hydration. Inappropriate antidiuretic hormone secretion and body potassium deficit have also been blamed.¹

While important details of the clinical features of Dr Pinnock's cases are missing, it does seem that his patients developed hyponatraemia in the absence of oedema or cardiac failure as diuretic treatment proved unnecessary. His letter confirms a suggestion that the elderly are particularly likely to develop hyponatraemia on diuretics² and emphasises the importance of awareness of this drug adverse effect.

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¹ Fichman, M P, *et al*, *Annals of Internal Medicine*, 1971, 75, 853.

² Roberts, C J C, *et al*, *British Medical Journal*, 1977, 1, 210.

SIR,—Further to Dr C A Pinnock's letter (7 January, p 48) experience in recent years has taught me that unexpected confusional symptoms and signs, especially with seemingly hysterical features, occurring in an elderly patient on loop diuretics may be due to iatrogenic salt and water depletion. Thus it seems that the key factor is the intensity of treatment rather than the type of diuretic.

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Overdose of Lomotil

SIR,—We would like to add to the discussion by Dianne Penfold and Dr G N Volans in their article (26 November, p 1401).

This year we successfully treated two 2-year-old children who had ingested large amounts of Lomotil. The first had taken 94 tablets prescribed for his father's chronic diarrhoea, each tablet containing 2.5 mg of diphenoxylate hydrochloride and 0.025 mg of atropine sulphate. One hour before admission the child had been found playing with the empty bottle, there being no evidence of spillage. On examination the child was slightly flushed, pupils equal and non-dilated, pulse 105/min, and respiratory rate 24/min. Gastric lavage was carried out, tablets being retrieved from the aspirate. We decided to try purgation with enemas and magnesium sulphate,¹ give intravenous fluids, practolol in the event of a tachycardia greater than 200/min, and naloxone for respiratory depression. Five and a half hours after ingestion the patient became increasingly drowsy, his respiratory rate had fallen to 12/min, his pupils were widely dilated, he was peripherally warm, with a tachycardia of 150/min, and was just responding to painful stimuli. He was given naloxone 0.12 mg (0.01 mg/kg) twice within a space of 2 min with good effect. After the second dose of naloxone his respiratory rate never fell below 15/min but was on occasions shallow. Five hours after becoming unconscious he woke up and asked for tea! Purgation was unsuccessful. The second child took about 22 tablets and some 5 h after ingestion she had a respiratory arrest with a tachycardia of 150/min. She had been given ipecacuanha at another hospital. This patient required three doses of naloxone (0.14 mg) and went home 48 h later.

We would agree with the authors about the dangers of Lomotil; there were over one million prescriptions for Lomotil in the United Kingdom in 1976.² However, there is no mention of the necessity of inducing vomiting, particularly if the patient is known to have

taken the tablets recently; in the first of our cases we believe this was life-saving.³ We feel the ineffectiveness of purgation might have been due to under-treatment or to the side effects of the atropine in drug. Finally, naloxone seems to be a fairly safe drug and might be used by general practitioners faced with such a case, a dose being given before or during transfer of the patient to hospital.

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¹ Meadow, S R, and Leason, G A, *Archives of Disease in Childhood*, 1974, 49, 310.

² Information supplied by manufacturer.

³ *British Medical Journal*, 1977, 2, 977.

Vacuum pipelines for anaesthetic pollution control

SIR,—A recent meeting of the British Standards Subcommittee on Hospital Vacuum Pipelines (SGS/24/1) considered the use of medical vacuum pipelines for the removal of atmospheric pollution in areas where anaesthetics are administered.

Medical vacuum pipelines are designed for clinical use in the treatment of patients, often in emergency situations, while the removal of atmospheric pollution is for staff safety. The two purposes do not coincide. A medical vacuum pipeline may not be adequate for pollution control under all circumstances and most older installations of medical vacuum pipelines (perhaps 90% of hospitals in Britain) are below British Standard specifications. Their use for other than patient care would render them even less effective. In addition, there is a small, unquantifiable, hazard of ignition from the aspiration into the pipeline of higher concentrations of oxygen and of inflammable agents. In both old and new hospitals it was felt that a system for removing atmospheric pollution should be entirely separate from the medical vacuum pipeline.

For these reasons the committee could not support the use of medical vacuum pipelines for pollution control in this country other than in exceptional circumstances.

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British Standards Subcommittee
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Doxorubicin cardiotoxicity: role of digoxin in prevention

SIR,—It would be unfortunate if clinicians drew from the report of Mr D Guthrie and Dr A L Gibson (3 December, p 1447) the conclusion that it is proved that digoxin protects from doxorubicin (Adriamycin) cardiotoxicity. Evidence on the protective effects of digoxin given before doxorubicin in animal models is conflicting¹⁻³ and it is therefore reasonable to test its role in the prevention of doxorubicin cardiotoxicity in man. Unfortunately, this non-randomised study does not fully report the distribution of known risk factors:⁴⁻⁶ age, previous radiation to the heart, concomitant cyclophosphamide, and possibly preceding hypertension. Though electrocardiographic (ECG) criteria capable of predicting doxorubicin-associated cardiac failure have been reported,⁷ the criteria for the ECG

detection of doxorubicin cardiotoxicity are regrettably not reported in this study.

One patient in this report (case 1) developed ECG changes of doxorubicin toxicity "some weeks" after stopping her digoxin, the implication being that the cessation of digoxin precipitated a very rapid onset of cardiotoxicity. The use of serial endomyocardial biopsies in patients receiving doxorubicin has clearly demonstrated that there is a gradual and progressive drop-out of myofibrils which is detectable at very low doses when all other non-invasive tests are normal.^{5,8} It is, therefore, highly likely that this patient was gradually developing doxorubicin cardiotoxicity before stopping her digoxin.

The rate of administration of doxorubicin (immediately or over the whole drug course) may influence the incidence of cardiotoxicity,⁷ and the lack of data on the exact dose schedule for patients in each group further compounds the difficulty in analysing this study.

A controlled clinical trial with careful analysis of all important risk factors is necessary to show that digoxin can prevent doxorubicin cardiotoxicity. More sensitive methods^{5-7,9} of detecting cardiotoxicity, including prospective ECGs, phonocardiography (PEP/LVET), echocardiography, and possibly cardiac catheterisation and endomyocardial biopsy would greatly strengthen any conclusions. The achievement of a P value of 0.001 in a small non-randomised retrospective study does not constitute good evidence supporting the clinical use of digoxin for the prevention of cardiotoxicity. It should, however, stimulate further investigation in this field.

Despite the similarity between tetracycline and doxorubicin the inference that it can improve necrosis of myofibrils in doxorubicin cardiotoxicity is without foundation, particularly when digoxin is already being used for a similar mechanism of action.

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¹ Arena, G, *et al*, in *International Symposium on Adriamycin*, ed S Carter *et al*, pp 96-116. New York, Springer, 1972.

² Phillips, F, *et al*, *Cancer Chemotherapy Reports*, 1975, 6, 177.

³ Lefrak, E A, *et al*, *Cancer*, 1973, 32, 302.

⁴ Lenaz, L, and Page, J, *Cancer Treatment Review*, 1976, 3, 11.

⁵ Bristow, M, *et al*, *Proceedings of the American Society for Clinical Oncology*, 1977, 18, 334.

⁶ Gilladoga, A, *et al*, *Cancer Chemotherapy Reports*, 1975, 6, 209.

⁷ Minow, R A, *et al*, *Cancer*, 1977, 39, 1397.

⁸ Fridman, M, *et al*, *Proceedings of the American Association for Cancer Research*, 1977, 18, 179.

⁹ Rinehart, J J, *et al*, *Annals of Internal Medicine*, 1974, 81, 475.

Legionnaires' disease

SIR,—It would be incorrect to assume, as you do in your leading article (7 January, p 2), that no attention has been paid to sputum examination in patients with legionnaires' disease even though the organism has not yet been cultured from this source. In that treatment cannot be delayed until a specific diagnosis is made it is often similarly true that sputum from patients with pneumonia may be negative for other respiratory pathogens. There is the additional problem of seeking for minute, slow-growing bacterial colonies among other flora. Ultimately it is probable that a diagnosis may be established more rapidly and reliably by fluorescence or other immunochemical test.

Though legionnaires' disease may have "no characteristic clinical features" it has manifested itself in Britain as a severe lobar pneumonia with amnesia and rapid progression despite usually adequate chemotherapy. This, I suggest, points to a bacteraemia with general dissemination of the organism. Some support for this comes from recovery of the organism from pleural exudate during life.¹ Considerable efforts should be made to recover the organism from the blood by subculture on the recommended medium.

It is likely that materials for a retrospective serological diagnosis will shortly be available, so that it is worth collecting and storing serial samples of serum from suspect cases. This should be done up to and beyond the third week of illness. As well as culture, specific fluorescence of fresh or formalin-fixed lung tissue can establish a diagnosis when the outcome has been fatal.

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¹ *Morbidity and Mortality Weekly Report*, 1977, 26, 111.

Ferritin and iron overload

SIR,—Mr A K Li and Dr R G Batey (19 November, p 1327) describe a patient with a primary bronchial carcinoma which contained an "abnormal" ferritin. They suggest that this ferritin was the cause of iron deposition in the liver and spleen. We would like to comment on two aspects of their study.

Their method for determining isoelectric points by polyacrylamide gel electrophoresis is not given, but in any case the description of the tumour ferritin as being of isoelectric point (pI) 4.9-5.0 is not proof of abnormality. Ferritins of similar pI are normally present, for example, in heart muscle and in reticulocytes.

The demonstration that serum ferritin will bind ⁵⁹Fe in a similar way to transferrin needs very careful examination. Iron must normally be supplied as Fe²⁺ for incorporation into the iron core of ferritin. Even with Fe³⁺ we have found that several serum proteins, including some of similar molecular weight to apoferritin, will bind iron. However, iron uptake by ferritin in crude or partially purified serum is negligible under these circumstances.

We feel that much clearer evidence for an abnormal ferritin is needed before any speculation about its role in iron metabolism is worth while.

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Plasmapheresis and myasthenia gravis

SIR,—The recent article on progress in myasthenia gravis by Dr C W H Havard (15 October, p 1008) makes brief mention of the role that circulating antibodies to acetylcholine receptor plays in the pathogenesis of this disease. However, no comment is made of plasmapheresis as a means of reducing the titre of the antibody in patients who are refractory to conventional therapy, particularly after surgical removal of the thymus gland.

Experience at the Hammersmith Hospital¹ has drawn attention to the benefits that may follow the use of plasma exchange in myas-

thenia gravis and a similar experience is reported by Finn and Coates.² Our own experience with one case (to be published) underlines the important fact that plasmapheresis may not only be of benefit early in the course of treatment but may need to be continued for at least six weeks. Our observations support the suggestion² that an immediate response might not occur if the myoneural junction had been structurally damaged by long exposure to antibody attack and that a variable period of time might be needed for end-plates to recover.

On the basis of available evidence together with our own experience we believe that plasmapheresis requires further study in the treatment of patients with myasthenia gravis and that this may assume critical importance when ventilatory support is required. The beneficial effects¹⁻³ may also be related to the reduction in plasma cholinesterase activity⁵ and this should be combined with immunosuppressive drug regimens to avoid rebound phenomena.⁴

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¹ Pinching, A J, Peters, D K, and Newson Davis, J, *Lancet*, 1976, 2, 1973.

² Finn, R, and Coates, P M, *Lancet*, 1977, 1, 190.

³ Pinching, A J, Peters, D K, and Newson Davis, J, *Lancet*, 1977, 1, 428.

⁴ Nissenson, A R, *New England Journal of Medicine*, 1977, 296, 819.

⁵ Wood, G J, *British Medical Journal*, 1977, 2, 1305.

"Curing" minor illness in general practice

SIR,—I am just a bit doubtful about the complete rightness of Dr G N Marsh's article (12 November, p 1267) and of the letters of Dr M A Gilbert and others in this week's edition of the *BMJ* (10 December, p 1540). My reason is time and it is a conclusion reached after 25 years of general practice (I have recently retired). In my practice there seemed to be two classes of patients: those who were particularly "Welfare-State minded" and who would attend surgery for the most trivial of complaints, and the others—the so-called "good patient"—who loyally stuck to the 10 am rule, seldom asked us out at night or weekends, and in other words did everything they could to help.

I recall one particular case: a young child of one of these so-called "good families" was unwell one morning. Mindful of the suggested rules, no call for a visit was made and the feverish child was put to bed and treated with home remedies. During the day he worsened but still the parents felt that they must not call us out, although we were certainly not a practice which resented evening visits. At long last the parents sent at 4 am, when the doctor on duty went immediately, only to find the child dead of an acute pharyngolaryngo-tracheobronchitis, a pretty heavy penalty to pay for trying to "spare the doctor."

To me an essential of general practice is a knowledge among patients that one of the practice should be available at least to assess the urgency of the visit on the telephone (and far more harm and stress are caused by tearing a worried mother off a strip than at least by listening to her in as kindly, though objective,

a way as possible). She, or whoever was ringing, could well be satisfied with advice or would willingly wrap the child up and bring it round to the surgery, where I always kept a supply of emergency medicines. "Oldie" though I now am, I still feel as strongly about this as ever. I cannot forget the child who lost his life because his parents, out of the goodness of their hearts, hung on too long.

M C PLATTEN

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Powys

SIR,—I read Dr G N Marsh's article (12 November, p 1267) with interest. I do not wish to be drawn into an argument over what constitutes trivial illness but do find his aims of patient education, so that they may become more self-reliant and independent, quite appropriate. However, some of the means to that end are dubious and, in particular, that he tells patients before examining them, "From what you tell me, I don't think I will find anything"—a very dangerous thing to do. That he should be teaching it to his trainees is deplorable. Not only does it irritate, intimidate, and antagonise the patient but also, should the doctor be wrong, causes further embarrassment. Surely this is not part of patient education.

R MOODY

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Cervical herpes zoster and shoulder pain

SIR,—Your expert (17 December, p 1589) discusses the relation between cervical herpes zoster and a subsequent painful shoulder. I have somewhere in my collection an x-ray of a young woman of about 25 whose shoulder showed marked subluxation following herpes zoster; this was shown only when the patient was x-rayed in the erect position, when the head of the humerus was seen to drop significantly.

I was assured that this was due to an associated paresis of the deltoid and that minor muscle (that is, anterior horn) lesions may be found in herpes zoster as well as the characteristic posterior horn lesions. If such lesions are accepted as possibly occurring in herpes this might be the mechanism for the subsequent pain. A follow-up film some months later showed a return to normal. I would be interested to learn what he thinks of this possibility.

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*.*Our expert replies: "I was very interested to read Dr Eban's letter. Deltoid paresis would, of course, make disuse stiffness of the shoulder after herpes zoster a little more likely."—Ed, *BMJ*.

Surgical treatment of hiatus hernia

SIR,—I would like to comment on several surprising omissions from your leading article on this subject (3 December, p 1436). The article is headed "Surgical treatment of hiatus hernia," but in fact is entirely concerned with the symptomatology and treatment of sliding hiatus hernia, no mention being made at all of paraoesophageal hernia, and 10% of hernias through the oesophageal hiatus are