different clinical pictures, and it is not common to obtain the epidemiological history described in their letter.

I believe that a doctor should always be consulted if the child has never had croup before. It is, however, difficult to see what could have been done to alter the outcome in this case. It seems reasonable to keep children with acute viral croup or other acute viral infections away from their playgroups and nursery schools until symptoms have subsided.

I accept Dr J Couriel's comments (14 April, p 1162) that the severity of stridor is not helpful when attempting to distinguish between viral croup and acute epiglottitis. I have found that a lateral radiograph of the neck is sometimes useful but agree that this has its hazards, and the child must at all times be accompanied by a doctor who can carry out immediate intubation.

Dr P J Robb and Dr P D M Ellis recommend tracheostomy rather than intubation for children with staphylococcal tracheobronchitis (14 April, p 1162). In my limited experience intubation and frequent endotracheal suction have been sufficient to maintain an adequate airway. I would still recommend this as the first line treatment. If secretions become difficult to control an elective tracheostomy is a relatively simple procedure.

A D MILNER

Department of Child Health,
Queen's Medical Centre,
Nottingham NG7 2UH

SIR,—I was surprised to read Dr J Couriel's comment that in acute stridor "examination of the throat using a tongue depressor will rarely clarify the diagnosis" (14 April, p 1162). This is one procedure which will establish whether or not epiglottitis is present and therefore whether or not artificial airways are likely to be needed. Of course, it must be done where immediate intubation is possible. A bronchoscope should be handy, and personnel and equipment for carrying out an immediate tracheostomy must be present.

What action should the general practitioner take when he receives a telephone call about a child with appreciable stridor? Ideally he could advise the parents to take the child to the nearest appropriate unit, where the prewarned staff would be ready to receive the case. Few units in the United Kingdom have facilities for the accommodation of many stridulous children for the necessary periods of observation. Improvements in this direction would reduce the mortality rate from acute upper airway obstruction.

M HARDINGHAM

Ear, Nose, and Throat Department,
Gloucester Royal Hospital,
Gloucester GL1 3NN

Idiopathic ulceration of the small bowel

SIR,—Dr M J Glynn and others (31 March, p 975) suggest that recurrent intestinal bleeding may be due to idiopathic ulcers of the small bowel. Biopsy examinations of these show no inflammation, and they may be diagnosed only by intraoperative examination with a fibrescope.

Although we agree with the principle that such endoscopy may be useful in some cases,[1] we cannot help feeling that in the two cases presented the ulcers may have been due to trauma from the passage of the endoscope: it is common to see small bleeding lesions in the stomach on the "way out" of a gastroscopy which were not visible on the first passage. The fact that such ulcers were not seen in two other cases is not enough to prove that they were not traumatic. The crucial point is whether the ulcers were seen on the way down or only on the way up.

J F L SHAW
E MCDERMOTT

Department of Surgery,
Whittington Hospital,
London N19 5NF

[1] Spiller RC, Parkins RA. Recurrent gastrointestinal bleeding of obscure origin: report of 17 cases and a guide to logical management. *Br J Surg* 1983;**70**: 489-93.

**Dr Glynn and Dr Parkins reply below.—ED, *BMJ*.

SIR,—We agree that the exclusion of trauma is central to the diagnosis. In both cases the ulcers were certainly first seen on the downward passage of the endoscope. During an intraoperative endoscopy it is easier to carry out a detailed inspection of the whole mucosa during withdrawal of the instrument, but it is important to make an adequate examination during insertion to avoid confusion with traumatic lesions.

One other factor which we think is important is that during the second laparotomy on case 1, which demanded considerable division of adhesions and consequent handling of the bowel, there was no correlation between areas of maximum handling and those of maximum ulceration. In addition, the ulcers described were different from deliberate suction injury to the mucosa.

M J GLYNN
R A PARKINS

Gastrointestinal Unit,
Charing Cross Hospital,
London W6 8RF

Sexually transmitted diseases in pregnancy

SIR,—We were surprised at Dr Janina M Harvey's statement that acyclovir is a cytotoxic agent (14 April, p 1158). Inhibition of cell multiplication is not produced by acyclovir at concentrations 1000 times those which will inhibit herpes simplex virus replication.[1] Indeed, a unique feature of acyclovir as an antiviral agent is its selective capacity to kill the virus in human cells without affecting the viability of uninfected cells.[1] Dosage with acyclovir is constrained by pharmacokinetic factors rather than any evidence of cellular toxicity.[2 3]

Dr Harvey's view may have arisen from misinterpretation of the advice in the Zovirax intravenous data sheet that "as there has been no clinical experience of the effects of Zovirax intravenous injection on human pregnancy or the fetus, caution should be exercised in prescribing Zovirax to pregnant woman" or from similar advice in the Zovirax oral data sheet. This advice is given despite the fact that two generation reproduction-fertility studies in mice showed no evidence that acyclovir was embryotoxic or impaired reproduction or development processes. Studies in rats and rabbits also failed to produce evidence of teratogenic effects or fetal problems.[4 5]

Herpes simplex infections are a problem in pregnancy due to morbidity in the mother and the risk of neonatal infection. We are, therefore, planning the cautious evaluation of the safety, efficacy, and pharmacokinetics of acyclovir in the mother just before term. We would be very interested to be informed of details of the use of acyclovir at any stage in pregnancy and would hope that the outcome of the pregnancy would be followed.

A M BELL
S R KINGSLEY
A P FIDDIAN
W D BRIGDEN

Departments of Clinical Immunology and Chemotherapy,
Wellcome Research Laboratories,
Beckenham, Kent BR3 3BS

[1] Elion GB, Furman PA, Fyfe JA, de Miranda P, Beauchamp L, Schaeffer HJ. Selectivity of action of an antiherpetic agent (9-(2-hydroxyethoxymethyl) guanine. *Proc Nat Acad Sci USA* 1977;**74**:5716-20.
[2] Brigden D, Fowle S, Rosling A. Acyclovir, a new antiherpetic drug: early experience in man with systemically administered drug. In: Collier LH, Oxford J, eds. *Developments in antiviral therapy*. London: Academic Press, 1980:53-62.
[3] Brigden D, Rosling AE, Woods NC. Renal function after acyclovir intravenous injection. *Am J Med* 1982;**73**:182-5.
[4] Moore HL, Szczech GM, Rodwell DE, Kapp RW, de Miranda P, Tucker WE. Preclinical toxicology studies with acyclovir: teratologic, reproductive and neonatal tests. *Fund App Toxicol* 1983;**3**:560-8.
[5] Tucker WE Jr. Preclinical toxicology of acyclovir: an overview. *Am J Med* 1982;**73**(suppl 1A):27-30.

Self help in venereology

SIR,—Dr A D G Gunn (7 April, p 1024) offered some useful insights into patients' mutual aid organisations but did not state the features of the diseases which engender these organisations. These are chronic diseases which cause considerable distress and are not cured by current treatment.

Venereology can permanently cure most of the infections which fall under its aegis. It cannot, however, cure all, and four particular problems are herpes genitalis, pelvic inflammatory disease, acquired immune deficiency syndrome (AIDS), and hepatitis B.

The patient initiated support groups for sufferers from these diseases are listed here:

(1) The Herpes Association, c/o Spare Rib, 27 Clerkenwell Close, London EC1 0AT.

(2) PID Support Group, 32 Parkholme Road, Dalston, London E8.

(3) AIDS sufferers and at risk groups can contact the Terence Higgins Trust, c/o Gay Medical Association, London WC1N 3XX.

(4) The Hepatitis B support group: telephone 01-603 6516 or 01-373 6105.

DERMOT MURRAY

Department of Genitourinary Medicine,
Derbyshire Royal Infirmary,
Derby DE1 2QY

Patient information booklets

SIR,—Dr P J M Sloan (24 March, p 915) mostly restricted his list of educational literature for patients to the publications of pharmaceutical companies. This gives the wrong impression that there is little information available for the rheumatic patient.

The Arthritis and Rheumatism Council produces the following patient information handbooks: *Rheumatoid Arthritis*, *Osteoarthritis*, *Ankylosing Spondylitis*, *Gout*, *Lumbar Disc*

Disorders, and *Pain in the Neck*. These are free and can be sent in bulk to general practitioners and hospital doctors or individually to patients. Special booklets for the parents and teachers of children with juvenile arthritis, *When Your Child Has Arthritis* and *When A Young Person Has Arthritis*, have also been produced.

Other booklets—*Your Home and Your Rheumatism*, *Marriage*, *Sex and Arthritis*, and *Are You Sitting Comfortably?*—are available at a modest cost. All booklets and a publication list can be obtained from the Arthritis and Rheumatism Council, 41 Eagle Street, London WC1R 4AR.

RACHEL J INGLIS

Arthritis and Rheumatism Council,
London WC1R 4AR

Medicine in the Third World

SIR,—Professor J K G Webb's paper (14 April, p 1141) was most stimulating. I have recently returned from west Africa after two years in a rural mission hospital and have been reflecting on the worth of this enterprise. There seem to have been three main benefits. Firstly, the obvious benefits to me in broadening my experience of life and medicine.

Secondly, several hundred patients benefited directly from having been seen and treated by a doctor. Most of this work was curative, and often the results were dramatic despite limited resources and laboratory facilities. The major cost was not that of drugs, materials, or overheads, but of paying me. In fact since I was funded from the UK the care received was subsidised by my funding organisation. The overall cost benefit ratio was still impressive in comparison with the "first world." This reflects more on Third World deprivation than on "first world" inefficiency.

The third benefit was to the whole population. Here I believe I served as a lure. European and American trained doctors have a considerable reputation, and patients are drawn to them from great distances—often to the detriment of local clinics and hospitals. A positive side was that our hospital was able to run nurse and midwife training schools where the standards attained were adequate and, more importantly, appropriate. Without the stream of committed, mainly foreign, doctors to the hospital these schools would be threatened, and I suspect that my main contribution was this indirect support to the schools. This may encourage the deprofessionalisation of medicine, but the long term benefits will outweigh the care dispensed by quality conscious doctors.

C S BARCLAY

Northern General Hospital,
Sheffield S5 7AU

What carbohydrate foods should diabetics eat?

SIR,—Dr J I Mann attempts to explain the beneficial effect of cooked dried beans in terms of delayed digestion, fibre that forms gels, particle size, and integrity of structure of the food (7 April, p 1025). To the list of possible explanations should be added the effects of lectins, which are haemagglutinating proteins present in most plant products and especially prominent in legumes.[1] Some lectins are resistant to cooking and digestion and may appear in active form in the systemic bloodstream after feeding.[2] Kidney bean lectin binds to intestinal epithelium and interferes with the absorption of glucose and in higher concentrations causes gut damage that is occasionally manifest as severe gastroenteritis in incautious folk who cook their beans inadequately.[3]

Most relevant in this context is the insulin mimicking effect of certain lectins.[4] Wheat germ lectin binds to the insulin receptors of adipocytes and is as effective as insulin itself in enhancing glucose transport and inhibiting adenylate cyclase. A substantial effect occurs at lectin concentrations as low as 4·0 nmol/l. At such low concentrations wheat germ lectin also enhances the binding of insulin to its receptors—an effect noted by Dr Mann as resulting from a high carbohydrate diet. Although biochemists have known these findings for over 10 years,[5 6] no physician seems yet to have tried the therapeutic effect of deliberate lectin administration, perhaps because of the fearsome reputation of some lectins as poisons.[7] I have ingested the jackbean lectin concanavalin A many times experimentally and live to tell the tale; lectins are not as frightening as they may seem and should be investigated in diabetic therapy.

DAVID FREED

Department of Immunology,
University of Manchester,
Manchester M13 9TP

[1] Lis H, Sharon N. Lectins in higher plants. In: Stumpf PK, Conn EE, eds. *The biochemistry of higher plants*. Vol VI. New York: Academic Press, 1982.
[2] Pusztai A, Clarke EMW, Grant G, King TP. The toxicity of Phaseolus vulgaris lectin: nitrogen balance and immunochemical studies. *J Sci Food Agric* 1981;**32**:1037-46.
[3] Public Health Laboratory Service. An unusual outbreak of food poisoning. *Br Med J* 1976;ii:1268.
[4] Cuatrecasas P, Tell GPE. Insulin-like activity of concanavalin A and wheat germ agglutinin—direct interactions with insulin receptors. *Proc Nat Acad Sci USA* 1973;**70**:485-9.
[5] Smith JD, Liu AY-C. Lectins mimic insulin in the induction of tyrosine aminotransferase. *Science* 1981;**214**:799-800.
[6] Freed DLJ. Lectins, allergens and mucus. In: Bog-Hansen TC, ed. *Lectins—biology, biochemistry and clinical biochemistry*. Vol II. Berlin: Walter de Gruyter, 1982.
[7] Knight B. Ricin—a potent homicidal poison. *Br Med J* 1979;i:350-1.

Drug induced Parkinson's disease

SIR,—I want to make one important amendment to Dr J E C Hern's algorithm for the elucidation of tremor (7 April, p 1072). He suggests that if parkinsonian tremor is present and the patient is not taking phenothiazines, haloperidol, or tetrabenazine, then the patient should be treated for Parkinson's disease. This is an unnecessarily limited inquiry and will lead to some cases of drug induced parkinsonism being missed. We found four cases of occult drug induced parkinsonism,[1] and have added several more in the past two years. These were patients who presented with parkinsonism and in whom it appeared that no drug was responsible, but further detailed inquiry showed that they had been taking a drug capable of causing parkinsonian features.

Additionally, we have recently studied 48 cases of drug induced parkinsonism and found that while most lost their parkinsonian features between six weeks and three months a few retained these features much longer. One 88 year old lady became totally free of parkinsonian features only after nine months. She would have been caught in Dr Hern's algorithm and treated for parkinsonism—no doubt with excellent results.

Drug induced parkinsonism is much commoner (especially in the elderly) than most people have been led to believe. It is necessary to inquire minutely into all recent drug treatment before diagnosing idiopathic parkinsonism and in doubtful cases a careful "drug holiday" should be arranged at intervals of three months up to at least one year from starting levodopa or other antiparkinsonian treatment.

J WILLIAMSON

Department of Geriatric Medicine,
City Hospital,
Edinburgh EH10 5SB

[1] Murdoch PS, Williamson J. A danger in making the diagnosis of Parkinson's disease. *Lancet* 1982;i:1212-3.

Fractures and hypercalciuria: two markers of severe dependence in alcoholics

SIR,—We have read the contrasting data of Dr R D Johnson and others (4 February, p 365) and Lindsell *et al* (28 August-4 September 1982, p 597) about the presence of fractures on chest radiographs as an indicator of alcoholism in patients with liver disease. Dr Johnson and others are probably right that the differences between surveys may reflect the different populations studied.

Many patients with alcoholic liver disease have only mild if any alcohol dependence.[1 2] We work in a wine producing area where many heavy wine drinkers develop liver disease without major social, psychological, or behavioural problems. Our findings are that the presence of fractures is related only to the severity of alcohol dependence. We have found a similar relationship with urinary calcium excretion.

We studied 106 patients aged between 22 and 73 years (mean 43·8) admitted to the department of internal medicine and the alcohol treatment unit between 1982 and 1984 with a history of alcohol consumption of at least 160 g (5·6 oz) a day for 10 years or more. A history of alcohol consumption and inquiry of any trauma or fractures was elicited by one of the doctors using a standardised interview schedule. Dependence was assessed by an alcohol dependence questionnaire devised according to the guidelines of a World Health Committee.[3] A total of 53% of alcoholics had no or only mild dependence, while 47% were classified as severely dependent. A control group consisted of 150 patients matched for age and sex and referred with various diseases not related to alcohol. Fractures were more common in alcoholics (61 patients, 58%) than in controls (11 patients, 7%). Limb and rib fractures were the most common (31 patients, 29%; and 29 patients, 27%, respectively). Rib fractures were multiple in 24% and bilateral in 9% of alcoholics. Skull, nasal septum, pelvis, and clavicular fractures were less common (11 patients, 10%). Severely dependent alcoholics showed a higher incidence of fractures compared with those with mild dependence (39 patients, 78%, compared with 12 patients, 22%). No significant difference in age, duration of drinking, serum γ-glutamyl transpeptidase or mean corpuscular volume was found between the groups, but severely dependent alcoholics showed a slight decrease in serum calcium (2·25±0·11 mmol/l (9±0·44 mg/100 ml) versus 2·4±0·13 mmol/l (9·6±0·52 mg/100 ml); p=0·029).

We decided to study the renal handling of calcium in our patients. Fractional urinary excretion of calcium, calculated as the ratio of calcium clearance to creatinine clearance, and fasting urine calcium to urine creatinine ratio were measured within 48 hours of admission. Severely dependent alcoholics showed increased fractional urinary excretion of calcium compared with those with mild dependence (1·387±0·502 versus