or antagonists depending on the test condition.⁷ The 8-hydroxyquinolines may be acting as imitators of prostaglandin action in acrodermatitis.

It is possible that a prostaglandin deficiency may be involved in acne. Diphenylhydantoin is a drug whose long-term use is frequently associated with acne-like eruptions. Because we have shown that membrane-stabilising drugs can have prostaglandin antagonist actions⁷ and because diphenylhydantoin is known to be a membrane stabiliser we have recently tested it and shown it to be a competitive prostaglandin antagonist. We suggest that zinc is effective in acne because it stimulates prostaglandin synthesis and that this is also the basis of its action in acrodermatitis.

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¹ Brocks, A, Reid, H, and Glazer, G, *British Medical Journal*, 1977, **1**, 1390.

² *British Medical Journal*, 1977, **1**, 1493.

³ Neldner, K H, and Hambridge, K M, *New England Journal of Medicine*, 1975, **292**, 879.

⁴ Michaelsson, G, Juhlin, L, and Vahlquist, A, *Archives of Dermatology*, 1977, **96**, 283.

⁵ Michaelsson, G, Vahlquist, A, and Juhlin, L, *British Journal of Dermatology*, 1977, **96**, 283.

⁶ Evans, G W, and Johnson, P, *Lancet*, 1977, **1**, 52.

⁷ Manku, M S, and Horrobin, D F, *Prostaglandins*, 1976, **12**, 789.

Propranolol in thyrotoxic heart failure

SIR,—We welcome the important paper by Dr H Ikram (11 June, p 1505) showing that propranolol may be deleterious in patients with thyrotoxicosis and heart failure for we have recently had two patients who died in whom there was a probability that the administration of propranolol was a factor in their deaths.

Case 1—A coloured woman aged 58 of West Indian origin was admitted from the outpatient department with rapid atrial fibrillation 160/min, settling to 120/min in the ward. There was clinical evidence of tricuspid and mitral incompetence, with severe oedema to the top of the thighs and widespread expiratory wheezes in the chest but no basal crepitations. The blood pressure was 130/80 mm Hg. There were no abnormal eye signs and the thyroid was not palpable. She was known to have had atrial fibrillation and mitral incompetence, presumed due to ischaemic heart disease, for some years and there was also a history of chronic bronchitis being treated with salbutamol. Thyrotoxicosis had recently been diagnosed, serum thyroxine being 224 nmol/l (17.4 µg/100 ml) and triiodothyronine (T₃) 4.1 nmol/l (0.27 µg/100 ml). At 10 pm she was given digoxin 0.25 mg, propranolol 40 mg, salbutamol 4 mg, and nitrazepam 5 mg orally. Three and a half hours later she became urgently dyspnoeic and distressed owing apparently to severe asthma, which was treated with intravenous salbutamol and aminophylline with some improvement in her clinical state. After another three and a half hours—that is, seven hours after propranolol had been given orally—she had a sudden cardiac arrest, from which she was immediately resuscitated, but she ultimately died after a further five hours of aggressive treatment. After the cardiac arrest there was ECG evidence of myocardial infarction and the serum hydroxybutyrate dehydrogenase was 337 IU/l after the episode of cardiac arrest.

Case 2—A 21-year-old English girl was admitted as an emergency with a 12-h history of increasing drowsiness preceded by two weeks' history of shortness of breath and palpitations. On examination she was confused and disorientated, temperature 39°C, pulse 180/min. There was exophthalmos and a diffuse goitre was present.

There were focal signs of pneumonia in the right upper chest. A diagnosis of thyrotoxic crisis provoked by right upper lobe pneumonia was made and confirmed by chest x-ray. Serum thyroxine was 300 nmol/l (23.3 µg/100 ml) and T₃ 7.3 nmol/l (>0.48 µg/100 ml). The ECG showed sinus rhythm. At 2 pm on the day of admission she was given propranolol 40 mg orally and an intravenous infusion of dextrose saline was started. At 5 pm she was given intravenous ampicillin and 10 min later she collapsed with severe wheezing, hypotension, and skin mottling. Anaphylactic shock was diagnosed and she responded to intravenous hydrocortisone and antihistamine. However, she still had extreme agitation and tachycardia so 2 mg of propranolol was given intravenously. Immediately after this she had an exactly similar collapse and she subsequently required artificial ventilation. Carbimazole, digoxin, gentamicin, and potassium iodide were then given. The thyrotoxicosis came under control but neurological activity did not return and she had an asystolic arrest after 14 days on the ventilator.

It was unfortunate that in case 1 the patient was given propranolol in view of the history of chronic bronchitis, and, although it was over three hours before she had the acute episode of asthma, we feel that this was probably provoked by the administration of the drug. It is not possible to be sure of the exact cause of the subsequent sequence of events and unfortunately necropsy was refused, but in the light of Dr Ikram's paper it is quite possible that a fall in cardiac output following this single oral dose of propranolol was a factor that led to myocardial infarction and cardiac arrest and her ultimate death. In case 2 there was no previous history of allergy to penicillin or anything else and in view of the immediate collapse following intravenous propranolol we think that this drug was the more likely cause of both episodes and that the initial diagnosis of penicillin anaphylaxis was incorrect. The hypotension and the hypoxia at the time of the collapse were contributory to her death. Although these two patients were quite different in their clinical presentation it did seem that propranolol was an important factor in their deaths and we shall be interested to hear whether anyone else has similar cases to report.

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Mumps orchitis and testicular tumours

SIR,—We would like to comment on your leading article on mumps (11 June, p 1489), in which you suggest that there may be "at best a tenuous relationship" between mumps orchitis and malignant testicular tumours and urge an inquiry "in large numbers of patients."

We have investigated the incidence of testicular tumours in the population of Hamburg. During the period 1966-76 294 patients with such tumours were collected (3.0 cases per 100 000 males) in Hamburg and 200 further cases from other parts of Germany (total 494). Only two cases of unilateral mumps orchitis were found in which a testicular tumour had later developed: a man 76 years old with seminoma (orchitis in the same testicle at 22 years) and a patient 45 years old with the identical diagnosis (orchitis in the diseased testicle at 21 years). It is interesting that 24 patients with testicular tumours (4.9%) had suffered from epididymitis on the affected side some years ago (median 8.6 years). We could not find any patient with a

history of epididymitis in the non-affected testicle. Since it is well known that epididymitis can very occasionally be the only symptom of mumps¹ this seems worth recording. Sixty-one patients (12.3%) had simultaneous atrophy of the diseased testicle; in addition, 28 patients (5.7%) had a palpable atrophy of the contralateral testicle. This fact seems to be of interest since Beard *et al*² also found atrophic testes in their patients with previous mumps orchitis and testicular tumour.

From our data on a larger series of patients with testicular neoplasms we conclude that mumps orchitis itself is seldom found in the past history of these patients. On the other hand, if one speculates that the previous epididymitis in the testicle in which a tumour subsequently developed in 4.9% of our patients was in fact a manifestation of mumps the situation is different.

We have also checked 87 cases of mumps orchitis treated between 1960 and 1970 in Hamburg hospitals. On screening the records of the urology and x-ray departments of the city none of these patients was found to have developed a testicular tumour up to 1977.

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¹ Christie, A B, *Infectious Diseases: Epidemiology and Clinical Practice*, p 469. Edinburgh and London, Churchill Livingstone, 1974.

² Beard, C M, *et al*, *Mayo Clinic Proceedings*, 1977, **52**, 3.

Getting close to the patient

SIR,—Has any aspect of the reorganisation of the Health Service resulted in any benefit to anyone except those who appear to enjoy endless committee work? Were they not typical of the ineptitude of the system that has been created, the following excerpts from one of those circulars from the local area health authority would have been treated as a joke. Instructing hospital staff on the management of violent patients it says, after some initial platitudes about peaceful persuasion:

"If this fails [that is, if he refuses to co-operate] stay close to the patient. . . . If he has abandoned the weapon or instrument but appears likely to attack you or anyone else in the vicinity or do injury to himself, approach him, still talking to him, and when close enough grasp his arms at elbow level and pull him towards you. Quickly change your grasp and encircle him with your arms but continue to pull him towards you. . . . Then quickly get behind the patient, push him towards a nearby wall, retain your grip, lean on him, press him to the wall or floor and remove his footwear. . . . Make sure the relevant incident forms are filled in as soon as possible."

What can we do to alter the system?

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Diagnostic amniocentesis in early pregnancy

SIR,—You state in your leading article (4 June, p 1430) that "anti-Rh(D) immunoglobulin should be given [following amniocentesis] to patients with a significant fetomaternal transfusion as indicated by the Kleihauer technique." If this recommendation is followed some untreated mothers will become sensitised with serious risks to their babies.

I have recently cared for a 37-year-old patient in my practice, para 3+0, who had no antibodies at booking or in previous pregnancies. In this, her fourth pregnancy, she had an amniocentesis to exclude chromosomal abnormality, but no anti-D was given as the Kleihauer test was negative. At 32 weeks her anti-D titre was 1/512 and amniocentesis showed the bilirubin level to be in the severely affected category. The baby was delivered by caesarean section at 33 weeks but did well.

It has now become policy in Oxford to give anti-D to all rhesus-negative mothers having amniocenteses and to increase the dose if the Kleihauer test is positive. This policy is also recommended by the Department of Health and Social Security.¹

Amniocentesis is performed in early pregnancy to reduce the incidence of abnormal children. To cause an otherwise normal pregnancy to result in a handicapped child as a result of the test would be a tragedy. Anti-Rh(D) immunoglobulin is very effective and safe. It should surely be given in all cases, whether the Kleihauer test is positive or not.

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¹ Department of Health and Social Security, Standing Medical Advisory Committee, *Haemolytic Disease of the Newborn*, revised version, para 4.7. London, DHSS, 1976.

Vitamin B₁₂ for vegans

SIR,—Your expert errs in stating (11 June, p 1525) that "there are no measurable amounts of vitamin B₁₂ in any known plant." Seaweeds, including those growing round our shores, are good sources,^{1,2} about 100 g a day furnishing the daily requirement of the vitamin (as well as useful amounts of vitamins A and B₂ and arachidonic acid). The nodules on the roots of nitrogen-fixing plants are very rich sources of Vitamin B₁₂,³ although they are not usually eaten. Yeast from wine-making contains the vitamin, and a pint of cider may contain nearly 1 µg. Even vitamin D occurs, in water-soluble form, in some plants.

We would be happy to provide doctors and dietitians with our list of commonly available vegetarian and vegan sources of vitamin B₁₂.

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¹ Chapman, V J, *Seaweeds and their Uses*, 2nd edn. London, Methuen, 1970.

² FAO/US Department of Health, Education and Welfare, *Food Composition Tables for Use in East Asia*, 1972.

³ Blondeau, R, *Comptes Rendus Hebdomadaires des Séances de l'Académie des Sciences*, 1971, **272D**, 2781.

Tetracycline and toxoplasmosis

SIR,—Drs P L Grossman and J S Remington (25 June, p 1664) criticise our short report on toxoplasmosis (23 April, p 1064) because we did not give details of the oxytetracycline dosage used and because we ventured to suggest that one of the tetracyclines should be considered for the treatment of severe lymphadenopathic forms of toxoplasmosis.

In our article we were primarily concerned with the epidemiology and natural history of

the disease, but felt that the problems of treatment also deserved some attention. In the interests of brevity we omitted from our case descriptions whatever could be taken for granted. Thus the omission of all drug dosages clearly denoted that standard therapeutic doses were given. In the case of oxytetracycline the dosage was 500 mg four times a day. We prefer oxytetracycline to demethylchlorotetracycline (which was used in the experimental work we referred to) because the former is considerably less expensive and is less likely to cause gastric disturbance and photosensitisation. Nevertheless, possible differences in the activity of the various tetracyclines in human—as opposed to murine—toxoplasmosis should certainly be explored, as should the use of relatively large doses of these antibiotics.

We are not alone in our dissatisfaction with the standard treatment regimen, with or without corticosteroids.¹ We were therefore impressed by the rapid clinical improvement both with oxytetracycline used alone (but evidently for an inadequate length of time) and then when used for a longer period with prednisolone. This was in sharp contrast to the total lack of response in the two severely affected patients to a month's course of pyrimethamine and sulphadiazine, even though these drugs have proved so successful in murine toxoplasmosis.²

We were particularly interested in the probable source of infection in this small outbreak. The only communal meal eaten by the three affected men was also attended by four others who suffered no ill effects. This distribution was surprising but by no means unprecedented in infections due to ingested food. We have, however, subsequently elicited information on the varying degree of exposure to infection in the seven individuals. The unaffected four had already eaten well before the meat course and they ate only a small portion of inadequately grilled lamb on skewers. In contrast, the three men who developed primary toxoplasmosis eight days later had consumed large amounts of the meat and are assumed to have swallowed a relatively high infective dose of *Toxoplasma gondii*.

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¹ Beverley, J K A, *British Medical Journal*, 1973, **2**, 475.

² Eyles, D E, and Coleman, N, *Antibiotics and Chemotherapy*, 1955, **5**, 529.

Sclerosing peritonitis due to practolol

SIR,—Sclerosing peritonitis due to practolol therapy can present diagnostic difficulties and the excellent paper by Dr R P H Thompson and Mr B T Jackson (28 May, p 1393) is to be welcomed for recording the unfortunate fact that many cases are not diagnosed until laparotomy is performed. All six cases described by them had at least two laparotomies and one patient underwent six operations.

Dr Thompson and Mr Jackson do not say whether small-bowel barium studies were performed on their patients, but they do insist that they found radiology to be unhelpful. This has not been our experience at this hospital, where barium studies form part of the investigation of patients with subacute small-bowel obstruction.^{1,2} We have observed distinctive

radiological appearances in all the patients with symptoms due to practolol peritonitis whom we have examined. In two of our patients the disease was first suggested by the barium findings, and a past history of practolol therapy was elicited after the radiological investigation. There were no adverse effects due to barium examination.

In view of the poor results of unsuspecting laparotomy and the impaired cardiac status of many of these patients it is obviously important to make the diagnosis of sclerosing peritonitis before abdominal surgery. Small-bowel barium examination is indicated in patients with clinical evidence of practolol peritonitis to help confirm the diagnosis and to define its extent.

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¹ Lee, R E J, *et al*, *Clinical Radiology*, 1977, **28**, 119.

² Marshall, A J, *et al*, *Quarterly Journal of Medicine*, 1977, **46**, 135.

Solitary perforated diverticulum of ascending colon

SIR,—I would like to report an unusual case of perforated solitary diverticulum of the ascending colon.

A 29-year-old woman was admitted with a two weeks' history of epigastric pain, which later localised to the right iliac fossa. The clinical examination revealed an ill-defined, tender mass in the right lower abdomen with guarding. Rectal and pelvic examinations confirmed the presence of a mass in the right side of the abdomen, arising from the pelvis. She had had appendicectomy 10 years before this admission. The other relevant past history was that she had been having similar attacks of pain, though of less severe nature, for the past five years.

At laparotomy the pelvic viscera were normal. There was a hard, adherent mass in the ascending colon extending to the hepatic flexure upwards and to the pelvic downwards. An ulcer crater was felt in the centre of the mass. In view of the doubtful nature of the mass radical right hemicolectomy with end-to-end anastomosis was performed. Macroscopic and microscopic examinations established the diagnosis of perforated solitary diverticulum of the ascending colon with chronic pericolic abscess.

A review of the literature¹⁻³ revealed that perforated solitary diverticulum of the ascending colon is rare. The correct diagnosis is almost never made preoperatively. Even at laparotomy it is often difficult to recognise this abnormality; often malignancy is suspected, as in the above case. Although this is a rare clinical entity, surgeons should be aware of it so that major colonic resection is not needlessly undertaken.

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¹ Chughtai, S Q, and Ackerman, N B, *American Journal of Surgery*, 1974, **127**, 508.

² Wagner, D E, and Zollinger, R W, *Archives of Surgery*, 1961, **83**, 436.

³ William, K L, *British Journal of Surgery*, 1960, **47**, 351.

"Baby alarm" in hospital care of infants with pertussis

SIR,—With the controversy over pertussis immunisation causing a sharp decline in the numbers of infants immunised we may expect