was 14·4 mg./100 ml., and serum phosphorus 4·5 mg./100 ml. The serum alkaline phosphatase was 20 King-Armstrong units, and the 24-hour calcium excretion in the urine was 400 mg. Clinically he was markedly hyperthyroid, and propranolol 80 mg. q.i.d. was administered in an effort to ameliorate his symptoms. This was followed by a drop in the serum calcium to 10·6 mg./100 ml. by the fifth week and subsidence of the symptoms of hypercalcaemia with a return to a euthyroid state. An attempt to reproduce the hypercalcaemic state by stopping propranolol for two weeks failed, though the clinical features of thyrotoxicosis became overt. Financial considerations made the patient unwilling to remain in hospital, and he was given $^{131}I$ 4 mCi and discharged on propranolol 40 mg. q.i.d.

An Indian female, aged 58 years, was first admitted on 9 March to King Edward Hospital with a seven-year history of dyspnoea, palpitations, and loss of weight. Clinically she was found to have diffuse thyroid enlargement, exophthalmos, lid lag, fine tremor of the hands, proximal myopathy, hot sweaty palms, and a pulse rate of 120/min. Her P.B.I. 10·6 μg./100 ml., 24-hour $^{131}I$ uptake 86%, and latex particulate triiodothyronine $^{131}I$ 48%. No serum calcium study was done, but an E.C.G. excluded evidence of any calcium disturbance. She was treated with propranolol 40 mg. q.i.d. and $^{131}I$ 4 mCi was administered on 22 April. She was subsequently discharged, but readmitted on 1 May in congestive cardiac failure. An E.C.G. showed prolonged $QT_c$ interval (0·45 sec.), and the serum calcium was 6·6 mg./100 ml., serum phosphorus 4·4 mg./100 ml., and serum potassium 4·4 mEq/l. Propranolol was stopped on 23 May and two days later she died suddenly. Necropsy failed to reveal any definite cause of death.

Though propranolol has been shown to improve many of the clinical features of hyperthyroidism[1-4] it does not alter thyroid function per se, as evidenced by its lack of effect on P.B.I. and $^{131}I$ uptake by the thyroid gland.[4] Furthermore, propranolol produces no change in other metabolic derangements in hyperthyroidism—for example, the raised free fatty acid abnormal glucose tolerance or human growth hormone response.[4] Hence it is surprising to find reduction in serum calcium in patients treated with propranolol. Thus it seems worthwhile investigating the effects of propranolol on hypercalcaemia in hyperthyroidism noting that hypocalcaemia may be a dangerous side effect.—We are, etc.,

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## Peritoneal Dialysis in Pulmonary Oedema

SIR,—In reply to the letter of Dr. M. I. M. Noble (25 July, p. 224) on the above subject, we should like to make the following comments.

The four patients who underwent peritoneal dialysis were selected from 850 with proved myocardial infarction who passed through our coronary monitoring unit, so that the problem of resistant pulmonary oedema is a rare one. All four patients were given salt-restricted diets, and we agree that it may be valuable to observe serial weight changes during the patients' stay in coronary monitoring units. However, in a nine-bedded unit of a regional hospital it was not thought to be an economic proposition to have weigh-beds and, of course, such patients as we have described are usually too ill to get out of bed for routine weighing.

Our experience of large intravenous doses of frusemide for pulmonary oedema or pulmonary congestion after myocardial infarction is not impressive. Doses of 300-600 mg. have been administered to such patients with no greater diuretic response than that achieved with 100-200 mg., so as a consequence of this we now no longer tend to use such high dosage. It should also be appreciated that the patients who were dialyzed were critically ill, and it was thought that no more time should be wasted using routine diuretic methods, since the removal of fluid by peritoneal dialysis can be very rapid. Furthermore, this procedure allows some measure of correction of any metabolic disturbance.

All patients with pulmonary oedema after myocardial infarction are nursed well propped up in bed with the head of the bed raised and the foot of the bed lowered 6 in. (18 cm.)

Finally, peritoneal dialysis in capable hands, using rapid one-litre cycling, is not usually upsetting to the patient, and to date we have not experienced any worrying complications.—We are, etc.,

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## Anticoagulants and Cardiac Infarction

SIR,—There is ever-increasing literature on the subject of anticoagulants and cardiac infarction, with strongly varying views on its value even from clinicians with considerable experience and equal ability. Medical students are taught that, unless there is some definite contraindication in myocardial infarction, anticoagulation is part of the routine treatment for coronary thrombosis, especially in young men.

While early work on this subject appeared to make this a reasonable conclusion, the most comprehensive survey by the Medical Research Council, published in the *British Medical Journal* (8 February, 1969, p. 335) has shown that earlier hopes of the value of these drugs have not been justified, and on the over-all mortality they have made little or no difference. For some extraordinary reason this important paper seems to have been ignored and most students and newly qualified doctors still think this therapy is of the utmost importance, and almost regard people who do not give it as withholding essential therapy. In the paper mentioned it did seem to bring out, as have other papers, that the liability to thromboembolic phenomena is slightly reduced, and this of course is important, but such phenomena are by no means more comon in the young, and in my own experience seem to be related to the size of the infarct. If this is so, where a big infarct is suspected there would seem to be a greater indication for anticoagulant therapy, otherwise the value of these drugs seems in such grave doubt that it is extraordinary to me that they are still being regarded as a routine therapy.

I would be most interested to know if any of the team who took part in this extensive trial have any views on which patients are the most likely to get thromboembolic phenomena, or is it still impossible to assess this?

As a physician working at two regional hospitals dealing with very large numbers of these patients, I have been increasingly unimpressed with the value of this therapy but would not wish to withhold it entirely unless its lack of value is conclusive.—I am, etc.,

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## Entonox in the Ambulance Service

SIR,—Following the report on the use of Entonox in the Ambulance Service by Drs. P. Baskett and A. Withnell (4 April, p. 41), we have analysed the 91 cases that have been treated in the first six months of the use of Entonox in the Brighton Ambulance Service. The analysis has been made under the same headings as those used by Drs. Baskett and Withnell, and are as follows, their figures being given in brackets:

| | | |
|---|---|---|
| Limb Injuries | 41 | (27) |
| Burns | 2 | ( 3) |
| Chest Injuries | 6 | ( 4) |
| Back Injuries | 5 | ( 2) |
| Cuts and Scrapes | 0 | ( 2) |
| Acute Abdomen | 13 | (13) |
| Myocardial infarction (Heart Cases) | 14 | ( 3) |
| Pneumonia and Pleurisy | 1 | ( 2) |
| Meningitis | 0 | ( 1) |
| Terminal new growth of stomach | 1 | ( 1) |
| Obstetric | 8 | ( 6) |

The distribution of cases is very similar, except for a much larger number of cases in the Brighton series when Entonox was used for myocardial infarction.

No follow up has been attempted on the lines of the Gloucestershire experiment, but the ambulance men were asked to record on a form, completed immediately after the incident, upon the degree of pain relief. In 75 cases relief was stated to be satisfactory, in 15 it was partial, and in one case only was no benefit gained from use of Entonox.

The experience in Brighton so far indicated that this is a method of treatment that is welcomed by the patients and ambulance men alike, and may be regarded as a most valuable addition to an ambulance service.—We are, etc.,

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## Scleroderma and Primary Biliary Cirrhosis

SIR,—In view of our own observations we were interested in Professor A. E. Read's comments on the association between systemic sclerosis and primary biliary cirrhosis (1 August, p. 278) and also the two case reports by Dr. I. M. Murray-Lyon and colleagues (p. 258).