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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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.