consider, however, that the risks of lactic acidosis make the use of phenformin undesirable.⁷ Stanozolol on the other hand has been shown to be effective when given alone for periods up to 30 months and it is relatively free from side effects.⁸ We should like to suggest that stanozolol is effective in reversing the deficiency of fibrinolysis found in Behçet's disease and that it deserves further consideration as long-term therapy to prevent the development of potentially serious thrombotic episodes in patients with this condition.

We would like to thank Dr B Colvin for advice and Professor R D Cohen for permission to report this case.

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Eyesight standard for driving

SIR,—As an ophthalmologist I am regularly seeing middle-aged patients who come in to have their reading glasses strengthened but who, I find, have been driving with visual acuity below the legal standard. None of these had realised that the distance vision was not good, so that there was no question of writing to the Vehicle Licensing Centre. There was also a driving instructor who worked for three months with substandard vision following the loss of his spectacles.

I would agree that poor vision is not one of the main causes of road accidents. However, I think that the legal standard is reasonable. It should be enforced by having the police carry out spot checks with the number-plate test in laybys when they check the lights, brakes, and tyres. We seldom hear of a prosecution for driving with inadequate vision. There could also be more publicity on this subject.

All my patients became "legal" by my prescribing a distance corrector for their hypermetropia.

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Hypotensive effect of propranolol and phenoperidine in tetanus

SIR,—Dr N Buchanan and others (22 July, p 254) describe a patient with tetanus who developed profound hypotension after receiving propranolol. I have recently seen a similar phenomenon in which both propranolol and the synthetic narcotic phenoperidine appeared to be implicated.

A previously very fit 71-year-old man developed tetanus six days after sustaining a penetrating injury of the forearm while gardening. Artificial ventilation

was established, D-tubocurarine and diazepam being used as relaxants. Heart rate and intra-arterial recordings of blood pressure were monitored continuously. Phenoperidine 2 mg was given intravenously for sedation on five occasions during the first 24 h, during which period the heart rate and blood pressure remained stable. On the second day after admission the heart rate rose gradually to 150/min with a blood pressure of around 150/70 mm Hg. After a short burst of supraventricular tachycardia propranolol 2 mg was given intravenously over 5 min. The heart rate fell to around 120/min with no change in blood pressure. An hour later phenoperidine 2 mg was given intravenously and the systolic blood pressure immediately fell to 30 mm Hg at a heart rate of 100-120/min. There was no evidence of reflex vasoconstriction and the hypotension persisted for 5-10 min until it was rapidly and completely reversed by the narcotic antagonist naloxone 0.4 mg intravenously. The patient appears to have suffered no ill effects during this episode.

There is little doubt that phenoperidine was the immediate cause of the fall in blood pressure in view of the sequence of events and the prompt response to naloxone. However, previous doses of phenoperidine had not produced hypotension in this patient and it appears that prior β -blockade with propranolol was an important contributory factor.

The autonomic manifestations of tetanus are complex and unpredictable. When a β -blocking drug is considered essential to control sympathetic overactivity it would seem advisable to use the minimum effective dose and the possibility of a synergistic hypotensive effect with narcotics should be borne in mind.

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Body weight and endocrine function in anorexia nervosa

SIR,—Dr R D G Leslie and his colleagues (19 August, p 526), in their study on the influence of weight gain on hypothalamic-pituitary-thyroid (HPT) function in anorexia nervosa report that pituitary thyroid-stimulating hormone (TSH) reserve, as assessed by a standard thyrotrophin-releasing hormone (TRH) test, was reduced at low body weight. In a previous letter (20 August 1977, p 521) they reported a similar finding for prolactin. They suggest that these results are in accord with the theory that there is excessive dopaminergic activity at central receptors in anorexia nervosa.

We have recently completed a study of 20 patients with anorexia nervosa with particular emphasis on interrelationships between body weight and concentrations of gonadotrophins, prolactin, TSH, and thyroid hormones.¹ Our results in relation to the HPT axis are in substantial agreement with those of Dr Leslie and his colleagues, as were the findings of normal resting prolactin concentrations, but we were unable to demonstrate any correlation between body weight and the prolactin response to TRH. Beumont *et al*² were also unable to show any effect of weight on prolactin responses to TRH. In our own study and that of Beumont *et al*, however, TRH tests were performed on patients of low weight only after they had been in hospital for at least five days and had resumed an adequate diet. It is important to know whether Dr Leslie and his colleagues studied their patients initially in the starvation phase or after a period of refeeding,

as it is known that prolonged fasting results in a blunting of prolactin response to TRH.³

Many of the endocrine changes in anorexia nervosa occur as a consequence of malnutrition and quickly resolve with either the resumption of a normal diet or restoration of normal weight.⁴ It is clearly inappropriate to accord such endocrine changes and their central mechanisms any primary significance in the aetiology of the disorder. Many patients, despite weight gain, retain the characteristic psychopathological features of anorexia nervosa. In order to attach primary aetiological significance to changes in endocrine function it would have to be demonstrated that such changes are not merely the secondary effects of a low body weight. The clearest evidence so far of failure to resume normal endocrine function after resumption of body weight relates to the hypothalamic-pituitary-gonadal axis.⁵ Findings in this area, however, are not yet sufficiently advanced to permit aetiological hypotheses in terms of central neurotransmitter activity.

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Phenothiazine-induced dystonic reaction while swimming

SIR,—We wish to call attention to a rather unusual but potentially very serious situation in which a 24-year-old woman who had been admitted to hospital with a diagnosis of acute schizophrenia developed a dystonic reaction during a swim with other patients. Eight days previously she had experienced a dystonic reaction involving eye, neck, and lower extremity muscles approximately 26 h after she had received 20 mg of oral fluphenazine hydrochloride. The dystonia quickly subsided after 2 mg of intramuscular benztropine mesylate, which was continued orally at 1 mg twice daily. Oral fluphenazine hydrochloride was resumed at a dosage of 10 mg once daily at 9 pm.

Approximately 19 h after the patient had received her evening dose of fluphenazine and 7 h after her morning dose of benztropine mesylate she felt her legs "tighten up" while swimming in the deep end of the pool. She went under several times but managed to call out for help. She was pulled out almost immediately; one observer noted that her eyes "rolled back" several times, an apparent oculogyric crisis. She reported that after swimming 1½ lengths her legs had gone into spasm, making it impossible for her to stay afloat. After approximately 20 min these symptoms subsided completely.

We believe that this patient developed a phenothiazine-induced dystonic reaction of the lower extremities. We do not think that there is any association between swimming and dystonic reactions because many patients have participated in our swimming programme during the past four years without an incident