tonic seizures associated with contralateral paraesthesia, they postulated transversely spreading activation between axons in the spinal cord as the mechanism. Similarly, Matthews[7] proposed that the underlying pathophysiology in paroxysmal attacks in multiple sclerosis is abnormal lateral axonal spread of excitation in plaques. A normally transmitted impulse, on reaching a plaque of demyelination, depolarises a neighbouring demyelinated axon. This ephaptic conduction leads to the attack, the nature of which is dependent on the afferent or efferent axon involved. Osterman and Westerberg[8] explain how transversely spreading activation of axons within a partially demyelinated plaque accounts for various paroxysmal phenomena by describing in detail the relevant anatomy of the spinal cord and brain stem.

Thus, paroxysmal attacks in multiple sclerosis are considered to be due to axonal discharge rather than neuronal discharge through usual anatomical and physiological connections. Although the beneficial effect of the anticonvulsant drug carbamazepine seems to suggest that paroxysmal attacks in multiple sclerosis might be due to focal epilepsy, carbamezapine has effects on the axonal membrane thereby blocking ephaptic conduction[9] as well as its better known effects on neuronal discharge.

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Development of HTLV-I associated myelopathy (HAM) in a seroconverted patient for antibody to HTLV-I

Sir: Seroconversion in recipients of transfused human T lymphotrophic virus type-I (HTLV-I) antibody positive blood was reported by one of the authors (KO) in 1984.[1] Osame *et al* suggested that infection transmitted by blood transfusion could be a basis for the development of HTLV-I associated myelopathy (HAM).[2,3] However, the development of HAM caused by blood transfusion has not been previously reported.

A 70 year old Japanese woman was admitted to our clinic on August 20 1988 in a lactic acidotic coma with hypoglycaemia caused by calcium-hopantenate.[4] She had had an operation for a DeBakey type I dissecting aneurysm and had been given a blood transfusion on May 22, 1985. She had gradually developed spastic paraplegia six months to one year after the operation. From July 1986 she had been bed-ridden with complete paraplegia as well as sensory and sphincter disturbances. No orthopaedic abnormality was detected. On this admission, she recovered from the coma within nine days but the spastic paraplegia, sensory and sphincter disturbances remained unchanged. Magnetic resonance imaging (MRI) revealed no abnormality in the spinal cord. A high anti-HTLV antibody titre (1280) in the serum and cerebrospinal fluid (CSF) (320) was detected by an indirect immunofluorescence method,[1] indicating HAM. After checking the transfusion history, it was revealed that the patient had been in a follow up study for transfusion-transmitted infection of HTLV-I started in this hospital in 1981.

Anti-HTLV-I antibody was not detected before her operation but she became positive (antibody titre = 5 at one month, 80 at two months later). During her operation the patient received one unit of anti-HTLV-I antibody positive packed red cell (PRC, antibody titre = 640) and one unit of antibody positive fresh frozen plasma (FFP, antibody titre = 640), as well as antibody negative 26 units of PRC and 10 units of FFP all of which were antibody negative. We believe this to be the first reported case of a patient developing HTLV-I HAM from a blood transfusion. To prevent similar occurrences a donor screening programme was set up in Japan in 1986.

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Parkinsonian symptoms in a patient with AIDS and cerebral toxoplasmosis

Sir: Extrapyramidal symptoms of bradykinesia, rigidity and tremor, have been reported as rare presentations of brain tumour,[1] subdural haematoma[2] and tuberculoma.[3] We report the case of an AIDS patient with parkinsonian features due to bilateral basal ganglia toxoplasma abscesses.

In 1983 the patient, a 66 year old white female, had a resection of a right cystic hamartoma of the bile duct with incidental right adrenalectomy. During her stay in hospital, she required seven units of blood. She did well postoperatively until June 1984 when she developed a disseminated petechial rash. The platelet count was 4,000/mm^3 and the laboratory investigations were consistent with an autoimmune thrombocytopenia. With high dose prednisone, the platelet count rose to 12,000/mm^3. Incidental toxoplasma titres were drawn and revealed an IgG of 1:4096 and an IgM of 1:128. In