Although occlusion of the basilar artery has been well studied and documented in adult patients,¹² particularly after the work of Kubik and Adams,¹³ it is very uncommon in childhood. It is characterised by sudden alterations to the state of consciousness with motor and neuro-ophthalmological anomalies in the majority of cases.^{6,12} In one third of all patients there are prodromal episodes with transient or reversible neurological disturbances.^{6,9} Occlusion of the basilar artery may give rise to the locked-in syndrome caused by ventral pontine ischaemia, although other sites of origin also have been described.¹⁴ Pontine lesions were confirmed in our patient by ocular bobbing and CT scan images of decreased density at the pons.

The diagnosis of occlusion of the basilar artery must be established through angiography. The examination of the four branches is advisable to rule out congenital vascular anomalies, laminar flow and spasm of the vertebral or basilar arteries.^{4,7,10} The prognosis for occlusion of the basilar artery generally is poor with a mortality rate reaching 85% in adults¹² and nearly 25% in children, while there may be serious sequelae in survivors.⁹ In our patient death was not directly attributable to the occlusion of the basilar artery.

Mild cervical injuries have repeatedly been held responsible for different types of lesions in the vertebral arteries, such as tearing of the intima, thrombosis, aneurysms and dissections, which in turn may be the cause of severe neurological disturbances.¹⁵ In such cases the occlusion of the basilar artery may be produced by the thrombosis extending from the damaged vertebral arteries or through embolisms originating at the site of the thrombosis.^{7,11,15} The location of the arterial injury is not restricted to the occipitotlantoaxial level but includes the course through the transverse foramina, with sudden lateral rotating movements either with or without forced extension of the neck being the most frequent cause for this type of lesion.^{3,7,11,15}

Our patient had no signs of arteritis, cardiac embolism, congenital malformation or cervical lesions and therefore craniocervical injury appeared to be the most probable cause for the occlusion in spite of the normal angiographic findings for both vertebral arteries.

When occlusion of the basilar artery is suspected in a child, the appropriate angiographic studies should be performed in selected patients to confirm the diagnosis with a complete study to determine its possible aetiological factors. A previous cervical injury must always be looked for.

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Multiple plasmacytoma presenting as raised intracranial pressure

Sir: Plasma cell tumours may cause compression of the spinal cord or nerve roots and are sometimes associated with peripheral neuropathy or involvement of the central nervous system. The skull bones are often involved but intracranial complications are rare. We report an adult male with multiple plasmacytoma who presented with raised intracranial pressure.

A 40 year old man presented with severe occipital headache for 10 days and diplopia on looking to the right. He gave a 3 month history of low back pain. Examination revealed bilateral papilloedema, partial right lateral rectus palsy and limited straight leg raising on both sides. A CT scan of the head was normal. Opening pressure at lumbar puncture was over 400 mm CSF; protein, glucose, and gamma globulin content were normal and no cells were seen. The blood urea was elevated at 10 mmol/l with normal electrolytes. Bone alkaline phosphatase, calcium, plasma protein electrophoresis, plasma protein and albumin were normal. The ESR was 10 mm in the first hour, haemoglobin 12.0 g/dl, white cell count was $1 \times 10^9/l$ (normal distribution) with normal platelet count. A 24 hour urine collection revealed normal creatinine clearance with protein loss of 3.2 g.

A radiograph of the sacrum showed a large osteolytic lesion. Small "punched-out" lytic lesions were seen on radiographs of the skull and left clavicle and multiple deposits in sacrum, pelvis, ribs and spine were evident on the bone scan. Two bone marrow aspirates gave normal appearances and bone marrow trephine showed no evidence of metastases or myeloma. Biopsy of the sacrum revealed plasmacytoma. One month after admission, free kappa light chain globulins were found in the urine and accounted for 3.9 g in a 24 hour protein loss of 5.2 g. Soon afterwards, plasma protein electrophoresis revealed kappa chains plus a significant fall in plasma immunoglobulin levels. CSF electrophoresis and plasma viscosity were normal. CT scan of head at this stage revealed no intracranial abnormality; however, there was a large lytic lesion in the left temporal bone.

Despite melphalen, dexamethasone, allopurinol and radiotherapy, he deteriorated over the course of the following year with numerous pathological fractures and infections. He developed severe hypercalcaemia and later succumbed to a chest infection.

The diagnosis of multiple plasmacytoma was based on biopsy and radiological evidence of multiple bone lesions without

Fig High resolution agarose gel electrophoresis showing an abnormal protein band in patients serum and urine. (PAP3 = normal control pool serum sample).

cytological change in the bone marrow.¹ Multiple extramedullary plasmacytoma is a rare tumour with an incidence of about 1% of all plasma cell tumours.² Cranial and intracranial plasma cell tumours are also extremely rare. Cushing³ found that among 2000 intracranial tumours, only four were intracranial myelomas but he did not record the details.

Clarke⁴ reviewed 24 cases of cranial and intracranial plasma cell tumours from the literature and added four of his own. He distinguished three separate syndromes; multiple cranial nerve palsies, intracranial tumour formation, and a constellation of signs due to invasion of the orbit by plasma cell tumour. Intracranial abnormalities are thought to be due to plasmacytomas arising in the base of the skull.⁴ Alternatively it has been suggested that increased intracranial pressure could be due to abnormal globulin production inducing a hyperviscosity syndrome with protein deposition in the central nervous system.⁵ In our patient, CT scan showed that there was no significant mass lesion and plasma viscosity was normal. Dural involvement around the left mastoid may have lead to sinus thrombosis and consequent raised intracranial pressure.

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Abducent palsy after rapid shrinkage of a prolactinoma

Sir,—The sixth cranial or abducent nerve, by virtue of its anatomical course, is particularly susceptible to damage in pathological conditions affecting the cavernous sinus.¹ An expanding pituitary tumour may produce a sixth nerve palsy by lateral displacement and stretching of the cavernous portion of the nerve, often with accompanying third and fourth nerve damage.² We report the occurrence of a transient sixth nerve palsy in association with the rapid reduction in size of a pituitary prolactinoma induced by bromocriptine, a relationship not previously documented.

A previously well 31 year old man presented with a two-month history of blurring of vision of the left eye, and diminished libido and potency. He had no associated headache, diplopia or other symptoms. Examination of the left eye revealed a visual acuity of 6/12, an upper temporal quadrant field loss and pallor of the optic disc. The right eye was normal, as was the remainder of neurological and the general physical examination.

Skull radiographs showed massive expansion of the pituitary fossa, with destruction of the dorsum sellae and almost complete replacement of the adjacent sphenoid sinus. A large, partly cystic pituitary tumour with suprasellar, cavernous sinus and sphenoidal sinus extension was delineated by axial and coronal CT scanning (fig. a). Visual evoked potentials showed bilateral prolongation of latencies with attenuation of the major positive peaks, these abnormalities being more severe on the left. The serum prolactin, as measured by radioimmunoassay, was 11,300 ng/ml (normal less than 25). Other parameters of endocrine function were normal, including serum TSH, thyroxine, FSH, LH, growth hormone, cortisol and testosterone levels. Bilateral carotid and left vertebral angiograms confirmed significant suprasellar extension, with elevation of the A₁ segment of the anterior cerebral artery, and lateral displacement of the cavernous portions of the internal carotid arteries. No evidence of intracranial aneurysm formation or of arterial encasement was present.

Oral bromocriptine was commenced at a dosage of 2.5 mg nocte, gradually increasing to 15 mg per day. The patient's condition remained unchanged until 4 weeks after the institution of treatment, at which time he developed increasing horizontal diplopia over 4 days. There was no associated headache or changes in visual acuity. Exam-