drugs. A prostaglandin E₁ analogue reverses the deleterious effect of giving corticosteroid, as we previously showed for indomethacin.

Piper *et al* concluded that their study might have missed an effect of corticosteroids alone on peptic ulcer disease as less than 8% of the patients they studied were taking doses of over 30 mg/day. Though we do not yet have data from humans, particularly on the rate of regenerative repair of peptic ulcers during treatment with corticosteroid, we do not believe that a clinically relevant effect of these drugs on human peptic ulcer disease in the absence of concomitant non-steroidal anti-inflammatory treatment has yet been ruled out. On this, as on the potential protective effects of prostaglandins and acid reducing agents, insufficient data are available.

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1 Guslandi M, Tittobello A. Steroid ulcers: a myth revisited. *BMJ* 1992;304:655-6. (14 March.)
2 Piper JM, Ray WA, Daugherty JR, Griffin MR. Corticosteroid use and peptic ulcer disease: role of non-steroidal anti-inflammatory drugs. *Ann Intern Med* 1991;114:735-40.
3 Levi S, Goodlad RA, Lee CY, Stamp G, Walport MJ, Wright NA, *et al*. Non-steroidal anti-inflammatory drugs inhibit the processes of gastric mucosal repair which lead to healing of gastric ulcers. *Lancet* 1990;336:840-3.

Octreotide and urinary glycosaminoglycan in Graves' disease

SIR,—We reported that octreotide is effective in Graves' ophthalmopathy and pretibial myxoedema.¹ P Perros and colleagues suggest that it will be more convincing if a reduction in circulating insulin-like growth factor-1 concentrations could be shown.² Although insulin-like growth factor-1 has been postulated to be related to the pathogenesis of Graves' ophthalmopathy,³ its concentration in serum was not raised in untreated patients (unpublished data). An increased accumulation of glycosaminoglycan in retrobulbar tissue from patients with Graves' ophthalmopathy and in the dermis of patients with pretibial myxoedema has been reported.⁴ Urinary excretion of glycosaminoglycan is increased in these patients and decreased after treatment with prednisolone.⁵ We find that octreotide also decreases urinary excretion of glycosaminoglycan in these patients.

We treated six patients with Graves' ophthalmopathy and pretibial myxoedema with octreotide 50 µg thrice daily for two days followed by 100 µg thrice daily. A 24 hour urine collection was made before and after treatment with octreotide for one week. Urinary glycosaminoglycan excretion was determined by the method of Kahaly *et al*⁶ except that the supernatant reacted with cetylpyridinium chloride and the isolated glycosaminoglycan was centrifuged for 30 minutes at 15 000 g. Urinary creatinine excretion was measured with an automatic multichannel blood chemistry analyser (Hitachi 736, Japan). Excretion of glycosaminoglycan was expressed as µg/mg creatinine. To avoid interassay variation, samples from each patient obtained before and after treatment were determined within the same assay. We also measured urinary excretion of glycosaminoglycan in three other patients with Graves' ophthalmopathy and pretibial myxoedema, 15 patients with Graves' ophthalmopathy, and 14 normal subjects; they all served as controls.

Urinary excretion of glycosaminoglycan was higher in the patients with Graves' ophthalmopathy, with and without pretibial myxoedema, than in the normal controls ($p<0.005$ and $p<0.05$, respectively). In the patients with Graves' oph-

Urinary excretion of glycosaminoglycan before and after one week's treatment with octreotide in patients with Graves' ophthalmopathy and pretibial myxoedema

Case No	Urinary glycosaminoglycan (µg/mg creatinine)	
	Before treatment	After treatment
1	11.3	10.2
2	7.0	5.4
3	32.8	12.1
4	11.8	7.5
5	20.5	13.7
6	60.1	45.2
Median (range)	16.2 (7.0-60.1)	11.2 (5.4-45.2)*
Median (range) in controls:		
Normal subjects (n=14)	6.8 (1.8-12.7)	
Graves' ophthalmopathy (n=15)	9.9 (4.4-26.0)†	
Graves' ophthalmopathy with pretibial myxoedema (n=9)	13.4 (7.0-60.1)‡§	

* $p<0.05$ Compared with value before treatment (Wilcoxon's signed rank test).
† $p<0.05$, ‡ $p<0.005$ Compared with normal subjects; § $p<0.05$ compared with Graves' ophthalmopathy (Mann-Whitney U test).

thalmopathy with pretibial myxoedema, urinary excretion of glycosaminoglycan significantly decreased after a week's treatment with octreotide ($p<0.05$; table).

Lymphocytes may produce lymphokines that stimulate production of glycosaminoglycan,⁶ and octreotide can inhibit responsiveness of T lymphocytes.⁷ Octreotide may therefore directly inhibit the release of lymphokines, resulting in inhibition of the production of glycosaminoglycan and a decrease in urinary excretion of glycosaminoglycan.

We thank Sandoz Pharmaceuticals for supplying the octreotide, Dr R Teoh for his encouragement and help, and Dr K S Tsai, Miss J C Lin, and Miss Sandy J H Huey for technical help. This study was supported by a grant from the National Science Council, Republic of China (NSC-80-0412-B002-176).

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1 Chang TC, Kao SCS, Huang KM. Octreotide and Graves' ophthalmopathy and pretibial myxoedema. *BMJ* 1992;304:158. (18 January.)
2 Perros P, James RA, Kendall-Taylor P. Octreotide and Graves' ophthalmopathy. *BMJ* 1992;304:507. (22 February.)
3 Hansson HA, Petruson B, Skottner A. Somatomedin C in pathogenesis of malignant exophthalmos of endocrine origin. *Lancet* 1986;ii:218-9.
4 O'Brien JF. Glycosaminoglycan and exophthalmos. In: Gorman CA, Waller RR, Dyer JA, eds. *The eye and orbit in thyroid disease*. New York: Raven, 1984:43-7.
5 Kahaly G, Schuler M, Sewell AC, Bernhard G, Beyer J, Krause U. Urinary glycosaminoglycans in Graves' ophthalmopathy. *Clin Endocrinol* 1990;33:35-44.
6 Tsao M, Ferrarini M, Evans C, Ambro A, Whiteside TL. Purification of a glycosaminoglycan-stimulatory lymphokine from supernatants of in vitro-activated human mononuclear cells. *Clin Immunol Immunopathol* 1989;50:122-31.
7 Malec P, Zeman K, Markiewicz K, Tchorzewski H, Nowak Z, Baj Z. Short-term somatostatin infusion affects T lymphocyte responsiveness in humans. *Immunopharmacology* 1989;17:45-9.

Reversing vasectomy

SIR,—I was disappointed that Anjan K Banerjee and Alan Simpson did not mention in their editorial the best pregnancy rates that have been achieved after reversal of vasectomy in the United Kingdom.¹ Jenkins and Blacklock reported a pregnancy rate of 64.5%.² They used a simple technique that required a prolene stent, which ensures that the lumina are accurately apposed; four sutures; and, perhaps most importantly, milking the epididymis of all obvious secretion before anastomosis so as to decrease the risk of

leakage of sperm and subsequent granuloma. This potential complication is arguably the most important for the long term results of this operation.

Banerjee and Simpson's comments questioning the desirability of performing this kind of surgery in a hard pressed NHS are valid. I suggest that adoption of Jenkins and Blacklock's technique would save operating time and obviate the need for expensive microsurgical equipment to be used, especially as there is no evidence that pregnancy rates are improved by increasing the complexity of the procedure.

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1 Banerjee AK, Simpson A. Reversing vasectomy. *BMJ* 1992;304:1130. (2 May.)
2 Jenkins IL, Blacklock NJ. Experience with vasovasostomy: operative technique and results. *Br J Urol* 1979;51:43-5.

Advanced trauma life support

SIR,—In his letter on advanced trauma life support Andrew J Mowat describes the focus of this method of managing trauma as being x ray investigation, diagnostic peritoneal lavage, urinary catheterisation, and rectal examination.¹ As a successful candidate on an advanced trauma life support course he will realise that x ray investigation, diagnostic peritoneal lavage, and rectal examination are part of the secondary survey.² Urinary catheterisation is part of the resuscitation phase. Although resuscitation can be started at the scene, urinary catheterisation is clearly not essential to the patient's survival before admission to hospital.

I came away from an advanced trauma life support course with the focus of the system ringing in my ears—namely, "A, B, C, . . ." This is the primary survey:

- A: Airway maintenance with control of the cervical spine;
- B: Breathing and ventilation;
- C: Circulation with control of haemorrhage;
- D: Disability: neurological status.²

Resuscitation begins while the elements of the primary survey are reassessed. Once the patient's condition is stable, and only then, the secondary survey begins.

Advanced trauma life support is one method of trauma care. If one method was adopted by all doctors caring for patients with trauma, life would be easier for us and perhaps safer for our patients. I would have thought that any immediate care doctor treating a patient at the scene of an accident could follow the "ABCD" above and begin appropriate resuscitation as taught by the advanced trauma life support course. I would certainly be happy to take on the care of such patients in an accident and emergency department and would be grateful to the doctor concerned.

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1 Mowat AJ. Advanced trauma life support courses. *BMJ* 1992;304:1114-5. (25 April.)
2 American College of Surgeons. *Advanced trauma life support course manual*. Chicago: American College of Surgeons, 1989.

SIR,—Andrew J Mowat comments on the need for better immediate care in the United Kingdom.¹ Deaths before admission to hospital are usually due to brain injury, haemorrhage, and airway problems. In addition, 15-35% of patients in whom a head injury is diagnosed before admission have secondary brain injury from hypoxia or hypovolaemia.²

If certain measures (care of the cervical spine, intubation and ventilation, setting up an intravenous line, splinting fractured limbs) are performed at the scene of the accident and the critically injured patient is transferred to the appropriate hospital by the appropriate means within the "golden hour" morbidity and mortality can be reduced. Other interventions (such as fluid resuscitation and applying military antishock trousers) that entail a philosophy of "stabilise then mobilise" are less beneficial than those entailing a philosophy of "scoop and run," especially in urban areas. This may not be true, however, if the transfer to hospital takes longer than 20 minutes.

The Royal College of Surgeons of Edinburgh holds courses for the diploma in immediate care, and there are British textbooks and other courses specifically on this subject. The Royal Army Medical Corps runs a battlefield army trauma life support course based on the principles of advanced trauma life support. It caters for the field management of casualties without the sophistication of hospital care and thus meets the criteria of the immediate care module suggested by Mowat.

It is not only, however, the immediate care of patients with trauma that needs to be addressed but also the continuum of care (from the scene of the accident to rehabilitation) and accident prevention. Successful care is care that returns an injured patient to society quickly.

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- 1 Mowat AJ. Advanced trauma life support courses. *BMJ* 1992; 304:1114-5. (25 April.)
- 2 Trunkey DD. Prehospital care. In: Trunkey DD, Lewis FR, eds. *Current therapy in trauma*. Philadelphia: B C Decker, 1991:3.

Behçet's disease

SIR,—B Wechsler and J C Piette have mysteriously omitted to mention chlorambucil as a treatment in Behçet's disease.¹ This alkylating agent may have a useful role in preventing recurrent episodes of uveitis and encephalopathy in the disease.^{2,3}

Recurrent ocular symptoms may progress to loss of useful vision within 10 years in a considerable proportion of people.⁴ Death and permanent deficit after Behçet's disease affecting the central nervous system have been well documented.^{5,6} As corticosteroids may not ultimately prevent blindness or death,⁷ chlorambucil should be considered in appropriate patients despite its small risk of oncogenicity.

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- 1 Wechsler B, Piette JC. Behçet's disease. *BMJ* 1992;304: 1199-200. (9 May.)
- 2 Mamo JG, Azzam SA. Treatment of Behçet's disease with chlorambucil. *Arch Ophthalmol* 1970;84:446-50.
- 3 O'Duffy JD, Robertson DM, Goldstein NP. Chlorambucil in the treatment of uveitis and meningoencephalitis of Behçet's disease. *Am J Med* 1984;76:75-84.
- 4 Benezra D, Cohen F. Treatment and visual prognosis in Behçet's disease. *Br J Ophthalmol* 1986;70:589-92.
- 5 Wolf SM, Schotland DL, Phillips LL. Involvement of nervous system in Behçet's syndrome. *Arch Neurol* 1965;12:315-25.
- 6 Chajek T, Fainaru M. Behçet's disease; report of 41 cases and a review of the literature. *Medicine* 1975;54:179-96.
- 7 O'Duffy JD. Behçet's syndrome. *N Engl J Med* 1990;322:326-7.

Diagnosing pulmonary embolism

SIR,—We welcome N W Morrell and W A Seed's pragmatic approach to the difficult problem of reliably diagnosing pulmonary emboli.¹ Ventilation-perfusion scans are often requested

and relied on to confirm or exclude pulmonary embolus. Misgivings about the reliability of the test led us to audit the scans obtained in our hospital and assess the consistency of reporting among radiologists.

During 1991, 608 lung scans were obtained, of which we located 567. Altogether 162 (29%) were reported as being "high probability" scans and 330 (58%) as "low probability" scans, which is a much lower proportion than that in the studies quoted by Morrell and Seed.^{2,3} In our study only 15% of the patients would have required a further procedure to determine whether they had pulmonary emboli by Morrell and Seed's criteria.

When five consultant radiologists who regularly report isotope lung scans were presented with 10 scans, clinical information, and chest x ray films—simulating the normal reporting conditions—inconsistent results were produced. In each case the majority opinion was assumed to be correct. On this assumption, the original scan had been misreported in four of the 10 cases in a way that would have altered the diagnosis and subsequent management. The original report had been of low probability in three cases; one scan initially reported as high probability was reported by the consultants as low probability. If these results are representative, up to 40% of reports may be misleading.

A practical strategy for dealing with scans that fall between low and high probability scans is useful, but we need to be sure that the initial categorisation is correct more than 60% of the time.

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- 1 Morrell NW, Seed WA. Diagnosing pulmonary embolism. *BMJ* 1992;304:1126-7. (2 May.)
- 2 Hull RD, Hirsh J, Carter CJ, Raskob GE, Gill GJ, Jay RM, et al. Diagnostic value of ventilation-perfusion lung scanning in patients with suspected pulmonary embolism. *Chest* 1985;88:819-28.
- 3 PIOPED Investigators. Value of the ventilation/perfusion scan in acute pulmonary embolism. *JAMA* 1990;263:2753-9.

Free For All: "Cancer positive"

SIR,—Jeffrey Tobias is entitled to his opinion of our programme, but he should not mislead readers over facts.¹

Firstly, he seems to suggest, incorrectly, that patients treated at the Bristol centre who were included in the study reported in the *Lancet* were not having orthodox treatment.

Secondly, he says that these patients "still seemed to do worse in the long run." This was never stated, even by the researchers: Bristol patients were observed for an average of only six months.

Thirdly, with regard to overall survival, the press release put out by the Imperial Cancer Research Fund and the Cancer Research Campaign in September 1990 quite properly stated, "it is still too soon to compare overall survival." Even if the study was continuing this would still be true: some patients were entered into the study less than five years ago.

Fourthly, the woman who said that some doctors would "throw you out of the room" at the mention of the Bristol centre also said of other doctors: "They'll be, oh, great, especially the younger ones. . . . They're very keen and ask you all about it." Tobias seems anxious to polarise the discussion. We would like the two sides to come together, and we believe that orthodox and complementary medicine can and should gel, as they already do in many NHS hospitals.

Fifthly, it was not Penny Brohn who said that "the charities' publicity machine made sure it was made public" but ourselves, in our commentary.

The Bristol Cancer Help Centre had no hand in making the programme.

Sixthly, the report did not analyse the effect of treatments on offer at Bristol; indeed, the researchers made no use of the questionnaires which would have documented compliance.

Finally, the "sensational story" was not "gobbled up" by the tabloid papers any more than by the quality press. The *BMJ* gave it 39 column inches in September 1990, the *Times* 20, and the *Sun* only three.

It seems to need restating that, in the words of Sir Walter Bodmer himself, "women going to Bristol had more severe disease than control women." He was good enough to apologise on camera for the distress caused, saying: "I am very sorry that a mistake was made." A further important point is that the Bristol women were younger than the controls, and evidence shows that breast tumours tend to grow faster in younger women.²

What scientific conclusions, if any, may be drawn from such a study is, in our view, a matter for an independent inquiry. We agree that a review committee should have pointed out major flaws. The problem was that no review committee did so.

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- 1 Tobias J. Publish and be damned. *BMJ* 1992;304:991. (11 April.)
- 2 Cheung CWD, Johnson AE. Carcinoma of the breast: measurement and the management of treatment. *Br J Radiol* 1991;64: 121-32.

Improving postgraduate training in ophthalmology

SIR,—J H Sheldrick and colleagues sought to obtain information on undergraduate and postgraduate ophthalmic training by analysing diagnostic accord between general practitioners and an ophthalmologist.¹ Their finding of agreement in nearly 60% of cases suggests that training in ophthalmology must be reasonably good. Though they acknowledge that this was a self selected group of general practitioners working under abnormal conditions, I think that they have seriously underestimated the effect that this has on any conclusions that may be drawn from their study.

The quality and nature of referrals from primary care to ophthalmic casualty and outpatient departments are valid reflections of the adequacy of training. The aims of training should be to instruct in the management of simple eye conditions (such as blepharitis) and to provide awareness of more serious problems (usually visual) requiring referral. In a recent audit of 102 referrals to the clinic at St Paul's Eye Hospital I found that in 34% of cases the urgency of the outpatient appointment could not be assessed because inadequate information had been provided. In only 55% were symptoms described, and in only 23% was the duration of symptoms mentioned. It is more important for a referral letter to include the history and results of simple examination (including visual acuity) than for it to include a suggested diagnosis.

Standard referral forms have been advocated.² Issuing general practitioners with such forms, with examples, could be an important step in postgraduate training. Improving the quality and promptness of the hospital's reply to the general practitioner is another potential opportunity for education. Bringing the ophthalmologist into the general practitioner's surgery, as proposed by some fundholders,³ may be the ideal solution but is probably feasible on only a small scale.

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