severe, even fatal, pelvic actinomycosis can develop in association with usage of the IUCD.^{3,4} Further investigation and follow-up of asymptomatic patients with actinomycosis-positive smears may provide some answers concerning the role of anaerobic bacteria in the pathogenesis of pelvic inflammation. Studies with these aims are being conducted in centres in the United Kingdom and the United States.

M CHARNOCK

Joint Academic Unit of Obstetrics,
Gynaecology, and Reproductive
Physiology,
London Hospital Medical College,
London E1 4DG, and
Medical College of St Bartholomew's
Hospital,
London EC1A 7BE

¹ Gupta, P. K., Hollander, D., and Frost, J. K., *Acta Cytologica* (Baltimore), 1976, **20**, 295.

² Spence, M. R., et al., *American Journal of Obstetrics and Gynaecology*, 1978, **131**, 295.

³ Charnock, M., and Chambers, T. J., *Lancet*, 1979, **1**, 1239.

⁴ Hager, W. D., and Majmudar, B., *American Journal of Obstetrics and Gynaecology*, 1979, **133**, 60.

What is to be done with the XYY fetus?

SIR,—Five years ago the *Lancet*¹ carried a leading article entitled "What becomes of the XYY male?" Its conclusion concurred with that of other critical reviews of the literature²⁻⁴ appearing about that time—namely, we cannot answer that question in the absence of better-designed studies. Regarding the myth of the XYY male as an aggressive inborn criminal, however, it was concluded that the evidence already seemed reassuring. Whatever the reason for the reported excess of XYY males in maximum security special hospitals, the evidence did not support a direct causal explanation. Studies of selected samples of the 99.9% of XYY males who exist outside special hospitals did not produce any convincing evidence of increased susceptibility to aggressiveness, violent criminality, or any other serious behavioural deviancy. In most surveys where XYY males were identified, the only distinguishing feature was their increased height.

This state of knowledge has not been changed by later studies, which are referred to your leading article (9 June, p 1519). Before debating the question of whether or not to "tell the parents" about an XYY fetus, is it not necessary first to establish whether there is anything of significance to tell other than our lack of knowledge? If selective abortion of XYY fetuses is to be considered purely on the basis of a statistical association between presence of a particular genotype and incarceration in a special hospital, then the next question which we must ask is "What is to be done with the XY fetus?" The ratio of male to female convicted "violent" offenders in 1977 in England and Wales was 9:1,⁵ and for those entering penal institutions 26:1.⁶ However, even at the height of the eugenics movement it is improbable that society would have tolerated selective abortion of males on the grounds of gender differences in crime statistics.

The implications of a doctor counselling expectant parents about termination of pregnancy when the fetus is XYY on the basis of the "facts" presented in your article are far reaching in that (a) the evidence relating to these facts is at best equivocal; (b) the expectant parents are unlikely to have any personal experience or other sources of sound knowledge about the XYY male on which to

base their decision about termination; and (c) the problem, which it is suggested might be prevented through selective abortion, is not a medical problem but a social problem.

Your article left unanswered the question "what if the parents then refuse to agree to an abortion?" yet there are firmer grounds for speculating about the behavioural prognosis of this fetus than of one who escapes prenatal or postnatal detection of an XYY genotype. In contrast to the lack of evidence for directly attributing any type of behavioural deviancy to the XYY genotype, the effect of adult expectations on a child's behaviour has been experimentally demonstrated⁷—and it does not cease after the age of 4 years, as might be inferred from your leading article.

W FARRANT

Institute for Social Studies in
Medical Care,
London NW3 2SB

MAJ HULTEN

Regional Cytogenetics Laboratory,
East Birmingham Hospital,
Birmingham B9 5ST

¹ *Lancet*, 1974, **2**, 1297.

² Hook, E. B., *Science*, 1973, **179**, 139.

³ Borgaonkar, D. S., and Shah, S. A., in *Progress in Medical Genetics*, vol. X, ed A. G. Steinberg and A. G. Bearn, p. 135. New York, Grune and Stratton, 1974.

⁴ Beckwith, J., and King, J., *New Scientist*, 1974, **64**, 474.

⁵ *Social Trends* 9, p. 204. London, HMSO, 1979.

⁶ *Social Trends* 9, p. 205. London, HMSO, 1979.

⁷ Rosenthal, R., and Jacobson, L., *Pygmalion in the Classroom*. New York, Holt, 1968.

SIR,—With regard to your leading article (9 June, p 1519) on the XYY fetus, I would like to comment on the Edinburgh study¹ that you mention. Although this work is currently in press, I believe that the control group used was based on the first child born on a Monday morning. I feel that this control group is totally unrepresentative of the general population and so the high mean IQ of this group of 115.7 is an interesting finding.

I feel it important to note that the first baby born on Monday morning is more likely to have had a full-term, non-induced delivery following few complications during pregnancy. Unfortunately this means that the comparisons that the Edinburgh study made between XYY boys and controls are biased in favour of there being more deviance amongst the XYY group in view of the privileged position of the control group, which is not representative of the general population.

SIMON WILKINSON

Royal Edinburgh Hospital Young
People's Unit,
Edinburgh EH10 5HF

¹ Ratcliffe, S. G., Axworthy, D., and Ginsborg, A., in *Birth Defects Original Article Series*, in press.

Pneumoperitoneum associated with artificial ventilation

SIR,—Dr Bruce Summers (9 June, p 1528) attributed the pneumoperitoneum in three of his four patients to leakage through minute ruptures in alveoli subjected to the stress of artificial ventilation.

We reported a similar combination of complications including pneumoperitoneum in two children.¹ We came to the conclusion that the important event was damage to the mucous membrane at the insertion of the endotracheal tube, so that air was pumped into the interstitial tissue by the ventilation.

We agree that this complication is likely to

be more common than reports suggest. It deserves to be more widely known so that unnecessary deaths can be prevented by discontinuing ventilation through the endotracheal tube and using alternative means.

J K SCOTT

Chest Clinic,
Pinderfields General Hospital,
Wakefield, West Yorks WF1 4DG

¹ Scott, J. K., and Viner, J., *Postgraduate Medical Journal*, 1975, **51**, 654.

"Diuretic escape" and "rebound oedema"

SIR,—The short report by Dr M K Chan and others (16 June, p 1604) leads me to believe that they do not fully understand the principles of diuretic treatment in oedematous states. It also introduces two terms, "diuretic escape" and "rebound oedema," which can only lead to confusion and would best be forgotten.

It is axiomatic that the urinary sodium excretion of a patient treated with frusemide does not exceed the sodium intake indefinitely. If it did, death from sodium depletion would be inevitable. In fact a new steady state is reached at which sodium excretion equates with intake, due to the homeostatic mechanisms which the authors mention. Often this new steady state is reached when oedema has resolved, and the diuretic treatment is considered successful. The figure in Dr Chan's report illustrates this well. During continuous frusemide treatment the urine sodium excretion returned convincingly to baseline levels on day 42, and it then oscillated around this value. The authors term this "diuretic escape," and I suppose one could say that "renal escape" had occurred by analogy with the phenomenon observed during mineralocorticoid treatment, although I doubt whether this would serve a useful purpose. However, the term "diuretic escape" is distinctly misleading, because the patient has not "escaped" from the therapeutic effect of frusemide. Approximate calculation from the figure (assuming constant sodium intake) reveals that the patient had lost at least 1600 mmol sodium. There is nothing in the report to suggest that he would not have maintained this new steady state indefinitely had frusemide been continued, and the figure in fact depicts an eminently satisfactory diuretic response.

If those factors which led to oedema formation have not been modified it is entirely predictable that sodium retention will follow discontinuation of frusemide, and that oedema fluid will reaccumulate. There seems no need to call this inevitable event "rebound oedema." The patient has certainly become "dependent" on the diuretic in the sense that he will be oedematous without it, but he was equally dependent on frusemide before he was given the drug! There is no evidence that "oedema was perpetuated" by the diuretic. The factors which led to oedema formation had not been modified, and it would be just as logical to incriminate frusemide in oedematous patients who have never taken the drug.

I agree that long-term diuretic treatment has hazards and that over-treatment should be avoided. It is sensible to attempt gradual reduction of potent diuretic treatment from time to time, but if the factors promoting oedema have not altered the attempt will fail. If oedema is severe enough to warrant symptomatic treatment frusemide is a very

effective drug, and there is no evidence that patients will escape from its therapeutic effect, or that oedema will be perpetuated, during prolonged treatment.

LAWRENCE E RAMSAY

Department of Therapeutics,
Hallamshire Hospital,
Sheffield

*. *We sent a copy of this letter to the authors, whose reply is printed below.—Ed, *BMJ*.

SIR,—We agree entirely with Dr Ramsay's "axiom." However, it is obvious that Dr Ramsay has failed to grasp the points we wished to make in our short report (16 June, p 1604).

It was not our intention to detail the principles of diuretic therapy. We merely pointed out the peculiar behaviour of renal allograft recipients in respect of diuretics, a phenomenon which appears to be more complicated than Dr Ramsay has appreciated. We have shown not only that urinary sodium excretion did not exceed intake indefinitely but that it actually returned to about baseline levels in about two weeks. The dose of the diuretic had to be increased to achieve the same saluresis in the presence of unchanged sodium intake, suggesting "escape" from the therapeutic effect of the drug.

Oedema often develops acutely during rejection episodes in renal allograft recipients. Since causative factors are usually removed if the rejection responds to immunosuppressive treatment, it is imperative to try to wean the patient off the diuretic after the rejection crisis as the maximal therapeutic effect is achieved in a short time. Patients who are inappropriately left on the diuretic after the rejection crisis will become dependent on the drug, since homeostatic mechanisms of salt retention are stimulated by the inappropriate use of the diuretic. Sudden withdrawal of the diuretic even when all causative factors have resolved (as was the case in our report) frequently produces severe yet transient "rebound" oedema.

We therefore wish to emphasise that frequent reassessment of individual requirements for diuretics is needed, and that one should not be dissuaded from weaning patients off potent diuretics after treatment of the cause of the oedema, since the "rebound" fluid retention is transient.

O N FERNANDO
J F MOORHEAD
M K CHAN
P SWENY
Z VARGHESE

Department of Nephrology and
Transplantation,
Royal Free Hospital,
London NW3 2QG

Prostaglandins and impotence

SIR,—The answer to the question about impotence as a recognised side effect of indomethacin suppositories (16 June, p 1608) needs further elaboration.

According to Karim and Hillier,¹ the spontaneous activity of strips of corpus cavernosum muscle from human penis in vitro is abolished by inhibitors of prostaglandin synthesis (aspirin and indomethacin); this implies a role of endogenous prostaglandins in the activity of this tissue. They also have pharmacological effects on the muscle of human penis in vitro, producing relaxation or stimulation depending on the compound used. It has been speculated that a com-

bination of smooth muscle stimulation and vasodilatation brought about by prostaglandins may be involved in the physiology of erection and ejaculation.

Prostaglandins, being among the most potent biologically active substances, play a major role in the reproductive process. Assessing the in-vivo effects of indomethacin and other inhibitors of prostaglandin synthesis on the basis of information obtainable from subfertility or rheumatology clinics will provide us with better answers.

P M SREENIVASA RAO

Lister Hospital,
Stevenage, Herts

¹ Karim, S M M, and Hillier, K, in *Prostaglandins and Reproduction*, ed S M M Karim, p 23. Lancaster, MTP Press, 1975.

Mental handicap and the BBC

SIR,—I was most interested to read the correspondence between my colleague Dr Bernard Laurance and Mr Ian Trethowan (30 June, p 1785), since, unknown to Dr Laurance, I have had similar correspondence.

The things that disturb me most about the forthcoming play are the very rare cause of handicap in the child (anaesthetic negligence) being portrayed and that they were looking for a handicapped child to exhibit in the play.

Like Dr Laurance I received a very bland and quite unsatisfactory letter from Mr Trethowan and in it is the fact that the disquiet of paediatricians about this programme is not going to influence the BBC, who clearly intend that it should go on.

R B JONES

Queen Elizabeth Hospital for
Children,
London E2 8PS

Drugs for epilepsy

SIR,—We applaud the advice of Dr P S Mehta (9 June, p 1565) to follow the accurate diagnosis of epilepsy by the use of a suitable single anticonvulsant drug in therapeutic dosage. However, the young epileptic described in our article (14 April, p 1000) had been considered resistant to single-drug therapy several years before he presented to our community clinic in clinical pharmacology, by which time he was receiving three different anticonvulsant preparations.

There is scant evidence to suggest that anticonvulsants given in combination have effects which are additive. Our initial objective therefore was to adjust the dosage of one drug (phenytoin) to an optimal level and then, once seizures had been controlled, to reduce the complexity of the regimen. Regrettably, he died before this strategy could be followed through.

We accept the point made by both Dr Mehta and Dr D P Addy (5 May, p 1217) regarding the merits of carbamazepine. It was not our intention to advocate the wider use of phenytoin, but rather to demonstrate how the dosage of this drug may best be adjusted.

J C MUCKLOW

University Department of
Pharmacological Sciences,
Newcastle upon Tyne NE1 7RU

D F SMITH

Whickham Health Centre,
Newcastle upon Tyne

Drug-induced oesophageal injury

SIR,—Mr F J Collins and others (23 June, p 1673) clearly demonstrate the ability of emepromium bromide to cause oesophageal ulceration, but do not remark on the long latent period before symptoms occur. I myself have seen three cases of dysphagia caused by this drug, which I would like to describe briefly.

Case 1—A 9-year-old girl was admitted to the Bristol Royal Hospital for Sick Children with a 48-hour history of painful dysphagia for both solids and liquids. She has been taking emepromium bromide for three months for enuresis. Barium swallow showed no abnormality and all symptoms disappeared over three days with conservative treatment with intravenous fluids and mucaine. Emepromium bromide was discontinued.

Case 2—A 24-year-old woman presented at the accident and emergency department of Bristol Royal Infirmary complaining of retrosternal discomfort on swallowing. She had been taking emepromium bromide for 10 days for frequency following a urinary tract infection. It is of note that she was "of no fixed abode." The dysphagia had resolved completely two days after stopping emepromium bromide therapy.

Case 3—A 79-year-old man was given emepromium bromide for urgency following a prostatectomy. After two weeks he developed dysphagia. This resolved on stopping the treatment with no adverse effect on his frequency.

The common factor in these three cases was that they were no longer taking an adequate quantity of fluid with their tablet. In cases 1 and 3 this was because of a desire to avoid a fluid load last thing at night and in case 2 because of the patient's general life style.

Patients who take emepromium bromide should be warned to take a good volume of fluid when swallowing the tablets and a reminder is appropriate when represcribing the drug. Neglecting this precaution while continuing to take the drug is probably the explanation of the latent period that occurs between starting therapy and dysphagia occurring.

ROBIN HUGHES

Department of Surgery,
Bristol Royal Infirmary,
Bristol BS2 8HW

SIR,—With reference to the paper on drug-induced oesophageal injury by Mr F J Collins and others (23 June, p 1673), we wish to bring to the attention of your readers two more cases of oesophageal ulceration due to emepromium bromide (Cetiprin).

Case 1—A 26-year-old dental nurse was prescribed emepromium bromide (200 mg three times daily) for urinary frequency. She had taken the drug uneventfully for five days when she developed severe midretrosteral pain shortly after swallowing two tablets with a little water. She took no more tablets but continued to have painful dysphagia. Seven days later she was referred to this hospital, where fibreoptic oesophagoscopy showed a large, superficial ulcer in the mid-oesophagus. With the help of symptomatic treatment she made a full recovery from the symptoms and repeat oesophagoscopy two months later confirmed complete healing without scarring.

Case 2—A 30-year-old housewife had been taking emepromium bromide for 10 days when she suddenly developed retrosternal pain and dysphagia a few minutes after swallowing two of the tablets with a little water. For the next eight days she continued to take the tablets in the prescribed dose of 200 mg three times daily but could swallow them with only small amounts of water because of painful dysphagia. Fibreoptic oesophagoscopy nine