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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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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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