THERAPY FOR DIARRHŒA

Sir,—I refer to your leader of Jan. 11 on oral glucose/electrolyte therapy for acute diarrhoea. In this country we have summer epidemics of severe diarrhoea accompanied by vomiting. I have found that dehydration can be avoided, and vomiting prevented, in many instances, by giving the patients teaspoons (as you advise) of iced water, with the ice cubes still in the glass. Where even this is not tolerated, one tablespoonful of brandy or whisky to a glass (200–300 ml) of iced water has prevented vomiting in all cases I have seen so far. I shall in future add the electrolytes and glucose you recommend.

P.B. 8130, Jerusalem 91080, Israel.

A. YUVAL.

MORTALITY AND WATER HARDNESS

Sir,—We hope that the negative results obtained by Dr Meyers (Feb. 15, p. 398) do not discourage other investigators from pursuing the hypothesis that the composition of drinking-water may influence mortality from ischaemic heart-disease (I.H.D.).

As Dr Meyers has himself pointed out, when dealing with a multifactorial disease such as I.H.D. it is difficult to find populations that differ in only one variable, and it is possible that in some situations the effect of the "water-factor" (assuming it exists) is hidden by differences in other, more potent, risk-factors.

It is also possible that the importance of the water-factor (or factors) will differ from one area to another because of interaction with other variables. Thus if a deficient intake of magnesium leads to a higher I.H.D. death-rate, the beneficial effect of water-borne magnesium could only be expected to show up in those populations whose basic dietary intake of magnesium was less than adequate. Similarly, if toxic metals such as lead or cadmium were responsible for increasing the I.H.D. death-rate, the beneficial effect of water-borne magnesium in some soft-water areas, a high dietary intake of protective substances such as calcium or zinc might, in other areas, obscure the effect.

From the practical point of view it would be tragic if, for lack of trying, we failed to identify a water-factor that did in fact exist, since there is a reasonable chance that, once this was identified, a safe and generally acceptable modification of water (or food) could be used as a preventive measure. This would compare favourably with our present state of frustration over risk-factors such as smoking and physical inactivity which, although well defined, are very difficult to modify.

Department of Epidemiology and Biometrics,
School of Hygiene,
University of Toronto,
Toronto, Canada M5S 1A1.

TERENCE W. ANDERSON
DAVID HEWITT.

THE OPEN UNIVERSITY AND MEDICAL STUDIES

Sir,—It is to be hoped that the " other quarters " from which Sir Walter Perry (March 1, p. 518) will wait for overtures will include not only the U.G.C. but also the D.H.S.S.—that is, if the Department seriously wishes to remedy the geographical maldistribution of British medical graduates. The absurd expedient of redistribution of registrars is the failure it was bound to be. Cannot someone in the D.H.S.S. now show the sort of imagination that the Open University should not at least be attempted.

Medical School,
43 Woodstock Road,
Oxford.

JOHN POTTER.

DOES T4 TOXICOSIS EXIST?

Sir,—We were very interested in the letter by Dr Turner and his colleagues (Feb. 15, p. 407) on the existence of T4 toxicosis. During the past four months we have treated a 70-year-old woman for thyrotoxicosis.

She was admitted with a transient left-sided hemiparesis and atrial fibrillation. Serum-T4 was 15.8 µg per 100 ml (normal 5.5–12.5), free-T4 index 719 (150–371), and serum-triodothyronine (T3) 119 ng per 100 ml (37–153). Serum-cholesterol was 158 mg per 100 ml (160–350). The 4-hour T3 uptake in the thyroid gland was 36% (10–30%) and the 24-hour uptake 67% (20–60%). The thyroid scan showed a multinodular goitre. Liver-function tests were normal. She was treated with propylthiouracil (300 mg daily), and 14 days later serum-T4 was 9.0 µg per 100 ml, free-T4 index 418, and serum T3 27 ng per 100 ml. Three months later, on treatment with propylthiouracil (100 mg daily), she was discharged euthyroid: serum-T4 7.6 µg per 100 ml, free-T4 index 210, and serum-T3 72 ng per 100 ml. Serum-thyrotropin (T.S.H.) was < 1·0 µu per ml (normal < 4.9 µu per ml).

The sera have been reassayed for serum T4 and T3, with similar results, and the values given are means of the two determinations. In order to exclude a non-specific influence of the sera on the T4 radioimmunoassay, dilution and recovery experiments were performed. No such influence could be demonstrated, since the curves could be superposed on the standard curve.

Our data suggest that thyrotoxicosis with serum-T4 within 95% normal range and serum-T3 exceeding 95% normal range exists and requires treatment. Our findings probably reflect an extreme biological variation in the T4/T3 ratio; or they may be a result of diminished extra-thyroidal conversion of T4 to T3, though this patient did not have a severe chronic illness.

C. KIRKGAARD
K. STERSBAEK-NIELSEN
TH. FRIIS
P. ROGOWSKI.

T.R.H. TEST IN SUBACUTE THYROIDITIS

Sir,—Dr Papapetrou and Dr Jackson (Feb. 15, p. 361) reported 3 cases of " silent " thyrotoxicosis with hyperthyroidism. They emphasised the importance of thyroidal uptake of 131I as a routine diagnostic aid in thyroid disease, despite the newer diagnostic tools for thyrotoxicosis, such as T1 and free T4 and T3 levels. I agree with their statement, but I am surprised that the thyrotrophin-releasing hormone (T.R.H.) test was not mentioned. The T.R.H. test is the most sensitive method of detecting all forms of hyperthyroidism, even in the preclinical stage.1