ties for using it are considerably less than is generally believed, common though the condition is. Consequently, intensive outpatient treatment for those who stand to benefit from it is probably a realistic policy within National Health Service resources. Although in absolute numbers more women have strokes than men, it is likely that considerably more men than women are suitable for intensive outpatient rehabilitation.

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Requests for reprints should be addressed to Dr T W Meade.

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SHORT REPORTS

Status epilepticus treated by barbiturate anaesthesia with continuous monitoring of cerebral function

Status epilepticus is a hazardous condition with a mortality of 6% to 18%.[1] In most cases it is controlled by intravenous diazepam or phenytoin or other anticonvulsive drugs. In severe tonic-clonic status epilepticus unresponsive to anti-epileptic drugs the induction of general anaesthesia has been suggested, although the anaesthetic agent of choice has not been defined.[1]

The anticonvulsive properties of short-acting barbiturates are well known, and in status epilepticus thiopentone sodium has been reported to be effective in low doses that do not depress the level of consciousness.[2] We have found, however, only one report of the use of barbiturate anaesthesia in the treatment of severe status epilepticus.[3] We report the use of general anaesthesia induced with intravenous thiopentone sodium in five patients with prolonged status epilepticus. These patients had failed to respond to ordinary treatment, including diazepam and phenytoin.

Patients, methods, and results

In 1979 the casualty department of the University Central Hospital, Helsinki, dealt with 743 cases of convulsions. Forty-seven of these patients developed status epilepticus, defined as repetitive convulsions over at least 30 minutes or as repeated generalised seizures without return to consciousness in between. Five patients failed to respond to ordinary anticonvulsive therapy within 48 hours. These patients were admitted to the intensive care unit. All underwent endotracheal intubation, with assisted ventilation when necessary. Cerebral function was monitored by a Devices cerebral function monitor using three silver-silver chloride disc electrodes at sites P3 and P4 and a ground electrode on the forehead.[4] In two patients relaxation with curarisation was tried before the induction of general anaesthesia, but cerebral monitoring still showed seizure activity. Thiopentone sodium was given as an initial intravenous dose of 100 to 250 mg, followed by 50 mg at intervals of two to five minutes until no epileptic discharges were seen on the monitor. The infusion rate was regulated according to intra-arterial blood pressure. Maintenance therapy with phenytoin or benzodiazepines was continued during the anaesthesia, and dexamethasone (5 mg intramuscularly every six hours) was given for brain oedema. The level of anaesthesia was adjusted with an intravenous thiopentone infusion (2500 mg in 500 ml isotonic saline given at a rate of 0·5 to 1·5 ml/min) to keep the patients free of epileptic discharges visible on the cerebral function monitor. The infusion was continued at a steady rate for at least 12 hours after the last paroxysm seen on the monitor. The infusion rate was then gradually reduced and stopped within the following 12 hours.

Four patients responded well without recurrence of the seizures (see table). Status epilepticus recurred in one patient, who was then successfully treated with concomitant infusions of chlormethiazole and lidocaine (case 5). No permanent sequelae attributable to status epilepticus were seen in any of these patients.

Comment

Several lines of evidence seem to argue in favour of the use of barbiturates as general anaesthetic agents in prolonged status

History and clinical details of the five patients

Case No	Age and sex	History of epilepsy and cause of status epilepticus	Duration of status epilepticus and total dose of drugs given before barbiturate anaesthesia
1	34 M	No previous epilepsy, psychiatric problems, chlordiazepoxide 150 mg/day changed to amitriptyline 40 mg/day	Duration 84 hours. Diazepam 420 mg, phenytoin 1750 mg, clonazepam 14 mg, paraldehyde 20 ml, chlormethiazole 2,000 mg, lidocaine 3000 mg, curarisation with pancuronium
2	21 M	Post-traumatic 10 years, clonazepam 4 mg/day changed to sodium valproate 1200 mg/day	Duration 56 hours. Diazepam 210 mg, phenytoin 1000 mg, clonazepam 6 mg, paraldehyde 24 ml, chlormethiazole 4000 mg, curarisation
3	57 M	No previous epilepsy, intracerebral haemorrhage	Duration 48 hours. Diazepam 210 mg, phenytoin 1550 mg, clonazepam 5 mg
4	35 M	Post-traumatic 15 years, alcohol abuse	Duration 48 hours. Diazepam 180 mg, phenytoin 1250 mg, clonazepam 6 mg
5	19 M	Post-traumatic 10 years, cause of status epilepticus unknown	Duration 50 hours. Diazepam 100 mg, phenytoin 1150 mg, paraldehyde 18 ml, clonazepam 5 mg, chlormethiazole 4000 mg, lorazepam 2 mg IV

Modes of drug administration were as follows: Diazepam 10-20 mg intravenously initially followed by infusion of 100 mg/500 ml 5% dextrose solution 0·5-2 ml/min. Clonazepam 1-2 mg slowly intravenously. Phenytoin 125-500 mg slowly intravenously. Paraldehyde 8-10 ml divided in two doses intramuscularly. Chlormethiazole (8 mg/ml) infusion 24-60 mg/min initially followed by 4-8 mg/min. Lidocaine (0·4% solution) 100-200 mg intravenously initially followed by infusion of 3-5 mg/kg/h.

epilepticus. Curarisation of the patient, although it stops the convulsions, does not arrest seizure activity in the brain, and so oedema and hypoxia develop further, producing possibly permanent brain damage. The long-term use of an anaesthetic gas, such as halothane, is unsuitable because of its general toxicity. Besides their anticonvulsive properties the barbiturates have been shown to protect the brain against anoxia and ischaemia and to lower the increased intracranial pressure.[5] Barbiturate coma has recently been successfully used to treat both severe traumatic brain damage and Reye's syndrome.[5]

The treatment of intractable status epilepticus by general anaesthesia requires an intensive care unit with equipment for controlled ventilation and continuous monitoring of arterial pressure and cerebral function. Although the cerebral function monitor only partially reflects the pattern of electrical activity of the brain seen on an electroencephalogram, it provides an adequate record of the epileptic discharges, a particularly useful feature being its ability to show the events of several days on a short strip of paper. Follow-up of the neurological status of the patient by mere physical examination is not possible during general anaesthesia. This underlines the importance of adequate diagnostic examinations, including computed tomography.

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Department of Neurology, University of Helsinki, SF-00290 Helsinki 29, Finland
MARKKU PARTINEN, MD, resident in neurology
JUSSI KOVANEN, MD, lecturer in neurology

Department of Anaesthesiology, University of Helsinki, Helsinki, Finland
EIJA NILSSON, MD, lecturer in anaesthesiology

Child abuse presenting as apparent "near-miss" sudden infant death syndrome

"Near-miss" sudden infant death syndrome refers to infants previously well who, during sleep, experience an episode of apnoea, limpness, and cyanosis or pallor that is terminated by vigorous stimulation or mouth-to-mouth resuscitation.[1] Several conditions such as sepsis, fits, aspiration, and oesophageal reflux may present a similar clinical picture and are excluded by appropriate investigations.

Two cases of child abuse simulating near-miss sudden infant death syndrome have been described.[2] In each case the mother had deliberately attempted to arrest respiration in hospital after admission to investigate recurrent apnoeic episodes. I describe a further case of this form of child abuse.

Case report

A 4-month-old boy was brought to hospital by his mother after an apnoeic episode at home. The history obtained was that, having found the baby apnoeic and cyanosed in his cot, his mother had resuscitated him by shaking him and slapping his face. The baby had previously been well. He had been born at 32 weeks' gestation and developed severe respiratory distress syndrome necessitating transfer to another hospital for ventilation. He was allowed home to his mother aged 32 days. His mother (aged 27 years) had bronchiectasis, which had necessitated her staying in hospital from the fifteenth week of pregnancy until delivery. She had suffered from depression with suicidal thoughts, but had no psychiatric history. On the day before the baby's admission his father had been admitted to hospital with haematemesis.

On admission the infant was pale and hypothermic but centrally pink. He was conscious but irritable with a generalised increase in muscle tone. His fundi were normal and there was no evidence of increased intracranial pressure. Respiratory system, cardiovascular system, and abdomen were normal. A small faint bruise was noted above his upper lip, which his mother attributed to her attempts at resuscitation. He otherwise appeared well cared for.

Investigations showed a metabolic acidosis (pH 7·24, $Paco_2$ 3·1 kPa (23 mm Hg), bicarbonate 10 mmol(mEq)/l) but normal serum urea, electrolyte, calcium, and blood glucose concentrations. Haemoglobin concentration was 9·6 g/dl and white cell count $28·5 \times 10^9$/l (34% neutrophils, 58% lymphocytes, 3% monocytes, and 4% eosinophils). No bacterial or viral pathogens were isolated from cultures of blood, urine, cerebrospinal fluid, faeces, and throat swabs. Chest *x*-ray films and skeletal survey were normal.

The baby was managed by gradual warming and intravenous antibiotics. Four hours later body temperature, colour, and muscle tone were normal and he took a bottle feed. Five days later he developed fever and a macular rash that persisted for two days, but recovery was otherwise uneventful. One week after his admission his mother was admitted to hospital after taking an overdose of diazepam. On recovery she was referred for psychiatric evaluation. She confessed that she had deliberately attempted to arrest the baby's respiration by holding his nose but had become frightened when he became cyanosed and apnoeic. The infant is at present placed with relatives.

Comment

This case and the two previously described[2] indicate that child abuse should be considered in the differential diagnosis of near-miss sudden infant death syndrome. In our case abuse occurred against a background of maternal depression related to prolonged hospital admission, early separation from the baby, and acute paternal illness. Thus adequate background information should be obtained in cases of apparent near-miss sudden infant death syndrome. This should include details of maternal psychiatric illness, marital or family stress, and any contact with a social worker. Data should be obtained as indirectly as possible so as not to exacerbate feelings of anxiety and guilt that will already be present in the parents. During physical examination careful inspection of the nose and mouth should be made for pinch marks, bruising, or trauma to the gums.

Appropriate management might entail home monitoring and instruction in resuscitation in the case of genuine near-miss sudden infant death syndrome,[3] whereas an infant already subjected to child abuse would clearly be at risk if discharged to the same home environment.

I thank Dr D Haigh for permission to report this case under his care.

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Bradford Children's Hospital, Bradford BD8 7QF
A M B MINFORD, MRCP, DCH, senior registrar in paediatrics

Are population-genetic mechanisms responsible for clustering of cases of Creutzfeldt-Jakob disease?

There is considerable indirect evidence that susceptibility to Creutzfeldt-Jakob disease may be genetically determined,[1] though the exact mechanism is still not clear. About 15% of patients with the disease are familial cases.[1] An increased incidence of the disease has been reported in some areas, with spatiotemporal clusterings in England[2] and Czechoslovakia.[3] A genetic hypothesis may be advanced for at least some of these observations: in familial Creutzfeldt-Jakob disease genes controlling susceptibility to the disease are shared with relatives, and the agent may be acquired by contact or vertical transmission or perhaps activated by genetically determined mechanisms. The frequency of genes or genotypes contributing to the disease may in some populations be increased by drift, founder effect, or inbreeding.

In 1976 we observed a clustering of cases of Creutzfeldt-Jakob disease in a rural district of south-east Slovakia. The high incidence of such a rare disease seemed unlikely to be coincidental so we studied the genetic structure of the area. Genetic isolates have