Letters

The latency of the anal reflex

Sir: There is debate of the latency of the spinal anal reflex. Henry and Swash originally gave a value averaging about 9 ms (range 6-9 to 11-5 for 10 women and 7-5 to 9-4 for three men) when recording by concentric needle electrodes, placed posterior to the midline of the anal verge, on bipolar stimulation of the perianal skin. This would give a surprisingly fast conduction velocity to and from the cord. Others have suggested that the real latency of the anal reflex is closer to 50 ms (range 30 to 60)—see also this volume page 767. The discovery that it is possible to stimulate the spinal cord directly with large anodal shocks applied to the skin over the back has given us the opportunity to measure the latency of the motor volley from the lower sacral segment to the external anal sphincter. A shock of 700 volts, decaying with a time constant of 100 μs applied with the anode over the spine of L1 evoked a biphasic action potential recorded from the external anal sphincter by a Post Office telephone jack in the anal canal (fig). The well-defined latency of this response is 7 ms, corresponding to a conduction velocity of roughly 60 m/s (the distance from this man’s L1 spine to the anal verge is some 40 cm). A similar latency was obtained in a second subject. These results suggest that the minimal monosynaptic latency for any spinal anal reflex is unlikely to be less than about 14 ms, and cutaneous polysynaptic reflexes would probably take much longer to appear.

CD Marsden
PA Merton
HB Morton
University Department of Neurology,

Figure

Action potential from the external anal sphincter on stimulation at three times threshold of the conus at the L1 spinous process. Trace length 50 ms shock artifact at 10 ms, onset of response at 7 ms, vertical calibration 0-5 mV. Subject PAM, 2 March 1982.

References

liver function tests and arterial blood gases were normal. Radiotherapy was administered for presumed neoplasm. Mental functions again failed accompanied by right hemiparesis. No changes were discernable by CT scan and asterixis could not be elicited. The patient's course terminated in severe dementia.

Since the original delineation by Adams and Foley, unilateral asterixis has been an accepted sign of metabolic encephalopathy of exogenous or endogenous origin. Uraemia, hepatic failure, hyponatraemia, hypercapnea and drugs have been implicated. The pathophysiology of the motor disturbance is, however, uncertain. Leavitt and Tyler have suggested that failure of integration of proprioceptive impulses promotes the motor disturbance.13 Shahani and Young suggest that dysfunction occurs in a central motor system for the maintenance of sustained muscle contraction.2 Unilateral asterixis has been described as a consequence of structural lesions in the brain. Vascular events in the mesencephalon, a thalamus and internal capsule,3 stereotactic thalamotomy,4 and parietal lobe lesions5,6,7 have been associated with unilateral asterixis of the contralateral extremity. Discrete lesions in mesencephalic, thalamic and parietal lobe structures may disrupt somatosensory impulse integration, attention to stimuli and facilitation of motor activity, thus producing unilateral asterixis.8

To the best of our knowledge, the unusual synchrony of loss of muscle tone observed in this case and the association of asterixis with obstructive hydrocephalus have not been previously described. The patient reported by Tarsey et al did not show asterixis in the period preceding ventricularperitoneal shunting for communicating hydrocephalus.9 Presumably, intraventricular pressure was normal in their case. Bilateral alternating asterixis in this patient was thought secondary to biventricular hydrocephalus caused by an anterior midline haemorrhage, possibly of neoplastic origin. Dexamethasone, cinetidine, or phenytoin, however, may have acted in concert with hydrocephalus to yield this sign. Patients recovering from unilateral stereotactic thalamotomy for Parkinson's disease may show asterixis contralateral to the lesion when challenged with phenytoin.9 Biventricular obstructive hydrocephalus may produce asterixis because intracranial hypertension affects brain functions globally as in metabolic encephalopathy. Alternatively, pressure on nearby brain structures, particularly the thalamus, may cause bilateral focal disturbances of the ascending reticular activating system.10 The contribution of plateau waves and pulsatile arterial pressure to the origin of this sign is unknown.

The authors are grateful to Mr Richard De Young for preparation of the figures and Ms Cynthia Smith for typing the manuscript.

WH WEINREB
JR PERRY
LR JENKYN

Division of Neurology,
Department of Medicine,
Roger Williams General Hospital,
Brown University,
Providence, RI, 02908, USA

References

Matters arising

Subacute sensory neuropathy

Sir: The interesting case of neuropathy associated with Hodgkin's disease described in your journal by Sagar and Read1 is difficult to classify. They reported the case under the title “subacute sensory neuropathy”, yet their patient's illness progressed to prevent walking within three weeks, remained unchanged for four weeks and then improved coincidentally with treatment of the Hodgkin's disease. The course of the illness and the slowed motor conduction (25 m/s in the legs) surely resemble the Guillain-Barré syndrome2 more closely than subacute sensory neuropathy3 despite the predominance of sensory involvement. The well recognised association between Guillain-Barré syndrome and Hodgkin's disease4,5 may be due to depression of cell-mediated immune responses which protect the body from herpes infections or autoimmune processes or both. So long as the nosological limits of Guillain-Barré syndrome2,6 and subacute sensory neuropathy3 remain poorly defined, disputes about diagnoses like this will continue. It would be preferable to discard the old terms and describe the disease in as much detail as investigations permit. Sagar and Read's case would become “acute demyelinating motor and sensory neuropathy”. This description would at least have the merit of accuracy, and could be abbreviated to ADMSN.

RAC HUGHES
Department of Neurology,
Guy's Hospital
St Thomas St
London SE1 9RT, UK

References