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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, 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 inef-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.

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1 Herskovits E, Blackwood W. Essential (familial hereditary) tremor: A case report. J Neurol Neurosurg Psychiatry 1969;32:509-11. 2 Dubinsky R, Hallet M. Glucose hypermetabol-

ism of the inferior olive in patients with essential tremor. Ann Neurol 1982;22:118

3 Llinas R. Rebound excitation as the physiological basis for tremor: a biophysical study of the oscillatory properties of mammalian central neurones in vitro. In Findley LJ, Capildeo R (eds). Movement Disorders: tremor. London: Macmillan, 1984:165-82.

4 Longo VG, Massotti M. Effect of tremorigenic

Longo VG, Massotti M. Effect of tremorigenic agents on the cerebellum. Int Rev Neurobiol 1985;26:315-29.
 McLeod NA, White LE. Trazodone in essential tremor. J AM Med Assoc 1986;256:2675-6.
 Cleeves L, Findley LJ. Beta adrenoreceptor mechanisms in essential tremor: a comparative single dose study of the effect of a non-selective and a beta-2 selective adrenoreceptor antagonist. J Neurol Neurosurg Psychiatry, 1984:47:976-82.

Psychiatry 1984;47:976–82.
7 Cleeves L, Findley LJ. Variability in amplitude of untreated essential tremor. J Neurol Neurosurg Psychiatry 1987;50:704-8. 8 Koller W. Trazodone in essential tremor: Probe

of serotonergic mechanism. Clin Neurophar-

of scotoletgic incentation. Oth Neuropharmacol 1989;12:134-7.
 Findley LJ. The pharmacological management of essential tremor. Clin Neuropharmacol 1986;9 (suppl 2):S61-S75.

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. Other causes of paraparesis were excluded by clinical and paraclinical inves-

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

	HTLV-1 HIV-I		HIV-II
Patients 1 to 15	_	_	_
Patient 16	+	_	-
Patient 17	+	_	+
Patient 18	+	_	+
Patient 19	_	+	+
Patient 20	-	_	+

tigations. The presence of HTLV-1, HIV-1 and HIV-II 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-II, one was positive for HIV-II and one was positive for HIV-I and HIV-II. 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 Seychelles (85%) 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 (200).

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 lack of HTLV-1, HIV-I and HIV-II 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).

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> Department of Bacteriology and Virology,† Hôpital Universitaire Dupuytren, Limoges, France Department of Neurology,‡ CHU d'Abidjan, Ivory Coast

1 Roman GC, Schoenberg BS, Madden DL, et al. Human T-lymphotropic virus type antibodies in the serum of patients w

tropical spastic paraparesis in the Seychelles.

Arch Neurol 1987;44:605-7.

2 Gessain A, Barin F, Vernant JC, et al.

Antibodies to human T-lymphotropic virus

Antibodies to human T-lymphotropic virus type-I in patients with tropical spastic paraparesis. Lancet 1985;ii:407-10.

3 Verdier M, Sangare A, Denis F, et al. Seroepidemiology of HTLV-I in Ivory Coast. Abstracts of the IV International Conference on AIDS, Stockholm (June 1988).

4 Gessain A. Epidemiologie du virus HTLV-I et des maladies associées en Martinique et Guyane Française. Thèse Doctorat en Médecine (Paris): 1987:83.

Médecine (Paris); 1987:83.

5 Hugon J, Giordano C, Dumas M, et al. HIV-2 antibodies in Africans with spastic paraplegia. Lancet 1988;i:189.

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 19.9×10^9 /l, 74° _o neutrophils, ESR 38 mm/l 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 Wegengranulomatosis 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-