fossa component involved the 3rd cranial nerve.

Plain radiographs were normal except in one case where there was erosion of the ipsilateral posterior clinoid process. As might be expected with a schwannoma, CT showed an isodense or slightly hyperdense mass with a well defined edge, which enhanced after intravenous contrast. When the tumour is embedded in the side of the brain stem at the level of the tentorial edge, it may be difficult to determine whether it is intrinsic or extrinsic until the scan has been repeated with intrathecal contrast, as in the present patient.

In all six patients who had surgery, a total removal was achieved with good functional recovery. In the case reported here, the tumour was removed from below via a suboccipital craniectomy. In the other patients the subtemporal transtentorial route was used.

It seems possible that schwannomas of the trochlear nerve and other nerves controlling external ocular movement may have been under-diagnosed in the past, being mistaken for trigeminal schwannomas. The curious fact that large "trigeminal" schwannomas may be associated with little or no disturbance of trigeminal nerve function is well known.[1] Before the invention of CT it was difficult to diagnose a tumour situated at the medial end of the petrous ridge before it became so large that it was difficult at the time of operation to determine with certainty the nerve of origin. Schwannomas of the oculomotor nerves arise close to the trigeminal sensory root and a large tumour, as in the second patient with a trochlear schwannoma described by Leunda *et al* may bridge the medial petrous ridge into the middle fossa like a trigeminal schwannoma.[4] Before operation the disturbance of external ocular movement may be minimal, as in the patient described in this report.

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An ependymoma involving the pituitary fossa

Sir: Ependymomas arising in the region of the third ventricle may occasionally cause visual loss by compressing the optic chiasm. Standard references of surgical neuropathology also refer to ependymomas in the differential diagnosis of intrasellar mass lesions, although we were unable to find any well documented cases. We therefore report a patient with clinical and radiological features of pituitary tumour which proved on transphenoidal removal to be an intrasellar ependymoma.

An 81 year old man had a six month history of progressive visual failure affecting particularly both temporal fields. There was a five month history of continuous headache felt over the right eye and three months of intermittent double vision with horizontal separation of images. His relatives had noticed a right ptosis. There was slight postural dizziness but no other symptoms to suggest endocrine dysfunction. Examination revealed visual acuities of 6/18 and 3/60 in the left and right eyes respectively, with a bitemporal hemianopia. There was a complete right third nerve palsy with preserved fourth and fifth nerve function. Blood pressure was 100/70 without postural drop.

He had a raised serum prolactin at 536 mu/ml (normal range 3–178 mu/ml) and a reduced free T4 (7 pm/l n > 8·8). CT scan (fig 1) showed a large enhancing mass lesion arising in the pituitary fossa and extending out of the sella, which was considered typical of a pituitary adenoma.

Because of the severity of the symptoms and the progressive visual failure, transphenoidal removal of the intrasellar tumour was carried out. The floor of the fossa was partially deficient and beneath a thin rim of pituitary the tumour was located and evacuated from the sella and suprasellar regions. Operation was uneventful, with no CSF leak noted, and was followed by immediate improvement in visual acuity in the right eye to 6/36. A few days following the operation he developed a fever and increasing confusion. Cerebrospinal fluid (CSF) removed at lumbar puncture contained 960 white cells/ml, with a protein of

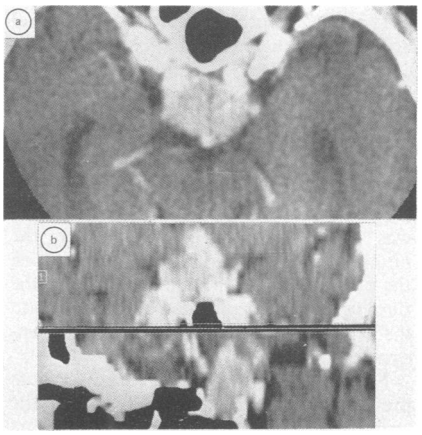

Fig 1 *(a) A high definition CT scan in the immediate suprasellar region; (b) computerised coronal and saggital showing a continuous intra-suprasellar lesion.*

580 mg% and 0·6 mm/l of sugar. Microscopy showed numerous gram positive rods and cocci. Despite treatment with intravenous cloramphenicol, metronidazole, penicillin and steroids he rapidly deteriorated and died three days later. Permission for post mortem was refused.

The pathological specimen consisted of several small fragments of grey tissue with a gelatinous consistency in some parts. Examination of paraffin sections stained for routine histology (fig 2) showed a tumour made up of elongated cells arranged in loosely packed rows and perivascular pseudorosettes. The tumour cells had eccentric pleomorphic nuclei, coarse chromatin and there were moderate numbers of mitotic figures. Glial fibrillary acidic protein, a marker for astrocytic-ependymal differentiation, was present in the processes of the tumour cells. This appearance is diagnostic of a moderately malignant ependymoma.

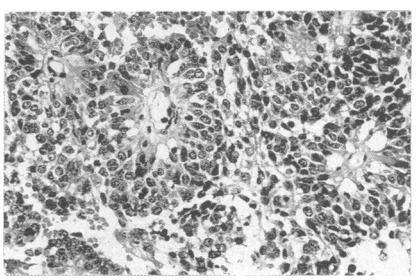

Fig 2 *Photomicrograph showing tumour cells arranged to form perivascular rosettes around thin capillaries (haematoxylin and eosin *300).*

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.[1] 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.[2] 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[3] and primary ependymomas have been described in extra-axial soft tissue locations.[4,5] 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.[6] 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,[1] and such episodes may be the presenting feature of this disease.[2] In common with a variety of different paroxysmal symptoms seen in multiple sclerosis, the episodes are characteristically sudden in onset, brief in duration, stereotyped for an individual patient and tend to remit over a period of time. We report a case of paroxysmal dysarthria and ataxia and the associated finding on magnetic resonance imaging (MRI).

A 31 year old physiotherapist presented with a two week history of episodes of incoordination of the left arm and left 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 symptoms. 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 potentials. The cerebrospinal fluid (CSF) contained 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 hemisphere, abutting without deforming the fourth ventricle and dorsal aspect of the pons. No other abnormality was seen. (fig)

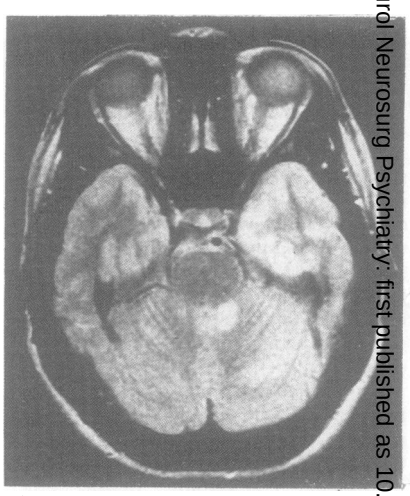

Fig *MRI showing high signal from lesion in left cerebellar hemisphere.*

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 discontinuing the carbamazepine, the number of paroxysms increased to the previous frequency of up to 20 per day. Reintroduction of the drug completely suppressed the attacks.

Using the classification proposed by Poser *et al*,[3] 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 Poser *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 pathognomonic for this disease,[4] that an alternative diagnosis is unlikely.

The symptoms responded sucessfully to treatment with carbamazepine but this dramatic response of paroxysmal attacks in multiple sclerosis to carbamazepine has been previously well described.[5] The MRI lesion in our patient, which probably represents a solitary plaque of demyelination, demonstrated anatomically a correlation with symptoms of paroxysmal dysarthria and ataxia.

The mechanism of ephaptic conductions is widely believed to be the cause of paroxysmal attacks. When Ekbom *et al*[6] reported