performance rating 3.2/3.0. Reported side effects on trazodone were tiredness (4 patients), constipation (1 patient) and drowsiness for the placebo (1 patient).

Due to the high drop out rate, the final number of patients in the study group was smaller than the minimum number we have recommended for clinical trials in essential tremor.⁷ However, our data were highly consistent for all patients and are similar to those of Koller⁸ who found there were no effects of trazodone in a similar group of 10 patients studied in the United States.

Despite an earlier optimistic report,⁵ these findings indicate that trazodone is ineffective in the control of ET. It seems unlikely therefore that serotonergic transmission is directly involved in the genesis of ET. Drugs increasing dopamine, acetylcholine and GABA transmission have been similarly ineffective.⁹ Further neuropharmacological probes may be helpful in exploring the origin of ET. However, it would seem unlikely that a deficiency in a single neurotransmitter system represents the primary pathology in ET.

LYNN CLEEVES,
LESLIE J FINDLEY
MRC Neuro-Otology Unit,
National Hospital for Nervous Diseases,
Queen Square,
London WC1E 3BG.

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Low prevalence of HTLV-1 antibodies in the serum of patients with tropical spastic paraplegia from the Ivory Coast

A high prevalence of human T-lymphotropic virus type I (HTLV-1) antibodies has been reported in the serum of patients with tropical spastic paraplegia (TSP), from various parts of the world. This disease is also present in West Africa where, as in other tropical regions, no obvious aetiology was discovered.

To discover the possible link between TSP and HTLV-1 antibodies in West Africa, the serum of 20 patients from the Ivory Coast was collected. All patients fulfilled the clinical diagnostic criteria previously described for TSP.¹ Other causes of paraparesis were excluded by clinical and paraclinical inves-

Table Results of HTLV-1, HIV-1 and HIV-2 antibodies in the serum of 20 TSP patients from the Ivory Coast (ELISA and western-blot methods)

	HTLV-1	HIV-1	HIV-2
Patients 1 to 15	-	-	-
Patient 16	+	-	-
Patient 17	+	-	+
Patient 18	+	-	+
Patient 19	-	+	+
Patient 20	-	-	+

tigations. The presence of HTLV-1, HIV-1 and HIV-2 antibodies was determined by ELISA and western-blot methods. The results are summarised in the table. One patient was positive only for HTLV-1, two patients were positive for HTLV-1 and HIV-2, one was positive for HIV-2 and one was positive for HIV-1 and HIV-2. High HTLV-1 antibody titres were found ranging from 1/5000 to 1/10 000.

The total prevalence of HTLV-1 positivity among TSP patients was 15%. This observation contrasts with the high prevalence of HTLV-1 positivity reported in the other tropical regions, particularly in the Seychelles (85%)¹ and in Martinique (59%).²

The seroprevalence of HTLV-1 among healthy controls in the Ivory Coast (1.6%)³ is similar to that observed in the French West Indies (2%).⁴

These findings confirm that in West Africa antibodies against retroviruses other than HTLV-1 are present in the serum of patients with TSP, as we reported previously.⁵ The lack of HTLV-1, HIV-1 and HIV-2 antibodies in 75% of patients with TSP indicate that different aetiologies may be linked to TSP in West Africa, such as other viruses, toxins or malnutrition.

Our results underline the need for a larger study of TSP in Africa to evaluate the role of various aetiological factors with the geographical distribution of the disease throughout the continent.

This work was supported by a grant from The French Ministry of Cooperation (DPR RS423).

J HUGON,*
JM VALLAT,*
M DUMAS,*
M VERDIER,
F DENIS,†
F AKANI,‡
YF BOA,‡
C GIORDANO,‡
Institute of Tropical Neurology and
Department of Neurology,*
Faculté de Médecine, Limoges,
Department of Bacteriology and Virology,†
Hôpital Universitaire Dupuytren,
Limoges, France
Department of Neurology,‡
CHU d'Abidjan, Ivory Coast

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Accepted 1 September 1989

Wegener's granulomatosis presenting as peripheral neuropathy: diagnosis confirmed by serum anti-neutrophil antibodies

The case described by Kirker, Keane and Hutchinson¹ illustrates that neuropathy may occur early in Wegener's granulomatosis in the absence of more classical pulmonary and renal findings, leading to delay in diagnosis. Even if the diagnosis is suspected, histopathological confirmation may be difficult. We describe a patient presenting with rapidly progressive peripheral neuropathy and inconclusive biopsy findings for whom the demonstration of serum autoantibodies to neutrophil cytoplasm antigen (ANCA) permitted early diagnosis and therapy.

A 57 year old man developed rapidly progressive numbness and weakness of both hands and the right leg over a period of 10 weeks. He had right wrist and foot drop and marked wasting of both hands. Right elbow, wrist, hand, ankle and foot movements were MRC grade 2-3; limb power elsewhere was grade 4. Fine touch and pinprick sensation were diminished over both hands, right foot and the lateral aspect of the right leg; other sensation was unimpaired. He had a vesiculopapular rash on his elbows and hands but no other abnormality, and urinalysis was negative.

Urea, electrolytes, haemoglobin and chest radiograph were normal. White cell count was $19.9 \times 10^9/l$, 74% neutrophils, ESR 38 mm/hour, and serum C-reactive protein 45 mg/l. Serum alkaline phosphatase was 1031 U/l, alanine transaminase 211 U/l, and aspartate transaminase 73 U/l. Urinary creatinine clearance was moderately reduced at 58 ml/min. Rose-Waaler was positive (1 : 1280). Serum B12, blood lead, abdominal ultrasound scan and CSF were normal. Screening for porphyria, hepatitis viruses and antinuclear and antimitochondrial antibodies were negative, digital renal arteriography showed no aneurysms, and the only abnormality on CT scanning of the skull, thorax and abdomen was fluid in both maxillary sinuses. ENT assessment revealed a granular mass on the right postnasal space. Biopsies of this area and of the skin lesions showed non-specific chronic inflammation. Despite unhelpful histopathological findings, Wegener's granulomatosis was suspected and the presence of serum ANCA was confirmed on indirect immunofluorescence (titre 1 : 80).

The patient was started on cyclophosphamide 2 mg/kg and a reducing dose of prednisolone. Six months later right wrist extension and finger movements were grade 3; limb power was otherwise normal. Sensory impairment was limited to the fingertips and the right sole. ESR, C-reactive protein, white cell count and creatinine clearance were normal, Rose-Waaler negative, and ANCA not detectable.

Histopathological confirmation of Wegener's granulomatosis may be difficult. Parlevliet et al² obtained a firm histological diagnosis in only 2 of 11 patients with typical symptoms and signs of the disease. While a presumptive diagnosis is acceptable when the presentation is typical, the decision to start cytotoxic therapy with its attendant morbidity is difficult in atypical cases.

Savage et al³ showed that ANCA was not present in a small series of patients with polyarteritis nodosa and Churg-Strauss syndrome, but was highly sensitive and specific for Wegener's granulomatosis and micros-

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.

W DICKEY

Department of Neurology,
Royal Victoria Hospital
Belfast BT12 6BA.

WJ ANDREWS

Whiteabbey Hospital,
Doagh Rd, Newtownabbey BT37 9RH.

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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.

NARINDER KAPUR
Wessex Neurological Centre,
Southampton

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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.