has (save for operative delivery in cases of known disproportion) distinct advantages: (1) it is easily made; (2) it is associated with relatively little bleeding; and (3) the repair is easy because the parts fall into accurate apposition, which is certainly not the case with a posterolateral episiotomy. Against the central episiotomy is the alleged danger that any extension will involve the anal sphincter or even the anal wall. This is true; but it is also true that, after making the orthodox posterolateral episiotomy, the operator is often dismayed to find that the anal sphincter is badly torn at one side by the passage of the baby. Fortunately, the repair of a damaged sphincter seldom presents difficulty to the experienced operator.

Finally, it is scarcely necessary to add that a clean episiotomy is a much easier wound to repair than is a ragged perineal tear. In my experience the former is also much less likely to be associated with severe perineal pain.—I am, etc.,

J. CHASSAR MOIR

Charlbury, Oxford

1 Baker, S., *A Survey into Post-natal Perineal Discomfort*. London, S. Maw and Sons, with the Royal College of Midwives, 1973.
2 Munro Kerr's *Operative Obstetrics*, 8th edn., by J. C. Moir and P. R. Myerscough, p. 881. London, Baillière, Tindall, 1971.

SIR,—Dr. Robyn L. Pogmore's allegation (5 January, p. 37) that midwives and obstetricians show little interest in the puerperal perineum may be relevant in these days of 48-hour discharge from maternity hospitals. That allegation would have been quite inappropriate in the days of my own training. We were very much concerned, and certain lessons then learned may be worth restating.

Sutures in the perineum, whether called for by tears or by incision, are never comfortable. The pain may originate either at skin level or in the deeper tissues. In each situation the main reason is tissue tension, in minimizing which particular care has to be taken to reduce the number of sutures to that which is quite essential for tissue apposition and for the elimination of dead space. Allowance must be made for oedema occurring in the next few days, and here a generous infiltration of local analgesic will mimic the later tissue reaction and help to prevent unnecessarily tight stitching. I cannot agree with the depreciation of immediate repair with local as distinct from (or in supplement to) regional analgesia.

There must be no confusion between round-bodied needles and cutting needles; skin stitching demands the latter. Much needless discomfort is produced by the stiff ends of skin sutures which become entangled in dressings or prick the adjacent tissue. Chromic catgut at skin level is almost as irritating as the non-absorbable material once thought mandatory. To cut the sutures very short does not overcome this difficulty. It is preferable to leave the ends of the stitches initially very long, up to 10 cm, and then to gather them together and secure them very firmly with a simple knot, after which the protruding ends are snipped off leaving no sharp projections.—I am, etc.,

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Fife.

Oncological Centres

SIR,—Your leading article (5 January, p. 2) relating to the development of oncology centres in Britain expressed some of the fears now felt by many junior hospital doctors who have already committed themselves to cancer medicine as a specialty.

We concur with the opinion voiced in your leader that the Department of Health should now unequivocally declare its intensions with regard to the establishment of these centres. If this is not done it will be felt that the proposals made 12 months ago represented an emotive but empty political gesture.—We are, etc.,

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Psychiatric Safeguards

SIR,—"Registered medical practitioners do not hold a monopoly of the art of healing." So begins your leading article (22 December, p. 689). An unexceptionable statement which is then followed by an attempt to prove the opposite. Surely if, as a free citizen, I choose to consult someone other than a doctor about my health and emotional problems, someone such as a pharmacist, a priest, a chiropodist, a dentist, or even a psychologist, that is my legitimate right. If that professional advises me within his competence and within the law even you, Sir, would allow that all is well; he may even advise me to see a doctor if he thinks his own skills are inappropriate. What right then has the American Psychiatric Association, the Royal College of Psychiatrists, the *B.M.J.*, or any other body to say that they are strongly opposed to trained clinical psychologists practising as independent professionals? Furthermore, what right has a psychiatrist to say that the facilities provided by the clinical psychologist should not be available to other doctors unless he, the psychiatrist, untrained in clinical psychology, says so? Have clinical psychologists at any stage suggested that proper medical consultation for patients is something they are opposed to and would try to prevent? Are trained clinical psychologists likely to be highly incompetent in knowing when to refer to their medical colleagues; and are psychiatrists infallible in knowing when to seek a psychological opinion? Have psychologists ever refused to examine their profession courses to seek to eliminate deficiencies?

Your leading article is headed "Psychiatric Safeguards." Safeguards for whom, may I ask? Perhaps a better title would be "Restrictive Practices." There seems to be little genuine concern for patients in your arguments. Modern psychological assessments and treatments are complex and time-consuming. Demand far exceeds supply. We should welcome professional collaboration at all levels if we are to supply a comprehensive service equal to modern needs. With the complexity and diversity of modern psychology the only way the psychiatrist could genuinely claim to be in the omniscient position you are advocating would be to undertake a further two- or three-year course and become a clinical psychologist as well as a psychiatrist. Would that help him or any of his patients?—I am, etc.,

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SIR,—Certain implications of your leading article (22 December, p. 689) cannot be ignored.

Responsibility for the patient rests squarely on the general practitioner, for the former is "registered" with the latter, never with a consultant. The general practitioner delegates clinical responsibility to his hospital colleague. The legal responsibility of the hospital doctor is shared with the hospital authority, as many court actions have shown.

Of course, psychiatric assessment is essential before referring a patient to a clinical psychologist, but I contest most strongly the principle that a general practitioner should be barred from making such referral. Many G.P.s are quite as expert in psychiatric evaluation as specialists in psychiatry. The clinical and ethical strength of the general practitioner lies in his knowing his limitations; he knows when to refer for advice from a specialist colleague. In the final analysis, it is the general practitioner who, by the terms of his legal contract, carries responsibility for his patient. He should have access to all investigation and treatment facilities.—I am, etc.,

H. CAIRNS

Cardiff.

Cancer and the Patient

SIR,—It was not our intention to add to the correspondence in your columns regarding the attitude of patients to the diagnosis of cancer. Nevertheless, it seems clear to us that a difficult situation is not helped by exaggerated claims for certain forms of treatment and we feel that Dr. E. L. Lloyd's letter (15 December, p. 674) on the use of hyperthermia to treat pain in cancer requires comment. In our opinion it borders on the naive to suggest, as Dr. Lloyd does, that a greater awareness and use of a treatment technique which at best is only temporarily successful in a proportion of cases will alter significantly the image that cancer has among patients. Anyone engaged in the treatment of patients with cancer is well aware that pain is one (but only one) aspect of the disease requiring skilled management, but few would argue that treatment should be the same for each and every patient as Dr. Lloyd appears to do.

Secondly, it is important to emphasize that Henderson and Pettigrew[1] presented no data comparing hyperthermia and other therapy (such as morphine) in the control of pain in their cases. It is therefore difficult to see how Dr. Lloyd can assert that hyperthermia is superior to other forms of treatment. We would argue, for example, that relief of localized pain can frequently be achieved by radiotherapy with virtually no side effects and negligible morbidity. On the other hand, the permanent hoarseness and intense dysphagia we have seen in patients following hyperthermia perhaps explains why some patients refuse to undergo a second course of this treatment.

Lastly, we must point out that when hyperthermia is given together with cytotoxic drugs, as is current practice in Edinburgh, the combined treatment is associated with a definite mortality. It therefore appears to us that further controlled studies and considerably more data are required before hyperthermia can be accepted as having a significant role to play in cancer management.—We are, etc.,

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1 Henderson, M. A., and Pettigrew, R. T. P., *Lancet*, 1971, **1**, 1275.

Glucagon Therapy in Acute Pancreatitis

SIR,—Your leading article (1 December, p. 503) and the subsequent letter from Mr. C. W. Imrie and Professor L. H. Blumgart (5 January, p. 38) reflect a surge of interest in the treatment of acute pancreatitis. Though there are disagreements about many aspects of this condition, there is no dispute that its mortality rate is unacceptably high.

Unfortunately, very few trials of methods of treatment have been designed in ways which can lead to scientifically valid conclusions. As a consequence many misleading claims for different drugs have been made. It is for this reason that the Medical Research Council has set up a working party which is about to start a randomized, controlled clinical trial to compare glucagon, aprotinin (Trasylol), and a placebo in the treatment of acute pancreatitis.

One of the difficulties in assessing the treatment of acute pancreatitis in Britain is the fact that no single centre has the opportunity to treat sufficiently large numbers. Therefore our trial will be conducted on a multicentre basis and many colleagues have already indicated their willingness to participate. We know that when our findings are reported they will be subjected to close scrutiny and that they may be criticized on the grounds of faulty dosage. We therefore wish to stipulate at this stage that the purpose of the trial is to test current claims for glucagon and aprotinin. If these prove to be wrong, it will be possible to test alternative dosages and alternative drugs.—We are, etc.,

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Co-ordinating Secretary

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Amphotericin Pharmacophobia and Renal Toxicity

SIR,—Professor W. St. C. Symmers (24 November, p. 460) has emphasized the need to treat systemic fungal infection with amphotericin B. He also quotes evidence that the drug is nephrotoxic and that this fact has deterred physicians from prescribing it. Renal toxic manifestations of amphotericin B tend to return to normal on cessation of therapy, particularly if the total dosage is less than 5 g.[1] Winn[2] has reported irreversible renal toxicity in patients who received total doses of 14 g, 16·7 g, and 21 g respectively of amphotericin. Reports of irreversible renal toxicity with total doses of less than 5 g are rare. There is evidence[3,4] that a total dose of at least 2 g and preferably 3 g is necessary for cure of systemic fungal disease. Drutz *et al.*[5] have criticized this recommendation and have successfully treated 13 patients with a variety of mycotic diseases using daily serum levels as a guide to therapy, adjusting dosage to achieve twice the minimum inhibitory concentration against the causative organism. Five of Drutz's 13 patients, however, in fact received a total dose of at least 2 g of the drug. The rapid infusion[6] of moderate doses (never more than 45 mg daily) of amphotericin over a prolonged period, to achieve a total dose of 2·5-3 g, is probably the best way to use this drug. Renal toxicity should not give cause for anxiety until the blood urea reaches 100 mg/100 ml and should not lead to premature cessation of treatment before this.—We are, etc.,

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1 Abernathy, R. S., *Medicine*, 1973, **52**, 385.
2 Winn, W. A., *Medical Clinics of North America*, 1962, **47**, 1131.
3 Tolhurst, J. C., Buckle, G., and Williams, S. W., *Chemotherapy with Antibiotics and Allied Drugs*, p. 115. Canberra, Australian Government Publishing Service, 1972.
4 Edwards, V. E., Sutherland, J. M., and Tyrer, J. H., *Journal of Neurology, Neurosurgery, and Psychiatry*, 1970, **33**, 415.
5 Drutz, D. J., Spickard, A., and Koenig, M. G., *Antimicrobial Agents and Chemotherapy*, 1966, **6**. 202.
6 Fields, B. T., Bates, J. H., and Abernathy, R. S., *Applied Microbiology*, 1971, **22**, 615.

Toxoplasmosis and Embryopathy

SIR,—The letter from Drs. Jean M. Scott and M. Layinka Swinburne (17 November, p. 422) and the resolution of the 13th Congress of Medical Women's International Association on Toxoplasmosis have oversimplified the problem as it is known in the U.K. Ruoss and Bourne[1] followed up 3,700 pregnant women throughout their pregnancies. Seven women converted from serologically negative to positive with the dye test; all seven produced normal uninfected babies. The Public Health Laboratory Service performs almost all the tests for toxoplasma antibody in England and Wales and finds about 50 congenital toxoplasmosis cases per year, which is equivalent to 1 in 14,000 pregnancies. Ross *et al.*[2] estimate an incidence of 1:30,000. It is reasonable to suppose, however, that cases may be misdiagnosed and that the incidence is higher. There is good evidence that the incidence in the U.K. is between 1 in 4,000[1] and 1 in 14,000. This is lower than in France and Germany and probably lower than the incidence of rubella and even cytomegalovirus disease. Clearly more work needs to be done to find the incidence more accurately.

If it is decided that this incidence is too high, what can be done about it? The therapy of toxoplasma infection during pregnancy is not as effective as that against syphilis so that the testing of sera during pregnancy, which would be expensive, would be unlikely to be very beneficial in preventing cases. It is also doubtful whether treating congenitally infected newborn babies is very rewarding. Might it not be more useful to protect women of childbearing age by instructions in avoiding the eating of raw meat and contact with the oocysts from infected cat faeces? More information is needed about the route of the oocyst; does it travel from soil, uncooked food, or flies to man?

In the long run protection by infection before pregnancy may be best. Is this to be achieved by vaccines? Such vaccines as have been tried in animals are of very limited value. Rubella is controlled by the use of an effective vaccine, but the immunity mechanism is probably different from that in toxoplasmosis. Perhaps we should persuade the veterinarians to make a vaccine for cats, or simply encourage contact between cats and young girls so that the childbearing population is protected before pregnancy.

I apologize for raising so many unanswered questions and speculations, but I hope this letter illustrates that the time is not yet ripe for a publicity campaign to the public when the medical profession has not yet enough knowledge to give sensible advice.—I am, etc.,

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1 Ruoss, C. F., and Bourne, G. L., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1972, **79**, 1115.
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High-dose Frusemide in Renal Failure

SIR,—We wish to make some comments about the paper of Dr. F. Cantarovich and his colleagues (24 November, p. 449) concerning the beneficial use of high-dose frusemide in established acute renal failure, since our own results appear different from those reported.

We conducted a single-blind randomized study on 66 patients with acute oliguric renal failure between 1971 and 1973. Criteria for retaining the patients in the study were as follows. (1) Presence of established acute renal failure with initial urine output less than 500 ml/day and remaining less than 20 ml/hr after correction of shock and/or hypovolaemia when present; low urinary urea concentration; normal or high sodium concentration and/or urine:plasma osmolality ratio less than 1·1. (2) Absence of obstructive uropathy; absence of glomerulonephritis or systemic disease involving the kidney.

Plasma urea levels were maintained below 200 mg/100 ml, using haemodialysis when necessary. To 33 of the patients a first dose of frusemide (3 mg/kg) was given intravenously and followed every four hours by doses ranging from 1·5 to 6·0 mg/kg, according to the diuretic response. The maximum daily dose was 1,200 mg. If no diuretic response was observed after three injections (diuresis <20 ml/hr) frusemide was temporarily discontinued, but further treatment with the same protocol was attempted every five days until diuresis occurred. The remaining 33 patients did not receive frusemide and served as controls; this group did not differ significantly in respect of aetiology of acute renal failure, sex ratio, initial urine output, or mortality from the treated group. No significant differences in the results of treatment were seen between the two groups (see table).

The differences between the results obtained by Dr. Cantarovich and his colleagues and our own lead us to point out the salient