



Cervical vestibular-evoked myogenic potentials in patients with multiple sclerosis: sensitive in detecting brainstem involvement?

Gökçe Kavasoglu¹ · Figen Gökçay¹ · Nur Yüceyar¹ · Neşe Çelebisoy¹

Received: 21 March 2017 / Accepted: 2 December 2017 / Published online: 15 December 2017
© Springer-Verlag Italia S.r.l., part of Springer Nature 2017

Abstract

Cervical vestibular-evoked myogenic potentials (cVEMPs) are accepted to demonstrate the vestibulo-collic reflex. However, the brainstem pathway is still not fully understood. The aim of the study was to evaluate the contribution of cVEMPs to detection of brainstem involvement in multiple sclerosis (MS). Thirty patients fulfilling the criteria for definite MS were included in the study. All were newly diagnosed cases, admitted due to an attack with active lesions on MRI. Thirty-one age- and sex-matched healthy controls constituted the control group. The latencies of peaks p13 and n23 and peak-to-peak amplitude of p13–n23 were measured. Brainstem lesions on MRI were present in 13 of the patients (43.4%). Comparison of the overall results recorded from patients with the healthy controls did not reveal a statistically significant difference in any of the parameters studied ($p > 0.05$). A significant inter-side difference was not also present between groups ($p > 0.05$). When p13 and n23 latencies exceeding 2.5 standard deviations (SD) were taken into consideration, it was seen that there were seven patients (23.3%) with prolonged latencies mainly involving the p13 peak. Five of them had brainstem signs on examination and had brainstem lesions on MRI. In the other eight patients with abnormal MRI, normal results were recorded indicating that in only 38% of patients with brainstem lesions, cVEMPs were altered. Absence of a correlation between cVEMPs and brainstem clinical or MRI lesions defies their role in identifying lower brainstem involvement.

Keywords Cervical vestibular-evoked myogenic potentials · Vestibulo-collic reflex · Multiple sclerosis

Vestibular-evoked myogenic potentials (VEMPs) are used to assess the otolithic pathways [1–8]. Cervical VEMPs (cVEMPs) are inhibitory electrical potentials generated after a sound stimulus (clicks or pure tones), originated in the sacule, and conducted by the lower portion of the vestibular nerve all the way to the central nervous system (CNS), generating inhibitory electrical responses picked up by electrodes placed on the sternocleidomastoid muscle (SCM) and demonstrate the vestibulo-collic reflex [2].

VEMPs have particularly been studied in the differential diagnosis of peripheral vestibular lesions involving vestibular neuritis, benign paroxysmal positional vertigo, Meniere's disease [9], bilateral vestibulopathy, or vestibular schwannomas [3]. Although it is known that lesions of the brainstem also

lead to VEMP alterations [10], the brainstem pathway of VEMPs is still not fully understood. Previous studies involving circumscribed brainstem lesions [10, 11] have shown that VEMPs may be a useful diagnostic tool to identify lower brainstem lesions especially in the lateral lower pons and the upper medulla oblongata. However, rostral brainstem infarctions up to the mesencephalon have also been reported to impair cVEMPs suggesting descending modulatory pathways for cVEMPs in the brainstem [12].

The aim of the study was to evaluate the contribution of VEMPs to detection of brainstem involvement in multiple sclerosis (MS).

Material and methods

Thirty patients fulfilling the criteria for definite MS according to the revised McDonald's criteria [13] with normal audiometric testing and no previous history of a peripheral vestibular disorder were included in the study. Thirty-one age- and sex-matched healthy controls constituted the control group.

✉ Neşe Çelebisoy
nese.celebisoy@ege.edu.tr

¹ Department of Neurology, Ege University Medical School, Bornova, 35100 Izmir, Turkey

Approval from the ethics committee of Ege University Medical School was obtained, and all the patients gave their written informed consent for the procedure. Brain MRI scans were performed in all patients during the study to check the presence of brainstem lesions.

cVEMPs were recorded by using a Synergy device (Medelec; Oxford Instruments Medical Inc., UK). To record the surface EMG activity, an active electrode was placed on the upper half of the sternocleidomastoid muscle (SCM) ipsilateral to the stimulation, with the reference electrode placed on the upper third of sternum and the ground electrode on the middle of the forehead. Patients were seated on an armchair and were asked to turn their head contralaterally to the ear being tested to achieve maximal activation of the SCM. Two stimulation sequences consisting of 100 sound stimuli were given. The acoustic stimuli were clicks at an intensity of 100 dBnHL (normal hearing level) of 0.1-ms duration, delivered at a frequency of 5 Hz through a headphone unilaterally to each ear. The EMG signal was bandpass filtered from 10 to 1000 Hz and averaged during a 100-ms interval. The amplifier gain of the recording system was 2000. Initial positive/negative polarity of the waveform with peaks was termed p13 and n23 on the basis of respective latencies. The latencies of peaks p13 and n23 and peak-to-peak amplitude of p13–n23 were measured. To achieve independence from the level of background activation, the amplitude of the VEMPs was expressed as the ratio of peak-to-peak amplitude divided by a mean prestimulus-rectified EMG measured during the recording [3].

Inter-side differences, defined as the ratio between the differences of the corresponding parameters measured from the right and the left SCM and the mean of these two values, were also studied.

SPSS 23 (IBM Corporation, Armonk, New York; USA) was used for the statistical analyses. Regarding numerical data conforming normal distribution, arithmetic mean, standard deviation, and 95% confidence intervals (CIs) were used. Independent samples *T* test was used to compare means. Mann–Whitney *U* test was used to compare inter-side differences of the patients with the healthy controls that did not confirm normality. Significance was assumed at $p = 0.05$.

Results

The MS group consisted of 18 women and 12 men with a mean age of 30 years (range 18–45 years). There were 19 women and 12 men with a mean age of 30 years (range 19–48 years) within the control group. All the MS patients were newly diagnosed cases defining neurological symptoms beginning within the last year, admitted due to an attack with active lesions on MRI. On examination, brainstem signs were present in eight patients (26.7%), ataxia in three, internuclear

ophthalmoplegia in two, facial hypoesthesia on one side in two, and gaze-evoked nystagmus in one.

Brain MRI scans performed during the study showed that brainstem lesions were present in 13 of the patients (43.4%).

cVEMPs were recorded from both sides in all healthy subjects and patients. For the healthy controls, right-sided p13 and n23 latencies and amplitude ratios were 12 ± 0.9 ms, 20.6 ± 1.95 ms, and 7.7 ± 3.1 , respectively. These figures were 12.1 ± 0.9 ms, 20.76 ± 1.6 ms, and 7.6 ± 3.0 for the left side. In MS patients, right-sided p13 and n23 latencies and amplitude ratios were 12.9 ± 2.1 ms, 21.2 ± 1.78 ms, and 7.1 ± 2.8 , respectively. These figures were 12.9 ± 2.2 ms, 21.4 ± 2.1 ms, and 7.0 ± 2.5 for the left side (Table 1, Figs. 1 and 2). When the results recorded from patients with MS were compared with the results of the healthy controls, statistically significant difference could not be noted in any of the parameters taken into consideration ($p > 0.05$). Inter-side difference for p13 and n23 latencies and amplitude ratios of the patients and healthy controls is given in Table 2. No significant difference between groups was present regarding any of the parameters ($p > 0.05$).

However, as the *p* values gathered by comparison of the p13 latencies of the healthy controls and MS patients were 0.052 for the right and 0.057 for the left side, latencies exceeding 2.5 standard deviations (SD) were also studied. There were seven patients (23.3%) with prolonged latencies (bilateral in four and unilateral in three). It was interesting that n23 latencies were prolonged in just two of these seven patients with prolonged p13 latencies, one unilaterally and one bilaterally. None of the healthy controls had p13 and n23 latencies exceeding 2.5 SD.

In seven patients with prolonged latencies, five were admitted with signs of brainstem involvement on examination (two ataxia, two facial hypoesthesia, and one gaze-evoked nystagmus) and had brainstem lesions on MRI (unilateral inferior cerebellar peduncle in two, bilateral pons and medulla in one, unilateral pons in one, and unilateral medulla in one). Figure 3 shows bilateral delayed p13 and n23 potentials in a patient with left pontine lesion. The other two patients with prolonged latencies did not have brainstem lesions on MRI. One had optic neuritis and the other had paraparesis.

On the other hand, in eight patients with brainstem lesions on MRI, cVEMPs were normal indicating that cVEMP abnormality in patients with brainstem lesions was 38.5% (5/13). In three of the eight patients with abnormal MRI and normal cVEMP, brainstem signs were present on examination; two had internuclear ophthalmoplegia and one had ataxia. Figure 4 shows normal cVEMP responses gathered from a patient with multiple brainstem lesions on MRI. Table 3 summarizes the clinical features and the VEMP results of patients with brainstem lesions on MRI.

Comparison of the p13 and n23 latencies and amplitude ratios recorded from patients with and without brainstem

Table 1 p13 and n23 potential latencies and amplitudes recorded from the healthy controls and the patients

	p13 latency (ms)		n23 latency (ms)		Amplitude	
	Right	Left	Right	Left	Right	Left
HC	12 ± 0.9	12.03 ± 0.96	20.6 ± 1.95	20.76 ± 1.63	7.7 ± 3.1	7.6 ± 3.0
MS	12.9 ± 2.1	12.9 ± 2.2	21.2 ± 1.78	21.4 ± 2.1	7.1 ± 2.8	7.0 ± 2.5
<i>p</i>	0.052	0.057	0.17	0.23	0.39	0.38

HC healthy controls, MS multiple sclerosis, *p* *p* values gathered by comparison of the healthy controls with MS patients

lesions on MRI did not reveal a significant difference (*p* > 0.05) (Table 4).

Discussion

There are several studies on VEMPs in multiple sclerosis [14–25]. The diagnostic sensitivity of the test in these studies varies between 18 and 70%. Sartucci and Logi [14] have found delayed p13 latency in 53.3% of their MS patients. Versino et al. [15] have reported abnormal VEMPs in 31% of their MS patients and in 11.8% abnormality concerned latencies. Reduction of p13–n23 amplitude was the main abnormality. Bandini et al. [16] studied patients with and without brainstem involvement and reported delayed p13 latencies in 62% and delayed n23 latencies in 31% of their patients with brainstem involvement. These figures were 25 and 1%, respectively, in patients with normal brainstem on MRI. They have reported that VEMPs are able to detect brainstem

dysfunction in MS patients with normal MRI. They have also reported a good correlation of p13 latency with the clinical severity of the disease. Alpini et al. [17] have reported abnormal VEMPs in 70% of their patients. Patko et al. [18] have found absent responses, longer latencies, and lower amplitudes in their MS patients significantly related with brainstem lesions. Eleftheriadou et al. [19] have reported p13n23 abnormality in 50% of their patients; in 32%, no brainstem lesions were present on MRI. Gazioğlu and Boz [20] have studied both ocular and cervical VEMPs and have found that n1 latency of ocular vestibular-evoked myogenic potentials (oVEMPs) and p1 latency of cVEMPs were prolonged in MS patients and were significantly correlated with Expanded Disability Status Scale. However, the correlation with brainstem clinical or MRI lesions was not significant. An abnormality rate of 18% has been reported for cVEMPs. Ivankovic et al. [21] reported oVEMP abnormality in 37.5% and cVEMP abnormality in 31% of their patients. Brainstem involvement on MRI was present in 43.8%. No correlation

Fig. 1 Bar graph of the p13 and n23 potential latencies recorded from the healthy controls and MS patients

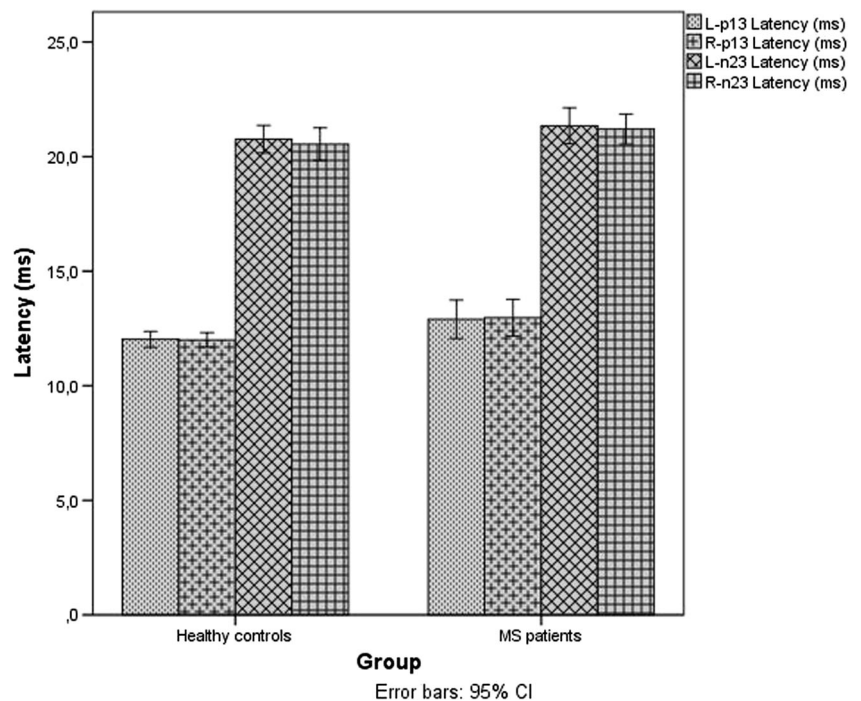
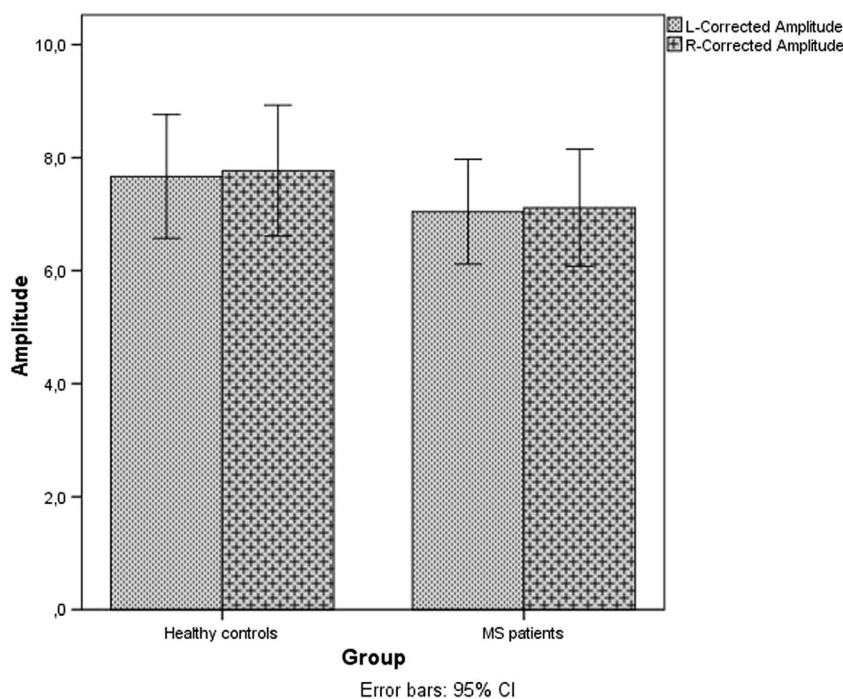


Fig. 2 Bar graph of the p13 and n23 potential amplitudes recorded from the healthy controls and MS patients



between the study variables has been reported. Garcia et al. [22] have found increased p13 and n23 latencies. However, a correlation with the clinical findings and abnormal VEMP responses was not present; patients with clinical signs and symptoms and abundant MRI lesions had normal VEMP results whereas asymptomatic patients with few lesions on imaging showed greatly altered potentials. Güven et al. [23] have reported 48% of MS patients had VEMP abnormalities seen as absent responses and/or prolonged latencies and have reported that n1 latency prolongation was significant in MS patients with brainstem lesions on MRI.

In two studies, one dealing with clinically isolated syndrome [24], a VEMP score derived from the evaluation of oVEMP and cVEMP latency, amplitude, and morphologies has also been used [24, 25]. In patients with clinical signs of brainstem involvement, VEMP score has been reported to be higher and correlated with disability and disease duration [25].

Table 2 Inter-side differences recorded for p13 and n23 latencies and amplitudes recorded from the healthy controls and the patients

	p13 latency Median (min–max)	n23 latency Median (min–max)	Amplitude Median (min–max)
HC	0.061 (0.000–0.238)	0.057 (0.000–0.237)	0.077 (0.000–0.437)
MS	0.095 (0.009–0.497)	0.063 (0.000–0.353)	0.092 (0.019–0.688)
<i>p</i>	0.074	0.627	0.224

p *p* values gathered by comparison of the healthy controls with MS patients, *HC* healthy controls, *MS* multiple sclerosis

In the study on clinically isolated syndrome, dominant hand function was found to be correlated with oVEMPs [24].

In our study, brainstem lesions on MRI were present in 13 patients (43.4%). The overall comparison of the p13 and n23 latencies and amplitude ratios of the patients and the healthy controls did not reveal a significant difference ($p > 0.05$). Inter-side difference for all the parameters recorded from the patients was not also different from the healthy controls ($p > 0.05$).

However, as the *p* values gathered by comparison of the p13 latencies of the healthy controls and MS patients were 0.052 for the right and 0.057 for the left side, latencies exceeding 2.5 standard deviations (SD) were also studied and seven patients (23.3%) with prolonged latencies were found. The main abnormality was the delay of the p13 potential recorded bilaterally in four and unilaterally in three patients. n23 latency prolongation was present unilaterally in one and bilaterally in one of these seven patients. This finding is in accordance with some previous studies reporting mainly delayed p13 latencies [14, 16, 20]. In five of the seven patients with abnormal cVEMPs, clinical signs of brainstem involvement as well as brainstem lesions on MRI were present. The other two patients with delayed latencies had no brainstem lesions on MRI. One had optic neuritis and the other had paraparesis.

On the other hand, in eight patients with brainstem lesions on MRI, cVEMPs were normal. In three of them, brainstem signs were present on examination; two had internuclear ophthalmoplegia and one had ataxia.

Comparison of the p13 and n23 latencies and amplitude ratios recorded from patients with and without

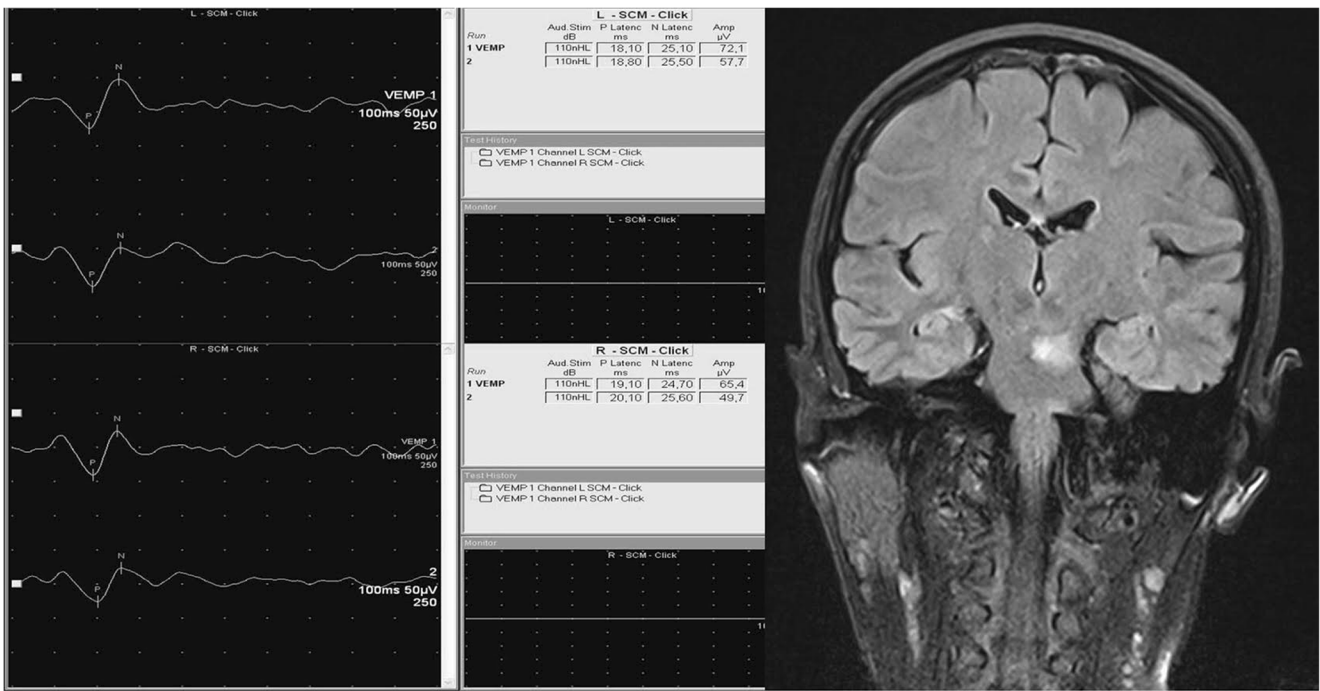


Fig. 3 Bilaterally delayed cVEMP responses in a patient with a left pontine high-signal lesion on sagittal fluid-attenuated inversion recovery (FLAIR) sequence of MRI

brainstem lesions on MRI did not reveal a significant difference as well ($p < 0.05$). These results are similar with the results of Gazioglu and Boz [20], Ivankovic

et al. [21], and Garcia et al. [22] who were also unable to find a correlation between VEMPs and brainstem clinical or MRI lesions.

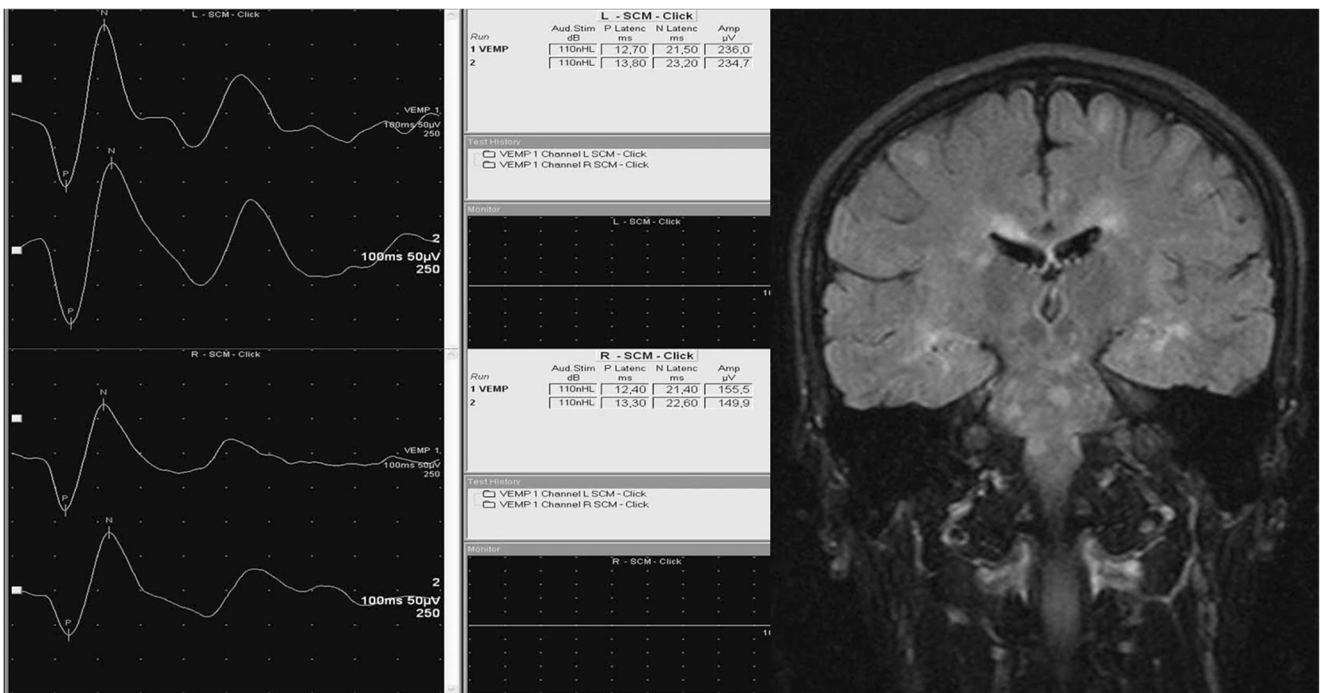


Fig. 4 Bilateral normal cVEMP responses in a patient with scattered bilateral pontine and medullary high-signal lesions on sagittal fluid-attenuated inversion recovery (FLAIR) sequence of MRI

Table 3 Symptoms and signs and cVEMP responses of patients with brainstem lesions on MRI

Brainstem lesion on MRI	Symptom and sign	cVEMP
Right inferior cerebellar peduncle	Ataxia	Delayed on the left side
Left medulla	Ataxia	Delayed on the left side
Left pons	Facial hypoesthesia	Bilaterally delayed
Bilateral pons, medulla	Facial hypoesthesia	Bilaterally delayed
Left inferior cerebellar peduncle	Gaze-evoked nystagmus	Delayed on the left side
Right pons	Optic neuritis	Normal bilaterally
Left medulla	Numbness of the legs	Normal bilaterally
Right medulla	Left hemiparesis	Normal bilaterally
Bilateral pons, medulla	Internuclear ophthalmoplegia	Normal bilaterally
Left pons	Right hemiparesis	Normal bilaterally
Right mesencephalon	Internuclear ophthalmoplegia	Normal bilaterally
Bilateral pons	Paresis of the right arm	Normal bilaterally
Left middle cerebellar peduncle	Ataxia	Normal bilaterally

Three previous studies on MS have taken brainstem auditory evoked potentials (BAEPs) into consideration in addition to VEMPs [14, 15, 21]. It was Sartucci and Logi [14] who reported BAEP and VEMP abnormalities in 53.3% of their MS patients. However, the study involved just 15 individuals. Versino et al. [15] reported BAEP abnormalities in 38%, Ivankovic et al. [21] in 21.9% of their patients. cVEMP abnormalities were present in 31% in both studies [15, 21]. A correlation between BAEP and VEMP abnormalities could not be detected [14, 15]. The occurrence of brainstem MRI lesions did not correlate with the instrumental abnormalities as well [15, 21].

According to the abovementioned data, we can say that studying cVEMPs in MS is not a sensitive way of documenting brainstem involvement. Prolonged latencies mainly involving p13 were present in 23.3% of our patients. Even in patients with brainstem lesions on MRI, cVEMPs were found to be delayed in only 38%. Though reported to be a useful diagnostic tool in identifying lower brainstem lesions especially involving the lateral lower pons and the upper

medulla oblongata [10, 11], of our eight patients with brainstem involvement on MRI, six had pontine and medullary lesions and still had normal cVEMPs. Studies involving larger number of patients with brainstem lesions seem to be essential in understanding the brainstem pathways for cVEMPs.

Compliance with ethical standards Approval from the ethics committee of Ege University Medical School was obtained, and all the patients gave their written informed consent for the procedure.

References

- Colebatch JG, Halmagyi GM (1992) Vestibular evoked potentials in human neck muscles before and after unilateral vestibular deafferentation. *Neurology* 42(8):1635–1636. <https://doi.org/10.1212/WNL.42.8.1635>
- Colebatch JG, Halmagyi GM, Skuse NF (1994) Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J Neurol Neurosurg Psychiatry* 57(2):190–197. <https://doi.org/10.1136/jnnp.57.2.190>
- Welgampola MS, Colebatch JG (2005) Characteristics and clinical applications of vestibular evoked myogenic potentials. *Neurology* 64(10):1682–1688. <https://doi.org/10.1212/01.WNL.0000161876.20552.AA>
- Rauch SD (2006) Vestibular evoked myogenic potentials. *Curr Opin Otolaryngol Head Neck Surg* 14(5):299–304. <https://doi.org/10.1097/01.moo.0000244185.65022.01>
- Honaker JA, Samy RN (2007) Vestibular evoked myogenic potentials. *Curr Opin Otolaryngol Head Neck Surg* 15(5):330–334. <https://doi.org/10.1097/MOO.0b013e3282ef7d0d>
- Welgampola MS (2008) Evoked potential testing in neuro-otology. *Curr Opin Neurol* 21(1):29–35. <https://doi.org/10.1097/WCO.0b013e3282f39184>
- Rosengren SM, Welgampola MS, Colebatch JG (2010) Vestibular evoked myogenic potentials: past, present and future. *Clin Neurophysiol* 121(5):636–651. <https://doi.org/10.1016/j.clinph.2009.10.016>

Table 4 p13 and n23 potential latencies and amplitudes recorded from the patients with and without brainstem lesions on MRI

	Brainstem lesion on MRI		<i>p</i>
	Absent (17 patients) Mean ± SD	Present (13 patients) Mean ± SD	
p13 latency (left)	12.8 ± 1.8	12.9 ± 2.1	0.73
n23 latency (left)	21.25 ± 1.94	21.29 ± 2.11	0.82
Amplitude (left)	7.21 ± 2.73	6.44 ± 2.37	0.43
p13 latency (right)	12.8 ± 2.2	12.9 ± 1.9	0.89
n23 latency (right)	21.24 ± 2.18	21.93 ± 2.2	0.33
Amplitude (right)	7.38 ± 2.99	6.48 ± 2.65	0.39

8. Eleftheriadou A, Koudounarakis E (2011) Vestibular evoked myogenic potentials eliciting: an overview. *Eur Arch Otorhinolaryngol* 268(3):331–339. <https://doi.org/10.1007/s00405-010-1408-7>
9. Hong SM, Yeo SG, Kim SW, Cha CI (2008) The results of vestibular evoked myogenic potentials, with consideration of age-related changes, in vestibular neuritis, benign paroxysmal positional vertigo, and Meniere's disease. *Acta Otolaryngol* 128(8):861–865. <https://doi.org/10.1080/00016480701784981>
10. Deftereos SN, Panagopoulos G, Eleftheriadou A, Korres S, Georgonikou D, Kandiloros D, Karageorgiou CE (2008) Using vestibular evoked myogenic potentials to localize brainstem lesions. A preliminary report. *B-ENT* 4(4):215–219
11. Itoh A, Kim YS, Yoshioka K, Kanaya M, Enomoto H, Hiraiwa F, Mizuno M (2001) Clinical study of vestibular-evoked myogenic potentials and auditory brainstem responses in patients with brainstem lesions. *Acta Otolaryngol Suppl* 545:116–119
12. Heide G, Luft B, Franke J, Schmidt P, Witte OW, Axer H (2010) Brainstem representation of vestibular evoked myogenic potentials. *Clin Neurophysiol* 121(7):1102–1108. <https://doi.org/10.1016/j.clinph.2010.02.007>
13. Polman CH, Reingold SC, Banwell B, Clanet M, Cohen JA, Filippi M, Fujihara K, Havrdova E, Hutchinson M, Kappos L, Lublin FD, Montalban X, O'Connor P, Sandberg-Wollheim M, Thompson AJ, Waubant E, Weinshenker B, Wolinsky JS (2011) Diagnostic criteria for multiple sclerosis: 2010 revisions to the McDonald criteria. *Ann Neurol* 69(2):292–302. <https://doi.org/10.1002/ana.22366>
14. Sartucci F, Logi F (2002) Vestibular-evoked myogenic potentials: a method to assess vestibulo-spinal conduction in multiple sclerosis patients. *Brain Res Bull* 59(1):59–63. [https://doi.org/10.1016/S0361-9230\(02\)00842-0](https://doi.org/10.1016/S0361-9230(02)00842-0)
15. Versino M, Colnaghia S, Calliecoa R, Bergamaschib R, Romanib A, Cosia V (2002) Vestibular evoked myogenic potentials in multiple sclerosis patients. *Clin Neurophysiol* 113(9):1464–1469. [https://doi.org/10.1016/S1388-2457\(02\)00155-4](https://doi.org/10.1016/S1388-2457(02)00155-4)
16. Bandini F, Beronio A, Ghigliione E, Solaro C, Parodi RC, Mazzella L (2004) The diagnostic value of vestibular evoked myogenic potentials in multiple sclerosis. *J Neurol* 251(5):617–621. <https://doi.org/10.1007/s00415-004-0378-3>
17. Alpini D, Pugnetti L, Caputo D, Cornelio F, Capobianco S, Cesarani A (2004) Vestibular evoked myogenic potentials in multiple sclerosis: clinical and imaging correlations. *Mult Scler* 10(3):316–322. <https://doi.org/10.1191/1352458504ms1041oa>
18. Patkó T, Simó M, Arányi Z (2007) Vestibular click-evoked myogenic potentials: sensitivity and factors determining abnormality in patients with multiple sclerosis. *Mult Scler* 13(2):193–198. <https://doi.org/10.1177/1352458506070940>
19. Eleftheriadou A, Deftereos SN, Zarikas V, Panagopoulos G, Sfetsos S, Karageorgiou CL, Ferekidou E, Kandiloros D, Korres S (2009) The diagnostic value of earlier and later components of vestibular evoked myogenic potentials (VEMP) in multiple sclerosis. *J Vest Res* 19:59–66
20. Gazioglu S, Boz C (2012) Ocular and cervical vestibular evoked myogenic potentials in multiple sclerosis patients. *Clin Neurophysiol* 123(9):1872–1879. <https://doi.org/10.1016/j.clinph.2012.01.022>
21. Ivankovic A, Madaric VN, Starcevic K, Skoric MK, Gabelic T, Adamec I, Habek M (2013) Auditory evoked potentials and vestibular evoked myogenic potentials in evaluation of brainstem lesions in multiple sclerosis. *J Neurol Sci* 328(1-2):24–27. <https://doi.org/10.1016/j.jns.2013.02.005>
22. Garcia VE, Carratala IL, Alborch MO, Algarra JM (2013) Vestibular evoked myogenic potential findings in multiple sclerosis. *Acta Otorrinolaringol Esp* 64(5):352–358. <https://doi.org/10.1016/j.otoeng.2013.10.009>
23. Güven H, Bayır Ö, Aytaç E, Özdek A, Çomoğlu SS, Korkmaz H (2014) Vestibular-evoked myogenic potentials, clinical evaluation and imaging findings in multiple sclerosis. *Neurol Sci* 35(2):221–226. <https://doi.org/10.1007/s10072-013-1483-9>
24. Cmosija L, Skoric MK, Gabelic T, Adamec I, Brinar V, Habek M (2015) Correlation of the VEMP score, ambulation and upper extremity function in clinically isolated syndrome. *J Neurol Sci* 359(1-2):197–201. <https://doi.org/10.1016/j.jns.2015.10.049>
25. Gabelic T, Skoric MK, Adamec I, Barun B, Zadro I, Habek M (2015) The vestibular evoked myogenic potentials (VEMP) score: a promising tool for evaluation of brainstem involvement in multiple sclerosis. *Eur J Neurol* 22(2):261–269. <https://doi.org/10.1111/ene.12557>