Prevention of simple febrile convulsions

Sir,

I should like to comment on Ngwane and Bower's paper.[1]

First of all, their statement that phenobarbitone takes 72 hours to achieve an adequate blood level is an often repeated inaccuracy. Phenobarbitone, like any other drug, will only achieve so-called therapeutic blood levels if an adequate dose is given. We have achieved this level within 90 minutes, orally and intramuscularly, after a dose of 15 mg/kg.[2]

I was intrigued that about 75% of their 265 children did not meet their criteria for simple febrile convulsions. The significance of their definition is not clear, and it is unfortunate that so few were left on whom to perform the trial.

It would appear that the untreated group, even allowing for their age at onset, had very severe febrile seizures if 6 out of 21 children had prolonged fits lasting 25 minutes or longer within the follow-up period. In our study of 83 unselected children with febrile convulsions, to whom no prophylaxis was given but only strict instructions for the prevention of prolonged seizures, only 2 children went on to have prolonged fits in at least a one-year follow-up.[3] Is the untreated group therefore a satisfactory one for comparison, particularly as it was not designed as a control group?

Although not commented on in the text, their results also show that phenobarbitone is not statistically better than no treatment in preventing further fits. Blood levels in the 4 treatment failures at the time of the fit would have been interesting.

Sodium valproate did appear to be more effective than no treatment. However for those therapeutic nihilists, like myself, who feel that prolonged seizures can generally be prevented by other means, Ngwane and Bower performed one valuable service; they suggested that sodium valproate was just as harmful to children as phenobarbitone. Again however the numbers were small, and it would have been interesting to know the exact nature of the side effects.

References

1 Ngwane E, Bower B. Continuous sodium valproate or phenobarbitone in the prevention of 'simple' febrile convulsions. Comparison by a double blind trial. *Arch Dis Child* 1980; **55**: 171–4.

2 Pearce J L, Sharman J R, Forster R M. Phenobarbital in the acute management of febrile convulsions. *Pediatrics* 1977; **60**: 569–72.

3 Pearce J L, Mackintosh H T. Prospective study of convulsions in childhood. *NZ Med J* 1979; **89**: 1–3.

J L Pearce
Department of Paediatrics, Wexham Park Hospital, Slough SL2 4HL

Use of isoprinosine in subacute sclerosing panencephalitis

Sir,

Isoprinosine has been used therapeutically in subacute sclerosing panencephalitis (SSPE) for some years although opinions on its value range from cautious optimism to sceptism. We report our observations on 4 children with SSPE who are currently being treated with isoprinosine. In each some degree of remission seems to have been achieved.

Clinical details of the patients are given in the Table. The diagnosis of SSPE and its clinical staging were established using the criteria of Jabbour *et al.*[1] None of the children had been immunised against measles and all had cerebrospinal fluid measles antibody titres greater than 1 in 32. Case 1 had been recognised to have SSPE for 11 months when isoprinosine in a dose of 100 mg/kg per day was introduced. At that time he had bulbar paralysis, intractable myoclonic seizures, and minimal cognition. Within 3 weeks of starting this treatment there was a pronounced reduction in seizure frequency, he became more responsive and alert, and partial oral feeding became possible. Other drugs were reduced or stopped and home nursing became possible after some months in hospital. This improvement was maintained for 9 months although currently the child again appears to be deteriorating.

The remaining 3 children started treatment with isoprinosine 100 mg/kg per day when their SSPE was confirmed. At this time each had intellectual and behavioural deterioration with seizures. Clonazepam was the only other medication. In all 3 the seizures have been adequately controlled and the children have maintained their pretreatment level of mental alertness although 2 are no longer mobile.

It is recognised that the course of SSPE varies and that some 10% of affected individuals have a protracted illness in which there may be prolonged remissions. This variability makes it difficult to evaluate the effect of isoprinosine although its use would appear to have some theoretical basis. Thus depression of cell-mediated immunity has been consistently demonstrated in SSPE while isoprinosine is an immune potentiator. Both in viral infections generally[2] and in SSPE specifically[3] improvement in cell-mediated immunity has been reported during treatment with this agent.

We clearly have no way of knowing whether the natural course of the disease has been modified in these 4 patients. The most obvious and consistent clinical feature however has been the maintenance of improved mental alertness. This is in our experience unusual and has been remarked on by all the parents. Even occurring in isolation such an improvement is most welcome given the usual relentless progress of this disorder. We would suggest therefore that continued trials with isoprinosine in SSPE, perhaps especially when the medication is introduced in the early stages of this disease, would appear to be justified.

References

1 Jabbour J T, Duenas D A, Modlin J. SSPE: clinical staging, course, and frequency (abstract). *Arch Neurol* 1975; **32**: 493–4.