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 epilepsy[3]; that the longer the duration of a seizure disorder the greater is the difficulty in controlling fits[4]; 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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[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 work[1-3] 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;i:474-6.
[2] Reynolds EH. *Lancet* 1978;ii:721-5.
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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 μmol/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 co-trimoxazole 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°.

Using a similar cot we calculated the angle to be 9°24'. Even on the highest tilt position provided by the manufacturers the angle is 15°33'. In a series of 24 measurements on eight babies in our nursery we found that even while asleep none could be tilted to 45° without sliding. The mean angle at which they slid was 37°34'. This would put the coefficient of static friction for infants in our nursery at 0·765 (similar to that for steel) and would suggest that without some form of mechanical support or velcro nappies 20° might be a working maximum.

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Ear syringing

SIR,—I was interested to read your article (9 February, p 374) on ear syringing by Dr Stuart Carne. May I comment on the question of the risk of damage to the drum or meatus if the patient is a child or a restless adult?

When I was young I was taught to syringe ears in the ear department at Barts, where the tradition was to use an 8-oz bladder syringe for the purpose. It is imperative that this, or any ear syringe, should be held correctly. The pinna of the ear should be held between the index and the middle fingers of the operator's left hand, with the hand resting against the side of the patient's head. The operator's left thumb should then be put in such a position that the ear syringe will rest on the thumb at the point where the nozzle joins the barrel.

In this way, however much the patient moves the operator's hand, the syringe will move with the head, thus reducing any risk to an absolute minimum. Anyone who damages an ear when syringing it and who has not taken this elementary precaution may well find that with the modern trend of damages for negligence he is being sued for damages.

A G S BAILEY

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Setting up a drip

SIR,—With reference to the article on setting up a drip (16 February, p 463), one factor that improved my success rate in finding a penetrable vein was the practice in suitable cases of placing the arm or forearm proposed in a bowl of fairly hot water.

In conjunction with the use of a tourniquet for about 30 to 60 seconds, the arm should be suspended at a lower level than the rest of the body. For example, in a supine patient the upper arm should be allowed to point toward the floor by the side of the bed. Before actual penetration the area should, of course, be dry and sterile.

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SIR,—In their excellent article on setting up a drip (16 February, p 463) Drs Barbara Bannister and C W H Harvard list as the first problem "No veins are visible or palpable," a common and daunting happening for the pressed junior doctor. Rather than proceeding to perform a "cutdown" or attempt to cannulate the subclavian or jugular vein, may I suggest a very simple and physiological way of increasing the blood flow in the arm and thereby dilating the superficial veins even in the most shocked patient?

If the patient's hand and forearm are immersed in hot water, at a temperature just bearable to one's own hand, and left to soak for approximately 10 minutes, the blood flow in the skin vessels is many times increased and the superficial veins become visible and easy to puncture. We have an arm bath available for this purpose but a bowl is suitable if large enough for immersing the hand and most of the forearm. The temperature of the water must be maintained by adding hot water.

I have used this method for many years and demonstrated and taught it to generations of house physicians, who are invariably impressed at the efficiency and simplicity of the procedure. It is an equally useful technique for venepuncture in blood sampling, and probably gives more reliable results as the sample is of free-flowing blood.

J HART

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SIR,—I was surprised to read that Drs Barbara Bannister and C W H Havard (16 February, p 463) neglect to use local anaesthesia for setting up a drip or inserting a central venous catheter. The intradermal injection of lignocaine with a 25-gauge needle is simple and quick, and has three advantages.

Firstly, it is not nearly as painful as having a 16-gauge needle and cannula pushed through unanaesthetised skin (I have made observations on myself and others). Secondly, any flinching that the patient is going to do occurs on injection of the local anaesthetic and not at the moment of insertion of the cannula. Thirdly, the 25-gauge intradermal needle can be used to break the skin at the site of injection. This allows a smooth and sensitive approach to the vein, enabling one to feel the different resistances of the tissues that the needle and cannula are passing through. It also prevents the cannula rucking up on the needle.

Finally, the use of paper tape to secure a cannula is an open invitation for the drip to be accidently torn out by the patient, the nurse, the trolley side, movement of the drip stand, etc. If it can come out it will, and always in the hypotensive patient, or the patient with no veins. There is no substitute for swathing the cannula, etc, in Elastoplast or other similar tape.

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Meningococcal infection

SIR,—Dr P Jan Geiseler (23 February, p 566) gives the results of his review of 1316 cases of bacterial meningitis in support of his contention that "the practice of pretreatment with suboptimal doses of antibiotics prior to hospitalisation is to be condemned."

The mortality rates for his two groups are, however, 7·1% (pretreated) and 8·7% (untreated). Though the decrease in mortality in the pretreated group does not reach statistical significance ($\chi^2 = 1{\cdot}15$; $p > 0{\cdot}1$) and the figures cannot be used as a simple justification of pretreatment, they certainly do not condemn it. It is indeed likely that the indication for pretreatment was the greater severity of the illness when first seen by the GP, and therefore that the prognosis of the pretreated group would have been far worse had the treatment been withheld pending hospitalisation.

Statistics can tell many tales, and perhaps greater caution is needed before condemning GPs out of hand.

CORNELIUS KATONA

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Cardiac tamponade

SIR,—Your leading article (23 February, p 505) on cardiac tamponade is timely, but the final sentence is irreconcilable with effective clinical practice. You rightly say that most physicians see cardiac tamponade only sporadically but that it constitutes a real emergency calling for prompt treatment. Most cases are going to occur in the district general hospital and will be the responsibility of the general physician. If he waits for the arrival of an experienced operator or relies on the availability of a thoracic surgeon most of the patients will not survive. May I suggest that your final sentence should read, "The prerequisites for aspiration are the strong possibility of the presence of a life-threatening amount of pericardial fluid and the presence of a punctilious operator."

In a previous leading article, "The physicians' dilemma" (24 February 1979, p 507) you commented on the decline of the general physician, and similar anxieties have been expressed on both sides of the Atlantic. Your approach to the management of cardiac tamponade highlights one of the main reasons for this decline—that is the progressive transfer from the generalist to the specialist of many of the activities which have previously fallen within the generalist's sphere. The training of general physicians should ensure that they have a working knowledge of all emergency procedures which may be required, albeit rarely, within the confines of the district general hospital and such procedures will include the management of cardiac tamponade.

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SIR,—I was interested to read the statement in your recent leading article (23 February, p 505) that "cardiac tamponade is rare in the postmyocardial infarction syndrome (Dressler's syndrome)." This conclusion is, I feel, somewhat misleading.

Dressler's syndrome is an infrequent complication of myocardial infarction, probably occurring after no more than 3% of cases.[1] However, in Dressler's syndrome, cardiac tamponade has been reported in at least three clinical situations. The first is tamponade due to non-haemorrhagic pericardial effusion.[2] This was mentioned in your editorial and is, as stated, unusual. Secondly, tamponade may follow leakage of a ventricular aneurysm. Recent reports have suggested that Dressler's syndrome is encountered more frequently when myocardial infarction is complicated by ventricular aneurysm,[3] and as these are prone to leakage or rupture[4] haemopericardium should be borne in mind as a cause of unexplained cardiac failure in Dressler's syndrome.

Thirdly, tamponade may follow inappropriate anticoagulant therapy. Although this is