clinics were held in the morning and one in the afternoon. The majority of the children were seen on the same day of the week at about the same time of day. Further blood samples were taken if a child was admitted to hospital febrile, with or without a fit. Our report examining the factors involved in compliance has yet to be accepted for publication. We welcome the confirmation that the investigation of such factors should not be regarded as "a minor footnote" to our main study.

Our use of χ^2 is on the basis of the independence of the six-month periods and the importance of age-dependence in vulnerability to febrile convulsions. We should have preferred the more direct calculation using numbers of children, but with a sample size of 108 children such an approach would require consistent medication in all children over 24 months. This methodological criterion was clearly clinically unattainable.

Dr J B P Stephenson (1 March, p 643) ignores the possibility that a recurrence of a febrile convulsion may be long and damaging^{1,2}; that repeated fits with fever predispose, in at least some children, to later epilepsy³; that the longer the duration of a seizure disorder the greater is the difficulty in controlling fits⁴; and that continuance of seizures may produce severe educational and employment problems in the future.⁵ Although most series report very low mortality rates, up to 11% of children with fits may convulse to death.⁶ Dr Stephenson notes that the incidence of reported fever per se is less in children receiving either phenobarbitone or sodium valproate, and comments on our indication that this may reflect a greater use of additional non-drug management procedures by this group. However, in his arguments against anticonvulsant medication he fails to note that one implication of the lack of difference between theoretically "optimal" and "sub-optimal" blood levels could be that "optimality" needs reconsideration.

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¹ Aicardi J, Chevrie JJ. In: Brazier MAB, Coceani F, eds. *Brain dysfunction in infantile febrile convulsions*. New York: Raven Press, 1976:247-57.

² Lennox-Buchthal MA. *Febrile convulsions: a reappraisal*. Amsterdam: Elsevier, 1973:59.

³ Nelson KB, Ellenberg JH. *New Eng J Med* 1976; **295**:1029-33.

⁴ Rodin EA. *The prognosis of patients with epilepsy*. Springfield: Charles Thomas, 1968.

⁵ Harrison RM, Taylor DC. *Lancet* 1976;ii:948-51.

⁶ Ekholm E, Niemineva K. *Acta Paediatr* 1950;**39**: 481-501.

Compliance and epilepsy

SIR,—We must take issue with the comments of Dr S D Shorvon and others (16 February, p 484).

In support of their statement "... poor compliance is the major cause of recurrence of seizures, the apparent failure of the first drug, and the subsequent escalation of polytherapy" they quote their own work.^{1,2} However, there is no evidence, even in their own published work, to support this. We can find no research published of the actual drug compliance in epilepsy.

Our own study among 64 epileptics taking anticonvulsants suggests that compliance with therapy is high. Eighty-five per cent of the

predicted number of prescriptions for 12 months were actually recorded as issued. This does not take into account prescriptions issued but not recorded (on home visits, etc.). In only three cases was failure to take tablets identified as the cause of the occurrence of fits.

Shorvon *et al* in their work¹⁻³ have demonstrated that the responsibility for failure in the treatment of epilepsy usually lies neither with the disease itself nor with the patient, but with the doctor. Our audit, which is soon to be completed, strongly supports this. While compliance is important, it has not been shown to be anything but a minor cause in the failure of treatment in epilepsy.

We do strongly support Dr Shorvon and colleagues in their advocacy of single-drug prescribing for epilepsy and the need to monitor serum anticonvulsant levels.

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¹ Shorvon SD, Chadwick D, Galbraith AW, Reynolds EH. *Br Med J* 1978;ii:474-6.

² Reynolds EH. *Lancet* 1978;ii:721-5.

³ Shorvon SD, Reynolds EH. *Br Med J* 1979;iii:1023-5.

Phenobarbitone and epilepsy

SIR,—Dr R H E Grant's comment in his letter (23 February, p 560) with regard to absence of side effects of phenobarbitone therapy in epileptics prompted me to write this note. Undoubtedly, phenobarbitone adequately controls epilepsy; but in long-term therapy biochemical osteomalacia,¹ evidenced by low calcium and phosphorus and raised alkaline phosphatase, associated with proximal myopathy and folate-deficient megaloblastic anaemia may occur. These side effects should be looked for as they can be adequately treated if not prevented.

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¹ Davies-Jones GAB. In: Dukes MNG, ed. *Side effects of drugs annual 2*. Amsterdam: *Excerpta Medica* 1978:62.

Retroperitoneal fibrosis associated with atenolol

SIR,—We wish to report a second case of retroperitoneal fibrosis in a patient receiving the beta-blocker atenolol (Tenormin). The patient had taken no other drugs in the six months prior to his presentation with abdominal pain.

A 48-year-old man suffered coronary thromboses in 1969 and 1974. After the second he became hypertensive and was treated with atenolol 100 mg daily. In June 1979 he was referred to hospital with right-sided abdominal pain and an intravenous pyelogram (1.6 mg/100 ml) revealed a non-functioning right kidney. Retrograde pyelography suggested a diagnosis of retroperitoneal fibrosis. Serum creatinine was 145 μ mol/l and the erythrocyte sedimentation rate was raised at 106 mm in one hour. The right ureter was explored by an extraperitoneal approach and freed from dense retroperitoneal fibrous tissue. A biopsy of this tissue showed infiltration with large numbers of chronic inflammatory cells and lymphoid follicles suggesting idiopathic retroperitoneal fibrosis. A postoperative intravenous pyelogram revealed a return of function to the right kidney.

The relationship between the beta-blocker practolol and intraperitoneal sclerosing fibrous

reactions is well known, and this drug has now been withdrawn. We report this case because of its similarity to an earlier case of retroperitoneal fibrosis in a patient receiving the more recently developed beta-blocker atenolol.¹

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¹ Doherty CC, McGeown MG, and Donaldson RA. *Br Med J* 1978;ii:1786.

Vomiting in the first year of life

SIR,—Dr H B Valman's excellent article (1 March, p 620) on Vomiting in the first year of life is, unfortunately, written from the experience of a hospital consultant. During my days in hospital I saw numerous cases of oesophageal atresia, but I have not seen one in over 25 years of general practice.

Dr Valman fails to mention that the commonest cause of anorexia or vomiting in infants between the ages of 1 week and 2 years seen in general practice is otitis media, which accounts for about 80% of cases. Ten per cent are probably due to virus infections and the remainder to other causes. I have been surprised that not one of the medical students who have come to me has been taught this, and they have been equally surprised to discover how common and how easily missed is otitis media, whether complicated by vomiting or not.

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Antibiotics for whooping cough

SIR,—Your otherwise excellent article on vomiting, in the "First Year of Life" series (1 March, p 620), suggests that erythromycin and ampicillin are equal alternatives for the reduction of infectivity in cases of whooping cough. This is not so. Erythromycin has been shown to be highly effective and ampicillin ineffective *in vivo* using culture and fluorescent antibody techniques.¹ This is supported by my own experience in a previously reported outbreak of whooping cough,² during which I isolated *Bordetella pertussis* from one child after a five-day course of ampicillin and from five children after seven-day courses of amoxycillin. The best available evidence suggests that either erythromycin or cotrimoxazole should be used for this purpose.³

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¹ Bass JW, Klenk EL, Kotheimer JB, Linneman CC, Smith MHD. *J Pediatr* 1969;**75**:768-81.

² Jenkinson D. *Br Med J* 1978;ii:577-8.

³ Arneil GC, McAllister TA. *Practitioner* 1977;**219**: 855-8.

The coefficient of static friction for infants

SIR,—We were interested to read Dr H B Valman's article (1 March, p 620) concerning the management of vomiting in the newborn. We would, however, like to question the mechanics of nursing a baby prone with the mattress at an angle of 30-45°.

The photograph accompanying the article shows a baby tilted at slightly less than 10°.