The clinical presentation of bitemporal field loss with a third nerve palsy was considered typical of a pituitary adenoma and this diagnosis was compatible with the CT scan appearance. Involvement of the third nerve is common in large pituitary tumours due to either compression against the interclinoid ligament or invasion of the cavernous sinus. The pathological finding was of an ependymoma involving the pituitary fossa. Although we cannot exclude the possibility that the tumour arose in the third ventricle and extended downwards into the sella, an intrasellar origin of an ependymoma may be possible.

Ependymomas are primary glial tumours presumed to arise from a cell related to the ependymal lining. Although these tumours are usually related to the ventricular system, a connection with the ventricular ependyma could only be identified in six out of 14 cases in one series and primary ependymomas have been described in extra-axial soft tissue locations. The presence of either embryological remnants of the ependymal cleft within the sella or heterotopic ependymal lining cells may explain the unusual location of the ependymoma in our case. In a 10 week (45 mm) human fetus the neurohypophysis develops as an elongated outpouching of neuroepithelial cells which encloses a cavity that is continuous with the cavity of the neural tube. The infundibulum at this stage consists of undifferentiated ependymal cell precursors. The ependymal cleft is readily identified at 13 weeks (60 mm) but recedes by 16 weeks (112 mm) and is not present at birth. Isolated ependymal cells could be left behind in the infundibulum and neurohypophysis, as rests or heterotopias and these may undergo subsequent neoplastic transformation.

We are not aware of any other similar examples of this very unusual presentation of a glial tumour.

We are grateful to Dr C J Earl and Mr N Grant for permission to report this case.

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Paroxysmal dysarthria and ataxia: associated
MRI abnormality

Sir: Paroxysmal dysarthria and ataxia has
been reported in multiple sclerosis, but such
episodes may be the presenting feature of this
disease. In common with a variety of
different paroxysmal symptoms seen in multi-
ple sclerosis, the episodes are characteristi-
cally sudden in onset, brief in duration, and stereotyped for an individual patient and
tend to remit over a period of time. We report a case of paroxysmal dysarthria and
taxia and the associated finding on mag-
ettomic resonance imaging (MRI).

A 31 year old physiotherapist presented
with a two week history of episodes of
incoordination of the left arm and leg with
simultaneous dysarthria. Each episode
occurred without warning and lasted up to
six seconds. The paroxysms occurred with
increasing frequency (up to 20 episodes per
day) until carbamazepine therapy was
initiated six weeks after the onset of symp-
toms. General and neurological examination
between attacks was completely normal.
During an episode, ataxia of the limbs and
dysarthria were observed, although full
examination was not possible due to the
brevity of the episodes.

Investigations revealed normal visual,
auditory and somatosensory evoked poten-
tials. The cerebrospinal fluid (CSF) con-
tained three lymphocytes/m\(^3\) and a total
protein content of 0.5 g/l. Oligoclonal bands
were present on CSF protein electrophoresis.
CT brain scan was normal, but MRI of the
brain revealed a solitary lesion in the deep
white matter of the left cerebellar hemi-
sphere, abutting without deforming the
fourth ventricle and dorsal aspect of the
cerebellum. No other abnormality was seen.

MRI of the brain was repeated three
months after the first scan and the solitary
lesion involving the left middle cerebellar
peduncle was unchanged.

Carbamazepine (100 mg tds) was started
one month after presentation and further
paroxysms of dysarthria and ataxia occurred
at a rate of only one per day. On disconti-
nuous the carbamazepine, the number of parox-
syms increased to the previous frequency of
up to 20 per day. Reintroduction of the drug
completely suppressed the attacks.

Using the classification proposed by Pose
et al, a diagnosis of multiple sclerosis cannot
be confirmed in this patient, despite the
supportive evidence of CSF oligoclonal bands—there are to date no symptoms, clinical nor paraclinical evidence (as defined by Pose et al) of a second lesion in the CNS of this patient. Nevertheless, paroxysmal
dysarthria is so characteristic of multiple
sclerosis, and has been described as patho-
nomonic for this disease, that an alternate
diagnosis is unlikely.

The symptoms responded successfully to
treatment with carbamazepine but this
dramatic response of paroxysmal attacks in
multiple sclerosis to carbamazepine has been
previously well described. The MRI lesion
in our patient, which probably represents
a solitary plaque of demyelination, demo-
strated anatomically a correlation with the
symptoms of paroxysmal dysarthria and
ataxia.

The mechanism of ephaptic conduction is
widely believed to be the cause of paroxys-
mal attacks. When Ekboim et al reported

Fig MRI showing high signal from lesion in left
cerebellar hemisphere.
tonic seizures associated with contralateral paraesthesia, they postulated transversely spreading activation between axons in the spinal cord as the mechanism. Similarly, Matthews 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 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, carbamazepine has effects on the axonal membrane thereby blocking ephaptic conduction 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. Osame et al suggested that infection transmitted by blood transfusion could be a basis for the development of HTLV-I associated myelopathy (HAM). 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 lac tic acidotic coma with hypoglycaemia caused by calcium-hypoparathyreosis. 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, 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.


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, subdural haematoma and tuberculosis. 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/mm3 and the laboratory investigations were consistent with an autoimmune thrombocytopenia. With high dose prednisone, the platelet count rose to 12,000/mm3. Incidental toxoplasma titres were drawn and revealed an IgG of 1:4096 and an IgM of 1:128. In