(2) If it does, then can it be controlled by drugs or other techniques?

(3) If it can, does it make any difference to the outcome?

We have previously shown that intracranial hypertension does occur after intrapartum asphyxia,² and in our most recent paper have established that mannitol can safely reduce intracranial pressure and improve cerebral perfusion pressure.¹

Unfortunately Dr Dear seems to have missed the point of our recent paper. The 'traditional' management of cerebral oedema includes relative dehydration and dexamethasone to prevent or reduce brain swelling. We have attempted to study the treatment of intracranial hypertension in a more scientific way. We did not state, as he suggests, that there was benefit in reducing pressure but merely asked whether it was possible to do so. Despite the apparent safety of this technique we feel it is not justified to insert a subarachnoid catheter (not subdural as Dr Dear states) unless treatment of raised intracranial pressure is planned.

How then can we answer our third question, and that of Dr Dear? We propose a controlled study of active management against traditional treatment. Infants fulfilling criteria for severe asphyxia would be allocated at random to one of two groups. Active treatment will involve insertion of a subarachnoid catheter and treatment of raised intracranial pressure. The control group will have no intracranial pressure monitoring, and supportive or symptomatic treatment of asphyxia undertaken as practised in most neonatal units. As Dr Dear points out, one hopes it is not possible for any one unit to generate sufficient numbers and such a study will require a multicentre approach. We would be more than happy to collaborate with the Leeds unit or any other in the country to plan this study further.

References

- Levene MI, Evans DH. Medical management of raised intracranial pressure after severe birth asphyxia. *Arch Dis Child* 1985;**60**:12-16.
- Levene MI, Evans DH. Continuous measurement of subarachnoid pressure in the severely asphyxiated newborn. *Arch Dis Child* 1983;**58**:1013-5.

Seizures and steroids

Sir,

As Dr Robinson rightly states in his excellent review,¹ infantile spasms are associated with a great variety of conditions, as long as these cause a severe disturbance of cerebral function and occur at a certain age. I am sure his last paragraph is of particular importance, as although it may not often happen, there is no doubt that steroids given within a few days of the onset of the myoclonus (sometimes mistaken for colic) can almost immediately stop this, with a return of the electroencephalogram to normal.

Admittedly, the situation is complex but it may be that among those patients with so called 'cryptogenic infantile spasms' there are children who are suffering from an allergic type of encephalomyelitis, perhaps due to virus infections and immunisations, and that in these children the steroids are not acting so much as anticonvulsants, but

as specific treatment for the underlying cause of the particular disease in that instance? I write this letter in response to the last sentence about the educational role of the paediatrician as I agree it is important to emphasise the occasional success of early steroid treatment when so many of the published trials confirm the lack of any effect of steroids in preventing mental handicap but omit to state the time interval between the start of the 'spasms' and the start of the treatment.

Reference

- Robinson RO. Seizures and steroids. *Arch Dis Child* 1985;**60**: 94-5.

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Sustained release theophylline in nocturnal asthma

Sir,

We read with interest the paper by Dr Elias-Jones *et al*¹ but must admit to being surprised by the unusual trial design, small numbers, and incorrect statistical analysis. The design and data analysis have defects which must question the validity of some of their conclusions.

Asthma, by its very nature, is a disease that shows considerable variation, and any trial must be long enough or study sufficient numbers to allow for this. Dr Elias-Jones *et al* studied 10 children only and analysed data from short active and placebo periods of 10 days duration. Their study design included a run in period during which the child was on active treatment, apparently to his and the doctor's knowledge. This may have biased the subsequent completion of diary cards during active and placebo trial periods as they would know what benefits could be expected from active treatment.

Inspection of symptom score, frequency of occasions when awake at night, and number of doses of beta agonists shows that these data are not normally distributed, have different standard deviations during active and placebo periods, and are therefore not suitable for paired *t* tests. It is also apparent that three of the 10 children showed no benefit from theophylline treatment. When the three who could not complete the trial are included, it would seem that six of the 13 children did not or could not benefit from theophylline.

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Dr Elias-Jones and co-workers comment:

Dr Davies and colleagues make a point of suggesting that studies of long duration are more appropriate when