examination. However, if the tail is cut with the loop of tail left in the uterus after insertion entanglement with tampons or introducers of vaginal preparations may result in the tail being pulled down to the introitus, with resultant vulval discomfort. The tail is then naturally cut shorter and may again disappear into the uterus as it reverts to the looped position. Confirmation of the presence of the device in the cavity now necessitates uterine sounding, ultrasonic scanning, or *x*-raying, and, if it cannot be changed otherwise, the risks of a general anaesthetic with impairment of patient confidence in the method.

Although tail complications are not usually serious, they lead to inconvenience and worry for the patient. The design of the tail and introducer is as important as the design of the device itself.

R A SPARKS

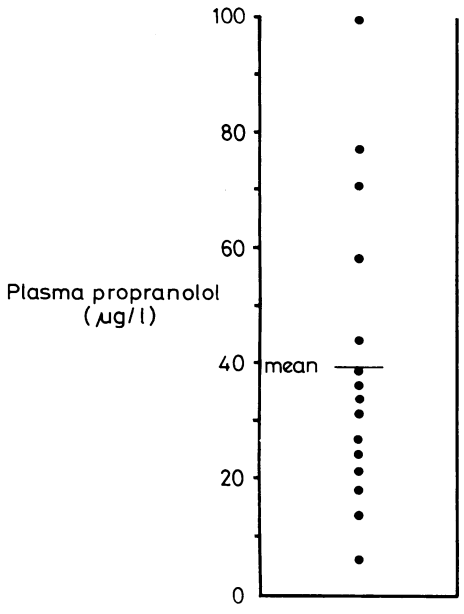
University Department of Human Reproduction and Obstetrics, Southampton General Hospital, Southampton

[1] Gräfenberg, E, in *The Practice of Contraception*, ed M Sanger and H M Stone, p 33. London, Baillière, Tindall and Cox, 1931.
[2] Tatum, H J, *et al*, *Journal of the American Medical Association*, 1975, **231**, 711.
[3] Sparks, R A, *et al*, in *The Uterine Cervix in Reproduction*, ed V Insler and G Bettendorf, p 271. Stuttgart, Thieme, 1977.
[4] Oates, J K, *Lancet*, 1976, **1**, 751.

Beta-blocking drugs and thyroid function

SIR,—Your recent leading article (22 October, p 1039) on beta-adrenergic blocking drugs and thyroid function once again questions the usefulness of these agents in hyperthyroidism. The largest published series[1] reported favourably on the use of propranolol to prepare patients for surgery, there being a marked reduction in preparation time (and in outpatient attendances) and a reduction in gland vascularity. The postoperative course was similar to that in conventionally prepared patients and thyroid crisis has not occurred in a series[2] of 150 patients prepared with propranolol alone. However, in a smaller series[3] of six patients increasing thyrotoxic symptoms were noted postoperatively in two patients, but this study may be criticised on the basis of inadequate dosage of propranolol, particularly in the perioperative period. In our series of some 30 patients prepared with propranolol we have not seen thyroid crisis.

One reason for the large discrepancy in the reported clinical experience with propranolol in hyperthyroidism may lie in the wide individual variation in the metabolism of propranolol. The figure shows the steady-state plasma propranolol concentration at 4 h post-dosage, measured by a specific gas liquid chromatographic method in 15 hyperthyroid patients who received propranolol 40 mg six-hourly (conventional dosage) for at least one week. Drug compliance was apparently satisfactory. There is obviously a wide (20-fold) interindividual variation in plasma propranolol steady-state levels in patients on the same propranolol dose regimen. Allowing for variability of assay methods, plasma levels of propranolol in excess of 50 μg/l[4] are generally required for maximal beta-blockade, whereas levels of less than 30 μg/l may be inadequate[5]; 40% of the patients in our study had levels below 30 μg/l.



Plasma propranolol concentration 4 h post-dosage in 15 hyperthyroid patients receiving 40 mg 6-hourly.

We feel therefore that patients receiving beta-adrenergic blocking agents for hyperthyroidism need to have the dosage requirement carefully assessed and frequently reviewed.

JOHN FEELY W F D HAMILTON
A L FORREST IAN H STEVENSON
ANDREW GUNN JAMES CROOKS

Ninewells Hospital Medical School, Dundee

[1] Michie, W, *et al*, *Lancet*, 1974, **1**, 1010.
[2] Michie, W, personal communication.
[3] Ljunggren, J G, and Persson, B, *Acta Chirurgica Scandinavica*, 1975, **141**, 715.
[4] Shand, D G, *New England Journal of Medicine*, 1975, **293**, 280.
[5] Alderman, E L, *et al*, *Circulation*, 1975, **51**, 964.

SIR,—Your leading article (22 October, p 1039) on the relationship between the adrenergic nervous system and the thyroid highlighted the actions of beta-blocking drugs without intrinsic sympathomimetic activity on some of the peripheral effects of excess circulating thyroid hormones. Although the beneficial effects of propranolol on some of the symptoms of thyrotoxicosis are well recognised, I can recall no accounts of adverse effects when the drug is used in myxoedema.

A 58-year-old woman was seen in 1973 with a two-year history of hypertension treated unsuccessfully but without side effects with bethanidine, oxprenolol, and methyldopa. Propranolol 20 mg thrice daily was prescribed in combination with bethanidine and cyclopenthiazide, but within a week she discontinued propranolol "feeling awful." She was encouraged to try the propranolol again but once more said she felt generally unwell after a few days. When seen two weeks later her heart rate was about 70/min and there was no postural or exertional hypotension. She was then recognised as being myxoedematous, with a serum protein-bound iodine concentration of 78·8 nmol/l (1·0 μg/100 ml) (normal 292-638 nmol/l (3·7-8·1 μg/100 ml)).

Her hypertension proved easy to control on small doses of prazosin and a diuretic once she was euthyroid. The side effect, although nonspecific, was severe and seemed clearly related to propranolol, although she had previously tolerated oxprenolol.

It seems possible with this experience and on theoretical grounds that patients with a deficiency of circulating thyroid hormones may be particularly prone to certain side effects from beta-blockers without intrinsic sympathomimetic activity, such as propranolol and sotalol. Complaints of fatigue on these drugs should perhaps raise the possibility of underlying hypothyroidism.

B I HOFFBRAND

Whittington Hospital, Archway Wing, London N19

Liquorice-induced cardiac arrest

SIR,—Dr Barbara Bannister and her colleagues, in their report of a cardiac arrest due to liquorice-induced hypokalaemia (17 September, p 738), fail to comment on several aspects of interest.

Firstly, their patient was hypomagnesaemic, and the hypomagnesaemia was probably clinically significant, as attested to by the episode of tetany in face of a normal serum calcium concentration. In addition, although the serum bicarbonate level was high, no arterial pH value is given. In the presence of significant hypokalaemia the elevated serum bicarbonate is most likely a reflection of metabolic alkalosis, which can also produce tetany with a normal total serum calcium by decreasing the ionised calcium portion. Both hypomagnesaemia and metabolic alkalosis can cause severe disturbances of the cardiac rhythm, including ventricular fibrillation,[1 3] and they, independently or in conjunction with hypokalaemia, may have been the factors responsible for the development of cardiac arrest, which in the case reported cannot be solely attributed to hypokalaemia.

JESUS MONTOLIU

Rogosin Kidney Center, New York Hospital—Cornell Medical Center, New York

[1] Loeb, H S, *et al*, *Circulation*, 1968, **37**, 210.
[2] Chadda, K D, Lichstein, E, and Gupta, P, *American Journal of Cardiology*, 1973, **31**, 98.
[3] Lawson, N W, Butler, G H III, and Ray, C T, *Anesthesia and Analgesia: Current Researches*, 1973, **52**, 951.

**We sent a copy of this letter to Dr Bannister and her colleagues, whose reply is printed below.—ED, *BMJ*.

SIR,—In reply to Dr Montoliu we agree that both severe alkalosis and severe hypomagnesaemia can cause disturbances of muscle function, including cardiac dysrhythmias and tetany. However we should like to make three further comments.

Firstly, calcium, magnesium, and potassium balance are interdependent to some extent, perhaps because of interaction at membrane transport level. Hydrogen and potassium transport are certainly related in this way. The fact that potassium infusion appeared to precipitate tetany in our patient would be compatible with such interdependence. Viewed in this way hypomagnesaemia and a raised bicarbonate level may be an unavoidable accompaniment of prolonged hypokalaemia rather than a separate entity contributing to the cardiac arrest.

Secondly, the relative magnitude of the potassium abnormality was much greater than that of bicarbonate or magnesium, and the cardiac dysrhythmia and muscle weakness responded very promptly to infusion of potassium alone. This suggests that the potassium deficiency was the most significant metabolic defect.

Thirdly, hypomagnesaemia is a feature of hyperaldosteronism, and aldosterone is known to increase magnesium excretion.[1 2] Our