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 lead 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.

SIMON RUBEN
FARHAD AFSHAR
Department of Neurosurgery, St Bartholomew's Hospital, West Smithfield, London, UK

Correspondence to: Mr Ruben, Western Ophthalmic Hospital, London, UK.

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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-

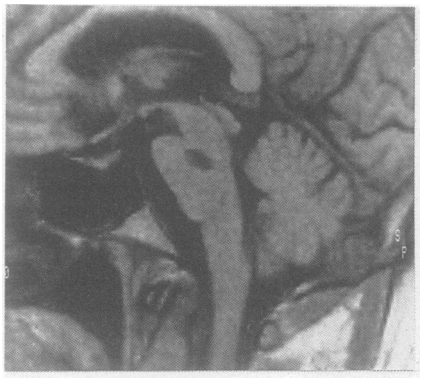

Figure Magnetic resonance, T-1 weighted sagittal image. Low intensity signal in the pons.

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 study[2] 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,[5] but to a loss of inhibitory impulses from the brainstem, due to the infarct in the pons.

P VERMERSCH
H PETIT
Service de neurologie, Hôpital B, Lille
MH MARION
Service de neurologie, Hôpital de Bicêtre, Paris
B MONTAGNE
Service de médecine interne, Hôpital V, Provo, Roubaix, France

Correspondence to: Dr Vermersh, Service de clinique neurologique, Hôpital B, CHU de Lille, 59037 Lille cedex. France

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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 partial 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

immediately instituted but over the following 25 minutes he developed recurrent episodes of ventricular tachycardia, ventricular fibrillation, bradycardia and asystole. Before ventilation his serum potassium was 4·3 mmol/L; at the time of the initial ventricular tachycardia his serum potassium had risen to 8·6 mmol/L. Following resuscitation he was comatose with fixed dilated pupils. The next day there was evidence of anoxic brain damage with myoclonic jerks. Dolls head eye movements were initially present but were then lost and all brain stem reflexes were lost gradually over the course of six days at which time brain stem death was confirmed.

The most probable cause of the ventricular arrhythmias in this patient was suxamethonium induced hyperkalaemia. Hypoxaemia was very unlikely to have been a contributory factor because the partial pressure of oxygen was normal and the patient was given 100% oxygen before the procedure. Autonomic dysfunction in the Guillain-Barré syndrome may be severe enough to cause sudden death.[3] Our patient had a sinus tachycardia before intubation but had no other signs of autonomic dysfunction. The ventricular tachycardia occurred before intubation so autonomic reflexes related to endotracheal intubation could not have caused this arrhythmia. Hyperkalaemia during the ventricular tachycardia after the suxamethonium administration indicates that suxamethonium induced hyperkalaemia was the probable cause of the arrhythmia.

Suxamethonium induced hyperkalaemia has been described in intracranial lesions,[1 4] spinal cord lesions,[5] peripheral nerve disorders,[1] muscular disease,[6] prolonged immobilisation[1] and many non-neurological conditions including infections,[7] burns[8] and cold injury.[9] Administration of suxamethonium causes a transient but slight increase in serum potassium in normal patients.[4] This may be a result of the release of potassium from the skeletal muscles during repolarisation.

In denervated or traumatised muscle, the muscle cell membrane becomes supersensitive to suxamethonium thus accounting for the sudden release of substantial amounts of intracellular potassium after suxamethonium is given. There appears to be a critical period between day five and day 15 when supersensitivity is maximum in traumatised muscle. This is less clear in denervated muscle when the sensitivity increases dramatically by day five but can continue for weeks and longer in the chronic and relapsing denervation syndromes.[10]

Our case clearly demonstrates that in clinical practice suxamethonium should not be used in chronic or relapsing polyneuropathies. In addition, because of the varying reports of the "at risk" time span and because of the incompletely understood mechanism of the aetiology of the hyperkalaemia, we suggest that suxamethonium should be avoided in all cases of Guillain-Barré syndrome.

M REILLY
M HUTCHINSON
Department of Neurology, Adelaide Hospital, Peter Street, Dublin 8, Ireland

Correspondence to: Dr Hutchinson

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Peripheral neuropathy associated with lithium toxicity

It is well known that lithium intoxication produces central nervous system damage,[1] but its effect on the peripheral nervous system is less well documented. We report a patient with generalised peripheral neuropathy following lithium intoxication, who recovered over several weeks. EMG and nerve conduction studies showed this to be an axonal neuropathy.

A 69 year old woman with long standing depression had been on 1000 mg/day of lithium carbonate for several years. She was admitted with a three day history of lethargy, withdrawal and depressed mood. She had stopped eating or drinking and on the day of admission become drowsy. On examination she was dehydrated, febrile at 37·5°C but vital signs were normal. She was obtunded but could move all four limbs to command against gravity and tendon reflexes were normal. Initial investigations showed impaired renal function (sodium 146 mmol/l, potassium 4·5 mmol/l, urea 48·3 mmol/l and creatinine 378 micromol/l), and a lithium level of 1·89 mmol/l (normal range 0·6–1·2 mmol/l). A full screen for infection was normal, and her pyrexia quickly settled.

Despite cautious correction of renal function with intravenous fluids, her conscious level continued to deteriorate, and she became unresponsive three days after admission. Her limbs were hypotonic, but still withdrew to pain, and she became areflexic with her plantar responses remaining flexor. With conservative management she slowly regained consciousness and ten days after admission was opening her eyes spontaneously. Gag reflex was present and she could swallow normally. Her limbs, however, remained flaccid and areflexic. There was distal loss of all sensory modalities in all four limbs. Haemoglobin was 11·3 g/dl, white blood cell count was 6,100/mm^3, serum albumin 32 g/l and glucose 5·6 mmol/l. Her renal function had now returned to normal (sodium 143 mmol/l, potassium 4·2 mmol/l, urea 3·2 mmol/l and creatinine 50 micromol/l). Other routine biochemical and haematological tests were normal, as were creatine kinase, serum vitamin B12 and porphyrin studies. The cerebrospinal fluid was normal (no cells, protein 0·5 g/l). Nerve conduction studies confirmed the presence of a peripheral neuropathy: sensory action potentials were absent in the legs and reduced or absent in the arms (left median, F2 and F3 to wrist, and left radial—all absent; left ulnar, F5 to wrist, 3 μV). Motor action potentials were reduced in all four limbs. Motor conduction velocities were at the lower end of the normal range (left ulnar 53 m/s-1; left common peroneal 41 m/s-1). EMG studies showed fibrillations distally in the legs and widespread polyphasic units. Nerve biopsy was not performed. The neurophysiological tests were consistent with an axonal neuropathy.

Over the next four weeks there was considerable return of limb function, so that she could feed herself and stand with support. Repeat neurophysiological testing at this stage indicated a mild improvement in motor action potentials but no significant sensory change. She continued to improve with physiotherapy and was discharged home after three months in hospital.

Whilst muscle fasciculations and paraesthesias are recognised clinical sequelae of lithium intoxication, frank peripheral neuropathy is rare and we have only been able to find six documented cases in the literature.[1 6] Temporal events and the exclusion of other causes of neuropathy incriminated lithium in our case, which appears to show many similarities with the other cases that have been reported. A polyneuropathy of the critically ill is well recognised,[7] but our patient was never ventilated and multiple organ failure was not present. In addition, no underlying infection could be found to explain the initial mild pyrexia.

She was encephalopathic at the time of presentation and her neurological condition deteriorated after admission. Brain lithium content is not always reflected by serum lithium concentration and worsening of neurological symptoms after discontinuation of lithium is well documented, even when serum levels are within the so-called "therapeutic" range.[8] An acute axonal dysfunction seems to be the hallmark of lithium-induced peripheral nerve damage but there is often considerable clinical recovery over a period of weeks to months.[1–3] It has been suggested that intracellular accumulation of lithium may interfere with the propagation of the action potential.[9]

Peripheral nervous system damage secondary to lithium is probably under-recognised. Abnormal nerve conduction velocities have been reported in manic-depressives and healthy volunteers taking lithium.[10] In addition, a mild neuropathy could easily be overlooked in acute toxicity where the central nervous system manifestations are usually much more dramatic. Lithium toxicity should thus be considered in any patient receiving this drug who develops evidence of a peripheral neuropathy, including muscle fasciculations or paraesthesia, even in the presence of central nervous dysfunction.

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SRD JOHNSTON
DJ BURN
DJ BROOKS
Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, Du Cane Road, London, UK