enough of the element. As a matter of fact many commercially available solutions and especially those of amino-acids are slightly contaminated with zinc and other elements,⁴ but the quantities present are far below actual needs. Some of the newer solutions for parenteral feeding do contain trace elements in adequate amounts, but nevertheless we still frequently have to add further 100-500 µg/day (1-5 ml of a solution containing 0.1 mg Zn/ml as ZnCl₂), mostly in surgical patients, to achieve positive nitrogen balance, normal wound healing, and growth.

Calculations of the actual amounts of other trace elements in human plasma lead to the same conclusions as to the rationale of its use as a source of trace elements for patients under parenteral nutrition. It is therefore mandatory actually to calculate even microgram amounts and stimulate the pharmaceutical industry to supply adequate solutions for general use.

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- ¹ Fox, H A, and Krasna, I H, *Pediatrics*, 1973, **52**, 14.
- ² Halsted, J A, and Smith, J C, *Lancet*, 1970, **1**, 322.
- ³ Ricour, C, and Nihoul-Fekete, C, *Archives Françaises de Pédiatrie*, 1970, **30**, 469.
- ⁴ James, B E, and MacMahon, R A, *Medical Journal of Australia*, 1970, **2**, 1161.

Cracking urinary bladder stones

SIR,—Your interesting leading article (9 July, p 79) suggests that electronic stone disintegration is a safe method of dealing with vesical calculi, though not without significant disadvantages. In my view the method is also accompanied by distinct hazards.

Each underwater spark produces not only the shockwave which you mention but also a bubble of vapour, which expands and contracts extremely rapidly in oscillatory fashion in the subsequent five or so milliseconds.¹ The maximum size of this bubble depends among other things on the electrical energy used, but in one instrument² (not the one you mention) an energy of 18 joules produces a bubble approximately 3 cm in diameter. Each time the oscillating bubble reaches a minimum volume it emits a pressure pulse and the first one or two pulses are comparable in destructive force to the preceding shockwave. The process has many similarities, except in scale, to the underwater detonation of high explosive.

Although the oscillating bubble, through its pressure pulses, adds significantly to stone destruction it is not widely appreciated that it also represents the major source of hazard from the technique, not from the pressure pulses (which, like the shock wave, pass relatively harmlessly through the bladder wall) but from the associated violent mass movement of water. Whereas a large stone shields the bladder wall from the vapour bubble and the fluid it rapidly displaces, a smaller fragment may be ejected into or even through the bladder wall if inappropriate power settings and techniques are used. These risks are not merely theoretical as damage to the bladder wall and frank perforation have been reported.³⁻⁵

The phenomena described make it clear why attempts to use existing disintegrators in the confined space of the ureter have resulted in damage⁴ and why their recent suggested use in the renal pelvis is inappropriate.

It may nevertheless prove possible to meet

the need, to which you direct attention, for instruments to tackle ureteric stones. Modifications to electrode design can produce focusing, enabling power (and hazards from the bubble) to be reduced without loss of effectiveness. It may also prove possible to produce a totally enclosed fluid-filled electrode, thus confining the bubble but permitting passage of the shock wave, but the engineering problems are considerable. Finally, further developments of the device described by Schuy and Schmidt-Kloiber⁶ in which the mechanical energy of external underwater electrical discharges is conveyed to the target calculus by a reciprocating flexible rod may also provide a solution.

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- ¹ Tidd, M J, et al, *Biomedical Engineering*, 1976, **11**, 5.
- ² Wallace, D M, Cole, P F, and Davies, K L, *British Journal of Urology*, 1972, **44**, 262.
- ³ Tidd, M J, et al, *Urological Research*, 1976, **4**, 49.
- ⁴ Alfthan, O, and Murtomaa, M, *Scandinavian Journal of Urology and Nephrology*, 1972, **6**, 23.
- ⁵ *Hospital Equipment Information*, 1972, **41**, 3.
- ⁶ Schuy, S, and Schmidt-Kloiber, H, *Biomedizinische Technik*, 1973, **18**, 17.

Tuberculous peritonitis with cirrhosis of the liver

SIR,—In your excellent leading article on abdominal tuberculosis in Britain (18 June, p 1557) it is suggested, among other things, that "ascitic fluid with lymphocytosis and high protein concentration should . . . increase the suspicion of tuberculosis." However, in tuberculous peritonitis complicating cirrhosis of the liver the protein concentration of the ascitic fluid may be low.¹ Tuberculous peritonitis in cirrhotics "is still encountered and is often unsuspected."² We should like to draw attention to such a diagnostic possibility, reporting a case of tuberculous peritonitis complicating cirrhosis in which the protein concentration in the ascitic fluid was low.

A 34-year-old woman was admitted to our department because of ascites under pressure, anorexia, abdominal and right hypochondrial pain, severe weakness, and pyrexia up to 38°C. The history had started two years previously with progressively deteriorating flatulent dyspepsia and weakness. Four months before admission pyrexia, abdominal pain, and ascites were added. On examination we found stigmata of liver disease, mild jaundice, ascites under pressure, and oedema of the ankles and legs. After we had aspirated about 1.5 l of ascitic fluid a large hard spleen with a sharp edge (about 10 cm below the left costal margin) and many small intra-abdominal lumps were felt. There was no hepatomegaly and no lymph-node enlargement. The blood count and film were normal and the erythrocyte sedimentation rate was 90 mm in the first hour. The serum albumin concentration was 30 g/l and that of globulins 57 g/l. There was mild elevation of the serum alkaline phosphatase and aspartate and alanine transaminase (SGOT and SGPT) activities. The plasma bilirubin ranged from 32.5 to 107.7 µmol/l (1.9 to 6.3 mg/100 ml) and was mainly conjugated. Tests for hepatitis B antigen were positive, for α-fetoprotein negative, and smooth-muscle antigen positive. The ascitic fluid was examined twice. The protein content was about 10 g/l and the Rivalta reaction negative; there were many leucocytes mainly lymphocytes. Tubercle bacilli were present in both the specimens. The chest x-ray was normal and a tuberculin skin test (1:100 000) was negative. A liver scan was compatible with cirrhosis.

The patient was treated with a conventional triple antituberculosis treatment. After some days the pyrexia declined and finally disappeared and progressively the appetite improved, the abdominal pain subsided, and the intra-abdominal

lumps almost disappeared. The patient was discharged from hospital but unfortunately died about three months later in hepatic coma.

Although the ascitic fluid in tuberculous peritonitis usually contains more than 25 g of protein/l and in cirrhosis less than 25 g/l, if the two diseases coexist values greater or less than 30 g/l have been reported.¹ The presence of pyrexia and abdominal pain in a cirrhotic raises the possibility of another disease. Hence the diagnosis of tuberculous peritonitis must be kept in mind when a cirrhotic patient presents with pyrexia and abdominal pain even if the ascitic fluid is a transudate. Thorough examination of the ascitic fluid for tubercle bacilli, including culture and inoculation, is mandatory.

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- ¹ Burack, W R, and Hollister, R M, *American Journal of Medicine*, 1960, **28**, 510.
- ² Sherlock, S, *Diseases of the Liver and Biliary System*, 5th edn. Oxford, Blackwell Scientific, 1975.

Vitamin B₁₂ for vegans

SIR,—We read your expert's reply (11 June, p 1525) and Mr Alan Long's letter (16 July, p 192) on vegan sources of vitamin B₁₂ with interest.

Beliefs that the comfrey plant (*Symphytum officinale*) is a natural source of vitamin B₁₂ persist and are repeated in the current catalogue of at least one firm of horticultural seedsmen and another specialist supplier of herbal products. We therefore extracted 12.5 g of freshly picked comfrey leaves by boiling in 500 ml acetate buffer (pH 5.0) containing 0.01% sodium cyanide in preparation for assay.¹ No vitamin B₁₂ was detected in the extract using the *Euglena gracilis* var *bacillaris* z-strain assay²; this implies a vitamin B₁₂ concentration of less than 10 ng/l of extract. Thus 1 kg (2.2 lb) of fresh comfrey leaves could at most have contained 400 ng (0.4 µg) of vitamin B₁₂.

We therefore conclude that comfrey leaves are not relevant as a source of vitamin B₁₂ in mixed, vegetarian, or vegan diets.

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- ¹ Gray, L F, and Daniel, L J, *Journal of Nutrition*, 1959, **67**, 623.
- ² Hutner, S H, Bach, M K, and Ross, G I M, *Journal of Protozoology*, 1956, **3**, 101.

Schistosomal myelopathy

SIR,—The report of a case of schistosomal myelopathy by Dr J Cohen and others (14 May, p 1258) is of great interest, but their conclusion that the cord damage is immunological is open to doubt. The presence of specific antibodies, whether in the serum or the CSF, indicates only that there is an immune reaction and not that pathological changes are due to it. In fact a study of previously reported cases of this condition would indicate that direct involvement of the cord

with schistosomal ova is an essential part of the syndrome and that the immunological response a consequence of the infestation and not the cause of the neurological lesion.

In 41 cases of schistosomal spinal cord disease I have traced in the literature¹⁻⁵ the neurological deficit was caused by cord compression due to a discrete granuloma in 13. It would be superfluous to implicate an immunological cause in these cases. Of the remaining 28 cases, in which cord compression was not reported, the spinal cord was examined histologically in 18 and ova found in all of these. I can find no recorded case of this condition in which ova were looked for in the spinal cord and not found. This is in remarkable contrast to schistosomiasis without clinical spinal cord involvement, in which ova are rarely found in the cord.⁶

As ova appear to be invariably present in the affected part of the spinal cord in these cases it is difficult to avoid the conclusion that the lesion is a direct consequence of their presence and there is no need to postulate an immunological cause.

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¹ Marcial-Rojas, R A, and Fiol, R E, *Annals of Internal Medicine*, 1963, **59**, 215.

² Bird, A V, *Neurology*, 1964, **10**, 647.

³ Levy, L F, and Taube, E, *Central African Medical Journal*, 1969, **15**, 52.

⁴ Rosenbaum, R M, et al, *American Journal of Tropical Medicine and Hygiene*, 1972, **21**, 182.

⁵ Herskowitz, A, *Journal of Neurosurgery*, 1972, **36**, 495.

⁶ Gelfand, M, *Schistosomiasis in Southern Africa*. Juta, Cape Town, 1950.

Poisoning with mianserin

SIR,—I would like to make the following point regarding the letter from Dr P Crome and Miss Belinda Newman (23 July, p 260) concerning overdosage with maprotiline and mianserin.

They report the death of a 72-year-old woman who took an overdose of 60 × 10 mg mianserin and an unknown quantity of lorazepam. On admission her plasma level of mianserin was 110 µg/l and that of lorazepam 500 µg/l. While the level of mianserin was in the therapeutic range, that of lorazepam was in overdose.

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Immunisation rates in Kent

SIR,—I was concerned to hear an extract from the BMA conference being reported on the national news in which Dr W J Appleyard quoted figures for the uptake of childhood immunisation in Kent. He stated that only 67% of children were being immunised against polio, diphtheria, and tetanus and only a third were being immunised against whooping cough.

The immunisation programme in Kent is computerised and I believe the computer's results to be suspect. I recently received a print-out showing that 46 children on my list had not been immunised. When these children were followed up it was found that six were either not on my list, had come on to it after their immunisations were complete, or mainly lived abroad. Twenty-three had been

fully immunised and in the remaining 17 cases the parents had elected not to have the whooping-cough component of the programme. Thus all the relevant children were accounted for. Discussion with several other GPs in south-east Kent revealed a similar picture. I reported these findings to the area health authority, but I believe that the quoted figures are still a gross underestimate of the true immunisation state of the county, which can be checked only by using the clinic and surgery records rather than relying on computer returns.

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Danger of saline emetics

SIR,—The *Daily Telegraph* of 2 July drew attention to the dangers inherent in the use of salt and water as an emetic. This specially arose in relation to the unfortunate death of a patient given such an emetic following an overdose of drugs. The news item stated that death was attributed to a dangerous surfeit of sodium in the patient's body which caused a brain haemorrhage. It appeared from the evidence given at the inquest that there is a general unawareness of the danger of salt emetics.

Some 14 years ago I described and discussed this hazard in a case study of fatal hypernatraemia after a saline emetic.¹ The practice of attempting to induce vomiting by this method has become especially dangerous since the advent of the phenothiazines as this group of psychotropic drugs is so frequently prescribed and so frequently figures in overdoses, and many of them are potent antiemetics. Perphenazine, for instance, is 16.6 times more powerful than chlorpromazine as an antiemetic. Nor is it always known with patients who have taken drug overdoses exactly what drugs or quantities have been taken.

All things considered it is probably safest to discard this practice from our therapeutic armamentarium.

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¹ Ward, D J, *British Medical Journal*, 1963, **2**, 432.

Dextrostix-Eyetone in the insulin hypoglycaemia test

SIR,—Dr M A Preece and Mr R G Newall (16 July, p 152) draw attention to the need for rapidly available blood glucose values during the insulin tolerance test (ITT) to avoid severe hypoglycaemia and to ensure a sufficiently low blood glucose concentration to elicit a growth hormone response. The Dextrostix-Eyetone procedure they describe goes a long way, in principle, to meet this need, but our own experience in routine rather than "research" conditions makes us urge caution.

We would make the following points: (1) Results improved with experience, even in the hands of one enthusiastic clinician. In practice, however, several of our housemen have had too little experience and time to develop the needed expertise. The very simplicity of the system encourages carelessness. (2) No quality control of the procedure is described. This is surely unacceptable for so important a test; the manufacturers themselves recommend the

regular use of Lab-Chek control materials. (3) Even under the favourable conditions of this evaluation the quoted precision is unimpressive. The error, so far as we can calculate from the figures quoted, is twice that of our routine laboratory method. (4) The major potential advantage, avoiding severe hypoglycaemia, is undermined by the one large, inexplicable error which the authors are honest enough to admit.

The Dextrostix-Eyetone system is tantalisingly close to being a major advance in monitoring of ITTs. We would stress the importance of adequate training and experience of the operator and regular quality control procedures.

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Getting close to the patient

SIR,—With reference to the letter from Dr J L Ogle (16 July, p 191), surely we require more information before we consider altering the system? It is my experience that the sort of advice which he quotes and ridicules, even when issued by an area health authority, has usually been prepared by or after consultation with practitioners in the relevant specialty. This practice is certainly neither new nor the creation of the reorganised Health Service.

If such advice is given to hospital staff which is not the result of consultation it should, of course, be repudiated. If staff are asked to participate in the preparation of such advice they should formally decline to do so if they consider the task inappropriate and make it known to the members of the health authority that the issuing of such advice is unnecessary.

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Debendox overdosage in children

SIR,—Following a previous report on Debendox overdosage in children¹ we would like to report a case treated here recently. It illustrates the dangers inherent in accidental ingestion of slow-release preparations when there is ignorance of their nature on the part of those for whom the drug is prescribed, particularly when the drug is commonly prescribed to an age group which is likely to have young children in the home, such as mothers suffering from vomiting of pregnancy.

A 3½-year-old boy was admitted from the accident and emergency department some time after midnight, having apparently been found by his mother 7 h earlier with an empty bottle which had contained an unknown quantity of Debendox tablets, each containing dicyclomine hydrochloride 10 mg, doxylamine succinate 10 mg, and pyridoxine hydrochloride 10 mg in slow-release form (57 were thought later to be unaccounted for). Because he seemed well at the time he had been put to bed and it was not until midnight that he became agitated and restless, precipitating his admission.

On arrival he was hallucinating and restless, with dilated pupils, a dry mouth, hot dry skin, and a tachycardia of 150/min. He was initially treated with pilocarpine 3 mg subcutaneously plus chlorpromazine 15 mg intramuscularly, with the oral admission of magnesium hydroxide as a purgative. He seemed to improve 90 min later after a further