results, peripheral vestibular system lesion (up to the root entry zone) was diagnosed in 36% of patients, central lesion in 16%, mixed lesion in 4%, and non-localizing in 8%.

Table 1 VEMP amplitude and threshold in vestibular migraine patients and controls.

	Patients		Controls		95% CI		*P*-value
	Mean	SD	Mean	SD	Lower limit	Upper limit	
p13 n23 Amplitude right (ms)	0.82	0.41	1.17	0.72	0.84	1.50	0.046*
p13 n23 Amplitude left (ms)	0.81	0.47	1.18	0.66	0.86	1.49	0.044*
Asymmetry ratio	0.16	0.11	0.079	0.06	0.049	0.108	0.005*

CI: confidence interval.
* P value $\leqslant 0.05$.

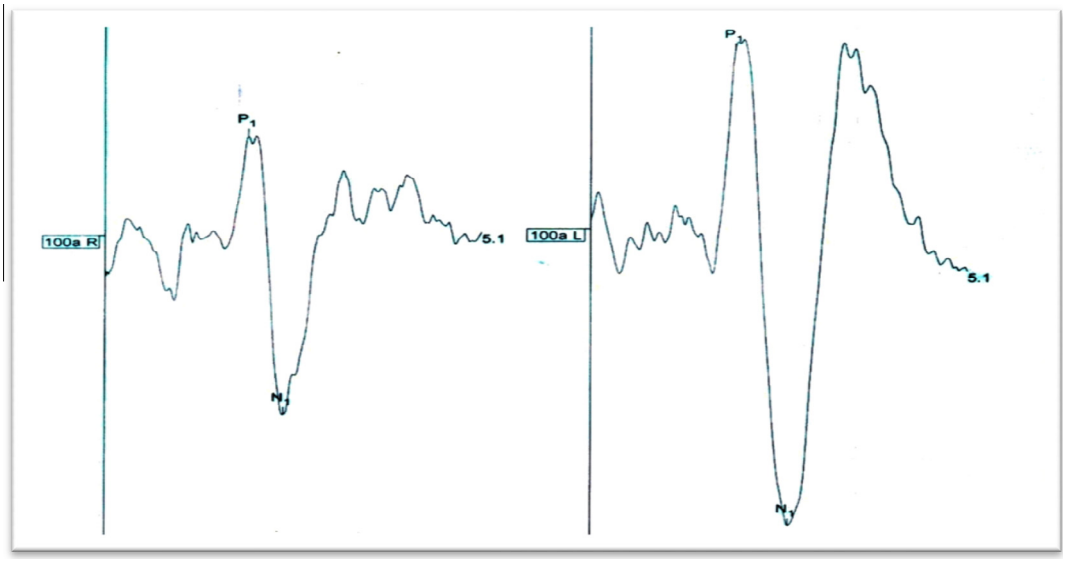

Figure 1 cVEMP recorded from a patient with vestibular migraine from the right and left sides. The corrected amplitude in the right side is reduced in comparison to the left side, the asymmetry ratio is 0.26.

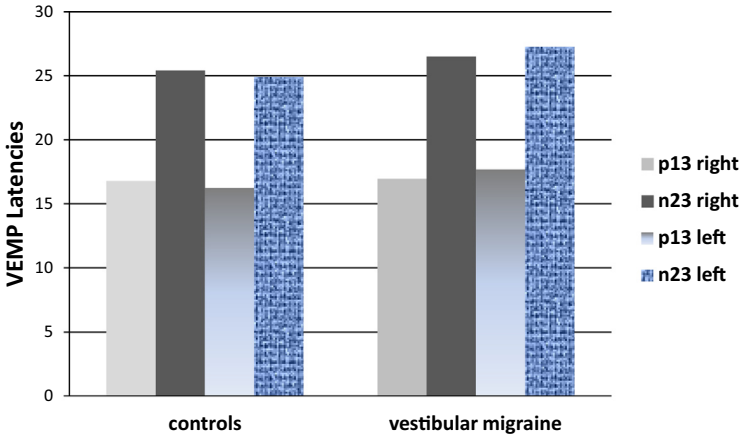

Figure 2 VEMP latencies (mean and standard deviation) of the p13 and n23 in patients with vestibular migraine compared to controls. No significant difference was seen.

Table 2 ENG findings in patients with vestibular migraine.

ENG tests	Abnormal (no)	(%)
Spontaneous nystagmus test	0	(0)
Gaze nystagmus test	0	(0)
Post head shaking test	4	(16)
Positioning & positional test	13	(52)
Unilateral caloric weakness	5	(20)
Saccade test	3	(12)
Pursuit test	3	(12)
Optokinetic test	2	(8)

ENG: electronystagmography.

4. Discussion

Cervical VEMP was successfully recorded from all control groups emphasizing that this potential is a reliable addition to the vestibular testing battery. This agrees with Welgampola and Colebatch, who reported presence of VEMP response in all normal subjects under the age of 60. After the six decade the amplitude of the reflex decreases progressively by 25–30% per decade.[28]

In this study, 24% of patients with vestibular migraine showed absent cVEMP response. Our results are in line with Boldingh et al. who reported that absent cVEMP response was more frequent among vestibular migraine patients (44%) and patients suffering from ordinary migraine (25%) than in healthy controls (3%).[6] The authors suggest that the absent response indicates a vestibulocollic dysfunction in vestibular migraine. Vitkovic et al. also addressed absent cVEMP response in 19.2% in patients with migraine related dizziness.[7] We found in the present study that 64% patients with VM showed reduced cVEMP amplitudes compared to those of sex and age matched controls. These results agree with Baier and Dieterich who reported that 68% of patients with VM showed reduced cVEMP amplitude and referred these results to saccular or saccular pathway dysfunction in patients with

VM.[11] The reduced cVEMP amplitude was also addressed by Allena et al. in patients with migraine; they suggest that this change was due to reduced serotonergic control of the saccular reflex pathways in the brainstem.[10] However other studies as Shalaby et al. reported no significant difference as regards p13–n23 amplitude between patients with VM and controls.[29] Alpini et al. attributed the variability in amplitude results between studies to a wide range of absolute amplitude measurements which depends greatly on the EMG level of sternomastoid contraction.[30] To overcome this, corrected amplitudes and asymmetry ratio were used in our study. To correct the amplitudes, the raw amplitudes were divided by the mean rectified EMG activity from the prestimulus period to correct for differences in muscle activation. Side to side differences in corrected reflex amplitudes were expressed as an asymmetry ratio (AR), it has been suggested that the most cautious interpretation of the potentials seems to be their ability to identify the asymmetry between left and right sides and therefore to suggest the likely side of the pathology.[31] The marked intersubject variability of absolute amplitude values is diminished by using this relative amplitude measurement. In the present study the AR of the control group subjects differ significantly from that of VM patients (Table 1). Iwasaki et al. reported that AR of amplitude might be a good reflectance of unilateral malfunction than the raw values.[32]

There was neither a group difference of the latencies for the p13 nor the n23 on both sides between VM patients and healthy controls. This agrees with other studies as Baier and Dieterich (2009) and Allena et al. (2007),[11,10] although, some studies found significant delay in p13 and n23 latencies in VM patients without amplitude reduction.[29,12] Most of the previous studies addressed cVEMP abnormalities in VM patients, either prolonged latency, or reduced amplitude, others reported absence of the response.[6,7,10–12,29] However, more recent work by Taylor et al. and Kandemir et al. reported no abnormalities in cVEMP response.[33,25] Therefore, the findings in the literature with regards to VM and cVEMP do not appear to be homogenous. This is because migraine is not a single disease entity and its pathophysiological mechanisms are areas for intense researches.

Current concept considered the migraine as an inherited vulnerability to a hyperexcitable cerebral cortex. Various stimuli may trigger episodes of cortical spreading depression, which in turn, initiates the process of localized neurogenic inflammation and sensitization of both the peripheral and central afferent circuitry, leading to migraine aura and activation of the trigeminovascular system.[34] Since the vestibular nuclei receive noradrenergic fibers from the locus coeruleus and serotonergic afferents from the dorsal raphe nucleus, it is likely that activation of this pathway during a migraine episode activates the central vestibular processing.[35] Dieterich and Brandt suggested that the processes in patients with vestibular migraine seem to affect both brainstem, and peripheral vestibular structures.[36] Celebisoy et al. reported higher incidence of peripheral vestibular dysfunction than central.[37] The peripheral vestibular dysfunction may be related to hypoperfusion-induced ischemia of the labyrinthine structures, or serotonergic induced extravasation.[11]

It was suggested from literature with regards to vestibular electropathophysiology, that cVEMP latencies were delayed in brainstem disorders, but not in peripheral dysfunction, this were attributed to slowing of conduction along the vestibulospinal pathways consequent to demyelination. While the reduction in amplitudes depends on peripheral vestibular dysfunction.[38] The significant reduction in cVEMP amplitudes, raised asymmetry ratio, and unaffected latencies are likely to suggest tendency for peripheral vestibular dysfunction in our patients group.

ENG is an important vestibular function test used to study site of lesion. A full test battery was done, 64% of vestibular migraine patients showed abnormality in at least one of the ENG tests. Other studies on vestibular migraine patients reported ENG findings in 55%, 73%, 75.6% of their patients groups.[39,40,9] Positional nystagmus was the most frequent finding in VM patients, it was recorded in 52% of the study group, this result agreed with Hazzaa et al.[41] who reported that positional nystagmus was the commonest finding and was recorded in 60% of VM subjects. In our study group, 20% showed unilateral caloric weakness. Our results consisted with other studies which reported unilateral caloric abnormalities in 20%, 25%, 20.8%.[42,11,25] However, in a study performed by Taylor et al.[33] caloric profile was normal. On the other hand, correlation analysis showed that the results of the caloric tests were not correlated with cVEMP amplitudes, this result agreed with Kandemir et al. and Baier and Dieterich.[25,11] The lack of correlation between Caloric test, and cVEMP results, because they assess different function of the vestibular system, with different vulnerability of the labyrinthine structures for ischemia, or serotonergic induced extravastion.[22,11]

According to ENG, peripheral vestibular system lesion was diagnosed in 36% of patients, central lesion in 16%, mixed lesion in 4%, and non-localizing in 8%. These results also suggest tendency for more affection of the peripheral vestibular system in our patients group.

The wide variation in results of cVEMP parameter study and localization of site of lesion in patients with vestibular migraine reflect that vestibular migraine is a heterogenous entity. It can affect peripheral, and or central vestibular system.

In conclusion, our study demonstrated reduction in cVEMP amplitudes, absent response, and insignificant shift of latencies in patients with vestibular migraine compared to healthy controls. The saccule, and or the sacculo-collic pathway are affected, with more tendencies for peripheral vestibular dysfunction in our patient group. cVEMP could be a useful complementary tool for testing vestibular function in vestibular migraine patients.

4.1. Recommendations

We advice to do vestibular rehabilitation therapy (VRT) for saccular dysfunction in VM patients with impaired VEMP response. Further study, comparing the effect of VRT for saccular dysfunction in VM patients, with and without impaired VEMP response is recommended.

Financial disclosures

No.

Conflict of interest

None.

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