In our experience, a single intramuscular injection of 150 mg medroxyprogesterone acetate administered during the early puerperium is a highly efficacious contraceptive measure, which in that period offers the extra advantages of not affecting lactation or predisposing the woman to thromboembolic complications. Therefore injectable hormones provide a rational approach for doctors wishing to give a contraceptive that will bridge the first postpartum trimester with optimal efficacy. However, as Dr Murphy clearly stresses, when initiated soon after birth (or abortion or miscarriage) the medroxyprogesterone treatment involves the risk of protracting vaginal bleeding without significantly affecting uterine involution. We also found, albeit more or less by chance, that in practice this sign appears to be highly distressing for some women.

For many years it has been our routine practice to delay surgical tubal sterilisation until the first postpartum menstrual period has occurred, at least in non-lactating women. To provide effective contraception during this interval we recommended an intramuscular injection of medroxyprogesterone administered at the time of hospital discharge. However, the results of the psychological evaluation conducted three and 12 months after voluntary sterilisation of 115 females and 70 males (185 couples) showed that the depot injection had led in some of the women to negative reactions which became manifest during the post-operative assimilation process. Even after active counselling and support, several women persisted in attributing their altered bleeding pattern to the sterilisation instead of to the contraceptive treatment. In a few of these women, who bled more or less continuously for more than six months, the concern about the metrorrhagia became pathological to a degree requiring psychiatric care. Consequently, we no longer recommend medroxyprogesterone for contraception in such cases, and though some of our patients in the puerperium still ask for it we delay the first injection until a menstrual period has occurred. If administered no later than on day 8 of the second postpartum cycle, this method has proved to be effective and the bleeding pattern does not differ from that usually observed with interval administration of this depot preparation.

A more recent psychological evaluation of a new series of women sterilised according to the same technique but not given medroxyprogesterone while awaiting partial tubectomy has shown that the adverse psychological reaction and the concomitant difficulties in assimilating the effects of the operation have no longer occurred.

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Cutaneous manifestations of hydrallazine toxicity

SIR,—We read with interest recent reports by Dr M W Kissin and Professor R C N Williamson (24 November, p 1330) and Dr R M Bernstein and others (19 January, p 156) of cutaneous manifestations of late toxicity to

hydrallazine, and wish to report a case currently under our care.

A 58-year-old man with severe hypertension had a bypass vein graft for left renal artery stenosis in May 1976 and commenced hydrallazine. The maximum dose was 150 mg daily, with a total dose to date of 150 g. In November 1978 he complained of polyarthralgia; the latex test for rheumatoid factor gave negative results. He presented in December 1979 with malaise, fever, bilateral pleuritic chest pains, haemoptysis, and mouth and penile ulcers. In addition he had vasculitic lesions on the buttocks, forearms, and hands, and a deep ulcer over the metacarpophalangeal joint of the right index finger. These have begun to heal since hydrallazine has been withdrawn and adrenocorticotrophic hormone started. At this time he was anaemic (Hb 8.3 g/dl) and had a raised sedimentation rate (114 mm in the first hour, Westergren) and increasing uraemia, with haematuria and albuminuria. The antinuclear antibody test is positive at > 1 in 400 (homogeneous staining), having been 1 in 10 in 1976.

This case fulfils several of the criteria of patients at risk of developing late toxicity to hydrallazine¹ including initial accelerated hypertension, a previously positive antinuclear antibody test, a cumulative dose of more than 100 g, and coexistent autoimmune disease (he has antibody-positive primary hypothyroidism). The cutaneous manifestations in this patient were widespread. With increasing use of hydrallazine, even at low doses, we must be vigilant for the sensitisation effects of such toxicity.

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¹ Perry HM jun. *Am J Med* 1973;54:58-72.

SIR,—Following the article of Dr M W Kissin and Professor R C N Williamson (24 November, p 1330), we would like to report on a case of the hydrallazine-related lupus erythematosus (LE) cell syndrome associated with extensive ulceration.

In 1971, when she was 21, this patient developed malignant phase hypertension with a blood pressure of 220/160 mm Hg, early papilloedema, and haematuria. Blood pressure control was difficult because of frequent postural hypotension and she was eventually controlled in 1976 on a regimen of 640 mg propranolol, 200 mg hydrallazine, and 10 mg bendrofluzide daily. In 1979 she came under the care of this hospital. After four miscarriages she was pregnant for the fifth time and was admitted for Shirodkar suture. For a few days prior to admission she had had irritation over the left arm and right leg and had small tender areas of purpura. The operation was performed uneventfully. The purpuric rash persisted and the LE cell test was found to be positive. Antinuclear factor was present, but antibodies to DNA were absent. Urea, electrolytes, and blood count were normal, and her hypertension was well controlled. Hydrallazine was discontinued, but her rash became very extensive and painful, and within five days started to vesiculate. Treatment with prednisolone was started at this stage. The rash did not extend any further; but the areas of confluent purpura and vesiculation became necrotic and extensive shallow ulceration developed over the whole affected area, requiring skin grafting. It finally healed completely after three months. The steroid was reduced abruptly at the time of the skin grafting and this precipitated an episode of pericarditis, with a friction rub and cardiographic changes. This settled as soon as the dose of steroid was increased. The blood pressure remained well controlled on 500 mg methyldopa and 10 mg bendrofluzide and the patient was delivered of a

Extensive ulceration associated with hydrallazine.

normal infant at 34 weeks. The LE cell test remained positive for three months after delivery but has now become negative, and her legs remain well healed. Her blood pressure is tending to rise and she requires a larger dose of methyldopa for control.

The remarkable feature about this woman's illness was the rapidity with which the purpura extended and became confluent. There was presumably an extensive underlying arteritis because these areas immediately became necrotic and gradually sloughed. Prednisolone appeared to prevent further extension of the purpura and it is possible that starting it as soon as the diagnosis was made may have prevented so much ulceration from occurring.

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Phenobarbitone and epilepsy

SIR,—We have recently learnt that it is official BMA policy to support the proposed legislation to include all 5,5-disubstituted barbiturates within the schedules of the Misuse of Drugs Act. We would like to comment on the effect that this would have on the prescription of phenobarbitone for the treatment of epilepsy.

Although the necessity to prescribe phenobarbitone has been reduced by more modern antiepileptic drugs, there are still a large number of people whose epilepsy is satisfactorily controlled by this drug. We feel that the inclusion of phenobarbitone within the provisions of the Act would expose many people with epilepsy to unnecessary inconvenience and possibly hazard. Many doctors would feel disinclined to comply with the extra demands of writing a prescription for a controlled drug and would attempt to withdraw phenobarbitone. The fact that many chemists would stop stocking the drug would increase the difficulty in obtaining it and would therefore encourage non-compliance. Furthermore, the concern of the patient over being

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