a convoluted pattern on dissecting microscope examination. On section there was a dense inflammatory infiltration of plasma cells and lymphocytes in the lamina propria and the surface cells of the villi were low columnar with a poor brush border towards the tips of the villi. The villous length to crypt depth ratio was approximately equal.

During the last year, during which she has had a further pregnancy, this patient has remained well on a normal diet. Further investigations show: haemoglobin 14.0 g/100 ml, red cells normal; serum folate 8.0 ng/100 ml; faecal fat excretion 3.3 g/24 hr. Xylose excretion was again low at 1.4 g. in 5 hrs. A tritium labelled folic acid (H^3FA) absorption test was carried out using an oral dose of 40 μ g/kg. body weight of H^3FA . Thirty-two per cent was excreted in the urine which is within normal limits.² A second small intestinal biopsy (Fig. 2) showed a mixed pattern of leaves and ridges under the dissecting microscope. The villous height to crypt depth ratio was 2 to 1. The villi showed a normal tall columnar surface epithelium with a normal brush border. The lamina propria contained scanty plasma cells and some lymphocytes.

Though there is clearly an association between folate deficiency and oral contraceptive therapy we feel that this case raises a number of problems in the assessment of such patients. Streiff has clearly shown that in healthy women on oral contraceptive therapy there is a defect of absorption of dietary polyglutamates but no defect in absorption of monoglutamate.³ He has postu-

lated an inhibition of folate conjugase activity in the hydrolysis of dietary polyglutamate to more readily absorbable monoglutamate forms. However, small intestinal biopsy appearances in his patients who became anaemic were not described, and it is possible that these patients could have had occult coeliac syndromes in which folate deficiency was precipitated by contraceptives. The failure of complete reversal to normality of the intestinal mucosa in our case and the continued abnormality of xylose excretion point to this possibility. In addition, the direct effect of the drug on the small intestinal mucosa cannot be discounted as there was definite improvement after stopping contraceptives and prolonged treatment with folic acid. Certainly folate deficiency per se as in nutritional or puerperal states has not been associated with mucosal changes.^{4 5} Other reports suggest that folate deficiency in women on oral contraceptives is by no means uncommon, and further descriptions of the small intestinal biopsy appearances would be of interest.—We are, etc.

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Dehydration and Fat Babies

SIR,—Your recent leading article (16 January, p. 125) on the dangers of hypernatraemic dehydration in obese infants prompts us to draw attention to a related topic. It was shown five years ago in the United States that once weaning started the American baby was likely to have his sodium intake doubled and soon trebled, as he gulped down increasing amounts of the proprietary strained foods so eagerly proffered him by his mother.¹ This seems to have evoked little attention in Britain, yet our estimates of the sodium and chloride content of three generally available and popular brands of similar food on sale in our shops make it unlikely that things are different here. The figures are as follows:

		Na ⁺ Cl ⁻ (mEq)	
		Na ⁺	Cl ⁻
Brand A 4½ oz (120g) tin ..	Beef broth	12	14
	Chicken and vegetable broth	11	12
Brand B Powder equivalent to 4½ oz (120g) tin ..	Vegetable, chicken, and tomato	21	18
Brand C 4½ oz (120g) jar ..	Vegetable and chicken	18	18

The majority of infants are now artificially fed (cow's milk containing nearly three times as much sodium chloride as human milk) and it is not at all unusual to

find solids added to the diet within a few weeks of birth.² During the second six months of life, if not earlier, an infant may consume more than one of the above brand tins, jars, or packs daily in addition to his cow's milk which alone supplies him with more than enough sodium and chloride for his needs.

Young rats, of a strain known to develop hypertension, did so earlier than controls when they were given a high salt intake (American infants' weaning diet) at weaning.³ Hypertension had earlier been shown to persist in similar rats after an excessive salt intake was stopped.⁴ Though there is no certainty as to whether there is a relationship between excessive salt intake and essential hypertension in humans,⁵ population studies attempting to answer this question have considered only the adult salt intake. We have reason to suppose that obesity in adult life is sometimes related to obesity—and by implication overfeeding—in infancy.

Perhaps we should be looking much more critically at feeding practices in the first year of the first decade where hypertension as well as obesity are concerned. It is often difficult to change established feeding patterns later, but Fomon⁶ has shown that young infants will readily accept unsalted weaning foods, which may be unpalatable to their mothers.—We are, etc.,

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Renal Tubular Necrosis after Gastroenteritis in Infants

SIR,—Dr. A. R. Chrispin and others (14 February 1970, p. 410) report that in the case of three infants who had gastroenteritis accompanied by severe dehydration the presence of renal tubular necrosis and renal insufficiency could be demonstrated. Having described the characteristic features of excretion urographic studies they recommend a method suitable in practice for the demonstration of change. The specific nature of the radiological differences was confirmed in experimental nephropathic studies in a more recent paper.¹ It was stressed that the cases would not have been recognized merely from clinical symptoms, and it was suggested that renal tubular damage is a relatively frequent complication in shock conditions in infants.

We should like to draw attention to our own observations, which provide a further basis for the supposition that renal tubular damage in the above diagnosis is more frequent than previously thought. In addition, our data suggest that the renal damage is initiated by urate nephropathy. In our first observations² we found that in severe

gastroenteritis and other toxic conditions in infants there was extreme hyperuricaemia—20 mg/100 ml was often recorded. With such levels precipitation of urate in the kidney had to be considered.

To investigate this possibility experimental production of the syndrome was effected in rats by blocking the uricase activity with oxonic acid.³ By application of sublethal tourniquet shock in these animals we regularly found hyperuricaemia and massive uric acid precipitation in the renal tubules.⁴

It is possible that "shock" kidney is provoked by this mechanism, and the particular purine metabolism of man may also be the explanation of his special proneness

to kidney damage in shock.—We are, etc.,
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Gold Excretion in Rheumatoid Arthritis

SIR,—Gold therapy is of proved merit in rheumatoid arthritis.¹⁻⁴ Smith *et al.*⁴ suggested that patients may be classified as normal, hypoeccretors, and hyperexcretors of injected gold. Whereas the hypoeccretor rapidly accumulates gold in the body leading to early toxicity, the hyperexcretor never achieves the therapeutic level. It is thus necessary to adjust therapeutic dosage to rates of excretion. We are at present engaged in investigating the biological half-life of injected gold salts in patients with rheumatoid arthritis. As a first step we have

measured urinary gold output by means of activation analysis.

Forty-eight patients were studied. In 18 patients 24-hour urine collections were made for seven days following an injection of sodium aurothiomalate (Myocrisin) 50 mg. In the other 30 patients a 24-hour urine collection was made during either the second, third, fourth, fifth, or sixth day after injection, selected at random. Aliquots of urine samples and of standard gold solution were irradiated for five minutes in a thermal neutron flux of 1.3×10^{13} neut-

rons/cm²/second in the DIDO reactor (UKAEA, Harwell). After allowing the ²⁴Na to decay to an acceptable level, the ¹⁹⁸Au radioactivity was measured by gamma ray spectrometry.

The seven-day excretion studies (18 patients) showed a remarkably flat curve (Fig. 1). In only one patient was a high excretion rate observed (Fig. 2). In this patient most of the injected gold was excreted within the first week, and probably a therapeutic level could not be achieved on conventional dosage. Apart from this patient, the maximum daily excretion of gold during a seven-day period of observation (Fig. 3), the mean daily excretion rate during the second to sixth day following injection, and the daily excretion rate of random 24-hour samples during the same period (Fig. 4) all showed variation within a relatively narrow range only.

We conclude that following an injection of sodium aurothiomalate for rheumatoid arthritis in most patients the gold is excreted slowly and fairly uniformly. In our experience, the type of hyperexcretion envisaged by Smith *et al.*⁴ is a rare occurrence.

We thank the East Anglian Regional Hospital Board who sponsored this research, and Messrs. May and Baker who contributed to the cost of the activation analysis.

—We are, etc.,

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Figure 1
Mean daily urinary gold excretion (+ or - S.D.) during seven days after injection of Myocrisin 50 mg.

Figure 2
Daily urinary gold excretion during seven days after injection of Myocrisin 50 mg in hyperexcretor.

Figure 3
Frequency distribution of maximum excretion.

Figure 4
Frequency distribution for urinary gold excretion in random and mean 24-hour samples.

Vaginitis and Tights

SIR,—I have been rather surprised by the considerable increase in vaginal and vulval infections in my practice over the past two or three years. It has struck me that this increase coincides to some extent with the present fashion of wearing nylon tights instead of stockings.

I would be interested to know whether any other doctors have noticed this possible correlation.—I am, etc.,

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Points from Letters

Tired Doctors

DR. S. STOWE (Leeds, York.) writes: Your article on jet pilots (26 December, p. 759) has provoked me to ask various questions about our profession. . . . Is there any doctor who can say he has never made a mistake due to fatigue? Must we have articles on jet pilots, however correct their content is, before any work is done on the problem of fatigue in doctors when their hours of work belittle the 33 hours mentioned in relation to jet pilots? They say that planes have automatic pilots and landing devices to help the pilot. But what does the young doctor get? And just as important, does not the patient deserve something better than a fatigued attendant? . . .