Figure a A lateral view of the cervical spine showing occipitalisation of the posterior arch of C1 with the anterior element apparently fused to C2. There is fusion of the posterior elements of C2 and C3 and of the anterior and posterior elements of C4, C5 and C6. Figure b CT scan showing a large midline hypodense mass lesion in the posterior fossa with peripheral flecks of calcification (solid arrow) and an associated midline defect in the occipital bone (open arrow).

commented upon in reviews of either condition¹⁸ and may well be under recognised. In view of the complications of these cysts, early recognition is important. Posterior cranial fossa dermoids should be added to the list of congenital abnormalities which must be sought in patients with Klippel-Feil syndrome.

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anterior communicating artery aneurysm filling from the right and measuring 12 × 8 mm projecting down and forwards (fig 1b).

The cerebral blood flow was measured intermittently throughout the admission using a radionuclide technique with deconvolutional analysis.¹ On the fifth day after the presenting haemorrhage this showed no regional derangement but a global reduction to 615 mls/minute (normal > 875 mls/minute).

On the sixth day she became increasingly drowsy and disorientated with the right pupil becoming dilated and transiently unreactive to light. A repeat CT scan showed absorption of the diffuse blood and again no evidence of hydrocephalus, a clot, or an aneurysmal mass. Her deterioration was therefore attributed to a worsening of her vasospasm. Over the subsequent four days she became increasingly alert, although remaining disorientated in time and place. At this point she began to complain of blindness.

Ophthalmological examination revealed there to be no perception of light in the right eye and only minimal in the left. The right optic disc was pale and there was mild macular oedema bilaterally. There was a right afferent pupillary defect. The CT scan was now normal and the global cerebral blood flow had returned to 1246 mls/minute with no regional disturbance.

The situation remained static over the subsequent days as did the cerebral blood flow and the CT scan appearances, and on the thirteenth day she had aneurysm surgery. A pterional approach was used and the aneurysm, parent vessels and optic apparatus were clearly displayed. The size of the aneurysm was in accordance with the angiographic findings. No blood clot was seen and there was no direct compression of the anterior optic pathways. The aneurysm was controlled with a single Sugita clip which was left lying clear of the optic chiasm.

She made a prompt recovery although remained mildly disorientated and with no change in her visual disturbance. Subsequent follow up at one year showed an improvement in the left visual acuity to 6/18 although the field remained restricted to a small central patch and there was complete blindness of the right eye.

Anterior communicating artery aneurysms, although close to the anterior visual pathways, rarely produce visual

Visual failure following subarachnoid haemorrhage from rupture of an anterior communicating artery aneurysm

Subarachnoid haemorrhage secondary to rupture of an intracranial aneurysm may lead to a wide spectrum of neurological disturbances. Although visual loss may occur, complete and permanent amaurosis is unusual unless associated with a large anterior communicating artery aneurysm. We report a case of total blindness with only minimal unilateral recovery following rupture of a small anterior communicating artery aneurysm which was associated with a documented period of reduction in global cerebral blood flow.

A previously healthy 56 year old woman was admitted to hospital following the sudden onset of severe occipital headache associated with dizziness, vomiting and neck stiffness. She was alert and orientated with no focal neurological signs, and both visual fields and acuities were normal. Lumbar puncture revealed uniformly bloodstained cerebrospinal fluid at a pressure of 18.5 cm. She was subsequently referred for neurosurgical assessment.

The following day she became drowsy although remaining orientated and otherwise

neurologically intact. Computerised tomography showed diffuse blood interhemispherically and in the sylvian fissures with no aneurysmal mass or clot visible (fig 1a). On the fifth day, four vessel angiography revealed an

Figure 1a CT scan on second day showing diffuse blood interhemispherically. 1b Angiogram showing anterior communicating artery aneurysm (12 × 8 mm).

failure.² Previous reports of permanent visual deterioration have all concerned large aneurysms of at least 12 × 15 mm.²³ The mechanism of visual loss has always been attributed to compression of the optic pathways either by aneurysm or by an associated haematoma with the pattern of loss being determined by the position of the aneurysm in relation to the optic nerves and chiasm.⁴

In this case, almost complete and permanent visual failure occurred six days after the primary haemorrhage. There was no evidence of further haemorrhage or clot on CT scans and the aneurysm was of moderate size with no signs of compression being seen at the time of surgery. The visual failure could have been produced by ischaemic damage to the anterior visual pathways secondary to vasospasm. A reduction in the cerebral blood flow over the period of onset of the visual failure was evident both clinically and by measurement.

Vasospasm has been postulated as a cause for transient ischaemic amaurosis before,⁵ but this is the first time that such an ischaemic event has led to permanent blindness and the vasospasm has been measured quantitatively during the evolution of the symptoms.

We suggest that ischaemia resulting from arterial spasm may play a role in the visual failure associated with anterior communicating artery aneurysms as well as direct compression of the visual pathways.

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Hemifacial spasm due to pontine infarction

Currently, hemifacial spasm (HFS) is thought to be due to a compression of the facial nerve at the root exit zone by blood vessels.¹ Compression by tumour, aneurysm or arteriovenous malformation has also been noted. We report a case probably due to a small infarct in the pons.

A fifty year old man, known to be hypertensive for 15 years, presented with a two year history of left hemifacial spasm. At the age of forty six, he had been admitted for a transient ischaemic episode probably in the right internal carotid territory. He recovered, but two years later, represented with a minor right hemiparesis and a transient left facial weakness. Two weeks later, he noted slight flutter-

Figure Magnetic resonance, T-1 weighted sagittal image. Low intensity signal in the pons.

ing of the lower eyelid which gradually increased in frequency and severity. The left hemifacial spasm was tonic, involved the orbicularis oculi, orbicularis oris and the platysma and was aggravated by light and emotional stress. The left limb reflexes were brisk. Therapy with botulinum toxin was effective.

The brainstem evoked potentials were impaired centrally on the left. Six months after the onset of the HSF, a blink reflex study² showed normal R1 (early component) latencies on both sides. The latencies of R2 (the late ipsilateral component) and R2c (the late contralateral component) were shorter on the left (28.7 and 30.1 ms) than on the right (33.4 and 35.1 ms) (control subjects: 33.2 + 2.7 and 34.8 + 2.9). With the paired stimuli technique, the R2 and R2c responses were obtained when the interstimuli interval was decreased to 100 ms on the left and to 200 ms on the right. In the control subjects, the inhibition of the R2 response was complete when the interstimuli interval decreased below 250 ms. The CT scan showed mild sub-cortical atrophy. MRI demonstrated an ectatic basilar artery and decreased signal on the T1-weighted MRI scan and increased intensity signal on the T2 weighted MRI scan in the right centrum ovale and in a small area just above and internal to the left facial nucleus (fig), suggesting a small infarct in the left pons.

To our knowledge, HFS due to a lacunar infarct has never been reported. The onset a few weeks after a regressive hemiparesis and the investigations support this view. The pathogenesis of HFS remains unclear. The ephaptic transmission hypothesis,³ due to a compression by a blood vessel on the root entry zone, is widely popular. Our case agrees with the claim that the physiological abnormality is situated in the facial nucleus area and that signs of HFS are caused by hyperexcitability of the facial nucleus.⁴ The results of the blink reflex of our patient, according to the study by Valls-Sole and Tolosa,² suggest an enhanced excitability of facial motor neurons and of those brainstem interneurons that mediate the blink reflex pathway. In our case, this hyperexcitability might not be due to antidromic impulses from compression of the root entry zone,³ but to a loss of inhibitory impulses from the brainstem, due to the infarct in the pons.

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Suxamethonium is contraindicated in the Guillain-Barré syndrome

Suxamethonium induced hyperkalaemia has been described in a variety of disorders.¹ Ferguson *et al*² described four patients with chronic/relapsing polyneuropathy who developed life-threatening arrhythmias following suxamethonium administration. The presumed cause was suxamethonium induced hyperkalaemia although this was not documented in their patients. We have recently seen a patient with relapsing Guillain-Barré syndrome who developed severe ventricular arrhythmia secondary to a documented suxamethonium induced hyperkalaemia. The potential danger of the use of suxamethonium needs to be emphasised in the neurological literature.

A 51 year old man was admitted with a two week history of tingling in his hands and feet and progressive weakness in his arms and legs. These symptoms had begun one week after a flu-like illness. Examination revealed a proximal muscle weakness with depressed deep tendon reflexes and normal sensation. Cerebrospinal fluid examination was normal but nerve conduction studies showed evidence of a demyelinating neuropathy. A diagnosis of Guillain-Barré syndrome was made and he was treated with plasmapheresis with significant improvement over the following ten days. He was discharged but was readmitted two months later with an exacerbation of his symptoms.

Examination revealed severe weakness in his arms and legs, absent deep tendon reflexes, bilateral mild facial weakness, mild dysphagia and dysarthria. Forced vital capacity was two litres. He was treated with nasogastric feeding and daily plasmapheresis without any improvement over the following ten days. On day 11 because of deteriorating pulmonary function, it was decided to electively ventilate him. Before ventilation, there was a sinus tachycardia of 126/min but no other evidence of autonomic dysfunction. The arterial pressure of oxygen was normal and he was given 100% oxygen for three minutes before the procedure. Anaesthesia was induced with thiopentone and he was then paralysed with suxamethonium.

Immediately after the suxamethonium was given and before intubation he developed a ventricular tachycardia followed by a cardiac arrest. Cardio-pulmonary resuscitation was