join the rota for experience, and the senior house officer on rotation."

Dr R J Brereton (15 October, p 1021) takes me to task for my careless sentence construction and asks for enlightenment. I had thought that it was obvious from the text that good antenatal supervision and intensive care after delivery, when necessary, contributed to an improved perinatal situation, whereas competent antenatal investigation would reduce the number of children born with disorders such as Down's syndrome and spina bifida.

GEORGE KOMROWER

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Names of drugs

SIR,—I would agree with your leading article (15 October, p 980) defending the non-use of proprietary drug names in medical articles.

Moving from British general practice to an academic job in the USA, I learnt the generic names, where I did not already know them, of all of the drugs I had become used to prescribing by proprietary names. Whether I had been right to do that is open to discussion, but patients and pharmacists (at least in my locality) found proprietary names often easier to use (and therefore less open to error, which is surely the main point). However, on arriving here, to my surprise I found that not only are the proprietary names different, as expected, but the approved names frequently are too (for example, paracetamol is acetaminophen), even with recently introduced drugs (for example, sodium cromoglycate is cromolin sodium).

Neither the *British National Formulary* nor the American Medical Association's *Drug Evaluations* gives these equivalents, and with increasing dissemination of medical personnel and literature I would say that a publication such as Dr G D J Ball suggests (15 October, page 1024), but listing the equivalent generic names as well as proprietary names of products still under patent or widely prescribed under their proprietary names, would be essential in any medical library that contains American as well as British publications (that is, most medical libraries). It would certainly have helped me.

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ECT and the media

SIR,—I have been interested by the comments concerning ECT and the media (22 October, p 1081, and 5 November, p 1215) as I am in the process of carrying out a study into the attitudes of patients to ECT and the effects of the media on these patients. My attention was drawn to this subject by a patient who refused to have ECT despite considerable morbid depression. He gave as his reasons the comments made on the television programme "Panorama." He had interpreted them as being an indictment of ECT. He was particularly worried that he would need repeated courses of ECT for many years and that his memory would be impaired, both being suggestions which came from the programme and both being very far from the truth, as several studies have shown.

I am sure that many psychiatrists have come across similar problems. The interesting point is, how much effect does this publicity, biased or not, have upon the people who are recommended for ECT? These people are almost all depressed by definition; they are therefore more likely to be hopeless and fearful about any treatment. They are also more likely to pick up, or misinterpret, hopeless messages about treatments for depression—"Nothing can really help me." It seems to me that if in fact the media are contributing to the worsening of a clinical condition (that is, depression), then they should rethink their policies.

I am pleased to report that the patient mentioned eventually, after several weeks of worsening depresssion, was persuaded to have a short course of ECT and is now happy, back at home, and grateful for the treatment.

J M BIRD

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SIR,—Dr J F Anderson and his colleagues (22 October, p 1081) complain of the unfavourable "image" of psychiatry conveyed by the media. They argue that especially in respect of ECT the picture painted is not only biased but is likely to deter patients who would benefit from this treatment. They themselves consider that ECT is the most consistently effective treatment for depression and they cite instances of patients being persuaded against it by relatives who had seen television programmes.

I think that these consultants may perhaps overestimate both the extent to which their optimism is shared by former recipients of ECT and the unwillingness of people in severe depression to take risks. In the latter regard one could describe cases of persons improvising their own methods of shock treatment that they know perfectly well are dangerous. The motive, however, is not suicidal but rather an attempt at self-help when all other remedies have apparently failed. Also there were those among us who, before anaesthetics were introduced, *asked* for ECT. This was despite the fact that each such treatment was almost like going to the electric chair.

In the former regard there are those who, when psychiatrists talk about ECT working "wonderfully well," think about Lourdes. We have had ECT, antidepressant drugs, and group therapy, and none of them helped. We believe we eventually got well not through these techniques but through "natural causes" assisted by some supportive commonsense talk.

It is true that psychiatry is sometimes unfairly attacked, even hysterically. But psychiatrists will not advance their own cause if they ignore a simple fact. This is that the experience of so-called mental illness can rarely be publicly discussed by those who have actually undergone it. They fear, often justifiably, that careers and social reputations will be jeopardised if it is known that they have had treatment, especially as inpatients. These people have, in consequence, very little outlet for their frustration if dissatisfied with psychiatry.

Perhaps this is why there are so many campaigns that are, from the point of view of the Cupar consultants, irrational and even "malign."

FORMER PSYCHIATRIC PATIENT

Gamma-glutamyl transferase in ascitic fluid in primary hepatoma

SIR,—One wonders if the high levels of γ-glutamyl transferase in the ascitic fluid of patients with hepatoma, as reported by Dr T J Peters and others (18 June, p 1576), are not simply secondary to the high serum levels of the enzyme which undoubtedly were present.

The inclusion of this information in their report would have permitted a better appraisal of the potential usefulness of this test in distinguishing hepatoma from other hepatic diseases which also produce greatly elevated serum levels of γ-glutamyl transferase.

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

SIR,—In answer to Dr Noll the *serum* levels of γ-glutamyl transferase were equally elevated in the patients with hepatoma and those with cirrhosis without hepatoma. Several patients with cirrhosis had high serum levels of γ-glutamyl transferase but low levels of the enzyme in ascitic fluid. It is well recognised that assays of serum γ-glutamyl transferase are not helpful in distinguishing patients with hepatoma from those with other forms of liver disease. It is therefore unlikely that the levels of the enzyme in ascitic fluid merely reflect serum enzyme levels.

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Problems with IUCD tails

SIR,—The assessment of intrauterine contraceptive device (IUCD) usage has naturally been mainly concerned with method failure, leading to pregnancy, and with side effects such as bleeding and pain, leading to IUCD removal. Although Gräfenberg[1] felt that the presence of a cervical appendage led to the introduction of bacteria into the uterine cavity, its possible role in the production of serious infection did not achieve prominence until the controversy over the multifilamentous tail of the Dalkon Shield.[2] Since then it has been shown that all types of IUCD tail result in the continued presence of small numbers of vaginal bacteria in the uterine cavity.[3]

Oates[4] has recently described a lesser-known problem. Long tails may become coiled and knotted. The regular use of vaginal tampons may lead to the adhesion of small particles of cotton wool to the knot and the formation of a small mass of foul-smelling material whose presence soon becomes apparent to the patient.

A more important problem is the disappearance of the Gravigard (Copper-7) tail with the device still in utero. The Gravigard is supplied partly loaded into the introducer with a loop of tail alongside it. If the tail is pulled down into the vagina after insertion and cut it is liable to withdraw into the uterine cavity and revert to the looped position it was accustomed to in the introducer. It can then no longer be seen on speculum

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

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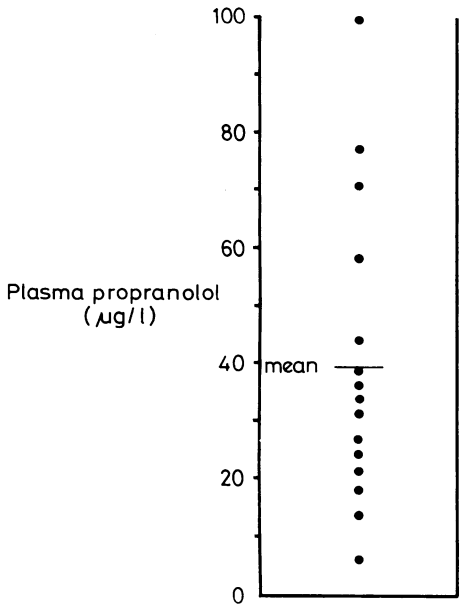
[1] Gräfenberg, E, in *The Practice of Contraception*, ed M Sanger and H M Stone, p 33. London, Baillière, Tindall and Cox, 1931.
[2] Tatum, H J, *et al*, *Journal of the American Medical Association*, 1975, **231**, 711.
[3] Sparks, R A, *et al*, in *The Uterine Cervix in Reproduction*, ed V Insler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

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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

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[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