rising incidence of invasive cancer in younger women?

Unscreened women need cervical smears from the age of 25, earlier if possible. Unscreened women under 35 need smears so that their preinvasive cancer is treated and cured with minimal morbidity. Unscreened women over 35 need smears so that their invasive cancer is treated, which by that age means an extensive hysterectomy or radiotherapy. Some will survive. Some may die sooner or later and become statistics.

The reason why deaths from cancer of the cervix show little decrease is a half-hearted screening programme, perpetuated by the DHSS, that boosts the figures for total screening (with 61% repeat smears) but does little to diagnose preinvasive cancer in unscreened women under 35.¹

SYLVIA W DAVIES

St Catherine's Hospital,
Merseyside L42 0LQ

¹ Davies SW, Kelly RM. *Br Med J* 1971;iv:525-6.

Work load produced by breast-cancer screening

SIR,—The recent article by Mr W D George and others (6 September, p 653) provides an excellent guideline for anyone who intends to set up a comprehensive breast-cancer screening programme. Their conclusions are derived from the analysis of the referral and biopsy rates of the women who actually accepted the invitation for screening. But it should be remembered that a screening campaign has a significant impact on non-responders and those who are outside the stipulated age limit. A proportion of these too become alerted about longstanding breast symptoms or fear about breast cancer and seek medical advice from general practitioners. This inevitably leads to increased hospital referrals and biopsies. The overall increase in the hospital work load due to a screening programme can therefore be calculated only by monitoring the increase caused by the screened population as well as the non-responders and women outside the age limit; in other words the entire female population.

J PHILIP

St Luke's Hospital,
Huddersfield HD4 5RH

Bladder cancer as a prescribed industrial disease

SIR,—Professor R A M Case (30 August, p 621) fears that my statement (17 May, p 1229) about the withdrawal from industrial use by the mid-'60s of the carcinogens responsible for urothelial tumours will tempt clinicians to ignore a patient's more recent working history when considering whether to advise that a PD 39 claim should be made. I should be surprised and disappointed if that were to happen. My letter was designed to help the claiming process, not to frustrate it. The benefit is available to anyone who is found to be suffering from the disease and who meets the prescribed conditions. Provided the individual satisfies these criteria the question whether or not any of the substances specified in the prescription are still in general industrial use is irrelevant.

The Industrial Injuries Advisory Council,

an independent body which advises the Government on the working of the industrial injuries benefits scheme, is currently reviewing the schedule of prescribed diseases. I am sure that their secretary (room 216, Friars House, 157-168 Blackfriars Road, London SE1 8EU, telephone 01-703 6380 ext 4111) would be pleased to receive from Dr Case or anyone else any evidence suggesting that the benefit conditions for PD 39 should be amended.

F J DARBY

Department of Health and Social Security,
London SE1 8EU

Site of action of intrathecal morphine

SIR,—Mr J Selwyn Crawford (6 September, p 680) suggests that intrathecal and extradural morphine produce their effects by diffusion in the cerebrospinal fluid up to the brain and points to such effects as nausea and respiratory depression as powerful evidence that this occurs. He also describes the lack of effect of a hyperbaric solution of morphine, which he says "would have produced a block to T10 had we injected a hyperbaric solution of local anaesthetic."

This is not evidence that morphine does not act on the cord, however. Local anaesthetics can act on nerve roots as they cross the subarachnoid space, but intrathecal morphine is said to act on cells in the substantia gelatinosa in the cord itself.¹ Since the spinal cord is much shorter than the vertebral column the highest segment of the cord at the level of T10 vertebra is the second sacral.² This anatomical point is not taken account of in many publications and may account for some failures of intrathecal or epidural morphine.³ Success would depend on cephalad diffusion at least to the level of the appropriate cord segments if not to the brain itself.

P J KNELL

North Manchester General Hospital,
Manchester M8 6RB

¹ Snyder SH. *Sci Am* 1977;235:44.

² Warwick R, Williams PL, eds. *Gray's Anatomy*. 35 ed.

Philadelphia: Saunders, 1973.

³ Magora F, et al. *Br J Anaesth* 1980;52:247-52.

SIR,—Mr J Selwyn Crawford's letter (6 September, p 680) on the site of action of intrathecal morphine contains a number of statements which I feel are unfounded. He states: "In almost every account the assumption is made that the drug has acted by virtue of a direct effect on cells in the spinal cord. Repetition appears to be establishing credence for this assumption in the opinion of many. Yet there appears to me to be no sound basis for this belief."

On the contrary elegant studies have been performed demonstrating specific opiate receptors in the spinal cord.¹ Furthermore, experiments in animals showed that the injection of small doses of narcotic analgesics into the spinal subarachnoid space produced a potent analgesia that could be antagonised by naloxone.² The narcotics acting only at spinal level changed cord function to block not only spinal reflexes but also the operant response to painful stimuli. This prompted Wang and co-workers to examine the analgesic effects of intrathecal morphine in human subjects with promising results suggestive of a spinal action.³

Subsequently a number of clinical reports have been published on intrathecal and extra-

dural opiates with mostly encouraging results. A few less comforting reports have of late been forthcoming, and at present doubt exists whether spinal opiates work. What is required now are carefully constructed and controlled clinical studies.⁴ It is apparent, however, that the basis for the use of intrathecal opiates is sound and rests on very much more than an assumption. It is not repetition that gives credence to the proposition of the spinal action of opiates but rather sound and enlightened research work backed up by an appropriate clinical response.

L JACOBSON

The Royal Victoria Infirmary,
Newcastle upon Tyne NE1 4LP

¹ Pert CB, Kuhar MJ, Snyder SH. *Proc Natl Acad Sci USA* 1976;73:3729-33.

² Yaksh TL, Rudy TA. *Science* 1976;192:1357-8.

³ Wang JK, Nauss LA, Thomas JE. *Anesthesiology* 1979;50:149-51.

⁴ Anon. *Lancet* 1980;ii:962-3.

Pleural aspiration and biopsy

SIR,—In emphasising the lack of myelo-suppression produced by intrapleural bleomycin as an advantage over intrapleural thiotepa in malignant pleural effusions, thus perhaps justifying the considerable extra cost of the former, Dr M J Ostrowski's evidence (6 September, p 681) seems incomplete. Although the theoretical risk of marrow suppression from systemically absorbed thiotepa is appreciated, I wonder how often this proves clinically important at the intrapleural doses and frequency used, and what observations may have been made concerning this when separated from the effects of simultaneously administered systemic cytotoxic chemotherapy?

I look forward to the results of the multi-centre study which he and his colleagues hope to complete in the near future, especially if it is randomised to include a group receiving intrapleural thiotepa, a group receiving perhaps mepacrine (which has also had its supporters), as well as a bleomycin group and a control group. I hope too that it is performed double blind with the prime emphasis on comparing adverse effects which actually lead to symptoms (as well as the length of interval between aspirations) in this context of incurable disease.

Finally, I did not suggest, as was stated, that one should not use bleomycin because it is four times as expensive as thiotepa, but merely that the cost (£93.20 for a 120 mg instillation) is borne in mind and weighed against any real advantages.

W T BERRILL

West Cumberland Hospital,
Cumbria CA28 8JG

Taking blood and putting up a drip in young children

SIR,—I read with interest the article "Taking blood and putting up a drip in young children" by Dr Andrew Whitelaw and Dr Bernard Valman (30 August, p 602). For anaesthetists involved in both operating-theatre and intensive-care work the problems of venepuncture and the siting of a reliable drip in a neonate or a premature baby are often compounded by previous multiple venepunctures and insertions of drips for intravenous therapy. This results in one being faced with a neonate with no visible peripheral veins. A technique in this hospital that has been employed with

great success in these difficult cases is to transilluminate the tissues with a powerful cold-light source such as the long flexible fiberoptic light used for cystoscopy, bronchoscopy, etc, which is usually readily available from theatres or intensive therapy units. By placing the light source in the palm of the patient's hand vessels on the dorsum can be clearly seen, where none have been previously visible, by inspecting the skin surface. A similar technique can be used for the foot. The wrist can also be transilluminated allowing easy access to the radial artery for cannulation and removing arterial blood to measure blood gases.

C G PETERS

Anaesthetic Department,
Sheffield Children's Hospital,
Sheffield S10 2TH

Intravenous urography

SIR,—Drs Benvon Cramer and Gerald de Lacey (6 September, p 661) discuss special circumstances in which the standard procedure for intravenous urography should be altered. One important modification that they do not stress is the value of taking delayed films up to 24 hours after the administration of contrast medium, if there is delay to the excretion of contrast by one, or both kidneys. This may give useful anatomical information as to the level of obstruction and may indicate the cause of that obstruction. It is particularly helpful in the management of renal and ureteric colic, and in cases of renal failure thought to be due to ureteric obstruction.

The delay in excretion gives a crude estimate of the degree of obstruction, but I would agree this is more accurately and better assessed by radioisotope methods. Intravenous urography, however, is readily available 24 hours a day in most hospitals and continues to be the first-line investigation for patients presenting as emergencies with obstruction to the upper urinary tract.

MARK HARRISON

Bradford Royal Infirmary,
West Yorkshire BD9 6RJ

Dietary fibre and calcium excretion in diabetes

SIR,—We read with interest the report by Mr P J R Shah and others (9 August, p 426) on the beneficial effect of bran in hypercalciuria. Our experience of the use of guar gum in maturity-onset diabetes supports their findings and suggests that a similar effect on calcium excretion may be achieved with various components of dietary fibre.

We studied the long-term effects of dietary supplementation with guar gum (21 g a day) taken in three divided doses at the main meals, in a double-blind cross-over comparison with placebo in nine insulin-independent diabetics. The main interest was in the effects on glucose and lipid metabolism, and the results will be published elsewhere. The serum and urinary calcium levels were monitored during the experiment, and it was found that after three months of guar gum supplementation the urinary excretion of calcium was significantly less (2.64 ± 0.67 mmol/24 h (106 ± 27 mg/24 h), mean $\pm$ SE) than during the control period (3.80 ± 0.74 mmol/24 h (152 ± 30 mg/24 h), $p < 0.025$). No difference was observed in mean serum calcium concentration between

the two periods. There was a concomitant reduction in mean glucosuria by about 50% during the guar gum period.

The observed reduction in calcium excretion is partially, but probably not entirely, explained by the reduced degree of glucosuria, because a positive correlation has been found between glucosuria and calcium excretion in diabetes.¹ The rather marked reduction in calcium excretion, however, which was evident in eight out of the nine diabetics studied, probably also reflects a reduction in calcium absorption due to the administration of guar gum. The possible mechanism by which guar gum could reduce calcium absorption is obscure, since in contrast to the phytate- and uronic acid-containing components of fibre, guar gum does not bind calcium *in vitro*.²

The study should be repeated and the results confirmed in non-diabetics before any general conclusions on the effects of guar gum on calcium excretion can be drawn. Our findings—although inconclusive—are in support, however of the results of Shah and co-workers and, in addition, suggest that different analogues of dietary fibre may be of practical importance in absorptive hypercalciuria.

ANTTI ARO
MATTI UUSITUPA
TIMO KORHONEN
ONNI SIITONEN

Department of Medicine,
University of Kuopio,
SF-70210 Kuopio 21, Finland

¹ McNair P, Madsbad S, Christensen MS, et al. *Acta Endocrinol (Copenh)* 1979;90:463-72.

² James WPT, Branch WJ, Southgate DAT. *Lancet* 1978;i:638-9.

Managing extrapyramidal disorders

SIR,—The issue of 6 September, (p 655 and p 690) contains references to the management of two extrapyramidal disorders, but the advice proffered in each case requires clarification.

Minerva quotes* a study from Boston¹ in which drug holidays were employed in patients with Parkinson's disease in an attempt to restore therapeutic benefit. While the success of this manoeuvre is open to question, the effect of stopping levodopa treatment can be quite dramatic. Next day the patient will have reverted to his usual Parkinsonian state, and a few days later he may deteriorate to become more immobile than ever before. Such patients may become mute, bedridden, and unable to swallow. Clearly this procedure is not to be undertaken lightly and is best carried out under supervision in hospital.

In response to a question concerning tests that might predict whether a child of a patient with Huntington's disease will develop the illness, your anonymous expert states that: "in descendants of families who have not yet developed the choreiform movements the use of levodopa at about 3 g a day will produce chorea in those who are at the moment asymptomatic." Certainly this is true in about a third of those at risk, but it is not yet known for certain whether such individuals will inevitably go on to develop Huntington's disease. In any case, the wisdom of undertaking such a predictive test is very questionable. There is no guarantee that the disease may not be precipitated earlier by doing so, and the psychological impact of provoking chorea in such an individual at risk can be disastrous; it is like passing a death sentence, a

burden many cannot carry. Until effective treatment for this disease is available, I, and many others concerned in the management of this disease, would proscribe the routine use of predictive tests. Elsewhere² I have argued that in the present state of therapeutic impotence it is reasonable to ask those at risk who are lucky enough not to carry the gene for the disease to share the burden of uncertainty with those who have inherited it, so as to provide the latter with some hope.

C D MARSDEN

University Department of Neurology,
Institute of Psychiatry,
London SE5 8AF

¹ Direnfeld LK, Feldman RG, Alexander MP, Kelly-Hayes M. *Neurology* 1980;30:785-8.

² Barette J, Marsden CD. *Psychol Med* 1979;9:327-36.

*.*We regret the page number quoted by Minerva was wrong.—Ed, *BMJ*.

Acupuncture and postherpetic neuralgia

SIR,—I was interested to read of the experience of Dr G T Lewith and Dr Jennifer Field (30 August, p 622) in the use of acupuncture for postherpetic neuralgia.

I use acupuncture in the pain relief clinic at Ipswich but have not found it very helpful in postherpetic neuralgia. It does seem logical to try to prevent this distressing complication, and there is some evidence that sympathetic nerve block given as early as possible in the acute attack of herpes zoster may diminish the incidence of neuralgia.¹ I have begun to use sympathetic or somatic nerve block in the acute phase, and I find that the pain is decreased and the attack seems to be reduced in length. In the few cases treated none have progressed to postherpetic neuralgia, but further experience is needed to evaluate this approach.

CLIVE JOLLY

The Ipswich Hospital,
Suffolk IP1 3PY

¹ Colding A. *Der Anaesthesist* 1967;16:172-4.

Percutaneous central venous cannulation

SIR,—The hazards of subclavian cannulation have again been rightly emphasised by Mr J L Peters and others (30 August, p 618). The suggested use of ultrasound to identify the subclavian artery and vein may be worthy of trial although we doubt if it will, in practice, reduce the incidence of pneumothorax. This is the major hazard of subclavian puncture particularly in inexperienced hands. A simpler alternative may be puncture of the internal jugular vein at the level of the cricoid. If the Vygon silicone catheter with detachable hub is used it is a simple matter to make a skin tunnel from the site of puncture down to the chest wall below the clavicle using the introducing cannula. This was originally described by Dr Powell-Tuck for the subclavian vein.¹ Puncture of the internal jugular vein is virtually free of the risk of pneumothorax and the use of the skin tunnel should prevent the problem of infection in patients' necklines. The major hazard is puncture of the carotid artery, but we suspect that this is less dangerous for the patient than tension pneumothorax occurring in the intensive therapy unit or on the operating table following subclavian puncture. We have now used this technique in a small number of patients—usually those on