SHORT REPORT

Blink reflexes and magnetic resonance imaging in focal unilateral central trigeminal pathway demyelination

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Abstract

The electrically elicited blink reflex allows quantitative analysis of the corneal reflex which traverses the trigeminal and facial nerves and the brainstem. Two patients presenting with symptomatic unilateral trigeminal lesions are described, in whom the blink reflexes showed conduction block and slowing at predictable sites in the central pathways, and magnetic resonance imaging confirmed precisely the clinical and electrophysiological localisation.

Unilateral electrical stimulation of the supra-orbital nerve (SON) evokes two separate contractile responses in the orbicularis oculi muscle. The early (R_1) component is evoked only on the side of stimulation as a pontine reflex, whereas the late (R_2) component is recorded bilaterally and is presumed to be relayed through polysynaptic pathways of the pons and the lateral medulla.¹⁻³ The normal values and variations of this reflex have been well established,²⁻⁴ and alteration of the reflex in a variety of neurological diseases has been recognised.^{1-3,5} We report two cases of focal central trigeminal pathway demyelination, each of which demonstrated a different pattern of blink reflex (BR) abnormality, and which correlated with the lesions identified by magnetic resonance imaging (MRI).

Methods

BR were recorded using the technique of Kimura *et al.*³ Eight responses were measured from each nerve and the onset latencies were compared to laboratory normal values, which were comparable to those established previously.^{3,4}

MRI was performed on both patients using a 1.5 Tesla Philips Gyroscan and T-1 and dual echo T-2 weighted axial and coronal images were obtained.

Case reports

Case 1 A 39 year old male presented with a one month history of left facial numbness. It started as a buzzing sensation and numbness along the upper lip which progressed over a

week to involve the maxilla and the lateral canthus of the eye, the frontal scalp, forehead, buccal mucosa and gums. There was no associated pain, thermolability or weakness of bite.

Examination showed a dense multimodal sensory deficit in the territory of the left ophthalmic division of the trigeminal nerve, including the cornea. In the maxillary division the preauricular and supra-auricular regions were spared. Over the mandible and chin there was hyperalgesia, pallanesthesia and hypoaesthesia. Two-point discrimination threshold was increased to 7.0 mm on the left upper lip. There was no motor involvement. The remainder of the neurological examination was normal with the exception of mild weakness of right ankle dorsiflexion and right lower limb hyperreflexia. A contrast cranial CT scan, VEPs and median and posterior tibial nerve SEPs were all normal. BAERs showed 0.3 ms relative prolongation of the I-V interpeak latency to left ear stimulation. The cerebrospinal fluid (CSF) protein was 0.47 g/l (0.15-0.45), the IgG 0.06 g/l (< 0.05) and the IgG/total protein ratio 13% (0-14) without oligoclonal bands. BR recordings (fig 1) showed an absent R_1 component to left SON stimulation with normal R_2 component latencies bilaterally.

Five weeks later, after symptomatic recovery, the R_1 component amplitude to left SON stimulation had normalised, but was considerably delayed (6 ms) compared to the right R_1 latency. R_2 latencies remained normal and comparable from both sides. A coronal dual echo T-2 weighted MR scan showed a small circular area of high signal intensity (fig 2), approximately 4 mm in longitudinal extent at the rostral margin of the left brachium pontis and ventral tegmentum. This lesion corresponded to the location of the principal trigeminal sensory nucleus and spared the intra-axial mixed sensory and motor trigeminal nerve fascicles.

Case 2 A 45 year old female who had an episode of acute left optic neuropathy two years earlier, presented with a three day history of right maxillary hypoaesthesia, which then extended to involve the medial aspect of the cheek, upper lip, buccal mucosa and gums followed by the periorbital region, the frontal scalp and forehead, but spared the chin. There

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Figure 1 Blink reflexes (re-traced) from the left and right orbicularis oculi muscles. Note the absent ipsilateral R_1 components from both patients at the initial study, with only Case 2 showing delay of the R_2 components, and the subsequent return of a delayed ipsilateral R_1 at the second study of Case 1.

was tactile and thermal hypoaesthesia and hyperalgesia to pinprick in the medial territories of the ophthalmic and maxillary divisions of the right trigeminal nerve including corneal and intraoral sensation. In the mandibular division tactile hypoaesthesia and hyperalgesia were confined to the lower lip. Left ankle hyperreflexia was the only other significant finding. BAERs were normal. BR recorded to right SON stimulation showed the R_1 to be absent and the R_2 components were delayed bilaterally. The R_1 and both R_2 respon-

ses were normal to left SON stimulation (table). MRI demonstrated a circular lesion (fig 2) in the right pons with a diameter of approximately 14 mm. This was situated ventral to the fourth ventricle and extended rostrocaudally between the midpons and high medulla and laterally to involve the principal sensory and spinal tract trigeminal nuclei.

Discussion

The dissociated and partly segmental facial

Figure 2 Coronal T-2 weighted (TR 2500 ms; TE 100 ms) images from Case 1 (left) and Case 2 (right) showing a small area of high signal intensity (arrow) at the rostral margin of the left brachium pontis and ventral tegmentum (Case 1) and a larger circular area of high signal intensity (arrow) in the right pons (Case 2). Note also other areas of hyperintense signal in the cerebral hemisphere of each case.

Table Mean latencies of early (R_1) and late (R_2) blink reflex components recorded from both orbicularis oculi muscles to unilateral supraorbital nerve electrical stimulation

	Mean Latency (msec)		
	Ipsilateral R_1	Ipsilateral R_2	Contralateral R_2
Case 1 (14.7.88)			
Right supraorbital nerve	10.7	30.7	34.3
Left supraorbital nerve	Absent	33.4	35.3
Case 1 (25.8.88)			
Right supraorbital nerve	10.3	32.4	32.1
Left supraorbital nerve	16.2	29.5	30.9
Case 2			
Right supraorbital nerve	Absent	43.5	48.8
Left supraorbital nerve	11.9	30.4	30.7

sensory loss accompanied by contralateral crural pyramidal signs in each patient implied a small localised central lesion in the ventrolateral caudal pontine tegmentum. This clinical localisation was confirmed in both cases by MRI and correlated with the BR findings, which in turn provided additional pathophysiological information on the effects of demyelination in the central trigeminal pathways. To our knowledge this is the first detailed anatomical and pathophysiological correlation of single lesions involving this pathway, and in Case 1, the resolution of conduction block enabled accurate quantitation of the degree of conduction slowing through the demyelinated oligosynaptic portion of the BR pathway.

The sensory nuclei of the trigeminal nerve comprise three different parts, together extending from the mesencephalon downward to the second cervical segment of the spinal cord. Rostrocaudally these are the mesencephalic, the principal and the spinal tract nucleus. The first is thought to mediate proprioceptive impulses from the muscles of mastication.⁶ The latter two nuclei are concerned primarily in the transmission of tactile sensibility, and pain and thermal sensibility of the face, respectively, and there is a rostrocaudal "onion-skin" somatotopy within the spinal tract and its nucleus.⁷ The fibres of the sensory root, on entering the pons, traverse its basilar portion, coursing dorsomedially, in the direction of the principal sensory nucleus. Many of the fibres then divide into short ascending rami ending in the principal sensory nucleus and long descending rami giving off collaterals to the spinal nucleus which extends through the medulla to overlap with Lissauer's tract. In our patients, there was clinical involvement predominantly of fibres projecting from the principal sensory nucleus in the pons. This resulted in tactile hypoaesthesia, with some involvement of axons of the rostral spinal tract and nucleus⁷ causing hyperalgesia and thermal hypoaesthesia.

Although the orbicularis oculi reflex was first described by Overend in 1896,⁸ it did not receive systematic evaluation until 1952.⁹ The precise central pathways are still in question. Tokunaga *et al*¹⁰ has suggested that the early reflex (R_1) is mediated via the principal nucleus, while the late responses are probably transmitted via multisynaptic pathways that include at least the spinal tract and nucleus. Hence delay of the R_1 response suggests an ipsilateral pontine lesion, provided the facial and trigeminal nerves are intact.¹ Delay of the

R_2 responses may also occur when the R_1 is delayed due to more extensive pontine lesions involving the descending axons projecting to the spinal nucleus and tract.¹ Kimura¹¹ reported results of analysis of the BR obtained from 260 patients with suspected multiple sclerosis. The most consistent and significant delays in R_1 in this and other studies^{11,12} were found in those patients with an internuclear ophthalmoplegia, suggesting that the reflex pathway of R_1 is located near the medial longitudinal fasciculus. In none of the patients studied by Kimura,¹¹ however, was there accurate visualisation of the lesions responsible, nor was more widespread involvement of brainstem pathways able to be excluded.

In Case 1 the initial BR study showed severe conduction block in the short latency pathway (R_1 component) with normal R_2 component latencies. The second study five weeks later, soon after symptomatic recovery, demonstrated the resolution of conduction block with the normalisation of the R_1 component amplitude, which together with the 6 ms delay of R_1 characterised the lesion as demyelinating. The restriction of the BR abnormality to the R_1 component indicated the lesion involved only the principal nucleus and/or its central connections in the oligosynaptic pathway and spared the spinal tract and nucleus as was shown by MRI. In contrast to this small selective lesion, Case 2 showed both an absent R_1 and prolonged R_2 latencies. This pattern of abnormality suggested the pontine lesion also involved the descending axons to the spinal tract and nucleus in addition to the principal nucleus and its connections which correlated with the larger MRI lesion. In other studies of pontine demyelination this pattern of R_2 latency increase comparable with the R_1 increment is the most commonly encountered.^{1,10,11}

Our cases illustrate some important points. Firstly, demyelinating lesions confined to a small CNS pathway can now be imaged and correlated with the electrophysiological changes to define the functional significance of such lesions.¹³ Secondly, Case 1 demonstrated the cardinal electrophysiological features of a primary demyelinating process, and the degree to which an isolated single lesion will prolong the latency of the short pontine reflex pathway immediately following the resolution of conduction block. This degree of conduction slowing is comparable to that which occurs in the recovery phase of demyelinating human¹⁴ and experimental¹⁵ optic neuropathy. Finally, the differential effects on conduction in the oligosynaptic and the polysynaptic BR pathways illustrate the selectivity of the small lesion in Case 1 and confirms the original observations of Tokunaga *et al*¹⁰ and Kimura¹ that the former traverses the principal sensory nucleus and the ipsilateral facial nerve, whereas the latter incorporates the descending spinal tract and nucleus and their projections to the adjacent reticular formation.⁶

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