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,[3] 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 report is produced routinely in all hospitals, then Vermeulen *et al*[1] 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

Drs 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 with 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 reported by Martin and Swash.[2] Interestingly, Z band disintegration was also observed in malignant hyperthermia[3] suggesting a common pathogenetic mechanism for these clinically indistinguishable conditions.

Our patient had a non fatal illness and the absence of a myopathy is probably due to the mild nature of the disease and the prompt initiation of therapy with dantrolene sodium. Assuming that a pre-existing myopathy had been excluded in Drs Jones' and Dawson's patient, the discrepancy between their findings and ours could be explained by the different disease severity in the two patients. On the other hand, postmortem changes probably account for the absence of hypercontractile muscle fibres in their case.

It would appear that there is a wide spectrum of pathological changes in NMS depending on disease severity. Muscle biopsies in a large number of patients with NMS will help to resolve this question.

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2 Martin D, Swash M. Muscle pathology in the neuroleptic malignant syndrome. *J Neurol* 1987;**235**:120–1.
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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 change 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 Drs 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.

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.

Unilateral paresis of the abdominal wall

I have read with interest the letter from FPJ Billet, H Ponssen and D Veenhuizen.[1] We do not agree with the authors when they say: "This radicular syndrome has not been described before." In fact LJ Benaim *et al* published two similar observations in 1986.[2,3]

We would like to point out the interest of EMG in these cases.[2,3] The study of the abdominal wall muscles allow 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.

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1 Billet FPJ, Ponssen H, Veenhuizen D. Unilateral paresis of the abdominal wall: a radicular syndrome caused by herniation of the L1 L2 disc? *J Neurol Neurosurg Psychiatry* 1989;**52**:678.
2 Benaim LJ, Papy JP, Servant JM, Givaudan JF, Acquaviva PC. Atteinte des deux premières racines lombaires et paralysie des nerfs abdomino-génitaux, données cliniques et électriques. Revue d'EEG. *Neurophysiol* 1985;**15**:251–4.
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BOOK REVIEWS

Manual of Clinical Problems in Neurology — 2nd Edition. Edited by J P Mohr, M.D. (Pp 400. Price: £9·95.) Little Brown Spiral Manual. Distributors: Edinburgh: Churchill Livingstone. 1989.

The 12 authors contributing to this text maintain a consistent quality and absence of stylistic unevenness, which is in itself remarkable. Since the first edition, four years ago, essential additions have been made to cover newly developing fields on brain imaging and AIDS and a successful attempt has been achieved in updating the text.

This manual was developed against the needs of internists preparing for board examinations in the specialty and also for busy clinicians in the specialties of neurology and internal medicine; awareness of the needs of senior medical students, electively studying neurology, was also taken into account. The manual succeeds on all these scores. It is refreshing to read. It emphasises conciseness and accuracy and although there are minor differences in emphasis in the transatlantic experience, most particularly in a more active or aggressive approach to therapeutics, this is as good and useful a manual of the clinical problems in neurology as any I have recently read.

Practical aspects of clinical neurology are comprehensively and interestingly covered. Appropriate background neuroanatomy and physiology are included where relevant, and are apt and precise. The inclusions of sections on paediatric neurology and laboratory studies are a welcome addition to a book of this kind and are reliably instructive for those not in everyday contact with the subject at specialist level. Similarly, the chapters on brain stem and cranial nerve disorders, which include a careful and well written appraisal of neuro-ophthalmological disorders, will appeal to those with an ophthalmological inclination. For the clinical neurologist, this provides a valuable clinical resumé especially