ongoing spike train. The rate of stimulation was less than once in 3 seconds. The stimulus intensities used were in the range of 60-70% of the maximal output of the stimulator, which was 12-15% lower than the threshold intensity for a compound muscle action potential recorded over the right FDI.

Peristimulus time histograms constructed off line demonstrated periods of zero firing preceding all three motor units. These had onsets after the stimulus of 34, 40 and 50 ms and durations of 47, 33 and 34 ms, respectively (fig). Neither primary nor secondary peaks were evident in any motor unit. The absence of any significant excitative response was confirmed by cuxum analysis.

In this patient motoneurons were suppressed from firing by motor cortical stimuli. This contrasts with the characteristic short latency excitation evoked by transcranial magnetic stimulation in the motoneurons from healthy subjects. The mechanism for this may have involved a transient withdrawal of excitatory drive from the motoneurons or there could have been an active inhibition of the motoneuron, either pre- or post-synaptically, from a pathway activated by the motor cortical stimuli. The findings suggest that this patient’s weakness could be attributed in part to this suppression.

It is becoming clear from studies of the unitary response to transcranial magnetic stimulation that abnormalities causing upper motor neuron lesions can involve several mechanisms, including abnormalities of spatial and temporal summation at the motoneuron and also, we now believe, suppression of motoneuron firing via a central mechanism.

S J Boniface
R R Mills
Clinical Neurophysiology Unit
University Department of Clinical Neurology
Radcliffe Infirmary
Oxford, UK

Correspondence to: Dr Mills


Control of epilepsy partialis continua and secondarily generalised status epilepticus with isoflurane

The majority of patients with status epilepticus can be adequately controlled with conventional drugs including benzodiazepines, phenytoin and phenobarbital, but some require more aggressive therapy. Various approaches have been used, most commonly to produce therapeutic serum concentrations but other anticonvulsants were stopped.

Twenty-four hours after the isoflurane was administered, she had several short lived focal seizures and then generalised isoflurane anaesthesia using volatile agents such as halothane or isoflurane has been advocated in patients who are either refractory to parenteral therapy or experience unacceptable side effects from anticonvulsant drugs. We report a patient whose status epilepticus and epilepsy partialis continua were controlled with the inhalational anaesthetic, isoflurane. A 30 year old woman had a six year history of increasing simple partial, complex partial and secondarily generalised seizures from the left hemisphere. A left posterior temporal corticectomy performed four years after onset of seizures did not result in any significant clinical improvement. Pathology showed mild gliosis. Two months before admission, despite high doses of carbamazepine, valproic acid and clobazam, she developed epilepsy partialis continua with constant twiching of the right face, progressive right hemiparesis and dysphasia. EEGs revealed multifocal spikes and frequent seizures in the left hemisphere. MRI showed postoperative changes in the left posterior temporal region. When she began to have three to five secondarily generalised seizures an hour as well as the epilepsy partialis continua, she was transferred to the intensive care unit, intubated and ventilated using a Narkomed 2A anaesthetic machine and "circle" breathing system. Insured gas was a mixture of oxygen and air to maintain an arterial oxygen saturation >90%. Positive pressure ventilation with 5 cm PEPE was adjusted to maintain normocapnia. Isoflurane was added to the fresh gas flow as required to control seizure activity and the end tidal concentration was monitored in conjunction with end tidal carbon dioxide. Other monitors included inspired oxygen concentration, intra-arterial blood pressure, high pressure and disconnet alarms on the ventilator. Small doses of morphine but no muscle relaxants were used following intubation with succinylcholine. The initial end tidal concentration of isoflurane was 0.5% which resulted in immediate clinical control of the seizures and marked EEG improvement (figure). Phenytoin and phenobarbital were continued at doses to

Figure EEG before (A), during (B) and following (C) treatment with isoflurane.
when isoflurane is used at higher concentrations. Isoflurane, unlike enflurane and to a certain extent halothane, has not been found to cause EEG or clinical seizure activation or to produce effects on cerebral energy stores and barbiturates, it is an effective agent for reducing cerebral metabolic rate. Isoflurane, however, can increase cerebral blood flow and intracranial pressure possibly by interfering with cerebral autoregulation.

Voluntary or intravenous general anaesthesia should be the last line of treatment for status epilepticus since prolonged anaesthesia is not without risks including hypotension, cardiac depression, hypothermia, venous thrombosis, pressure sores and increased susceptibility to infection.

Inhalational anaesthesia is costly and requires the availability of an anaesthesia machine and experienced medical and technical personnel to monitor the patient and equipment. The isoflurane vapouriser and carbon dioxide absorber need to be replenished periodically. In addition to continuous monitoring of vital signs and ECG, oximetry and capnography are essential in these patients and the ability to monitor end tidal isoflurane concentration useful. Muscle relaxants are not indicated and control of seizures can therefore be judged clinically as well as by intermittent or continuous EEG monitoring. With the proper facilities, inhalational anaesthesia may be effective for intractable status epilepticus and is relatively safe, easily titratable and rapidly reversible after the seizures have been controlled.

Intrinsic spinal cord haemorrhage due to streptokinase treatment for myocardial infarction

Haemorrhagic complications of streptokinase therapy for myocardial infarction (MI) occur in approximately 1% of patients. The most commonly reported neurological complication is intracranial haemorrhage. We report a case of spontaneous intrinsic spinal cord haemorrhage associated with streptokinase therapy for MI.

A 50 year old woman was admitted with a three day history of discomfort in the neck. In the early hours on the day of admission the pain became much worse. An ECG and enzyme studies supported the clinical diagnosis of myocardial infarction, showing elevation of creatine kinase and lactate dehydrogenase, with ST-segment elevation in the anterior and lateral ECG leads. She was given opiate analgesia and streptokinase, 1,500,000 units subcutaneously.

Late in the afternoon she complained of altered sensation and weakness of the legs. This progressed rapidly, and she was transferred to the regional neurological unit. She deteriorated rapidly, however, and by the time she arrived she was unable to move either leg and there was severe weakness of the arms. The weakness had started in the left arm, then the legs simultaneously, and the right arm. She had developed urinary retention during the afternoon and had been catheterised.

General medical examination was unremarkable. Neurological examination showed a flaccid quadriparesis, 0–3/5 in the arms, 0–5 in the legs. There was a sensory level to pinprick and light touch at C5, though there was some preservation of joint position sense in the arms. She was areflexic with upgoing plantars.

MRI of the cervical cord (figure) showed swelling of the cord from C3 to C7, with a central increased signal on the T2 weighted sagittal sections. There was no evidence of cord compression, and no hypointensity which might represent a focal haematoma, although it was possible that the appearances might represent oedema around petechial haemorrhage. Nerve conduction studies were normal. Clotting studies on the day of admission showed prolongation of the prothrombin time at 17s, prolongation of the partial thrombin clotting time at 16s with reduction of fibrinogen below the haemostatic level at 0.7 G/l. She was given 1 unit of cryoprecipitate, and the next morning the thrombin time was only marginally elevated at 10s, and other haemostatic parameters were within normal limits. Subsequent clotting studies were also normal.

At 11 am the day after admission she deteriorated suddenly, developing a complete quadriplegia, complete bulbar palsy, and she required ventilation. Over the next few days she deteriorated further, developing a gaze palsy to the left and a right internuclear ophthalmoplegia, she developed complete heart block, hypotension and a chest infection. On the ninth day of her illness she arrested and died.

Necropsy examination showed haemorrhage centrally in the cord extending from thepons down to T10. For most of its length the haematomas was narrow, between 2–4 mm, but there was a 5 mm haematoma in the lower pons.

Neurological morbidity from thrombolitic therapy in MI occurs in approximately 0–5% of patients and has been largely limited to small intracerebral haematomata with low mortality. To our knowledge, this is the first report of intrinsic spinal cord haemorrhage.

The administration of thrombolytic therapy to patients who subsequently appeared to have other disorders such as aortic dissection has been reported previously. Guillaume Barré syndrome (GBS) may present with chest or (more commonly) back pain, and the occurrence of pain in this circumstance is associated with a tendency for plasma creatine kinase level (CK) to rise. For this reason we felt it was necessary to carry out nerve conduction studies to rule out this disorder.

Transverse myelitis may also present with chest pain and abnormal ECG, and this diagnosis was also considered, particularly as the MRI appearances, though focal, would have been consistent with it. Given that neurological deterioration occurred between the time of the MRI and the necropsy findings, the reason for the atypical (for haematoma) MRI findings remain unclear.

Data regarding the duration of action of single dose streptokinase unaccompanied by heparin are scant, but do suggest that clotting may remain abnormal for more than 48 h after intravenous injection of 1 5 million units. At the time of her deterioration this patient’s clotting remained marginally abnormal despite having been given cryoprecipitate, though well within the usual haemostatic limits. Nonetheless, the acute nature of the deterioration makes it highly likely that it was indeed due to extension of the haemorrhage.

Where symptoms of spinal cord dysfunction following the administration of streptokinase cryoprecipitate should be given without delay, even when the clinical picture is compatible with primary neurological disease.

Letters to the Editor

G S CRUCKSHANK
R DUNCAN
DM HADLEY
1 BONE
Institute of Neurological Sciences,
Southern General Hospital,
Glasgow G51 4TF, UK


Figure T2 weighted sagittal image of the cervical and upper thoracic spinal cord, showing swelling of the cord between C3 and C7 (arrows) and increased signal in the central part of the cord.
Control of epilepsia partialis continua and secondarily generalised status epilepticus with isoflurane.  
D R Hughes, M D Sharpe and R S McLachlan  

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