copic polyarteritis. Both conditions require prompt treatment with cyclophosphamide, and the distinction between the two may be semantic only. We believe that early treatment with cyclophosphamide in this case, based on the finding of serum ANCA, prevented further deterioration in renal function and possible pulmonary involvement.

Serum testing for ANCA is a useful non-invasive investigation in patients who present with unusual neuropathies, particularly if there is evidence of sinusitis.

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MATTERS ARISING

Korsakoff's psychosis

The letter by Feinstein and Ron¹ raises the interesting diagnostic dilemma which occurs when alcohol abuse may be present alongside another neurological condition such as multiple sclerosis. I would, however, query the description of their patient as suffering from alcoholic Korsakoff's (AK) syndrome.

In the absence of a firm history of Wernicke's encephalopathy or residual signs of Wernicke's disease, the diagnosis of an AK syndrome is rather more difficult and, as is implicit in the Feinstein and Ron paper, relies mainly on current neuropsychological features of the patient's condition. Feinstein and Ron appear to consider two features of their patient's memory disorder as being especially important for this diagnosis—evidence of occasional confabulatory responses, and impaired anterograde memory test scores.

In the case of confabulatory responses, it is now generally accepted² that confabulation is not a diagnostic hallmark of the AK syndrome, particularly in its chronic phase, even if the severity of confabulation is more severe than the "elicited" form which was found by Feinstein and Ron. The fact that confabulation and poor memory can be functionally dissociated³ further limits the importance of this symptom as a reliable index of an amnesic syndrome.

In the case of anterograde memory test scores, the performance of their patient on the word recognition memory task was above the range of scores obtained by 11 amnesic

patients on this test,⁴ and was also higher than the scores obtained by the two AK patients reported by Mair *et al*⁵ or the six AK patients studied by Squire and Shimamura.⁶ Furthermore, neither the study by Mair *et al* nor that by Squire and Shimamura found the significant asymmetry of performance on words and faces memory testing which was reported by Feinstein and Ron. It is quite possible that the poor faces and picture memory scores of their patient were simply secondary to a visuo-perceptual/visuospatial impairment, as suggested by the patient's low Performance IQ, rather than related to a global memory disorder *per se*.

There is one important feature of AK patients' memory functioning which was not covered in the report by Feinstein and Ron, namely retrograde memory performance. In the case of their patient, this is critical for two reasons. Firstly, significant retrograde memory loss appears to be a hallmark of the AK amnesic syndrome. In my review of memory disorders,⁷ I was unable to find a single case of an AK patient with marked anterograde memory impairment who did not also have poor retrograde memory functioning. Although remote memory deficits may be present in some patients with chronic-progressive multiple sclerosis,⁸ the pattern of memory loss differs in the two conditions—a temporal gradient in the memory loss is present in AK patients under most test conditions, and severe retrograde amnesia in the absence of generalised cognitive dysfunction is a feature of the global memory disorder of many AK patients, but less so in the case of patients with multiple sclerosis, where remote memory loss in patients without significant dementia is only mild or moderate in severity.

Secondly, marked retrograde memory loss is a characteristic which also distinguishes AK patients from those with neuropsychological deficits resulting simply from chronic alcohol consumption alone.^{9,10} Feinstein and Ron's brief clinical observations of their patient's retrograde memory functioning suggest a rather patchy retrograde amnesia, with the patient reported as having poor memory for past personal events but intact memory for some details of his previous medical history, and if this suggestion of a limited retrograde amnesia was upheld by formal assessment it might be more in keeping with multiple sclerosis rather than an AK syndrome. Since marked anterograde memory deficits and other cognitive dysfunction can occasionally be present in patients with multiple sclerosis who do not have severe neurological disability,^{11,13} this diagnosis alone could explain most of the evidence reported by Feinstein and Ron.

While it is quite possible that a proportion of the anterograde memory impairment shown by Feinstein and Ron's patient may have been due to chronic alcohol abuse, it would have been useful (allowing for the normal gamma GT) to know the duration of abstinence to exclude the role of any recent, acute effects of alcohol consumption on memory functioning. It may also have been useful to document brain stem auditory evoked responses to examine the possibility of differences which have been noted between some Wernicke-Korsakoff patients and those with multiple sclerosis.¹⁴ In the case of other neuropsychological features of the case presented by Feinstein and Ron, I would agree that the Verbal-Performance IQ discrepancies, and the evidence of nominal dys-

phasia, are unusual for AK patients and probably reflect the impact of cerebral pathology related to demyelination (I assume that one of their statements was written in error—"In both Korsakoff's syndrome and multiple sclerosis verbal IQ's decline more than performance IQ's"—since their own supporting references indicate the reverse to hold!).

I would therefore consider that the case reported by Feinstein and Ron can at best be classified as impaired neuropsychological functioning resulting from multiple sclerosis, possibly exacerbated by the effects of chronic/recent alcohol abuse. Unfortunately, on the basis of the evidence reported, it cannot be reliably viewed as a case of Korsakoff's psychosis in the presence of multiple sclerosis.

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Xanthochromia in subarachnoid haemorrhage

We are pleased to see that the debate about the role of xanthochromia in subarachnoid haemorrhage continues (Vermeulen *et al*)¹ because lumbar puncture is still an important and commonly performed investigation in patients with sudden onset of severe headache. While a CT scan will reliably diagnose subarachnoid haemorrhage in 100% of patients who are confused or worse on the Glasgow Coma Score, it will be negative in 14% of orientated patients;² it is this group that represents the large number of patients with suspected subarachnoid haemorrhage.

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 hyperthermia³ 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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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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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