chose that drink to avoid the water he knew to be unsafe!

You suggest that water from the hot tap should be safe if a chlorinating tablet is added. In many places in Asia water from the cold tap is safe; Kuala Lumpur is a good example. But I would never advise anyone to drink water from the tap in India. Seventy years ago in Bombay the British laid the main water pipe and the main sewage pipe together. They still remain, though showing signs of corrosion. As the water supply is often intermittent negative pressures build up sufficient to suck in any leaking sewage. One of the responsible physicians in the infectious diseases hospital in Bombay boils his own water for drinking for 20 minutes. He hopes this way to deal with the infectious hepatitis virus, which is prevalent, and which means that not only is hot water not safe but that water-sterilising tablets would give a false sense of security.

When I was responsible for the health of 100 Europeans during a two-year tour of India, the Far East, and Australasia we gave specific rules for each country based on earlier conversations with local experts and examination of certain factories. In India we allowed people to drink tea, coffee, boiled water, Coca Cola, Fanta, Gold Spot, and Limca—and nothing else. Perhaps it is possible to be obsessional. But during the tour we were fortunate in having very few cases of diarrhoea, only one case of amoebic dysentery, and no cases of jaundice. Though I cannot back this up with exact figures it convinced me that while travellers' diarrhoea may be an entity, much that is labelled as travellers' diarrhoea can be avoided with more local knowledge.

J S LESTER

Tarporley,
Cheshire

SIR,—I am sure many of my colleagues will join me in complimenting you on your leading articles over the years. For the busy practitioner who has little time to delve into even a small part of the voluminous medical literature they perform a valuable service.

Although the topics are wide and ranging, there are certain recurring themes, one being affections of the gastrointestinal tract—in particular I refer to your leading article on travellers' diarrhoea (14 August p 385), in which you deal with the study of an outbreak of turista among gastroenterologists attending a conference in Mexico City.¹ You refer to the study of Loewenstein *et al*² in which an equally surprising group, microbiologists, were similarly afflicted at a conference in Mexico City in 1970. Although the main investigation in this latter work was on the country of origin of the sufferers, the other finding, the value of prophylaxis, was identical to that of Merson *et al*.¹

For over eight years I have been observing the behaviour of this type of gastrointestinal upset in the semiclosed community of a boarding school³ and in a university student health service. I would like to make several points: Firstly, it is not unusual for the traveller to suffer an attack of diarrhoea on his return to his homeland. Also, some individuals continue to suffer diarrhoea for weeks after they return home, with relapses precipitated by various stresses, including respiratory infections. Often a relapse may be responsible for new cases among those in the immediate environment. Secondly, the spread

appears to be from person to person with close contact. There is evidence for spread of the infection by aerosolisation of faeces in flushing the lavatory, as suggested by Darlow and Bale.⁴ Finally, the overall pattern suggests that the condition may in some cases be due to persisting infection by a microorganism (virus or bacterium) in which there exists a delicate balance between immunity and the invader. The stress of travel, associated with close contact, could easily disturb this balance.

In 1969-70 there was an epidemic of cold sores⁵ with a high incidence of cases among those in close contact with one another. This suggested that the lesion of a persisting infection such as the cold sore could be produced by infection from without in addition to the usual precipitating factors such as respiratory infections, menstruation, and sunlight.

H G NELSON

Student Health Service,
Trinity College,
Dublin

¹ Merson, M H, *et al*, *New England Journal of Medicine*, 1976, **294**, 1299.

² Loewenstein, M S, Balows, A, and Gangarosa, E J, *Lancet*, 1973, **1**, 529.

³ Nelson, H G, *Journal of the Irish Medical Association*, 1972, **65**, 136.

⁴ Darlow, H M, and Bale, W R, *Lancet*, 1959, **1**, 1196.

⁵ Nelson, H G, *Journal of the Irish Medical Association*, 1975, **68**, 527.

Diazoxide in treatment of hypertension

SIR,—Dr J C Petrie (7 August, p 359) is reluctant to use oral diazoxide in the treatment of severe hypertension for fear of worrying side effects.

Pohl *et al*¹ reported the successful use of oral diazoxide in the treatment of severe toxæmia of pregnancy. Hypertension in all patients was adequately controlled. Live babies were delivered. In patients with hypertension and renal failure there was a highly significant improvement in renal function in most cases on long-term diazoxide therapy.² Side effects were few and were easily controlled.¹⁻³

My patient with uncontrolled malignant hypertension (blood pressure 260/160 mm Hg) sustained encephalopathy, and brain damage. When diazoxide 50 mg was given her blood pressure dropped immediately to 150/75 mm Hg. She is maintained on 50 mg three times a day and continues to improve. Massive doses of apresoline, β -blockers, and methyl-dopa among other powerful drugs have been steadily withdrawn. The mild glycosuria appears to be transient and readily controlled with a suphonylurea. Mild oedema from sodium retention responds rapidly to small doses of spironolactone. I have noticed no extrapyramidal symptoms.

Diazoxide appears to be obligatory in life-threatening hypertension.

KENNETH T FARN

Grendon, Warwick

¹ Pohl, J E F, *et al*, *British Medical Journal*, 1972, **2**, 568.

² Pohl, J E F, and Thurston, H, *British Medical Journal*, 1971, **4**, 142.

³ Neary, D, Thurston, H, and Pohl, J E F, *British Medical Journal*, 1973, **3**, 474.

Dextropropoxyphene poisoning

SIR,—We read with interest the letters of Drs D J L and E D Carson (10 July, p 105) and Dr J A Vale and others (14 August, p 424)

concerning death after overdosage of analgesics containing dextropropoxyphene, in particular Distalgesic. We should like to report a case.

A 21-year-old man ingested an unknown quantity of Distalgesic tablets with alcohol. He was found convulsing in a telephone kiosk from which he had called for help. On arrival in the casualty department he was not breathing and the electrocardiogram revealed asystole. He was intubated and ventilated and external cardiac massage commenced. Following intravenous adrenaline and calcium chloride sinus rhythm was restored, with a satisfactory cardiac output. He subsequently developed complete heart block, which was successfully treated with a temporary endocardial pacemaker. There were still no spontaneous respirations two hours after admission and artificial ventilation was continued. Comprehensive urine and blood analysis revealed only paracetamol and dextropropoxyphene. The paracetamol level on admission was 1.3 mmol/l (196 mg/l) and 10 hours later 0.28 mmol/l (42 mg/l). Naloxone given repeatedly produced no respiratory stimulation. Though his cardiac status returned to normal, both pupils became fixed and dilated and there remained no spontaneous respiratory movements. Subsequently an electroencephalogram revealed no cerebral activity. Five days after ingestion of the Distalgesic tablets artificial respiration was discontinued and his kidneys were successfully used for transplantation.

In our case we believe that the combination of excess of alcohol and dextropropoxyphene produced first respiratory arrest and subsequently cardiac arrest. We were unaware of the potentially severe respiratory depressant effects of dextropropoxyphene so sensibly brought to our attention by the above authors. Though naloxone was unsuccessful in our case, it is likely that there was cerebral death from admission and we do not wish to throw doubt on its effectiveness.

We acknowledge with thanks the efforts of our biochemists and intensive care nursing staff and Dr Peter Crome, who advised us.

T J GRAY

G S BREAR

PENNY SCHOFIELD

T A D PIERSCIANOWSKI

GRAHAM JACKSON

Brook General Hospital,
London SE18

The Framingham study

SIR,—According to Dr J C Petrie (31 July, p 289), the Framingham study is "concerned with the natural history of a population with a wide range of blood pressures." In fact, however, as the director of the study has freely acknowledged in a personal communication, many of the participants receive unspecified treatment from their own physicians, who are informed of the pressure findings and are "at liberty" to use hypotensive agents. It cannot, therefore, be said that the study illustrates the natural history of hypertension.

I MCD G STEWART

Victoria Hospital,
Blackpool

Thyrotoxic vomiting

SIR,—Dr F D Rosenthal and his colleagues (24 July, p 209), in describing seven patients with thyrotoxicosis in whom vomiting was a prominent clinical feature, make no mention of serum calcium levels in the case histories or the discussion.

Some years ago I encountered within a few