Assessment of Cervical and Ocular Vestibular Evoked Myogenic Potentials in Multiple Sclerosis Patients

Original Article

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ABSTRACT

Background: Multiple sclerosis (MS) is a chronic neurological disease affecting the central nervous system and is the leading cause of disability due to brainstem affection. Cervical Vestibular Evoked Myogenic Potentials (cVEMPs) are a clinical demonstration of vestibulo-colic reflex which descends via the vestibulospinal tract through the lower brainstem while ocular VEMPs (oVEMPs) represent vestibulo–ocular reflex which ascends via the Medial Longitudinal Fasiculus through the upper brainstem.

Aim: To assess cVEMPs and oVEMPs in MS patients with and without brainstem lesion(s) compared to normal controls.

Patients and Methods: All subjects underwent history taking of clinical symptoms and Expanded Disability Status Scale (EDSS) score, audiometric testing, 500 Hz toneburst air conduction cVEMPs and oVEMPs and brain MRI. Latency and amplitude of cVEMPs (P13, N23) and oVEMPs (N10, P15) were recorded in 10 healthy controls (20 ears), 10 MS patients (20 ears) with brainstem lesion(s) and 10 MS patients (20 ears) without brainstem lesion(s).

Results: The cVEMPs and oVEMPs latencies in MS patients were significantly prolonged compared to controls. VEMPs latencies in MS with brainstem lesion(s) were significantly prolonged compared to patients without brainstem lesion(s). No correlation was found between the clinical state and VEMPs responses. A significant positive correlation was found between VEMPs latencies and EDSS in both MS subgroups.

Conclusion: VEMPs are of value in detecting silent brainstem lesions through evaluation of upper and lower brainstem. The combination of oVEMPs and cVEMPs to MRI and the correlation with the disability state provide comprehensive evaluation of brainstem involvement in MS patients.

Key Words: Brainstem lesions, Cervical Vestibular evoked myogenic potential, EDSS, multiple sclerosis, ocular Vestibular evoked myogenic potential.

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INTRODUCTION

Multiple sclerosis (MS) is a chronic inflammatory, autoimmune neurological disorder affecting central nervous system and it is the main cause of disability in young adults.[1,2] By attacking the myelin, which surrounds and protects the nerve fibers, MS leads to tissue scarring or sclerosis varying in degrees.[3,4]

MS affects females more than males with 2:1 ratio,[5, 6] it is mainly diagnosed from 15 to 45 years[5-9]. It is of unknown etiology, but several factors are attributed including: age, geography, environmental influences, genetics, and viruses.[5-10]

The clinical signs, symptoms, clinical disease course and success of treatment are dissimilar in each case. MRI is the main investigation in the diagnosis of MS, but it has a bad correlation with clinical findings. Nearly 65% of MS patients develop multiple clinical signs of brain stem (BS) involvement during the disease course.[11] The significance of BS involvement in MS has been emphasized in many studies showing good prediction of future disability in patients of MS.[12,13]

Anatomical site of BS lesion(s) on the MRI correlates poorly with clinical signs of BS involvement, nearly 60% of patients who have signs of BS involvement have consequent MRI lesions.[11,14] This can be explained by the low specificity of MRI in differentiation of heterogeneous pathophysiological mechanisms of tissue destruction, mainly neuroinflammation and neurodegeneration leading to clinico-radiological paradox. Clinico-radiological paradox consider that this discordance between clinical and imaging measures is mainly due to the own limitations of the T2 sequence because of its lack of histopathological specificity.[15-19] This paradox is mostly in MS patients with Expanded Disability Status Scale (EDSS) more than 4.0.[19]

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The EDSS is a known method of quantifying disability in multiple sclerosis with eight subscale measurements called Functional System scores (pyramidal / motor function, cerebellar, brainstem, sensory, bowel and bladder ,visual, cerebral or mental and other). The scale ranges from 0 to 10 in 0.5 unit increments that represent higher levels of disability. EDSS steps 1.0 to 4.5 refer to people with MS who are able to walk without any aid[20]

Hence additional tests are needed to recognize brainstem affection in MS patients and to supply neurologists with extra tools for detecting clinically and radiologically subclinical lesions. Evoked potentials (EP) such as Lower limb motor and somatosensory EPs together with visual EPs are most widely used in clinical practice aiming to reveal subclinical lesions.[21,22] VEMPs are biphasic short myogenic potentials that are evoked when the vestibular system is presented with high intensity sound. It can be evoked by acoustic, bone vibration or galvanic stimulation. The VEMPs represent otolith organs' response.[23,24]

1.1. Cervical vestibular evoked myogenic potentials:

These inhibitory myogenic potentials are picked up from the ipsilateral contracted Sternocleidomastoid muscle (SCM). Cervical VEMPs (cVEMPs) are composed of two components. The first component is a positive peak taking place at about 13 msec (P13), while the second one is a negative peak occurring around 23 msec (N23) after the stimulus.[25,26] Cervical VEMPs have become a vital test of the neuro-otological test battery as it is a clinical manifestation of vestibule-colic reflex which descends via the vestibulospinal tract throughout the lower brainstem.[27] It determines whether the saccule, the inferior vestibular nerve and central connections are intact.[28]

1.2. Ocular vestibular evoked myogenic potentials:

VEMPs can also be picked up from the contralateral inferior oblique extra ocular muscles and is termed ocular VEMPs. This ocular response is considered as a novel investigative tool in neuro-otology.[27,29] Ocular VEMPs (oVEMPs) to air conduction (AC) stimuli consist of an excitatory biphasic potential with an initial negative peak, at about 10 msec, (N10 or N1).[27] Followed by a positive peak at about 15 msec and is known as (P15 or P1).[30] Ocular VEMPs permit further evaluation of central vestibular pathways, particularly vestibulo–ocular reflexes that ascends in the Medial Longitudinal Fasciculus throughout the upper brainstem.[31]

Cervical and Ocular VEMPs were beneficial in the evaluation of brainstem involvement in MS patients with vestibular symptoms.[32] Therefore, VEMPs might be of value in MS patients as a complementary diagnostic tool to the brain MRI.

AIM:

The aim of this study was to investigate the ocular and cervical VEMPs tests in multiple sclerosis patients and to study the relation of ocular and cervical VEMPs with clinical and radiological findings.

PATIENTS AND METHODS:

3.1. Subjects:

The current study was done in the audiology unit of Alexandria main university hospital, Egypt, on 10 healthy controls (20 ears) with mean age 30.2 ± 9.75 years, 10 patients (20 ears) with definite MS according to the revised MC Donald criteria 2017 with BS lesion(s) with average age of 40 ± 12.34 years and 10 MS patients (20 ears) with definite MS without BS lesion(s) of an average age of 34.1 ± 6.52 years.[33] MC Donald criteria included MRI into the well-established diagnostic workup of patients, who present with a typical clinically isolated syndrome suggestive of MS, that concentrates on detailed neurological history, examination of patient and different laboratory examinations such as cerebrospinal fluid analysis. The diagnostic requirements for MS on an MRI must include the documentation of lesions disseminated in space (DIS) and disseminated in time (DIT). DIS requires one or more T2 lesions in two or more MS locations of the CNS. The MS typical locations are periventricular, juxtacortical, corpus callosum, and spinal cord. DIT can be established by the presence of a new lesion on a scan compared to a baseline scan at least 30 days after the onset of the initial clinical event.[33]

3.2. Methods:

The study protocol was approved from the Ethics Committee of Faculty of Medicine, Alexandria University in accordance with the International Ethical Guidelines for Epidemiological studies. For all subjects, complete history taking including clinical symptoms and EDSS score, otoscopy and audiometric testing and brain MRI scanning were done. Exclusion criteria included subjects with any disorders of the middle or external auditory system, those with limitation of neck rotation, weakness of sternocleidomastoid muscle or using vestibule-toxic or vestibular suppressant drugs within a month prior to evaluation.

3.2.1. Cervical VEMPs recording:

Cervical VEMPs test was done monaurally using the GSI Audera evoked system (GSI, USA) while subjects were seated and turned their head to a fixed target opposed to the side of the stimulus to put the ipsilateral SCM muscle in contraction. Active surface electrode was located on the superior one third of the ipsilateral SCM with a reference

electrode on the upper sternum and a ground electrode on central forehead after rubbing the skin.[34] Cervical VEMPs was recorded from a response to AC 500 Hz short tone burst of condensation polarity transmitted through supra aural headphones at 95 dBnHL. Stimuli were Blackman gated with a one-cycle rise-fall time and at two-cycle plateau. The stimulation rate was 5 cycles per sec. At least 2 runs were performed and 150 sweeps were averaged for each run with analysis time of 50 msec. EMG signal were amplified and band-pass filtered between 1 and 1000 Hz. The latency and amplitude of P13 and N23 were measured and the interaural peak asymmetry / difference in the form of IAD were calculated.

$$IAD(\%) = 100 \times (Amp[left] - Amp[right]) / (Amp[left] + Amp[right])$$

3.2.2. Ocular VEMPs recording:

Subjects were seated and were informed to look upward at a small fixed target > 2 meters from the eyes, with a vertical visual angle of approximately 30-35 degrees above the horizontal plane.[35] For each eye active recording electrode was located 1 cm below the center of contralateral lower eyelid and the reference was located at about 1-2 cm below it. Ground electrode was located on the forehead.[36]

Similar equipment and stimulus parameters as cVEMPs were used and the latency and amplitude of N10 and P15 was measured and IAD was calculated according to the aforementioned formula.

3.3. Statistical analysis of the data:

Data were analyzed using SPSS software package version 20.0. The Kolmogorov-Smirnov test was used to verify the normality of distribution. Significance of the obtained results was judged at the 5% level. *P-values* ≤ 0.05 were considered statistically significant.[37,38]

For demographic data:we used Chi-square test for normative data and Fisher's Exact or Monte Carlo correction when more than 20% of the cells have expected count less than 5.

For comparing VEMPs finding in the 2 subgroups of MS and control: F-test (ANOVA) was used for normally distributed quantitative variables, and Post Hoc test (LSD) for pairwise comparisons. While Mann Whitney test was used for abnormally distributed quantitative variables.

Spearman correlations were done between the measured parameters of both cVEMPs and oVEMPs and the clinical symptoms and EDSS scores.

RESULTS:

The pure tone audiometry showed normal level of hearing levels in the 10 healthy subjects, with hearing thresholds not exceeding 25 dBHL. In the MS group 4 patients had bilateral mild to moderate SNHL while the 16 remaining patients had normal hearing. None of the patients had an air-bone gap in the audiometry, which would have suggested the existence of a conductive hearing loss that might affect the VEMPs results.

4.1. Cervical VEMPs parameters:

Table (1) shows the distribution of cVEMPs parameters in the control and the 2 subgroups of MS patients. Cervical VEMPs were absent in 1 ear of an MS patient with BS lesions. The P13 and N23 latencies were significantly delayed in the patient groups compared with the control group with a significant increase in P13 and N23 latencies in MS group with BS affection compared to MS group without BS affection. There was no difference in cVEMPs amplitude or IAD between the 3 studied groups.

Normal range of P13 and N23 latencies were calculated using mean + 2 SD and the values were 15.93 msec and 26.64 msec respectively, the percent of abnormality were 63.2% and 31.6% in MS group with BS lesion and 30% and 5% in MS group without BS lesion.

Table 1: C VEMPs parameters in the 3 studied groups:

C VEMPs	Patient		Control	Test of sig.	*P*
	MS without BS	MS with BS			
P13 latency (msec)	(n= 20 ears)	(n= 19 ears)	(n= 20 ears)	F= 15.604	<0.001*
Min. – Max.	13.30 –19.0	13.80 – 19.0	11.83 – 15.70		
Mean ± SD.	15.21 ± 1.57	16.49 ± 1.49	14.05 ± 0.94		
Median	14.78	16.50	14.15		
Sig. bet. Groups	*p1=0.005*, p2=0.009*, p3<0.001**				
N23 latency (msec)	(n= 20 ears)	(n= 19 ears)	(n= 20 ears)	F=15.131*	<0.001*
Min. – Max.	20.34 – 28.15	22.40 – 29.35	19.0 – 26.20		
Mean ± SD.	23.97 ± 2.06	25.96 ± 1.93	22.36 ± 2.14		
Median	23.91	26.0	21.98		

Sig. bet. Groups	*p1=0.004*, p2=0.016*, p3<0.001**				
Amplitude (μV)	(n= 20 ears)	(n= 19 ears)	(n= 20 ears)	H=2.565	0.109
Min. – Max.	21.52 – 365.55	14.20 – 167.45	15.08 – 455.0		
Mean ± SD.	93.12 ± 78.01	66.25 ± 52.42	144.42 ± 122.73		
Median	65.75	41.50	110.0		
IAD	(n= 10)	(n= 9)	(n= 10)	H=0.000	1.000
Min. – Max.	2.10 – 42.50	4.10 – 46.20	0.10 – 33.0		
Mean ± SD.	20.76 ± 14.96	21.18 ± 16.24	9.89 ± 11.99		
Median	15.70	16.60	3.80		

F,p: F and p values for ANOVA test, Sig. bet. groups was done using Post Hoc Test (LSD) H,p: H and p values for Kruskal Wallis test

p1: p value for comparing between MS without BS and MS with BS p2: p value for comparing between MS without BS and control

p3: p value for comparing between MS with BS and control

*: Statistically significant at $p \leq 0.05$

4.2. Ocular VEMPs parameters:

Table (2) illustrates the distribution of oVEMPs parameters in the control and the 2 subgroups of MS patients. Ocular VEMPs were absent in 3 ears in MS patients with BS lesions. The N10 and P15 latency were significantly delayed in the patient groups compared with the control group with significantly increased latency of p15 in MS group with BS affection compared to MS group without BS affection. No difference in amplitude or IAD was found between the 3 studied groups.

Normal range of N10 and p15 latencies were calculated using mean+2 SD and the values were 11.93 msec and 17.11 msec respectively, the percent of abnormality were 58.8% and 52.9% in MS group with BS lesion and 25% and 10% in MS group without BS lesion.

Table 2: O VEMPs parameters in the 3 studied groups :

O VEMPs	Patient		Control	Test of sig.	*P*
	MS without BS	MS with BS			
N10 latency (msec)	(n= 20 ears)	(n= 17 ears)	(n= 20 ears)	H= 10.878*	0.004*
Min. – Max.	1.20 – 13.20	8.65 – 13.70	7.0 – 11.17		
Mean ± SD.	10.58 ± 2.50	11.43 ± 1.42	10.09 ± 0.92		
Median	10.75	12.0	10.10		
Sig. bet. Groups	*p1=0.232, p2=0.027*, p3=0.002**				
P15 latency (msec)	(n= 20 ears)	(n= 17 ears)	(n= 20 ears)	F= 9.829*	<0.001*
Min. – Max.	12.25 – 18.60	11.70 – 19.30	13.0 – 17.0		
Mean ± SD.	15.81 ± 1.42	16.86 ± 1.79	14.71 ± 1.20		
Median	15.80	17.40	14.30		
Sig. bet. Groups	*p1=0.036*, p2=0.022*, p3<0.001**				
Amplitude (μV)	(n= 20 ears)	(n= 17 ears)	(n= 20 ears)	H=0.231	0.891
Min. – Max.	1.0 – 7.61	1.0 – 17.10	1.0 – 5.40		
Mean ± SD.	3.22 ± 2.00	3.59 ± 3.82	2.76 ± 1.28		
Median	2.81	2.70	2.39		
IAD	(n= 10)	(n= 8)	(n= 10)	H=0.252	0.882
Min. – Max.	2.20 – 75.50	3.10 – 42.80	0.38 – 53.0		
Mean ± SD.	29.52 ± 26.43	22.23 ± 14.27	22.91 ± 17.84		
Median	24.10	17.85	18.90		

F,p: F and p values for ANOVA test, Sig. bet. groups was done using Post Hoc Test (LSD) H,p: H and p values for Kruskal Wallis test

p1: p value for comparing betweenMS without BS and MS with BS p2: p value for comparing betweenMS without BS and control

p3: p value for comparing betweenMS with BS and control *: Statistically significant at $p \leq 0.05$

4.3. Clinical symptoms of BS in MS:

Diplopia, facial sensory symptoms and balance problems were the most common brainstem-specific symptoms reported in MS patients as shown in Figure (1).

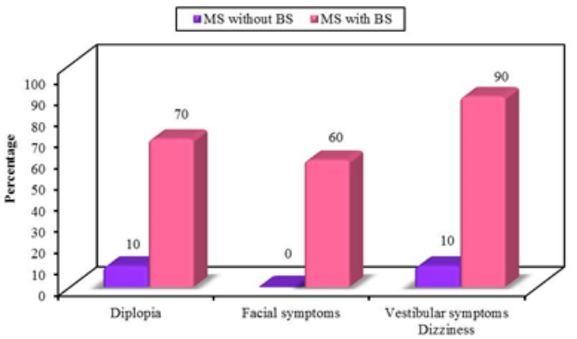

Fig 1: Comparison between the 2 MS subgroups according to clinical symptoms

Table (3) demonstrates the correlation between the clinical symptoms and the measured parameters of both cVEMPs and oVEMPs in the 2 subgroups of MS patients. No significant correlation was seen between the clinical symptoms and the VEMP parameters.

Table 3: Correlation between Clinical symptoms with C VEMPs and O VEMPs parameters:

	Clinical symptoms			
	MS without BS		MS with BS	
	r_s	p	r_s	p
C VEMPs				
P13 latency	0.087	0.811	-0.443	0.233
N23 latency	0.218	0.545	0.272	0.479
Amplitude	0.174	0.631	0.437	0.240
IAD	-0.435	0.209	-0.499	0.172
O VEMPs				
N10 latency	-0.174	0.631	0.572	0.107
P15 latency	-0.348	0.324	0.365	0.334
Amplitude	-0.435	0.209	0.535	0.138
IAD	0.000	1.000	0.517	0.190

r_s: Spearman coefficient

4.4. EDSS:

In the current study the MS without BS lesions subgroup had a mean EDSS score of 2.45 ± 2.14 while the MS with BS subgroup had a mean EDSS score of 4.76 ± 1.56.

A significant positive correlation between VEMPs latency and the level of the disability caused by the disease measured by the EDSS was found in both MS subgroups as shown in (Table 4) and Figures (2 &3).

Table 4: Correlation between EDSS and VEMPs latencies in the 2 studied MS subgroups:

	EDSS			
	MS without BS		MS with BS	
	r_s	P	r_s	P
P13 latency	0.501	0.002*	0.652	0.003*
N23 latency	0.565	0.012*	0.694	0.040*
N10 latency	0.809	0.002*	0.480	0.023*
P15 latency	0.461	0.002*	0.628	0.022*

r_s: Spearman coefficient
*: Statistically significant at $p \leq 0.05$

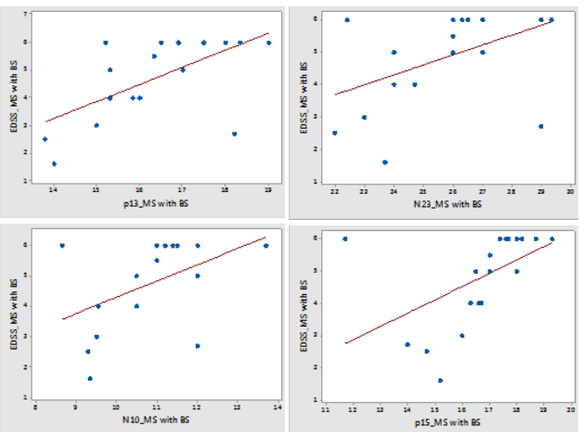

Fig 2: Correlation between EDSS and all latencies of all waves of both cVEMPs and oVEMPs in MS group with BS.

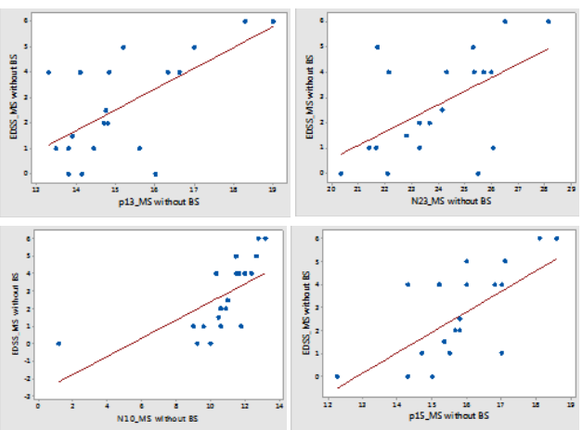

Fig 3: Correlation between EDSS and all latencies of all waves of both cVEMPs and oVEMPs in MS group without BS.

4.5. Percentage of VEMPs latency abnormalities in MS subgroups:

Table (5) shows the percentage of VEMPs latency abnormalities in the 2 subgroups of MS. P13, N23, N10 and P15 latencies were significantly prolonged in MS group with BS lesion compared to MS group without BS lesion.

Table (5):

	MS without BS	MS with BS	χ^2	P
Delayed P13	6(30.0%)	12(63.2%)	4.311*	0.038*
Delayed N23	1(5.0%)	6(31.6%)	4.674*	0.044*
Delayed N10	5(25.0%)	10(58.8%)	4.361*	0.037*
Delayed P15	2(10.0%)	9(52.9%)	8.111*	0.004*

χ^2, χ^2 and p values for Chi square test for comparing between the two groups
*: Statistically significant at $p \leq 0.05$

DISCUSSION

The aim of the current study was to evaluate cVEMPs & oVEMPs in MS patients with and without brainstem lesion(s) and compare the findings with normal controls.

The latencies of P13 and N23 were significantly prolonged in MS patients with and without Brain Stem lesion(s) compared to control subjects. Additionally significant increase in latency in BS lesion more than non BS lesion was found. The physio-pathological explanation of this phenomenon in the case of MS lesion, has been attributed to slowing down of the nerve signal conduction caused by the process of demyelination affecting the vestibulospinal tract.[39]

Previous studies have reported that the abnormality rate in cVEMPs response ranged from 31% to 60%.[40-44] Several studies proved that in patients with MS, cVEMPs were abnormal in up to 50% of patients[24, 41-45]. There was no significant difference in the mean amplitude of cVEMPs of both MS subgroups compared to normal subjects. The VEMPs amplitude parameter in MS patients is not a reliable diagnostic indicator because other variables such as muscle contraction and stimulus intensity can affect it.[44,46,47] Hence, amplitudes of VEMPs responses should not be used alone and should be interpreted together with latency values in MS patients. Bandini *et al*.[44] also reported that cVEMPs amplitude values make no significant diagnostic contribution in MS patients.

IAD was used to control inter subject variability and it showed no significant difference between both MS subgroups compared to normal subjects. Güven *et al*.[48] also reported that comparison of IAD of patients and healthy controls did not reveal any significant difference.

Prolonged latency of VEMPs are not specific for MS and cannot help distinguish MS from other etiologies[40, 44, 46]. Delays of latency in VEMPs have been seen in other neurological disease that affect brainstem such as stroke and tumors.[49]

Overall in the present study 63.2% MS patients with BS lesion showed P13 abnormality in the form of delayed latency and 31.6% had delayed N23 latency. Additionally one ear had absent response which is explained by extensive demyelination as shown on MRI of the patients. In MS patients without Brain Stem lesion abnormal increased latency of P13 & N23 were seen in 30% and 5% of patients, respectively. This suggests that cVEMPs may reveal a subclinical BS lesion not established by MRI.

The latency of N10 and P15 were significantly longer in MS patients with and without Brain Stem lesion(s) compared to control subjects. P15 latency is significantly prolonged in BS lesion than in MS patients with no BS lesions.

Slowing down of the nerve signal conduction is caused by demyelination affecting the vestibule-ocular tract. In the present study 58.8% had N10 abnormality and 52.9 % had P15 abnormality in MS patients with BS lesion and 3 ears had absent response due to extensive demyelination. Prolonged latencies of oVEMPs responses in MS patients have also been reported in several studies.[42, 46, 50, 51] Parsa *et al*.,[52] showed that 85.66% of their cases had some form of oVEMPs abnormality in MS patients with brain stem lesion.[52]

In MS patients without BS lesion(s), abnormality of N10 & P15 were seen in 25% and 10% of patients, respectively again proving advantage of oVEMPs over MRI in detecting subclinical lesions.

No statistical difference was found in amplitude and IAD analysis of oVEMPs between the 3 groups. Gazioglu and Boz,(46) Pollak *et al*.,[47] Bandini *et al*.,[44] also reported no significant difference in the mean amplitude of both oVEMPs & cVEMPs between MS patients with brainstem lesion(s) and MS patients without brainstem lesion(s).

In the current study there was discrepancy between radiological findings and clinical findings with one case of diplopia and one case of dizziness without radiological evidence of BS affection. Nakashima *et al*.,[11] also proved that the association

between clinical and MRI findings in the brainstem region is poor. This can be explained by the low specificity of MRI in differentiation of heterogeneous pathophysiological mechanisms of tissue damage, mainly neuroinflammation and neurodegeneration, the so called clinicoradiological paradox.

No relationship was found between the clinical state of the patient and the VEMPs responses. Versino *et al*.[41] also reported that brainstem clinical findings were not correlated with cVEMPs abnormalities. However a significant positive correlation between VEMPs abnormalities and the level of the disability caused by the disease measured by the EDSS was found. Guven *et al*.,[48] found that in patients with MS, the P13-N23 wave was absent in a higher frequency of ears in patients with greater EDSS score. The EDSS was the pivotal variable not the disease duration, as it measures the disability, hence the extent of pathology in the nervous system. Patko *et al*.,[42] have shown that conduction block is the most frequent pathology amongst MS patients and that conduction block of the cVEMPs is more frequent in patients with vestibular symptoms and a higher EDSS.[42]. In the current study mean EDSS in the group of MS without BS lesions was 2.45 ± 2.14 and in the group of MS with BS lesions was 4.76 ± 1.56.

VEMPs latencies of P13, N23, N10 and P15 were significantly increased in MS group with BS than in MS group without BS. In the MS group without BS affection 30% of patients had delayed P13, 0.5% had delayed N23, 25% had delayed N10 and 10% had delayed P15. A significant relation between both cervical and ocular VEMPs abnormality and BS affection was found. Several studies have also shown good relation between VEMPs abnormalities and brainstem lesions.[53, 54]

CONCLUSION

The trends found in this study may support the idea that the clinical symptoms of brainstem affection is not correlated with MRI finding. VEMPs are of value in detecting silent brainstem lesions through evaluation of both upper and lower brainstem. The combination of oVEMPs and cVEMPs as additional neurophysiological tests to MRI and the correlation with the disability and clinical state provide comprehensive evaluation of brainstem involvement in MS patients.

LIST OF ABBREVIATIONS

µV	Microvolt
AC	Air conduction
BS	Brain stem
cVEMPs	Cervical VEMPs
dBHL	Decibel hearing level
DIS	Disseminated in space
DIT	Disseminated in time
EDSS	Expanded Disability Status Scale
EP	Evoked potentials
Hz	Hertz
IAD	Interaural difference
MS	Multiple sclerosis
msec	Millisecond
nHL	Normalized Hearing Level
oVEMPs	Ocular VEMPs
SCM	Sternocleidomastoid muscle
SNHL	Sensory Neural Hearing Loss
VEMPs	Vestibular evoked myogenic potentials

CONFLICT OF INTEREST

There are no conflicts of interest.

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