Multiple sclerosis plaque related to abnormal somatosensory evoked potentials

W. B. MATTHEWS AND MARGARET ESIRI

From the University Department of Clinical Neurology, Churchill Hospital, and the Department of Neuropathology, Radcliffe Infirmary, Oxford

SUMMARY A patient with mild multiple sclerosis died from a ruptured intracranial aneurysm. It was possible to relate abnormalities of somatosensory evoked potentials recorded some months earlier to a plaque involving the root entry zone in the cervical spinal cord which had not resulted in clinical sensory abnormalities.

Averaged evoked potential techniques are being used increasingly in the diagnosis of multiple sclerosis. The underlying assumption is that persistent abnormalities of evoked potentials, in the absence of relevant clinical symptoms or signs, are evidence of persistent structural lesions of the central nervous system. Opportunities to verify this assumption must necessarily be rare and arise by chance. Death in multiple sclerosis usually occurs after a prolonged period of severe disability during which investigations are seldom performed. The identification at necropsy of symptomless plaques in such patients would in any case be impossible. No anatomical verification of the relationship of a multiple sclerosis plaque to abnormalities of somatosensory evoked potentials (SEP) has been published. A patient is described in whom death from other causes while mildly affected by multiple sclerosis allowed this relationship to be established.

Case report

The symptoms of this woman (CV) began in 1967 at the age of 39 years when she developed retrobulbar neuritis of the left eye and tingling in the fingers of both hands followed by recovery within a few months. In 1968 vision of the left eye was again affected, a central scotoma being documented on hospital attendance. In 1969 she developed paraesthesiae in the right foot and was found to have incoordination of the right lower limb but no other signs. This again recovered, but in 1970 there was a transient episode of weakness of the left leg. Remission persisted until March 1974 when the left leg again became weak, on this occasion with incomplete recovery.

She first attended the Radcliffe Infirmary in February 1977 having sustained a further episode of retrobulbar neuritis affecting the right eye in December 1976. Visual acuity was R 6/5, L 6/12. Both optic discs were pale. There was no nystagmus. She had no symptoms or abnormal signs in the upper limbs and, in particular, there was no sensory loss to stringent clinical examination. There was generalised weakness of the left lower limb with increased tendon reflexes and an extensor plantar reflex but again no sensory loss.

Visual evoked potentials (VEP) and SEP were examined on 21 March 1977. No measurable VEP could be recorded on stimulation of either eye with an alternating checkerboard pattern. A normal cervical SEP was recorded on stimulation of the right median nerve (Matthews et al., 1974; Small et al., 1978). On stimulation of the left median nerve the normal pattern of N11, 13, and 14 peaks (Jones, 1977) could not be recognised (Fig. 1). An early cortical wave (N20) was recorded with a latency of 23 ms on stimulation of the right median nerve but no early potential could be evoked from the left median nerve.

When seen in May 1977 the patient had experienced no fresh symptoms. On 25 October she was admitted in coma having sustained a subarachnoid haemorrhage. Decerebrate rigidity developed, and she died two days later. A necropsy was performed. The brain was sliced coronally after fixation. There was a saccular aneurysm on...
In the absence of histological evidence of fresh plaque formation or of clinical evidence of recent relapse it is reasonable to believe that the plaques found at necropsy were those present at the time of recording the evoked potentials seven months earlier. It was not possible to relate visual function and abnormalities of VEP with the anatomical lesions because the intraorbital portions of the optic nerves were not examined.

The abnormality of short latency SEP can,
however, clearly be attributed to the plaque in the cervical spinal cord involving the root entry zone and lateral aspect of the posterior column on the appropriate side and at the appropriate level. This finding cannot throw any light on the source of subcortical SEP, whether spinal cord or more rostral structures (Wiederholt, 1978), but confirms that the method can detect clinically silent plaques in the sensory pathways. The plaque was clearly responsible for the spastic weakness of the left lower limb but had caused no sensory symptoms or abnormal signs in the upper limb with the possible exception of tingling in the hands 10 years earlier. The basic assumption underlying the diagnostic use of somatosensory evoked potentials in multiple sclerosis is thus confirmed.

References


Multiple sclerosis plaque related to abnormal somatosensory evoked potentials
W. B. Matthews and Margaret Esiri

J Neurol Neurosurg Psychiatry 1979 42: 940-942
doi: 10.1136/jnnp.42.10.940

Updated information and services can be found at:
http://jnnp.bmj.com/content/42/10/940

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/