much challenged by the food manufacturers—that the loss of fibre is connected with some modern troubles of Western man. Like diverticular disease, which strikes at least one in four of us by the time we reach 70. And then there is appendicitis, cancer of the colon, obesity, piles, and constipation. So these days some of us have to take our fibre as medicine—like this, made by Reckitt and Colman, one of the biggest food and drug companies in Britain. Fybrogel selling well, overcoming some of the deficiencies associated with modern refined diets—that is what it says on the pack here.” The whole of the sequence was shot around an old-style privy with me (the reporter) poking my head out of the door and reading from the instructions on the packets. Yes, it was intended to “take the mickey” out of one of the “nonsenses” of modern society—taking something out of a natural food and replacing it with more expensive medicine.

CHRISTOPHER BRASHER
Producer,
Science and Features Department,
BBC Television

London W14

Treatment of meningitis and encephalitis

SIR,—Drs R J Fallon and J H Lawson (6 December, p 577) state that “intramuscular injection both in children and adults is perfectly adequate” in the treatment of meningococcal meningitis with penicillin. Indeed, they state that they “do not feel that there is any justification for intravenous therapy except on the grounds of causing less discomfort to the patient.”

Consider a typical 10-kg infant with meningococcal meningitis which one wishes to treat with a penicillin. Benzylpenicillin 400 000 U/kg daily will entail a total injection volume of 6-8 ml per day. Alternatively, ampicillin in a dose of 200 mg/kg daily requires a 6-ml injection volume per day (ampicillin 500 mg dissolved in 1.5 ml sterile water, given six-hourly). In addition, the infant may require hydrocortisone plus anti-convulsants.

An intravenous infusion is a wise “safety-line” in any ill child with bacterial meningitis. The administration of large intramuscular injection volumes to small infants requires frequent rotation of injection sites, occasionally produces sterile abscesses, and can cause considerable discomfort to the infant and to his attending nurses.

DENIS GILL

Children's Hospital,
Dublin

Smoking in the elderly

SIR,—Your leading article (13 December, p 607) rightly stresses the difficulties in advising old people in the matter of smoking. The general feeling is one of laissez-faire in those who have survived well beyond the induction phase of bronchial cancer and are coping with their arterial degeneration. Nevertheless, there are three other reasons which I would advance in advising against smoking in older age groups.

There are many instances where the expense of the habit is the reason for de-

privation of other needs, such as food, clothing, and the provision of heat in winter.

Socially, many old people are unattractive to their children and grandchildren but remain unaware that this is due to the poor oral hygiene resulting from smoking. They are sometimes relieved to be told by the family doctor that this is a reason for refusing intimate contact with them and not a general rejection on account of age.

Finally, smokers in general are insufficiently aware of the effects of carbon monoxide contained in the inhalant. The anoxia produced by a small concentration of carboxyhaemoglobin can be equated to that produced by a diminution in barometric pressure. Susceptibility is increased in the presence of anaemia and when the lean body mass is reduced, as in old age. It is a habit which encourages several of the symptoms commonly attributed to age itself, such as weakness, syncope, and cerebral symptoms when increased activity is called for. I have encountered toxic amblyopia, spasticity, and incontinence of a reversible type when heavy smokers have been persuaded to stop smoking, and dysrhythmias on the ECG returning to normal.

Thus abandoning the habit is, as you point out, unlikely to increase longevity very much. It will, however, nearly always improve performance during the years of retirement and, presumably, reduce dependency.

M KEITH THOMPSON

Croydon, Surrey

Free erythrocyte protoporphyrin

SIR,—We write with reference to the preliminary communication by Dr G M London and others (8 November, p 324) regarding free erythrocyte protoporphyrin levels and nerve conduction velocity in end-stage renal disease.

In the last paragraph of this paper there appears to be an inherent contradiction to which we wish to draw attention. While stating that “the free erythrocytic protoporphyrin level mainly reflects iron status” and that “iron deficiency . . . is common in dialysed patients,” the authors then go on to assert that there was an “absence of any real evidence of iron deficiency.” Their logic is difficult to follow and we believe that their discussion is confusing.

We have found comparable increases in erythrocyte protoporphyrin concentrations (that is, twice normal) in patients on maintenance haemodialysis, but the concentrations in our patients decreased to normal during intermittent iron therapy given parenterally over a period of nine months.¹ We would argue from this that an increase in erythrocyte protoporphyrin in patients on dialysis does indicate the presence of an iron deficiency component in their anaemia. The reason the erythrocyte protoporphyrin is not 10-20 times normal, as in non-renal iron deficiency anaemia, is possibly the lack in renal disease of an accompanying marrow stimulation commensurate with the degree of the anaemia.

However, we would not argue from this that there is any causal relationship between iron deficiency and changes in nerve conduction velocity. The biochemical picture in renal failure is a complex multiplicity of

increases and decreases in many disparate electrolytes and metabolites. In such circumstances it is not valid to argue either a direct relationship or a dependence on a common factor in any two without at least demonstrating that one varies specifically with the other under a common stimulus. A correlation of 0.5, as reported by Dr London and his colleagues, is not, after all, very convincing on its own.

Having ruled out iron deficiency in their patients (mistakenly, we believe) all the authors appear to be saying is that “the toxicity of uraemia” affects both protoporphyrin metabolism and nerve conduction velocity, which adds little to what is already known. To postulate defects in haem-synthetase activity and to suggest that this might be an underlying “common factor” hardly seems justified from their results.

W K STEWART
LAURA W FLEMING

Department of Medicine,
Ninewells Hospital,
Dundee

¹ Fleming, L W, Stewart, W K, and McConnell, J B, in abstracts of Proceedings of Scottish Society of Experimental Medicine, *Scottish Medical Journal*. In press.

Role of pelvimetry in active management of labour

SIR,—I thought the article by Mr D N Joyce and others (29 November, p 505) to be well conceived and showed well, to their surprise but not to mine, a clear correlation for both cephalic and breech presentation between obstetric conjugate and the size of the fetus that would deliver vaginally. What I could not understand, however, was their deduction from these findings that “antenatal pelvimetry is rarely justified except in breech presentation,” although they accept that the radiation risk to the fetus is only 1 in 30 000.

It is indeed true that the active management of labour has eliminated many of the problems associated with the prolonged labour that frequently occurs in the primigravida when the head is still above the brim at the onset of labour, and the better monitoring of labour and pain relief for the mother has made it possible to make correct decisions without the assistance of pelvimetry. This does not mean, however, because augmented labour delivers the baby in a reasonable time, that the pelvis cannot be contracted, and I think that cephalopelvic disproportion is one of the common causes of incoordinated uterine action. I would agree that such a labour should be augmented, but the conduct of that labour will be much safer for the fetus if we know accurately what difficulties it will have to negotiate. If the sacrum is flat and the outlet, as well as the inlet, small, too prolonged an augmentation of labour may result in a fetal head jammed in a pelvis that may make for difficulty in delivery upwards as well as downwards. In such cases we will be well advised not to indulge in fetal brinkmanship and delay caesarean section until there is evidence of fetal distress. If, however, the only narrowing is at the brim, with ample space below or no narrowing at all, we shall augment the labour with a lighter heart.

It is suggested by Mr Joyce and his

colleagues that routine pelvimetry should be performed only postpartum or after a caesarean section, difficult forceps delivery, or perinatal death. Is it not possible that if we had known about that nasty pelvis before we would not have traumatised or killed the baby delivering it vaginally but have made the correct decision to deliver it abdominally? Surely for a risk of only 1 in 30 000 it is well worth while having this extra piece of information with which to program our personal computer.

A L DEACON

Birmingham Maternity Hospital,
Birmingham

Oral dihydroergotamine in management of cluster headache

SIR,—Symonds¹ made a significant advance in the management of cluster headache when he introduced the prophylactic injection of ergotamine as mentioned in your leading article (22 November, p 425). However, we would advocate a trial of oral dihydroergotamine before resorting to parenteral injection.

We recently recorded² the case of a 55-year-old woman who had suffered intermittently from classical migraine since the age of 16. Her symptoms always began with a flickering light over the left visual field. This persisted for about an hour and overlapped with a right-sided throbbing headache. The headache lasted usually for a few hours but had persisted for up to 24 hours. A Cafegot suppository (ergotamine tartrate 2 mg, caffeine 100 mg, belladonna alkaloids 0.25 mg, isobutylallyl barbituric acid 100 mg) would usually relieve her headache within about two hours. She occasionally noticed at the height of her headache that the right eye would start to water and that her right nostril felt blocked. After 27 years of these very intermittent symptoms a cyclical pattern began to emerge. Her headaches would occur almost daily for spells of up to two months, with periods of freedom for up to a year. It proved possible to control her symptoms without recourse to injections. She has been well maintained on dihydroergotamine 1 mg twice daily.

We would agree with you that the distinctive clinical features of the condition "still often escape recognition." We suspect that this is especially true in women, in whom the time relationships are often less precise, the cluster of headaches may last longer, and the whole symptomatology may be less clearly defined than in men.³ It is nevertheless an eminently treatable condition. As you say, "in few conditions are patients so grateful for relief of their symptoms."

L HERZBERG
J A R LENMANUniversity Department of Medicine,
Ninewells Hospital,
Dundee

¹ Symonds, C P, *Brain*, 1956, **79**, 217.

² Herzberg, L, et al, *Journal of Neurology, Neurosurgery and Psychiatry*, 1975, **38**, 648.

³ Bickerstaff, E R, in *Handbook of Clinical Neurology*, ed P J Vinken, and G W Bruyn, vol 5, p 111. Amsterdam, North-Holland Publishing Co, 1968.

Serum creatine phosphokinase and malignant hyperpyrexia

SIR,—We must take issue with Dr M A Deborough (15 November, p 408) in his continued assumption that serum creatine phosphokinase (CPK) levels are an indica-

tive parameter in identifying patients susceptible to malignant hyperpyrexia.

Abnormal CPK levels have been found in a wide range of variegated disorders, including acute psychosis and epilepsy,¹ tetanus,² myocardial infarction and cardiac disease,³ prolonged coma or cerebrovascular disease,⁴ and muscular dystrophy and musculoskeletal disorders,⁵ and with muscular exercise.^{6,7} The administration of suxamethonium causes a marked and significant rise in serum CPK, which is potentiated by halothane in normal patients.^{8,9}

We have been studying the family¹⁰ of a man who died of malignant hyperpyrexia and whose daughter developed malignant hyperpyrexia on exposure to nitrous oxide,¹¹ although she had a normal serum CPK level. Little attention has been focused on the problems of normal serum CPK levels in susceptible families and it seems unwise to place too much emphasis on the measurement of this enzyme as a prognostic guide. Muscle biopsy and in-vitro testing remains the surest form of screening.

Until a simple diagnostic test is available unexpected cases will occur during anaesthesia and we must rely on the routine monitoring of temperature during anaesthesia and the availability of a standard treatment pack.

D W RYAN

Department of Anaesthetics,
Royal Victoria Infirmary,
Newcastle upon Tyne

T N APPELYARD

Department of Anaesthetics,
Hallamshire Hospital,
Sheffield

¹ Meltzer, H Y, et al, *Archives of General Psychiatry*, 1970, **22**, 398.

² Brody, I A, and Hatcher, M A, *Archives of Neurology*, 1967, **16**, 89.

³ Dreyfus, J C, et al, *Revue Française d'Études Cliniques et Biologiques*, 1966, **5**, 386.

⁴ Wright, N, et al, *British Medical Journal*, 1971, **3**, 347.

⁵ Walton, J N, *Disorders of Voluntary Muscle*. London: Churchill, 1964.

⁶ Vejajiva, A, and Teasdale, G M, *British Medical Journal*, 1965, **1**, 1653.

⁷ Rose, L I, *Journal of Applied Physiology*, 1970, **29**, 355.

⁸ Innes, R K R, and Strømme, J H, *British Journal of Anaesthesia*, 1973, **45**, 185.

⁹ Tammisto, T, and Airaksinen, M, *British Journal of Anaesthesia*, 1966, **38**, 510.

¹⁰ Ryan, D W, and Appleyard, T N, *British Journal of Anaesthesia*, 1975, **47**, 1001.

¹¹ Ellis, G R, et al, *British Medical Journal*, 1974, **4**, 270.

Neonatal-strength ampoules of nalorphine

SIR,—In correspondence with the makers of Lethidrone (nalorphine) Neonatal I have attempted to persuade them that they should discontinue production of the present multi-dose vial.

Although a recent article on neonatal resuscitation¹ does not mention the use of morphine antagonists, most practitioners in this field in Britain do recommend their use where there is presumptive evidence of depression of the baby due to opiate or pethidine administered to the mother. The ampoules of nalorphine for neonatal use contain 5 ml of solution with a concentration of 1 mg/ml. An individual dose for an infant should be of the order of 0.1 mg/kg body weight intravenously. Thus the present form of the vial exposes the infants to the twin risks of overdosage and infection. It seems irrational that we have largely rid ourselves of multidose ampoules because of the safety hazard to patients and yet we

allow the most vulnerable group at risk—the neonates with low Apgar score—to face the possibility of septicaemia or over-enthusiastic treatment of pethidine-induced respiratory depression.

In common with members of the division of paediatrics I feel that the time is overdue for the removal of multidose vials of nalorphine from the resuscitation trolley. Perhaps those of your readers who agree might care to contact the manufacturers. I have suggested as an alternative single-dose ampoules containing 0.5 mg nalorphine in 0.5 ml.

D G D DAVIDSON

University Department of Anaesthetics,
Royal Infirmary,
Manchester

¹ Gregory, G A, *Anesthesiology*, 1975, **43**, 225.

Nutmeg poisoning

SIR,—Following the report by Dr J A Barrowman and others (5 July, p 11) of the therapeutic use of ground nutmeg (*Myristica fragrans*) in doses of nine teaspoonfuls daily in controlling the diarrhoea associated with medullary carcinoma of the thyroid we would like to add a cautionary note on the dose recommended. Our own patient was given this dose and there was a satisfactory improvement in the number of motions passed daily. Within three days, however, he complained of dry eyes and mouth, blurred vision, dizziness, tingling, and feelings of depersonalisation and remoteness. They gradually resolved as the dose was reduced.

Nutmeg poisoning is not new. Green¹ cites descriptions given by De Lobel in 1576 and Purkinje in 1829. The subject aroused interest in the *BMJ* in 1906,² when the use of nutmeg as an abortifacient was reported; many subjects failed in their original intention and suffered side effects similar to those we have described. After a single dose of 18.5 g another patient reported periods of excitement and fear of impending death alternating with clouding of consciousness.¹ More recent reports^{3,4} cover the side effects intended in its use as a hallucinogen in the hippy subculture.

Should nutmeg or its active component, myristic acid, become a regular part of therapy for the diarrhoea associated with medullary carcinoma of the thyroid we would like to emphasise the side effects and remind people that at least one fatality has been recorded.¹

G S VENABLES
D EVERED
R HALLEndocrine Unit,
Royal Victoria Infirmary,
Newcastle upon Tyne

¹ Green, R C, *Journal of the American Medical Association*, 1959, **171**, 1342.

² Hammond, et al, *British Medical Journal*, 1906, **1**, 593; 1906, **2**, 778; 1906, **2**, 900; 1906, **2**, 984; 1911, **2**, 269.

³ Unwin, J R, *Canadian Medical Association Journal*, 1968, **98**, 402.

⁴ Panayotopoulos, D J, et al, *British Medical Journal*, 1970, **1**, 754.

Management of acute asthma

SIR,—I was interested in your leading article on this subject (11 October, p 65) and in Professor C M Fletcher's letter (8 November, p 345). I was particularly interested in his