

subsequently decreasing the dose according to the clinical response to achieve a maintenance level sufficient to suppress the signs of disease activity (usually 5-15 mg daily). If the response in the first 3-4 weeks is satisfactory we often then begin alternate-day rather than daily treatment. If there is no marked improvement in clinical signs or in the level of serum creatine kinase activity after two months of treatment with high doses of prednisone, then we add azathioprine 2.5-3 mg/kg per day in divided doses, adjusting the dose to lower the total lymphocyte count to about $1.0 \times 10^9/l$ ($1000/mm^3$). In a few cases it is necessary to maintain high doses of both drugs for up to six months before significant improvement is obtained, though most patients respond much more rapidly. After two years we attempt to tail off the treatment very gradually indeed, following the clinical state and the serum creatine kinase activity. If after any reduction there is evidence of recrudescence of disease activity higher dose levels are reinstated; slow withdrawal is again attempted every year until the disease has burned itself out.

W G BRADLEY
JOHN N WALTON

Regional Neurological Centre,
Newcastle General Hospital,
Newcastle upon Tyne

- ¹ De Vere, R, and Bradley, W G, *Brain*, 1975, **98**, 637.
² Benson, M D, and Aldo, M A, *Archives of Internal Medicine*, 1973, **132**, 547.
³ Metzger, A L, et al, *Annals of Internal Medicine*, 1974, **81**, 182.

Routine preoperative chest radiography

SIR,—Mr A M Rees and his colleagues (29 May, p 1333) make a strong case for re-examining the usefulness of preoperative chest radiography. It is worth bearing in mind, however, that, of all the *post*-operative chest complications, embolism is probably the commonest and can be very difficult to diagnose. The clinical signs may be equivocal and the lung scan ambiguous. A recent preoperative radiograph of the chest is then of great help in assessing minimal and often subtle *postoperative* changes, enabling a firm diagnosis to be made and anticoagulant treatment started. This applies to young as well as to older patients.

GORDON EVISON

Bath, Avon

Treatment of metabolic alkalosis

SIR,—Dr S E Williams (15 May, p 1189) describes a treatment for metabolic alkalosis that involves cannulation of a central vein and infusion of dilute hydrochloric acid. There is a simpler and no less physiological method that is illustrated by the following case.

A 55-year-old man was admitted in oliguric hypovolaemic renal failure (plasma creatinine 640 mol/l (7.25 mg/100 ml)) and with a metabolic alkalosis (Cl 60 mmol/l; HC03 54 mmol/l). He was confused and agitated, with frequent involuntary jerking of his limbs. Vigorous fluid replacement was undertaken, plasma and normal saline with potassium supplements being used. Despite restoration of blood volume and correction of other electrolyte levels the serum chloride remained low (65 mmol/l at 24 h). The neuromuscular irritability persisted and he continued to be disorientated. A rectal infusion of 2 l of 5% calcium chloride was given over six hours. During this time he became

rational, the twitching subsided, and the serum chloride rose to 90 mmol/l. Subsequent investigation showed a carcinomatous obstruction and a bypass operation was done.

Continuous rectal infusion (proctoclysis) was introduced by J B Murphy in 1908, but its use has declined with the development of intravenous techniques. It is particularly suitable for treating hypochlorhaemia because differential absorption of chloride ion occurs across the colonic wall, with little retention of fixed base.¹ The infusion is easily set up and supervised by nursing staff, is not hazardous, a sepsis is superfluous, and isotonic fluids are not required. What more could one ask for?

I am grateful to Dr A M Davison, St James's Hospital, Leeds, for permission to report this case.

TIMOTHY CHAMBERS

Derbyshire Children's Hospital,
Derby

- ¹ Parsons, F M, Powell, F J N, and Pyrah, L N, *Lancet*, 1952, **2**, 599.

Prostaglandins in depression

SIR,—The report by Drs Georgia Nikitopoulou and J L Crammer (29 May, p 1311) that changes in body temperature occur during the depressive phase of manic depression, if confirmed, is of great interest. There is a distinct possibility that changes in cerebral prostaglandin (PG) levels may explain changes in temperature regulation and in neuronal noradrenaline and serotonin levels in depression.

PGs of the E series increase temperature by an effect on the hypothalamus.^{1,2} Thus a change in temperature regulation may indicate a change in hypothalamic PGE₂ content or altered hypothalamic response to PGE₂. PGE₂,³ and possibly PGF_{2 α} are involved in the control of noradrenaline efflux from sympathetic nerves, and PGE₂ has recently been shown to inhibit noradrenaline and serotonin release from central neurones in the rat.³ Some time ago I suggested that prostaglandins might play a part in premenstrual depression.⁶ There is some evidence that prolactin may be involved in premenstrual depression⁷ and that some effects of prolactin may be mediated by PGF_{2 α} .⁸ May I urge those with research facilities to investigate the role of prostaglandins in depression?

G M CRAIG

London SW16

- ¹ Vane, J R, *Nature New Biology*, 1971, **231**, 232.
² Stitt, J T, *Journal of Physiology*, 1973, **232**, 163.
³ Hedqvist, P, *Acta Physiologica Scandinavica*, 1970, suppl 345.
⁴ Brody, M J, and Kadowitz, P J, *Federation Proceedings*, 1974, **33**, 48.
⁵ Masek, K, and Kadlec, O, in *Advances in Prostaglandin and Thromboxane Research*, ed B Samuelsson and R Paoletti. New York, Raven Press, 1976.
⁶ Craig, G M, *Postgraduate Medical Journal*, 1975, **51**, 74.
⁷ Horrobin, D F, et al, *Postgraduate Medical Journal*, 1976, **52**, suppl 2, p 80.
⁸ Rillema, J A, *Nature*, 1975, **253**, 466.

Erectile impotence

SIR,—Your leading article on this subject (29 May, p 1298) overlooks a very important aspect of the problem. While it must be accepted that the entrenched and consolidated case of erectile dysfunction is often resistant to treatment, every case of impotence passes through a mild and usually readily reversible

stage. Unfortunately this stage is rarely presented to the general practitioner unless he consciously searches for it. The majority of doctors are disinclined to uncover a condition which they feel incompetent to treat. The nub of the problem is therefore inadequate medical education in the field of sexual dysfunction.

My involvement in this field is at several levels. Firstly, as a general practitioner I am aware of the need to be sensitised to the hint, however gentle or disguised, of sexual anxiety. I know from long experience that the chance of successful treatment at this time is very high. One needs to take a little time and trouble in sorting out the ramifications of the disability and not to rush in with a prescription for behavioural tasks. It is as easy to prescribe so-called Masters and Johnson's therapy as it is to write a prescription for drugs. Both can be very effective at the right time. Both can be harmful if carried out before listening, before understanding, before interpreting.

My second hat is worn at my weekly psychosexual problems clinic at the Newcastle General Hospital. Here I am being consulted by patients referred by general practitioners, gynaecologists, family planning doctors, physicians, etc. My success rate is lower in this setting and yet I know this is not because the patients are suffering from a more intractable form of dysfunction. It is only too obvious that many of them are images of the patients relatively easily treated in general practice but now at a more advanced stage of the condition.

And so my third hat is an educational one, both at an undergraduate level (where the new curriculum at Newcastle will contain significantly more teaching in the area of sexual dysfunction and at postgraduate level) where a group of about 40 doctors is involved in a two-year course on managing psychosexual problems. In both cases the emphasis is on early detection and comprehensive diagnosis.

ROLAND FREEDMAN

Newcastle upon Tyne

Cervical smears

SIR,—The article by Mr G Brindle and others (15 May, p 1196) on the selection of women for uterine cervical smears is timely.

In this region, out of 41 863 women examined for the first time under 25 years of age, only 3 per 1000 were found to have carcinoma-in-situ and there were only two cases of microinvasion. Occasional clinical cases have occurred in this age group, but the incidence is very low compared with that of the older age groups. The majority of the cases detected were in women attending ante- or post-natal clinics. The detection rate at antenatal clinics was three times higher than at postnatal clinics. The high-risk women default from postnatal clinics.

The number of smears obtained from this under-25 age group increased threefold between 1966 and 1974, largely because of smears from family planning clinics. With the co-operation of the FP doctors smears are now taken only before a woman starts to take the contraceptive pill or has an intrauterine device fitted. Repeat smears are taken according to the normal routine.

To concentrate screening on the 40-year age group, where the yield is highest, would be ideal, but it is difficult to reach all of this group for the first time. The fact that this age group