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Using vestibular evoked myogenic potentials to localise brainstem lesions. A preliminary report

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Abstract. *Using vestibular evoked myogenic potentials to localise brainstem lesions. A preliminary report.*
Background: Vestibular Evoked Myogenic Potentials (VEMPs) are saccular responses to acoustic stimuli. They can be recorded from the sternocleidomastoid muscle ipsilaterally to the stimulated ear. Their reflex arc includes the ipsilateral vestibular nuclei.
Objective: To determine the usefulness of VEMPs in localising brainstem lesions.
Methods: We used VEMPs, Blink Reflex (BR) and Brainstem Auditory Evoked Responses (BAERs) to evaluate six patients presenting with acute ischaemic or haemorrhagic brainstem lesions, or basilar dolichoectasia.
Results: MRI in patient one revealed a dorsolateral medullary infarct on the right. VEMP amplitude was reduced ipsilaterally. The R2 BR component was delayed bilaterally upon stimulation of the affected side. Patients two and three had suffered a left lateral lower pontine infarct and a right lateral lower pontine haemorrhage. In patients four and five, MRA revealed dolichoectasia of the basilar artery exerting pressure on the lower lateral pons. VEMP amplitude was reduced ipsilaterally. Patient six had an ischaemic lesion in the right upper lateral pons. The R1, $R2_i$ and $R2_c$ BR components were delayed ipsilaterally. BAERs waves IV and V were absent on the right. VEMPs were normal.
Conclusions: VEMPs are affected by lesions of the lateral lower pons and upper medulla. Our results suggest that they may be a useful addition in the localisation of such lesions.

Introduction

The neurophysiological assessment of brainstem function mainly comprises the Blink Reflex (BR), the inhibitory masseter reflex, the jaw jerk and Brainstem Auditory Evoked Responses (BAERs), which involve pontine and medullary neural circuits.[1] In addition, the otoacoustic emission test can be used to study efferent pathways towards the cochlea,[2] while the stapedius reflex, mediated by a long neuronal pathway within the brainstem that starts from the auditory and ends at the facial nerve, is also affected in brainstem lesions.[3]

Vestibular Evoked Myogenic Potentials (VEMPs), on the other hand, are saccular responses to loud acoustic stimuli and are recordable from the sterno-cleido-mastoid muscle ipsilaterally to the stimulated ear.[4,5] Their reflex arc includes the ipsilateral vestibular nuclei located at the limit of the lower pons and upper medulla. We hypothesised that VEMPs could complement the above-mentioned evoked responses in localising brainstem lesions. We tested this hypothesis in six patients with lesions of this kind.

Methods

We studied six patients presenting with acute ischaemic or haemorrhagic brainstem lesions (confirmed by brain MRI) or basilar dolichoectasia (confirmed by brain Magnetic Resonance Angiography, MRA). The term dolichoectasia refers to the elongation and distention of the basilar artery, which may also have a tortuous course. The distended artery may compress brainstem structures and cranial nerves. VEMPs, blink reflex and Brainstem Auditory Evoked Responces (BAERs) were recorded within one week from the onset of symptoms, according to standard procedures.[4,6] More specifically, during VEMP recording, patients were lying supine and were instructed to raise their heads off the bed to activate the neck flexor muscles bilaterally and symmetrically. Raw EMG activity was displayed on an oscilloscope screen that was viewed by the participants. The participants were asked

to maintain the continuous EMG activity at the same high level (e.g. 50-100 uV) throughout the recording session. Electromyographic (EMG) activity was recorded from the sterno-cleidomastoid muscles bilaterally in a belly-tendon montage. Band-pass filter settings were 5 Hz to 1.5 kHz. The stimuli were rarefaction square wave clicks (duration 0.1 ms, intensity 140 dBSPL, frequency 5 Hz) and they were delivered to the left ear and right ear successively by a calibrated headphone. 250 unrectified EMG traces from 20 ms before the stimulus to 50 ms afterwards were collected and averaged using a Medelec Synergy T-EP EMG/EP monitoring apparatus (Medelec Synergy, Oxford Instruments Medical, Surrey, UK). Each recording was repeated twice to ensure reproducibility. In the recordings obtained we identified the first positive (p_{13}) and negative (n_{23}) peaks and we measured their onset latencies and the peak-to-peak amplitude of the p_{13}-n_{23} wave.[4]

Results

In case one, a 54-year-old Indian male, the brain MRI revealed a right dorsolateral medullary infarct (Figure 1). The amplitude of the p_{13}-n_{23} VEMP wave obtained from the right sternocleidomastoid was markedly reduced by comparison with the wave obtained from the left ear, while the latencies of these waves were similar in both ears and within normal limits. BAERs were normal on both sides. The latency of the R1 blink reflex response was normal on stimulation of either side. On stimulation of the left (unaffected) side, the latencies of the R2 ipsilateral ($R2_i$) and contralateral ($R2_c$) responses were also normal. However, upon stimulation of the right (affected) side, both these responses were delayed. Table 1 lists the values of specific evoked response parameters.

In cases two (a 60-year-old Caucasian female) and three (a 45-year-old Caucasian male) MRI revealed a left lateral lower pontine infarct and a right lateral lower pontine haemorrhage (Figure 1) respectively. The amplitude of the p_{13}-n_{23} VEMP wave obtained from the ear ipsilateral to the lesion was reduced in both cases, while BAERs, BR and the latencies of VEMP waves were not affected.

Table 1

Values of VEMPs, BAERs and Blink Reflex parameters in all subjects. Values are reported as right/left. Pathological findings are presented in bold

Case parameter (right/left)	**One**	**Two**	**Three**	**Four**	**Five**	**Six**
MRI findings	Right dorsolateral medullary infarct	Left lateral lower pontine infarct	Right lateral lower pontine haemorrhage	Dolicho-ectasia of the basilar artery	Dolicho-ectasia of the basilar artery	Right lateral upper pontine infarct
VEMPS						
P13n23 amplitude (μV)	**43**/170	82/**35**	**51.3**/205.6	**13**/60	**21**/110	143/130
P13 latency (msec)	12.7/12.2	13/12.8	13.2/13.6	13.2/13	13.0/13.1	12.8/12.6
N23 latency (msec)	19.8/19.6	20/19.2	22.1/22.4	19.4/20.0	20.1/20.0	23.1/23.0
BLINK REFLEX						
R1 latency (msec)	12.0/11.7	12.7/12.1	11.7/11.6	11.2/11.3	11.5/11.4	**21.0**/12.1
R2 ipsilateral latency (msec)	**48.2**/36	36.2/35.9	37.0/37.3	35.3/35.7	36.1/35.8	**53.0**/37.0
R2 contralateral latency (msec)	**48.4**/36.2	36.3/36.1	37.1/37.2	35.2/35.8	36.2/36.0	**55.4**/36.8
BAERs						
Wave IV present (+/-)	**+/+**	+/+	+/+	+/+	+/+	**-/+**
Wave V present (+/-)	**+/+**	+/+	+/+	+/+	+/+	**-/+**
I – III interval (msec)	2.2/2.3	1.9/1.8	1.9/2.0	1.8/1.9	1.9/1.9	**-**/2.1
III – V interval (msec)	1.8/1.9	2.2/2.1	1.8/1.7	2.0/1.9	2.0/2.1	**-**/2.2

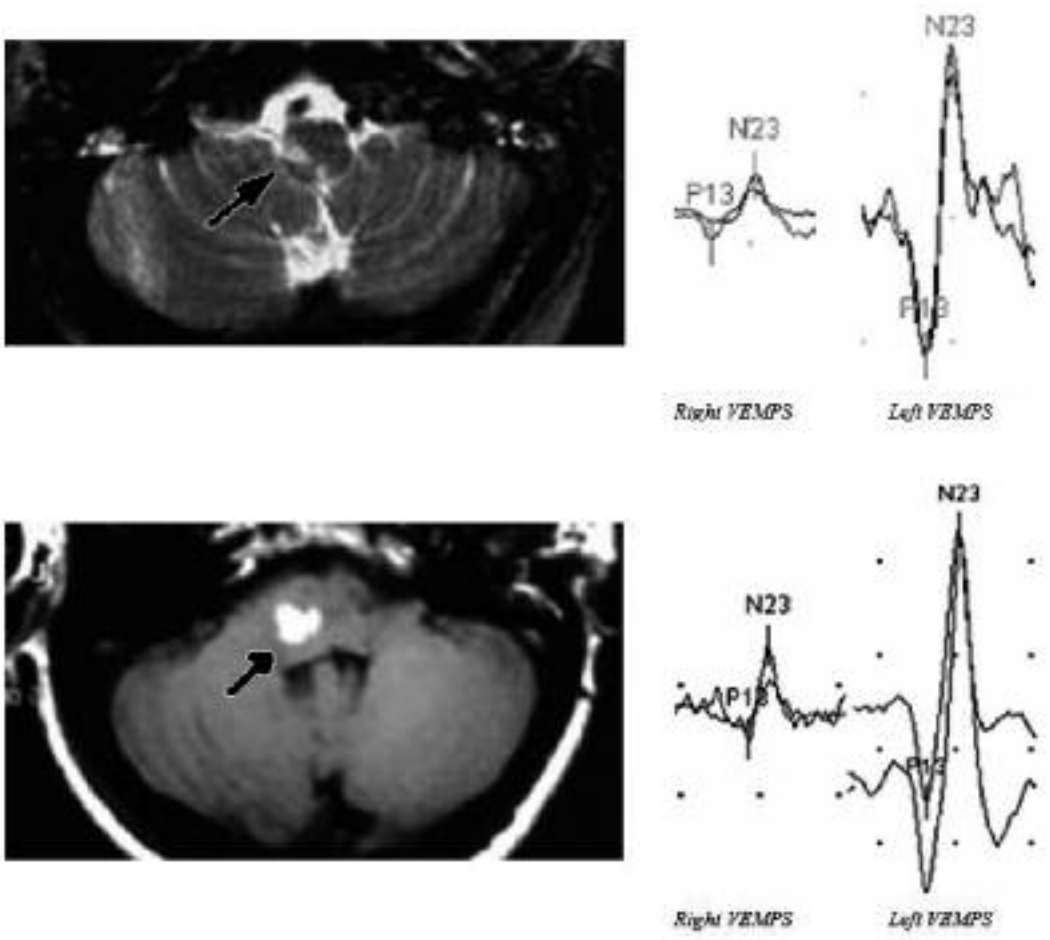

Figure 1
MRI and VEMP recordings of cases one and three, showing a right dorsolateral medullary infarct and a right lower pontine haemorrhage respectively. VEMP amplitude is reduced in the affected side.

In cases four and five, a 35- and a 43-year-old Caucasian male respectively, brain MRI was normal, but MRA revealed dolichoectasia of the basilar artery exerting pressure on the right lower lateral pons. In both cases, BR and BAERs remained unaffected, but the amplitude of the p_{13}-n_{23} VEMP wave obtained from the right ear was markedly reduced. VEMP latencies were again within normal limits.

In case six, a 55-year-old Caucasian male, the MRI revealed an ischaemic lesion of the right upper lateral pons. The R1, R2i and R2c BR components were delayed upon stimulation of the right side, whereas they were normal upon stimulation of the left side. Similarly, BAER waves IV and V were absent on the right but remained normal on the left. VEMPs were bilaterally normal.

Discussion

VEMPs represent myogenic responses evoked by acoustic stimulation delivered to the saccule, transmitted mainly via the inferior vestibular nerve to the ipsilateral vestibular nuclei, vestibulospinal tract and cervical muscles.[4] Vestibular evoked myogenic potential (VEMP) testing has become a well-established approach for the exploration of the sacculocollic pathways.[5]

It is currently used in the evaluation of peripheral vestibular disorders such as vestibular neuronitis, Meniere disease and herpes zoster oticus.[4] VEMPs have also been applied to the study of the central vestibulopathy caused by Multiple Sclerosis[7,8] and by Wallenberg syndrome.[9] VEMP amplitudes are raised and thresholds are pathologically

lowered in superior semicircular canal dehiscence presenting with the Tullio phenomenon.[10,11]

VEMPs are affected by brainstem lesions involving the vestibular nuclei, which are located dorsolaterally at the limit of the medulla and the pons.[9,12] BAERs, on the other hand, ascend the central auditory pathway in the brainstem via the pontine cochlear nuclei. They are affected by lesions involving the middle and upper pons.[12] Finally, the R1 blink reflex response is mediated by the ipsilateral principal nucleus of the trigeminal nerve and by that of the facial nerve, both located at the middle pons, while $R2_i$ and $R2_c$ are mediated by the medullary and spinal nuclei of the trigeminal nerve.[1] BR is therefore affected by middle/upper pontine and medullary lesions.

The Evoked Responses obtained in our patients were consistent with their anatomical lesions: in Wallenberg syndrome (patient one) both the lower pons and the medulla are involved. VEMPs and BR were therefore affected, as opposed to BAERs, which remained normal. In lower lateral pontine lesions VEMPs were the only affected evoked response (patients two, three, four and five). In upper lateral pontine lesions (patient six) BAERs and BR were affected, while VEMPs remained normal (Figure 2).

Our results support the value of VEMPs for the localisation of brainstem lesions; they provide information on the functional status of the lower lateral pons, which cannot be evaluated by BAERs and BR. Taken together, the three evoked responses make possible the neurophysiological evaluation of brainstem functionality up to the level of the upper pons.

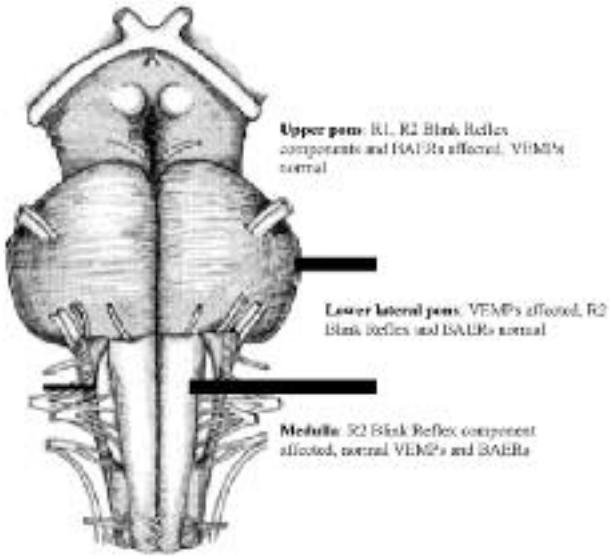

Figure 2
MRI and VEMP recordings of cases one and three, showing a right dorsolateral medullary infarct and a right lower pontine haemorrhage respectively. VEMP amplitude is reduced in the affected side.

It is of interest to note that, although the amplitude of VEMPs was reduced in the types of lesions examined in this study, their latency remained within normal limits (Table 1). Previous reports yielded conflicting results on this issue; VEMPs have been found to be absent, delayed[12,13] or diminished[12] in patients with brainstem infarct or haemorrhage. It has also been suggested that wave latencies are increased in demyelinating diseases, such as multiple sclerosis, in which there is a deceleration in the conduction of signals along neural axons.[7] Our cases favour the view that stroke, haemorrhage and mechanical pressure, which are characterised by neural cell destruction, mainly affect the amplitude of VEMP responses rather than their latency.

A limitation of VEMPs is that they require a certain degree of cooperation. The patient needs to be able to hold his head a few centimetres above the pillow for approximately 50 seconds, which is the duration of each recording. It may not therefore be possible to obtain them during the first few days after brainstem lesions, when such cooperation may not be possible. In all our cases, neurophysiological investigation became feasible within one week from the onset of symptoms.

In conclusion, our results suggest that VEMPs may be a useful addition to other existing neurophysiological techniques in the evaluation of brainstem function. VEMPs, BAERs and the blink reflex are mediated by different neural pathways and can therefore be combined to allow better localisation of brainstem lesions.

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