Orchidectomy versus oestrogen for prostatic cancer: cardiovascular effects

Sir,—With regard to Dr T W Meade’s comments (11 October 1986, p 953), the last 20 patients in the randomised study were evaluated with regard to activators and inhibitors of blood coagulation and fibrinolysis. Before treatment with prostatic carcinoma already had increased concentrations of fibrinogen and factor VIIc compared with healthy controls of the same age. During oestrogen treatment factor VIIc concentration rose significantly (p<0.001) and the antithrombin III concentration decreased significantly (p<0.001). With regard to fibrinolysis, plasminogen concentration increased and that of urokinase inhibitor fell (p<0.001) after treatment with oestrogen was started.

Thus patients with prostatic cancer receiving oestrogen treatment have increased concentrations of factor VII, factor VIIc, and fibrinogen. All these changes are identical with the findings that predicted death in patients with ischaemic heart disease in the prospective Northwick Park heart study. In addition, the patients’ concentrations of antithrombin III decrease and the fibrinolytic capacity seems to increase during oestrogen treatment. Fibrinolysis was also activated in many of the patients who received oestrogen, as judged by increased concentrations of a fibrinogen degradation product, the so-called D dimer, during treatment.

Department of Internal Medicine, Huddinge Hospital, S-141 86 Huddinge, Sweden

H. HEINRIKSSON
M. BLOMBÄCK
G. BRATT
O. EDHAG
A. ERIKSSON

Barrett’s oesophagus

Sir,—Dr R C Heading (21 February, p 461) cites many references that fail to explain the true nature of Barrett’s condition. First of all, is it indeed a separate anomaly as being acquired and refer to such things as short oesophagus, gastic lined lower oesophagus, Barrett metaplasia, and heterotopic gastric cells.

The title Barrett’s oesophagus is a misnomer, and Mr Heading failed to explain how the condition has been referred to in the literature, that it is metaplastic (that is, it is an epithelium that has replaced a different, earlier type) and that operative treatment that suppresses gastro-oesophageal reflux may allow reversion to a normal squamous oesophageal lining. One of the reasons for this is that there is no clear explanation of why some patients with gastro-oesophageal reflux develop Barrett’s oesophagus while others do not. There is perhaps an analogy in the gastric mucosa: it is not clear why some patients with gastritis develop extensive intestinal metaplasia, which does not occur in others. The proposal that Barrett’s oesophagus should be renamed congenital columnar lined lower oesophagus is logical if we are confident that the condition is indeed congenital. I believe, however, that most gastroenterologists do not share Mr Heading’s conviction and that the balance of evidence favours the view that Barrett’s oesophagus is an acquired lesion.

R C HEADING

University of Edinburgh, EH1 9YW

AIDS and intravenous drug use

Sir,—Dr J R Robertson and Carol Skidmore (28 February, p 571) produce a table relating the year of starting heroin addiction to the presence of antibody to the human immunodeficiency virus (HIV). They interpret the high prevalence among those who became infected in 1982-3 as being due to some other factor or factors operating in these years and suggest that “a minor epidemic of heroin use resulted in extreme and damaging behaviour, giving rise to ideal conditions for transmission of virus.”

We would interpret their data differently. In our study relating sexual lifestyle to HIV antibody state in homosexual men we found that those who had practised a gay lifestyle for five years or less had a significantly lower prevalence of HIV antibody than those who had practised for more than five years.1 Our figures showed that the 98 who were HIV seropositive, 91 (93%) had been homosexually active for more than five years, compared with 153 of 206 (74%) of those who were sero-
negative (p<0.001). Interpreted in a similar way, the data presented by Dr Robertson and Ms Skidmore show that 63 of 69 (91%) patients who were seropositive had been addicted for more than three years, compared with 34 of 54 (63%) who were seronegative (p<0.001).

In our paper we also showed that homosexual activity for more than five years was the strongest predictor of seropositivity, and we postulated that an additional factor may be necessary for HIV infection to become established. Such a cofactor may be present in those who have practised a high risk lifestyle for several years, which would account for the apparent failure of HIV infection to spread appreciably outside these groups. Perhaps the most likely candidate for this cofactor is another infection.

R A EVANS
K D MACKRE
West London Hospital, London W10 1DQ

Oral acyclovir in acute herpes zoster

SIR,—Dr M W McKendrick and colleagues make an astonishing statement in their letter (14 March, p 704) when they say "topical idoxuridine is thought to be effective, but we would dispute that this has ever been confirmed adequately." Years ago, my colleagues and I carried out a very elaborate double blind controlled trial. We concluded unequivocally that 40% idoxuridine in dimethyl sulfoxide applied continuously to an affected segment in zoster was highly effective (p<0.0003). The statistics were performed by Sir Richard Doll’s team and our findings have never before been disproved. Dr McKendrick and coauthors are also in their belief that 40% idoxuridine in dimethyl sulfoxide applied continuously on lint cannot be used to treat outpatient. We have treated hundreds of patients thus with very satisfactory results (35% idoxuridine still do just as well).

I am also surprised that Dr McKendrick and colleagues published the findings of their trial of high dose oral acyclovir without any follow up. The only thing that really matters in shingles is timing, one cannot be more specific. They did not refer to their paper to the Oxford double blind controlled trial of high dose intravenous acyclovir. We had expected that acyclovir might be only marginally effective against varicella zoster from in vitro results such as those published by Ellon. Varicella zoster requires 30–50 times the concentration of acyclovir to achieve inhibition similar to that found with most strains of herpes simplex virus. Though there was probably a quantitative difference between the placebo group and the patients who received 10 mg acyclovir/kg intravenously at long term follow up, some patients in both groups suffered from postherpetic neurolgia.

We know from in vitro experiments that vidarabine is several times more effective against many strains of varicella zoster than acyclovir. We treat complicated zoster (zoster of the trigeminal nerve and its branches, zoster of S2 and below, people with motor zoster, and the immunosuppressed) with vidarabine, provided that the patients are not aged over 65. (In older people there may be unpleasant extrapyramidal symptoms, and in such patients we always use acyclovir.) Straightforward, uncomplicated segmental zoster we treat on an outpatient basis with topical 35% idoxuridine applied on lint for four days. I do not believe that giving large doses of acyclovir by mouth is justified until it has been proved that this method of administration is as good as or better than intravenous administration of acyclovir. Unless we have the results of at least six months of follow up we will not know whether the oral treatment advocated by Dr McKendrick and coworkers can be justified.

We still need a really good drug to treat varicella zoster. Brumovinnyloxyuridin is one such drug, but alas it is not commercially available.

B E JUEL-JENSEN
Nuffield Department of Clinical Medicine, John Radcliffe Hospital, Oxford OX3 9DU


When a woman asks for a caesarean section

SIR,—I fear that Drs J G Thornton and R J Lilford (14 March, p 703) have read neither my leading article nor the paper on which it commented with sufficient care. Johnson et al advocated agreeing to perform a caesarean section, at the woman’s request, on dubious medical indications.2 Until I held that the obstetrician should advise caesarean section only on clear medical indications, taking a broad view of maternal and fetal mortality and morbidity risks. Thus I can hardly be suspected of promoting women’s choice as I adopted the more conservative stance.

Furthermore, I find it very strange that Drs Thornton and Lilford should have ignored in the fact that I was a “defence” witness in the Savage inquiry. My views were neither solicited nor expressed in that inquiry, and Drs Thornton and Lilford have had no other opportunity of which I am aware to ascertain my views.

Lastly, the Los Angeles trial of the delivery of the term frank breech should not be dismissed as “too small” as it did show a much greater maternal morbidity with caesarean section than with vaginal delivery, and as this was a prospective randomised control study its conclusions on the fetal outcome of vaginal breech delivery are valuable as most studies have been small retrospective case analyses subject to gross selection bias.

Marion H HALL
Department of Obstetrics and Gynaecology, University of Aberdeen, Aberdeen AB9 2ZD


Do adhesions cause pain?

SIR,—Mr John Alexander-Williams (14 March, p 659), discussing the possibility that adhesions resulting from abdominal surgery might cause pain, says: “The claim that adhesions are a major source of chronic pain in the neurotic patients themselves, who are desperate for an explanation for their symptoms that will protect them from the feared label of ‘neurotic’.”

As one who deals with many letters sent to newspaper and magazine problem pages by distraught patients who have been trying every avenue open to them to find relief for their very real symptoms of distress, I find this sentence very sad. It makes clear to me why so many patients have difficulty in obtaining help with their problems. The fact that symptoms are functional rather than organic does not make them any the less disturbing for the patient experiencing them. If doctors themselves regard only symptoms of organic origin as “interesting” and “real” and dismiss patients who have psychogenic pain as “neurotic” is it any wonder that patients themselves seek so desperately for physical explanations for their disease and fear that they may never feel better?

Perhaps if more doctors were more willing to regard their patients as whole people who have to function as best they can rather than as collections of organs, which may or may not operate smoothly, there would be fewer surgeons digging into abdomens looking for adhesions as sources of pain. If patients were allowed to admit that their pain might be psychogenic and were not made to feel guilty for doing so life would be pleasanter not only for the patients but also for the doctors.

CLaire RAYNER
Middlesex HA1 8BU