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 preceded by a motor unit or several motor units. These had onset 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 excitatory response was confirmed by cusan 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 influences onto the lower motor neurons. This upper motor neuron lesion 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.

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 general anaesthesia with continuous thiopental or pentobarbital infusion. Continuous infusions of diazepam, lorazepam and clorazepate have also been used. General anaesthesia using volatile agents such as halothane or isoflurane has been advocated in patients who are either refractory to paracentral 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 corticoectomy 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 twinning 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 had begun adduction of parenteral diazepam, phenobarbitone, phenytoin, phenobarbital and paraldehyde failed to control the seizures. She was transferred to the intensive care unit, intubated and ventilated using a Narkomed 2A anaesthetic machine and "circle" breathing system. Inspired gas was a mixture of oxygen and air to maintain an arterial oxygen saturation >95%. Positive pressure ventilation with 5 cm PEEP 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, intraarterial blood pressure, high pressure and disconnect 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 tics and marked EEG improvement (figure). Phenytoin and phenobarbital were continued at doses 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 the concentration was increased to 0.7%. Another EEG showed no seizure activity. After 48 hours, the isoflurane was discontinued and the patient awoke within 15 minutes at a measured end tidal concentration of 30 0 and 0.5%. She was extubated six hours later and transferred back to the ward the following day. She was maintained on phenytoin and phenobarbital and continued to have numerous daily focal seizures of the right face but only occasional generalised seizures. After a second corticoectomy of the left rolundic region, seizures were focal, brief and less than 10 a week. Chronic encephalitis was found pathologically.

This is the second patient with status epilepticus we have successfully treated with isoflurane anaesthesia, and the first with epilepsy partialis continua. The other patient has been previously reported as one of a small series of similar cases from North America.

The use of isoflurane anaesthesia has advantages over barbiturate coma which is the most commonly recommended "last resort" therapy for status epilepticus. The time to awaken after barbiturate coma is often several days due to the prolonged metabolism of the barbiturates, whereas our patient awoke within 15 minutes after 48 hours of isoflurane anaesthesia. The recovery from inhalational anaesthesia depends mainly on the concentration used, the duration of use and the tissue solubility of the agent. As isoflurane is the least soluble of the currently available volatile anaesthetics, recovery from anaesthesia is more rapid. Other advantages include a lack of metabolic side effects and, unlike halothane, there is no evidence to date of hepatotoxicity. Renal toxicity from inorganic fluoride production does not appear to be a problem, which is a significant advantage over enflurane or methoxyflurane. Overall metabolism of iso- flurane amounts to only 0-17% of the absorbed dose.

The use of isoflurane is not without potential risks. Hypotension requiring vasopressor treatment is a common problem with prolonged anaesthesia using barbiturates or inhalational agents. Although hypotension was not a problem in our patient, treatment with a vasopressor agent is often required.

![Figure EEG before (A), during (B) and following (C) treatment with isoflurane.](http://jnnp.bmj.com/10.1136/jnnp.55.8.739)
when isoflurane is used at higher concentrations. Isoflurane, unlike enflurane and to a certain extent halothane, has not been found to cause EOG or clinical seizure activation or to produce any effects on cerebral energy stores and like barbiturates, it is an effective agent for reducing cerebral metabolic rate. Isoflurane, however, can increase cerebral blood flow and pressure possibly by interfering with cerebral autoregulation.

Volatile 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, hypotension, 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 vaporiser 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 is 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.

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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 S-T segment elevation in the anterior and lateral ECG leads. She was given opiate analgesia and streptokinase, 1500 000 units intravenously.

Late in the afternoon she complained of altered sensation and weakness of the legs. This progressed rapidly, and she was transferred to the regional neurosurgical 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 1½ times normal and the thrombin clotting time at 16s with reduction of fibrinogen below the haemostatic level at 0·7 G1. 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 on the day after admission she deteriorated suddenly, developing a complete quadriplegia, complete bulbar palsy, and she required ventilation. On the next few days she deteriorated further, developing a general palsy to the left and a right internuclear ophthalmoplegia, 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 the pons down to T10. For most of its length the haematoma was narrow, between 2-4 mm, but there was a 5 mm haematoma in the lower pons.

Neurological morbidity from thrombolytic 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. Guillaum 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 atypical, 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.

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