## Letters

## The latency of the anal reflex

Sir: There is debate of the latency of the spinal anal reflex. Henry and Swash<sup>1</sup> 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. Others2 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<sup>3</sup> 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



Fig 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.

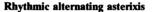
well-defined latency of this response is 7 ms, corresponding to a conduction velocity of roughly 60 m/s (the distance from this man's L1 spine to the anal verge is some 40 cm). A similar latency was obtained in a second subject. These results suggest that the minimal monosynaptic latency for any spinal anal reflex is unlikely to be less than about 14 ms, and cutaneous polysynaptic reflexes would probably take much longer to appear.

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## References

- <sup>1</sup> Henry MM, Swash M. Assessment of pelvicfloor disorders and incontinence by electrophysiological recording of the anal reflex. *Lancet* 1978;1:1290-1.
- <sup>2</sup> Pedersen E, Harving H, Klemar B, Tørring J. Human anal reflexes. J Neurol Neurosurg Psychiatry 1978;41:813-8.
- <sup>3</sup> Marsden CD, Merton PA, Morton HB. Percutaneous stimulation of spinal cord and brain; pyramidal tract conduction velocities in man. *Proc Physiol Soc* 1982;328:6P.



Sir: Asterixis commonly accompanies metabolic encephalopathy<sup>12</sup>and rarely focal brain lesions.<sup>3</sup> It is usually bilateral and ...ynchronous.<sup>12</sup> Unilateral asterixis has been described in patients without metabolic disturbances who harbour focal structural lesions.<sup>4-9</sup> The unusual finding of rhythmic bilateral alternating asterixis was observed in a patient suffering midline frontal haemorrhage and biventricular obstructive hydrocephalus. Asterixis resolved after decompressive surgery.

A 67-year-old woman was admitted to Roger Williams General Hospital after two weeks of confusion, difficulty in walking and falling backwards. Examination revealed mild hypertension (160/95 mm Hg), inattentiveness, retropulsion, and disinhibited glabellar blink and palmomental reflexes bilaterally. Computed tomography (CT) demonstrated a parasagittal blood density at the foramina of Monro, obliterating the anterior horn of left lateral ventricle and causing biventricular obstructive hydrocephalus (figure). amethasone, phenytoin, and cimetidine were administered regularly. Despite mannitol infusion for increasing lethargy, right hemiparesis evolved. Repeat CT scan showed enlargement of both lateral ventricles. Cerebral angiography revealed premature filling of a large draining vein in the wall of the left lateral ventricle. Examination one day later showed blunted affect

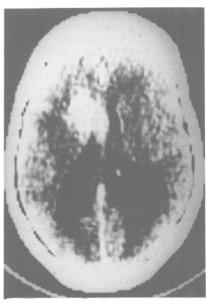


Figure Computed tomography with contrast shows a non-enhancing inhomogeneous blood density in the region of the head of the caudate nucleus with dissection into the midline, effacement of the anterior horn of the left lateral ventricle, and dilated bodies and posterior horns of both lateral ventricles.

without spontaneity of thought or action and strikingly rhythmic asterixis. With wrists and fingers extended, one hand lost tone then re-extended just as the other lost tone and fell. A distinctly sinusoidal pattern occurred that was regular in frequency, sustained in duration and difficult to mimic. A third CT scan suggested enhancement of the lesion with further enlargement of the lateral ventricles. Alternating asterixis persisted. Electroencephalography showed bifrontal intermittent rhythmic delta activity. After insertion of a ventriculoperitoneal shunt on the right, disappearance of the asterixis and a brighter, more spontaneous affect were noted. At surgery, ventricular cerebrospinal fluid was reported to be "under high pressure." Repeat CT scan showed collapse of the right lateral ventricle. Throughout the course, phenytoin levels remained constant, medication (including cimetidine) was unchanged and serum electrolytes, blood sugar, BUN, creatinine,