Figure 1 Brain CT of head on admission showing mass effect and midline shift.

head injury. Brain CT showed a left sided acute subdural haematoma with mass effect (fig 1). The haematoma was evacuated promptly and an intraventricular catheter inserted to measure the intracranial pressure. The mean pressure was initially raised and thiopentone was therefore given to maintain it at 20 mm Hg or below. The patient was mechanically ventilated for 21 days, but was weaned off barbiturates after 10 days. During weaning, an eight channel EEG was recorded from Ag/AgCl scalp electrodes placed according to the International 10/20 system with mastoids linked in common reference. After recovery from the barbiturate induced burst suppression pattern, the EEG was dominated by unorganised polymorphic slow activity, which was severely attenuated over the left hemisphere. Transcranial Doppler ultrasound of the middle cerebral arteries showed mean flow velocities of 58 cm/s on the right and 16 cm/s on the left, indicative of hypoperfusion on the left. An increase in intracranial pressure caused a reduction in peak to peak amplitude of the EEG, a further increase produced burst-suppression, and finally electrocerebral silence occurred seconds after the intracranial pressure increased to 30 mm Hg (cerebral perfusion pressure = 45 mm Hg; mean arterial blood pressure – intracranial pressure). In the absence of a mechanical cause, immediate hyperventilation reduced intracranial pressure and as cerebral perfusion pressure exceeded 50 mm Hg the EEG activity returned (fig 2). The child regained consciousness without hemiparesis, but still had neurological impairment one month after his injury.

Intracranial pressure is best interpreted with either a neurological examination or some measure of cerebral blood flow (for example, transcranial Doppler ultrasound) and metabolism (EEG or jugular venous oxygen).¹ This single case study in a young child shows a critical intracranial pressure of 20 mm Hg for cerebral electrical activity. This level marks the transition from a favourable to a poor outcome in severely brain injured adults² and children.³

It has been suggested that cerebral perfusion pressure is the critical variable that determines outcome in severe brain injury.⁴ In adults the optimal level is generally considered to be 60 mm Hg, although recent findings indicate that it should be main-

Figure 2 Amplitude of EEG plotted against cerebral perfusion pressure.

tained above 70 mm Hg.⁴ A study in head injured children found a critical lower limit of 40 mm Hg, but the authors suggested that the principal aim of treatment should be to achieve a cerebral perfusion pressure of 50 mm Hg or more.⁵ The changes in the EEG in this child provide functional evidence for a critical value of 50 mm Hg and therefore support such a protocol.

In children several investigators have reported persistent cerebral blood flow and glucose metabolism in brain-dead children with electrocerebral silence.⁶ This dissociation of cerebral blood flow and metabolism from electrical activity has been explained by the expansile skull and fontanelles in young children and the activity of inflammatory microglial cells in the ischaemic cortex. The EEG may therefore be an acceptable alternative to other measures of cerebral blood flow and metabolism for the functional assessment of intracranial pressure, and the rationale for its treatment in young children.

In conclusion, these findings support the need for multimodality monitoring to interpret intracranial pressure measurements,^{1,4} and highlight potential advantages of the EEG in children. The aim of treatment in brain injured children should be to maintain intracranial pressure below 20 mm Hg and cerebral perfusion pressure above 50 mm Hg to optimise outcome.

We are grateful to Miss CA Rowlands and the neuroradiological department at Frenchay Hospital for their technical expertise, and Mr Steven Gill for his permission to report this case.

NICHOLAS M KANE

Burden Neurological Institute, Stoke Lane, Stapleton, Bristol, BS16 1QT, UK

TIMOTHY J GERMON

RICHARD J NELSON

Department of Neurosurgery, Frenchay Hospital, Frenchay, Bristol, BS16 1LE, UK

Correspondence to: Mr NM Kane.

- Pickard JD, Czosnyka M. Management of raised intracranial pressure. *J Neurol Neurosurg Psychiatry* 1993;56:845–58.
- Miller JD, Butterworth JF, Gudeman SK, Becker DP. Further experience in the management of severe head injury. *J Neurosurg* 1981;54:289–99.
- Michaud LJ, Rivara FP, Grady MS, Reay DT. Predictors of survival and severity of disability after severe brain injury in children. *Neurosurgery* 1992;31:254–6.
- Chan KH, Dearden NM, Miller JD, Andrews PJD, Midgely S. Multimodality monitoring as a guide to treatment of intracranial hypertension after severe brain injury. *Neurosurgery* 1993;32:547–53.

- Elias-Jones AC, Punt JAG, Turnbull AE, Jaspan T. Management and outcome of severe head injuries in the Trent region 1985–90. *Arch Dis Child* 1992;67:1430–5.
- Medlock MD, Hanigan WC, Cruse RP. Dissociation of cerebral blood flow, glucose metabolism, and electrical activity in pediatric brain death. *J Neurosurg* 1993;79:752–5.

Unusual presentation of a germ cell neoplasm

According to two reports, a metastasis to the CNS is a common complication of disseminated germ cell tumours of the testes. Metastatic disease occurred in 16% of 242 patients treated in one series¹ and in 15.1% in another.² The prognosis for patients with brain metastases from non-seminomatous germ cell tumours of the testes has improved over the past 10 years, as more effective chemotherapeutic protocols have been found.³

We document a case in which a patient presented with an intracerebral haemorrhage initially thought to be due to an arteriovenous malformation. A biopsy of the lesion taken at the time of evacuation of the haematoma, however, showed that this was due to an underlying carcinoma.

A previously fit and well 35 year old man presented with a sudden onset of headache associated with nausea and vomiting, while a passenger in his car. On admission he was restless, with a severe headache, but orientated and obeying commands. Neurological examination showed a left hemiplegia. His condition rapidly deteriorated and he showed signs of acute raised intracranial pressure including a dilated, non-reacting, right pupil.

He was initially resuscitated with 20% mannitol and ventilated. A brain CT showed a large right hemispheric intracerebral haemorrhage (fig 1).

An urgent craniotomy was performed. Old and fresh clots were evacuated and there were abnormal arteriolised veins in the periphery of the tumour bed surface

Figure 1 Brain CT showing a large right parieto-occipital haemorrhage.

Figure 2 (A) Haematoxylin and eosin and (B) uncounter stained immunohistochemical preparation of the choriocarcinoma composed of syncytiotrophoblasts showing strong positive immunoreaction with BHCG. Antibody dilution 1:100; originally $\times 250$.

together with prominent cortical veins. Haemostasis was achieved with some difficulty, and without the benefit of angiography no attempt was made to remove the lesion. Nevertheless small biopsies were taken.

After operation, the patient was ventilated, sedated, and paralysed. A subsequent chest radiograph showed that there was a well circumscribed 4×4 cm lesion in the upper left lobe.

Histology of the clot and tissue taken showed that the brain lesion was a metastatic deposit (fig 2A). The primary was initially assumed to be a carcinoma of the lung, which had metastasised to the brain. The patient was a heavy smoker and the prognosis was initially thought to be extremely poor. Ensuing examination, however, showed a firm nodular right testicle. Direct questioning of his wife later confirmed that this had been present for at least three months.

Grossly raised serum HCG was detected (13 815 IU) and immunocytological examination of the original cerebral tissue confirmed the presence of a choriocarcinoma (fig 2B). On the basis of this the patient was started on intravenous etoposide and cisplatin. At review two months later his condition had improved. Although he had a residual left hemiparesis and left homonymous hemianopia, he had retained full higher faculties. Tumour markers have since declined to 4 IU.

This case serves to illustrate several points: firstly, an intracerebral haemorrhage in a young person may represent an underlying neoplasm; secondly, a careful history from relatives and a meticulous examination of an unconscious patient may produce information of great relevance; thirdly, the importance of biopsy in vascular lesions of undetermined aetiology; and fourthly, the prognosis of a choriocarcinoma metastasising to the brain is relatively good.

All tumours metastasising to the brain have the potential to bleed, especially malignant melanomas. In a similar case report to this one, where two patients with known

brain metastases from a testicular tumour were diagnosed, intratumoural bleeding led to a right hemiparesis with sensory involvement and eventual death in one patient, and a left hemiparesis and eventual death in the other. These deaths occurred despite the early commencement of chemotherapy.⁴ Another case study reported complete remission of the growth of a metastatic teratoma from malignant testicular tumour, using salvage chemotherapy, despite an intratumoural haemorrhage.⁵

Pulmonary metastases in a patient with a known germ cell tumour should stimulate a search for further metastases in the CNS. Several studies show that nearly all patients diagnosed with brain metastasis from a testicular germ cell carcinoma already had radiologically detectable pulmonary metastatic disease.^{1,2,6,7}

One problem highlighted from the literature is that the efficacy of chemotherapy against brain metastasis is restricted by poor penetration of the blood-brain barrier. Radiation therapy is also limited and cannot eradicate a tumour. This has prompted some authors to advocate surgery in the management of a brain metastasis before chemotherapy,⁸ or if no response to chemotherapy was achieved.⁶ Elective surgical removal of accessible brain metastases larger than 1 cm, as indicated by Jelsma and Carroll,⁸ would avoid the possibility of a spontaneous intratumoural haemorrhage, or massive tumour lysis after chemotherapy associated with a haemorrhage,² which can lead to such devastating results in an otherwise treatable condition.

We acknowledge the Department of Medical Illustration, Greenwich District Hospital, London.

J TIMOTHY
A SOFAT
M SHARR
B DOSHI

Regional Neurosurgical Unit,
Brook General Hospital,
Woolwich, London

Correspondence to: Dr Jake Timothy, Department of Neurosurgery, Brook General Hospital, Shooter's Hill Road, Woolwich, London SE18 4LW, UK.

- 1 Vulgrin D, Cvikovic E, Posner J, Hajdu S, and Golbey RB. Neurological complications of malignant germ cell tumors of testes. *Cancer* 1979;44:2349-53.
- 2 Williams SD, Einhorn LH. Brain metastasis in disseminated germinal neoplasms. *Cancer* 1979;44:1514-6.
- 3 Athanassiou A, Beagent RHJ, Newlands ES, Parker K, Rustin GJS, Bagshaw KD. Central nervous system metastasis of choriocarcinoma. 23 years' experience at Charing Cross Hospital. *Cancer* 1983;52:1728-35.
- 4 Nishizaki T, Orita T, Tsuha M, Wakuta Y, Fujii M, Ito H. Brain metastasis of testicular tumor with massive hemorrhage—Report of two cases. *Neurol Med Chir (Tokyo)* 1991; 31:586-9.
- 5 Suwa H, Hanakita J, Nishi S, Nishihara K, Ohta F, Sakaida H, Iihara K. Complete remission of metastatic teratoma from malignant testicular tumor using salvage chemotherapy with VP-16 (Etoposide), CDDP, and ANCU—Case report. *Neurol Med Chir (Tokyo)* 1991;31:277-82.
- 6 Kobayashi T, Kida Y, Yoshida J, Shibuya N, Kageyama N. Brain metastasis of choriocarcinoma. *Surgical Neurology* 1982;17: 395-403.
- 7 Rustin GJS, Newlands E, Bagshaw KD, Beagent RHJ, Crawford SM. Successful management of metastatic and primary germ cell tumours in the brain. *Cancer* 1986;57:2108-13.
- 8 Jelsma RK, Carroll M. Brain metastasis from nonseminomatous germ cell tumours of the testes: case report and review of the role of surgery. *Neurosurgery* 1989;25:814-9.

Intracranial fusiform aneurysms in von Recklinghausen's disease: case report and literature review

The patient was a 27 year old man who presented to our hospital with symptoms of left sided weakness—initially involving only the arm, but subsequently also the leg—during the preceding two years. Three months before admission he began to experience spontaneous clonus of his left arm and leg that impaired his ability to work and thus precipitated his presentation. He also complained of an occipital headache (sometimes associated with vertigo) as well as vertical diplopia. He denied any seizure activity or symptoms attributable to bowel or bladder dysfunction. There was no family history of note.

On examination he was a well looking man with pronounced axillary freckling as well as multiple (more than 10) large (many larger than 3-4 cm in diameter) café au lait spots distributed over his trunk and limbs. He had features in keeping with an upper motor neuron weakness of his left arm and leg as well as impaired sensation to all modalities over the same area. He also had a sensory deficit over the area supplied by the trigeminal nerve on the left side. His left pupil was 2 mm larger than his right and he was unable to raise his left eye in the abducted position, suggesting weakness of the superior rectus muscle. He had an ataxic nystagmus as well as incoordination of his left arm and leg. His gait was ataxic. His blood pressure was 130/80 mm Hg and examination of his cardiovascular, respiratory and other systems was entirely normal. He had no Lisch nodules.

It was considered that a lesion in the upper midbrain, involving the spinal, medial, and trigeminal lemnisci (carrying crossed sensory fibres from the body and face), the corticospinal tracts (carrying crossed motor fibres), and the superior cerebellar peduncle could account for most of the neurological signs shown by this