HAM/TSP attributable to blood transfusion

A multi-centre case-control study was carried out to clarify possible environmental factors related to the onset of HTLV-I-associated myelopathy/tropical spastic paraparesis (HAM/TSP) in northern Kyushu, Japan, which comprises one of the most prevalent areas of HTLV-I in the world. The frequencies of blood transfusion before the onset of HAM/TSP were 33% (6/18) among male patients and 18% (12/67) among female patients, which were significantly higher than 8% in males and 9% in females in the general population. The age-adjusted summary odds ratios with 95% confidence intervals were 7.0 (2.9-17.0) for males and 2.4 (1.3-4.5) for females. The percentages of population attributable risk of HAM/TSP attributable to transfusion were estimated to approximately 29% for males and 11% (1-21) for females.

The fraction of HAM/TSP attributable to transfusion after the introduction of blood screening for HTLV-I, in effect since 1986 in Japan, was definitely smaller than that before the programme. Our observations seemed compatible with a marked decline in newly diagnosed HAM/TSP patients after its introduction, which may be part of the benefit of blood screening. Neither smoking nor drinking was related to the risk of HAM/TSP. The prevalence of HAM/TSP in a health check-up programme was significantly lower in male patients.

References:

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 probability in motor or arm 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 cymus 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 acting through cortically projecting 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.

It is becoming clear from studies of the unitary response to transcranial magnetic stimulation that abnormalities caused by lesions in the upper motor neuron 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.

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, lidocaine, paraldehyde and more recently, etomidate and propofol have also been used. General anaesthesia using volatile agents such as halothane or isoflurane has been advocated in patients who are either refractory to paracental 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 twitching of the right face, progressive right hemiparesis and dysphasia. EEGs revealed multifocal spikes and frequent seizures in the lefthemisphere. 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 re-investigated. Administration of parenteral diazepam, pentytoin, phenobarbital and paraldehyde failed to control the seizures. She was transferred to the intensive care unit, intubated and venti lated 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 >94%. 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, intra-arterial 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 seizures 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 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 between 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 corticectomy of the left Rolandic 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 time 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 isoflurane 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 isoflurane 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.