electroencephalogram the morning after hypnotics or placebo were taken. However, blood samples were taken from the tubing of a catheter and could often be withdrawn without waking the subject. Furthermore, the study was double-blind and thus similar blood sampling took place after placebo, in comparison with which temazepam and chlormethiazole were shown to be effective hypnotics.

Drs Oswald and Adam would also have preferred that a battery of psychomotor tests had been used. The "e"-deletion test was used because it is a simple psychomotor test; there was no suggestion that performance in this test correlates with piloting an aircraft or driving down a motorway. The test was used, in conjunction with other measures, as a means of comparing drug and placebo effects. The "e"-deletion test was previously sufficiently sensitive to detect hangover effects up to 36 hours after a single dose of nitrazepam in elderly subjects.¹ In the present study there was a significant difference (Wilcoxon signed ranks test) between the performances of the old and the young ($p < 0.01$), and within each group between those found 4 and 11 hours after dosing ($p < 0.005$). We have found (unpublished observations) that "e"-deletion correlates well with more complicated tests such as the paced auditory serial addition test² in young subjects, but that older subjects found the latter test too difficult to perform and gave up before completing it. Other workers have shown that elderly women with a history of falls on rising from bed had a significantly greater degree of sway measured by ataximeter than their peers.³ Thus it is relevant that sway was not increased by hypnotics in our subjects. Again, the measurement was sufficiently sensitive to detect a significant difference in sway between the age groups ($p < 0.01$). Finally, Drs Oswald and Adam criticise our failure to use analysis of variance and accuse us of ignoring the element of chance in our analysis of pharmacodynamic tests. Non-parametric tests were deliberately used to avoid making assumptions about the distribution of the data, even though levels of statistical significance are less easily achieved with such tests. In the table in our paper 44 measurements after drugs and placebo were compared, of which 15 (34%) showed significant differences. It is a strange probability theory that can attribute this to chance.

With regard to the comments of Dr A F Macklon and others (22 March, p 861), we have again checked the elimination half life of temazepam from the logarithmic plot of the mean plasma concentration time curve; this cannot be interpreted as showing an elimination half life as long as 26 hours for either group. As we stated, there was marked variability in plasma concentrations between subjects, and insufficient estimations were made in many to allow individual beta elimination half lives to be calculated. This variability would almost certainly be increased in those suffering from illness at any age, and thus caution is necessary when extrapolating the data from normal volunteers to patients.

There is ample evidence that many hypnotics, including the commonly prescribed long-acting benzodiazepines, are unsatisfactory, particularly in the elderly—as reiterated in a subsequent leading article (15 March, p 743). While temazepam and chlormethiazole are not ideal for all subjects, it is surely justifiable to claim that they are an improvement on most alternatives.

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¹ Castleden CM, George CF, Marcer D, Hallett C. *Br Med J* 1977;ii:10-12.
² Gronwall D, Wrightson F. *Lancet* 1974;ii:605-9.
³ Overstall PW, Exton-Smith AN, Imms FJ, Johnson AL. *Br Med J* 1977;ii:261-4.

Prophylaxis against febrile convulsions

SIR,—Paediatricians and general practitioners should attempt to differentiate on the one hand between a simple febrile convulsion and on the other a convulsion precipitated by fever in a child who will later have epileptic fits. This is more than a matter of semantics as the prognosis and therefore the management of the two conditions are different. The label "febrile convulsion" should be reserved for patients between the age of 12 months and 5 years who have a generalised fit lasting not more than 10 minutes occurring once during an episode of fever. There should be no pre-existing history of neurological disorder, nor should there be any abnormal neurological findings.

In contradistinction, any history of cerebral birth injury, current neurological abnormality, or evidence of prolonged convulsion should alter the diagnosis to one of an epileptic fit precipitated by fever. Wallis and Aldridge-Smith (9 February, p 353) blur this distinction and confuse the issue by including in their group of children with "febrile convulsions" those who have obvious neurological disease.

We accept that further fits in epileptic children should be prevented where possible and that sodium valproate and phenobarbitone are two drugs which have been used successfully for this purpose. There is at present, however, little evidence that sodium valproate is effective in the prevention of simple febrile convulsions,¹ although phenobarbitone given in doses which result in therapeutic serum concentrations may be.² Furthermore, Wallis and Aldridge-Smith minimise the side-effects of drugs unduly. Phenobarbitone frequently produces unacceptable effects, particularly on behaviour. Although sodium valproate is relatively safe it can effect the haemopoietic system and liver function, with occasional irreversible hepatic failure.³ It is therefore important to determine the need for long-term anticonvulsant therapy before starting any treatment.

If paediatricians and GPs use the simple criteria stated above, they should save a large number of children from unnecessary treatment.

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¹ Williams AJ, Evans-Jones LG, Kindley AD, Groom PJ. *Clin Pediatr (Phila)* 1979;18:426-30.
² Faero O, Kastrup KW, Lykkegaard Nielsen E, Melchior JC, Thorn I. *Epilepsia* 1972;13:179-285.
³ Suchy FJ, Balistreri WF, Buchino JJ, et al. *N Engl J Med* 1979;300:962-6.

SIR,—Dr Sheila J Wallace and Mr J A Smith (22 March, p 864) says that "up to 11% of children with fits may convulse to death." What an alarming assertion, enough to make any parent or paediatrician break out in a cold sweat. Your readers may, however, find reassurance in the fact that the paper quoted¹ was written 30 years ago about 91 children admitted to hospital in Finland from 1920 to 1941 and that all that was said about them is that they had convulsions associated with infection and 10 of them died. Nowhere in the paper is it said that the children "convulsed to death." It seems very likely that the cause of death was the infection rather than the convulsion in most, if not all, of those children. Among 1706 American children with febrile

convulsions in the 1960s there were no deaths or persistent motor deficits.²

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¹ Ekholm E, Niemineva K. *Acta Paediatr* 1950;39:481-501.

² Nelson KB, Ellenberg JH. *Pediatrics* 1978;61:720-7.

Intravenous chlormethiazole in status epilepticus

SIR,—We were interested to read the article by Dr S Lingam and others (19 January, p 156) on problems with intravenous chlormethiazole in status epilepticus. We have found a further problem associated with this drug given by the intravenous route in that the droplets tend to be of much smaller size than equivalent droplets of electrolytes, etc. Therefore the volume delivered by any pump system that measures drops per minute will in fact be considerably less than expected. This can lead to underdosage with this drug.

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Unrecognised dehydration during parenteral nutrition?

SIR,—It is unfortunate that your "Lesson of the Week"—"Unrecognised dehydration during parenteral nutrition"—by Drs R W G Prescott and J C Stoddart (22 March, p 847) is inaccurate in title, fact, deduction, and conclusion.

The osmolality/osmolality of Glucoplex is 2387 mmol/l (2387 mosmol/kg; 2072 mmol/kg) not 3125 mmol/l, and the free water is 0.745 g water/ml not 0.764 g/ml as stated (personal communication, Messrs Geistlich Sons Ltd). These figures are, of course, irrelevant. If the glucose in the hyperosmolar solutions is "burnt," it represents no osmolar load. Metabolic water is produced giving a total of 0.985 g water/ml. "Correction" for fractional water content is obviously incorrect. If, however, the mass of glucose given exceeds the mass metabolised, glucose or its metabolites will accumulate. The intracellular osmolality rises and water is transferred from the extracellular compartment to the intracellular compartment. The extracellular sodium therefore rises. The extracellular osmolality reflects the intracellular environment.

In addition to the high serum sodium concentration, the hyperosmolar syndrome due to carbohydrate overload has other features. (1) The carbohydrate calorie input exceeds the measured carbon dioxide output by more than 25%. (2) The glucose level usually becomes more difficult to control. (3) Urinary sodium excretion paradoxically drops and may be less than 10 mmol/l in extreme cases. (4) Oliguria is usually obscured by clinical management. (5) The mean corpuscular volume rises, reflecting the increase in size of the intracellular compartment, which is due partly to a similar increase in size of the red cells themselves and partly to the method of measurement.¹

On the basis of the authors' figures, cases 1 and 3 were in positive fluid balance and negative sodium balance, yet had large rises of the serum sodium. Calculation suggests a shift of extracellular water into the intracellular compartment of 21% in case 1 and of 16% in case 3. Diagnosis: Overprovision of carbohydrate calories. Management: Stop intravenous nutrition until the small molecules