It is in this large group of patients therefore that the interpretation of the lumbar puncture result is critical, because failure to diagnose a ruptured aneurysm can be fatal.

The point of contention is the definition of xanthochromia. In the paper by MacDonald and Mendelow,¹ xanthochromia was determined by direct vision; in Vermeulen's paper, xanthochromia was determined by spectroscopy. Since the vast number of reports issued in the West of Scotland were based on visual inspection, and since most laboratories in the North of England similarly base their reports on visual inspection and not spectroscopy (15/15 laboratories recently surveyed), then the absence of xanthochromia cannot be taken as excluding a subarachnoid haemorrhage. If practice in Holland is such that a spectroscopic test is produced routinely in all hospitals, then Vermeulen et al² are correct in their environment, but their conclusions would be invalid in many hospitals in the United Kingdom, where visual inspection remains the normal practice.

Care should therefore be taken in interpreting their paper, and a ruptured aneurysm cannot be excluded on the basis of absent xanthochromia unless a spectroscopic examination has been shown to be negative.

It will also be important to know the long term fate of the nine patients who they failed to subject to angiography: four years is a relatively short follow-up period for a suspected subarachnoid haemorrhage.

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Pathology of neuroleptic malignant syndrome

Dr Jones and Dawson reported myopathic changes consisting of increase in muscle fibre size, vacuolation, segmental necrosis and regeneration in a fatal case of neuroleptic malignant syndrome (NMS).¹ We have recently studied the histopathology of this disorder and our observations are at variance with these findings. The most conspicuous feature in our case was excessive and irregular contraction of muscle fibres within mild oedema but no muscle necrosis or evidence of regeneration. Histochemical staining was normal except for mild depletion of glycogen and lipid, probably due to utilisation. Electron microscopy showed disintegration of Z bands, the remaining ultrastructure being normal. There was no primary myopathy in our case. Oedema and glycogen depletion in NMS (in addition to muscle necrosis) were also observed by Drs Bakheit and Behan, but myopathy alone may reflect the difference in severity and outcome of the illness in the two patients, rather than a difference in underlying pathology.

We agree that study of muscle biopsy in more patients with NMS is needed to better identify the changes that occur; this concurs with Dr Harriman's conclusions, in his review of MH myopathy, of the benefits of histopathology.

Drs Dawson and Jones reply

Our publication aimed, in the discussion of the pathogenesis of NMS, to focus attention on muscle rather than the central nervous system, as we found a striking picture of toxic myopathy in skeletal muscle but only non-specific changes in the brain. The observation, by Drs Bakheit and Behan, of pathological change in the Z-bands in muscle in their patient, supports this shift in focus.

The changes we found in muscle led us to support suggestions that a common mechanism underlies both NMS and malignant hyperthermia (MH). Drs Bakheit and Behan's observation of muscle Z-band disintegration on electron microscopy, and reference to the same changes being seen in MH, supports our views again.

There was no clinical indication of a pre-existing myopathy in our case. The differences in the muscle changes reported by us, and those observed by Dr Bakheit and Behan, may well reflect the difference in severity and outcome of the illness in the two patients, rather than a difference in underlying pathology.

Unilateral paresis of the abdominal wall

I have read with interest the letter from FPJ Billet, H Ponssten and D Veenhuizen.¹ We do not agree with the authors when they say: "This radical syndrome has not been described before." In fact LJ Benaim et al² published two similar observations in 1986.²³ We would like to point out the interest of EMG in these cases.² The study of the abdominal wall muscles allows us to affirm the peripheral neurogenic character of the pseudo-eventration of the lower and lateral part of the abdomen. The study of the paravertebral higher lumbar muscles, when they show positive sharp waves, suggests the radicular origin of the symptoms.

Unilateral paresis of the abdominal wall.

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