prescribed a controlled drug, and therefore by implication a drug of addiction, is also likely to cause many to stop taking their phenobarbitone. All these factors would expose the patient to the risk of status epilepticus. Even the slow withdrawal of the drug does not in our experience protect patients from this eventuality, which still carries a high mortality and morbidity.

Phenobarbitone is not a drug that is abused and, because of its pharmacokinetic properties, it is unlikely to be. Its inclusion in the proposed legislation would lead to the anomalous situation whereby the prescribing of this compound would be controlled, and yet that of the pro-drug primidone (Mysoline) would be unaffected. Abuse of primidone has been reported in the USA.

It has been argued that inclusion of phenobarbitone in the legislation would facilitate analytical work by the forensic laboratories, as a simple screening test would suffice rather than specific identification of individual compounds. In fact, it is relatively easy to separate out the various barbiturates by a variety of chromatographic techniques. But we feel particularly that the blanket inclusion of phenobarbitone purely for laboratory convenience is not a satisfactory reason for legislating against a drug of non-abuse. This action would cause considerable embarrassment and inconvenience to people with epilepsy if they were found in possession of a controlled drug which, in fact, was being prescribed for sound medical reasons. The discrimination against people with epilepsy is extensive enough without adding to it in this way.

Although as ex-CURB campaigners we fully support the attempts to restrict the abuse of the hypnotic barbiturates, we do not feel that this should be achieved at the expense of a large number of people handicapped by epilepsy.

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* * * The Secretary writes: "In the publicity the BMA has given to the inclusion of barbiturates in the Misuse of Drugs Act it has emphasised that phenobarbitone remains one treatment of choice for epilepsy. Surely it is not unreasonable to ask doctors to write phenobarbitone prescriptions for those of their patients who need them and withdrawal of the drug because of this change seems unlikely. The BMA sounded the views of all branches of the profession before deciding on this policy, though it could well be that no representative of the Medical Advisory Committee of the British Epilepsy Association was personally approached, an omission the BMA regrets."—Ed, *BMJ*.

Epileptic drugs and the police

SIR,—May we add our voice to that of Dr J A Riddle (12 January, p 113) in appealing to doctors to supply their patients with a statement if they are in possession of a driving licence?

We could add to the case he quotes several others concerning people (usually young

people and often male) who have been kept overnight in police cells without medication after having been found in some sort of epileptic seizure. These people also could benefit from a medical statement. We agree with your correspondent's statement that an official form would be preferable, but in view of the length of time it takes to produce such a form it would be extremely helpful if doctors could fill the gap. This association has in fact written to the Driver and Vehicle Licensing Centre in Swansea to ask for co-operation in this matter.

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Diabetic impotence

SIR,—We are extremely interested in the results reported by Dr D J Hosking and others (1 December, p 1394) but concerned about their conclusion.

On the basis of an overlap between their diabetic and normal groups, they conclude that impotence in most diabetics is likely to be psychological rather than organic. This appears to be based on the following assumptions: (1) The occurrence of nocturnal erections distinguishes between organic and psychogenic cases. While an interesting possibility this remains to be demonstrated. The assumption that those cases in which normal sleep erections occur are psychogenic has introduced a circularity into much of this reasoning. (2) All types of organic pathology would produce the same effects on nocturnal erections. (3) Nocturnal erections during sleep in normal men are full erections and sufficient for intercourse.

None of these assumptions can be justified with the evidence at present available. While these results suggest that complete impairment of erections is not necessarily associated with diabetic impotence, the effects of either autonomic neuropathy, peripheral vascular disease, or some other pathology could interfere with the attainment of full (that is, stiff) erections and could consequently lead to anxiety about performance, which in its turn produces further impairment. There is still much to be learnt before we understand erectile failure, diabetic or otherwise.

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Intermittent venous sampling during exercise

SIR,—Drs M J G Farthing and Anna M Mattei (3 November, p 1112) and Drs D P Mikhailidis and R A Hutton (12 January, p 111) have reported experience using different techniques for intermittent venous sampling in hospital patients. When samples are required from subjects undertaking active exercise there is the additional problem of "the moving target." Using butterfly needles (gauge 19-21, tubing length 305 mm) (Abbott Laboratories) we have been able to obtain multiple samples over periods of up to two hours from subjects exercising on a cycle ergometer.¹ The butterfly

is carefully positioned in a superficial vein of the forearm away from a joint. The tubing is occluded with a compression clip. After sampling the system is flushed with sterile 0.9% saline.

The flexible narrow-diameter tubing attached to the butterfly needle is of sufficient length to allow the subject reasonable freedom of movement at the time of sampling, and also provides a system of small volume which may be flushed easily. We have not encountered troublesome clot formation in the lumen of any catheter well flushed with saline after blood sampling. This simple inexpensive technique has been used successfully in more than 30 subjects exercising at mild, moderate, or severe work intensities.

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¹ Ratcliffe RJ. BSc thesis, University of Manchester, 1978.

Eating and ulcers

SIR,—It is true, as you say in your leading article (26 January, p 205), that much dietary advice to patients with peptic ulceration "is more often based on impression and prejudice than on fact." Moreover, what little sound evidence does exist suggests that special diets are ineffective.¹ But this may well be because the wrong sorts of diet have been used.

Experience with the use of a low-carbohydrate diet for the treatment of obesity² led to the unexpected observation that any concurrent dyspepsia, associated, for example, with peptic ulceration or hiatus hernia, was in many patients relieved as soon as the diet was adopted. As a result, we instituted a controlled trial in which alternate chronically dyspeptic patients with or without demonstrable ulceration were instructed to follow either the standard hospital "gastric diet" or a diet in which carbohydrate was restricted to 60 g or 70 g a day. At the end of three months the diets were reversed.

As we have reported in a brief communication³ to the Nutrition Society, of the 41 patients who completed the trial, two reported that their symptoms were improved more with gastric diet, 11 found no difference, and 28 found that they were better with the low-carbohydrate diet. The chief difference between the diets was the high proportion of starch and sugar (sucrose) in the gastric diet and the low proportion of these constituents in the low-carbohydrate diet. In order to see whether it was the reduction of dietary starch or the reduction of dietary sugar that was the more likely cause of the symptomatic improvement with the low-carbohydrate diet, we examined the gastric juice of seven healthy young men who were volunteers in an experiment in which their dietary sugar was increased and their dietary starch decreased by a similar amount.⁴ A test meal⁵ carried out before breakfast revealed that, after two weeks on the high-sugar diet, there was an increase in peak gastric acidity by about 20% and an increase in peak pepsin activity by about 250%.

Thus we can now say that, while it is true that conventional dietary treatment is ineffective in relieving the symptoms of chronic dyspepsia, it is not true that all dietary treatment is ineffective. But it remains to be seen whether a diet that is low in carbohydrate, and

more specifically low in sugar, is associated with the healing of any ulceration that may accompany the dyspepsia, as well as relieving the symptoms.

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¹ Doll R. *Scott Med J* 1964;9:183-96.

² Yudkin J. *Med Press* 1961;246:64-8.

³ Yudkin J, Evans E, Smith MGM. *Proc Nutr Soc* 1971;31:12A.

⁴ Szanto S, Yudkin J. *Postgrad Med J* 1969;45:602-7.

⁵ Hunt JN. *J Physiol* 1951;113:169-84.

SIR,—In your leading article "Eating and ulcers" (26 January, p 205) the advice offered appears to be that there is nothing to be done except keep on taking the tablets. While agreeing that conventional dietary regimens have little significance in the management of European peptic ulcers, we would like to draw attention to the increasing evidence of a relationship between diet and the incidence of duodenal ulceration in India.¹

Animal experiments in peptic ulcer cannot be dismissed quite so easily as you suggest. Their relevance to man in establishing the regulatory mechanisms of gastric secretion is surely acceptable. The fact that there is no difference in the acid output between patients on different diets from high- and low-incidence areas should not be used to dismiss diet as irrelevant, but rather to indicate that its effects are on some aspect of the aetiology other than acid.

Animal experiments based on dietary manipulation to resemble that of subjects from high- and low-incidence areas also show a correlation which may explain the geographical distribution in peptic ulcer.² Our experiments with the pylorus ligation technique in the rat clearly show that prefeeding the animals with diets similar to those eaten in low-incidence areas confers considerable protection against experimental ulceration.³ There is increasing evidence that foodstuffs from low-incidence areas contain a protective factor which is a lipid or liposoluble substance. If this is the case, no doubt a suitable therapeutic agent can be developed. However, a more logical approach would be appropriate modification of the diet. No diet exists with an inherent H₂ antagonistic activity, but diets probably already exist in India which protect against ulceration. We are not yet at the death of dietary regimens.

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¹ Tovey Frank I. *Gut* 1979;20:329-47.

² Tovey FI, Jayaraj AP, Clark CG. *Postgrad Med J* 1975;51:366-72.

³ Jayaraj AP, Tovey FI, Clark CG. *Postgrad Med J* 1976;52:640-4.

Compliance or unnecessary prescribing?

SIR,—The two themes of your editorial (10 November, p 1168) are well illustrated by the drug treatment of epilepsy.

There is no doubt that poor compliance with anticonvulsant therapy, even the omission of one or two doses, does matter. The patient is thus exposing himself to the possibility of withdrawal seizures, the perpetuation of his problem, and occasionally to such disasters as the burning described by Drs W F Durward

and M G Harrington in the same issue (p 1218). This applies throughout the many years of such therapy and imposes particular difficulties on patients who are often asked to take complicated regimens of polytherapy several times a day. However, it is especially important in the early years of therapy as poor compliance is the major cause of recurrence of seizures, the apparent failure of the first drug, and the subsequent escalation to polytherapy.^{1,2}

On the other hand, there is no satisfactory evidence that polytherapy is more effective in the control of seizures than a single drug used to its maximum potential with the assistance of blood level monitoring.^{2,3} The aim of treatment should be to use one or at the most two drugs per seizure type, with the maximum of compliance and ensuring if necessary optimum blood levels. If the patient fails to comply with unnecessary polytherapy he may be at no disadvantage, provided he does not indulge in intermittent therapy.

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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.

Legal aspects of child injury or neglect

SIR,—We would like to correct a technical point in our paper "Legal aspects of child injury or neglect" (13 October, p 910).

In the final paragraph under the heading "Care order" (p 911) it is stated that a local authority or voluntary body can appeal to the Crown Court if they are concerned about the failure to obtain a care order, or are concerned by the termination of a care order by a juvenile court on the application of a parent. This statement is inaccurate in that, under the Children and Young Persons Act 1969 (Section 2 (12)), only the child in question can appeal to the Crown Court.

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Prolonged intravenous use of chlormethiazole (Heminevrin)

SIR,—We were interested to read the article by Dr S Lingham and others (19 January, p 155). Since 1974 we have found intravenous chlormethiazole (Heminevrin) to be effective in the treatment of status epilepticus in children who did not respond to conventional anticonvulsant therapy, including infusions of diazepam and lignocaine. Although rarely required, prolonged chlormethiazole infusion is sometimes both necessary and possible, as illustrated by the following case.

A girl presented at age of 27 months with progressive right hemiparesis and right hemiconic seizures. Six months after onset the seizures became exceedingly frequent and were then generalised as well as partial. Various antiepileptic therapies, including all available standard drugs, adrenocorticotrophic hormone, levo-dopa, and ketogenic diet were ineffective. Two computed

tomography scans and a carotid angiogram did not reveal a focal lesion, but on electroencephalography the seizures consistently arose from the left anterior head region. A left frontal brain biopsy showed "focal subacute panencephalitis" but no virus was visible on electron microscopy or detectable serologically.

Intravenous infusion of a 0.8% solution of chlormethiazole through a paediatric giving set (Travenol Laboratories Ltd, Norfolk) at a rate of 10-18 mg/kg/h was started when all conventional anticonvulsant therapy had failed to control the seizures. Treatment with this drug was continued for 43 days before the effect of partial section of the corpus callosum allowed us to tail off the infusion. Throughout most of the treatment period, the patient remained conscious and responsive. Thrombophlebitis was a problem on only two occasions and was simply dealt with by changing the infusion site. No haematological, renal, or hepatic toxicity was observed.

Although we agree with Dr Lingham and his colleagues that care is essential, patients should not be denied prolonged chlormethiazole infusion when no other therapy is possible, and particularly when later epileptic surgery is contemplated.

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Whooping-cough immunisation

SIR,—I was not surprised at the reasons that Professor R S Illingworth (19 January, p 179) quotes for parents' refusing immunisation. I think, however, that there is in fact a different explanation for the sources of the myths he mentions.

In our practice more than three-quarters of the mothers of 7-month-old children express misgivings about the safety of immunisation and in particular the pertussis component of the triple vaccine. When asked why they are worried they quote friends, the lady down the road, parents, a man at work, and often a doctor on television. What is so interesting is that the reasons are the same as Professor Illingworth gives as coming from doctors and health visitors in his sample. Dare I suggest that he has yet to learn not to believe all he is told by parents?

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Training for hospital practitioners

SIR,—As a contribution to the solution of the problems of staffing structure, the employment in specialist departments of general practitioners in the hospital practitioner grade has considerable merit and helps to bridge the division between the two major branches of the profession. However, general practitioners are now largely recruited from among those doctors who have primary care as their aim from the time of graduation, rather than from those who have spent a considerable period in specialist hospital work. The new arrangements for vocational training emphasise short but wide-ranging experience in hospital medicine. Thus there will be few principals in general practice with sufficient qualifications to undertake a hospital role in specialist clinics without further instruction.

Some encouragement must therefore be given to general practitioners with an interest in a specialist subject and anxious to forge a