Whether Drs Brooks and Harrington's patient developed malignancy after the diagnosis of coeliac disease was made and some clinical improvement was obtained after gluten withdrawal or whether it was present initially, precipitating overt manifestations of gluten intolerance, it is not possible to say.

It is certainly believed that failure of a patient with newly diagnosed coeliac disease to respond to gluten withdrawal or relapse occurring after previous control by diet is suggestive of the development of malignancy.⁶ The view that intestinal malignancy may cause flattening of the jejunal mucosa and present as "the coeliac syndrome" is no longer held.⁷

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- ¹ Harris, O D, Cooke, W T, and Thompson, H. *American Journal of Medicine*, 1967, **42**, 899.
- ² Holmes, G K T, et al, *Gut*, 1974, **15**, 339P.
- ³ Holmes, G K T, et al, *Gut*, 1976, **17**, 612.
- ⁴ Creamer, B, and Hellier, M D, in *The Small Intestine*, ed B Creamer, London: Heinemann, 1974.
- ⁵ Creamer, B, in *The Small Intestine*, ed B Creamer, London: Heinemann, 1974.
- ⁶ Stokes, P L, and Holmes, G K T, in *Clinics in Gastroenterology*, vol 3, No 1, ed W T Cooke and P Asquith, Philadelphia, Saunders, 1974.
- ⁷ Austad, W I, et al, *American Journal of Digestive Diseases*, 1967, **12**, 475.

Effects of ethanol-induced hypoglycaemia

SIR,—We were most interested in the paper by Dr B I Joffe and others (10 September, p 678) which emphasises that plasma insulin levels are not completely suppressed in ethanol-induced hypoglycaemia (EIH). We have reported a similar finding in a 7-year-old boy with EIH.¹ Furthermore, other additional glucoregulatory mechanisms should be considered. In our report we described a delayed pituitary-adrenal activity in the pathophysiology of EIH. If this is confirmed in further studies it could be of interest in the treatment of patients with EIH. We suggest that plasma growth hormone and cortisol levels should be included in studies on the mechanism of EIH.

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- ¹ Rubiés-Prat, J, et al, *Annales d'Endocrinologie*, 1975, **36**, 223.

1- α -Hydroxycholecalciferol for renal osteodystrophy

SIR,—Dr A M Brownjohn and his colleagues have provided a great deal of useful information in their paper (17 September, p 721), which also confirms our two-year experience of 1- α -hydroxycholecalciferol for renal osteodystrophy.

We have used 1- α -hydroxycholecalciferol in doses ranging from 1 μ g to 2 μ g daily in 16 patients on regular dialysis treatment (10 female and 6 male) and would like to add that in addition to pruritus "red eyes" is a common feature of severe hypercalcaemia (> 3 mmol/l (> 12 mg/100 ml)). Our four patients developed a varying degree of conjunctival injection and dilatation of blood vessels. Though these patients complained of sore eyes, crystal deposits were not visible and slit-lamp

examination was not performed. This condition was reversible on temporary cessation of therapy, reduction in dosage, or both.

We concur in that no ectopic calcification was observed, but prolonged severe hypercalcaemia should be avoided and we endorse the need for careful monitoring not only of plasma calcium but also of plasma inorganic phosphorus and its meticulous titration with phosphate binders.

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Return to work after aortofemoral bypass surgery

SIR,—May I congratulate Mr K J Waters and his colleagues on their short report on this subject (27 August, p 556)? The ability of patients to take up former work or to return to modified or alternative work is something which is very often forgotten when clinical follow-up studies are designed. It is my belief that this ability is an extremely important measure of outcome, particularly when any kind of treatment, medical or surgical, is being evaluated.

On behalf of the Employment Medical Advisory Service and in co-operation with different clinical departments I am at present engaged in some similar studies detailing aspects of present and past "work history" in different physical conditions with special reference to advances in treatment. I would like to make use of your correspondence columns to urge any physician or surgeon undertaking long-term follow-up studies to include occupational status among the criteria of outcome examined. Such data, which are rarely specifically sought, would be of the greatest value not only to our own organisation but surely to clinicians whose responsibility to their patients should include informed advice on future return to work.

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Depot tranquillisers for disturbed behaviour

SIR,—The treatment of elderly people showing "antisocial behaviour" is often difficult. It is vital to exclude possible remediable factors which might be contributing to confused behaviour, both of a physical nature (for example, bronchopneumonia) or of a psychiatric nature (for example, depression). There are, however, many elderly people whose behaviour is so disturbing to relatives and neighbours and to other residents in homes and other patients in hospitals that some form of psychotropic medication seems indicated. This medication should primarily be used to ameliorate or abolish the patient's symptoms for their own benefit, but this primary obligation often blurs into a response to demands to control the patient's behaviour as a general social need and minimise pressures on other people with whom they are in contact.

Long-acting tranquillisers are occasionally useful in such patients. While they have the major disadvantage that once given side effects may develop inexorably, they may sometimes produce the very rewarding situation described by Dr H G Kinnell (27 August, p 578) in

younger patients. The elderly person becomes much calmer and happier and may not need any additional medication until the next injection of fluphenazine decanoate is required. Although this drug has to be given by injection, the patient and attendants are often very pleased that no further medication has to be given by mouth or injection for some considerable time. Our usual experience has been that doses of the order of 12.5 mg fortnightly are adequate and very rarely cause side effects.

I would reiterate the need to choose psychotropic drugs very carefully in the elderly confused patient but record that phenothiazines are sometimes indicated despite their side effects and that depot injection fluphenazine may sometimes be a very useful alternative to the more usual phenothiazines.

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Bruising in thyrotoxicosis

SIR,—In reply to Dr E N Wardle (24 September, p 828), clinically significant bruising is uncommon in thyrotoxicosis, although the finding of a positive Hess test is quite common. Studies¹ have shown that capillary fragility is increased in thyrotoxicosis and decreased in hypothyroidism. The platelet count is usually not affected.

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- ¹ Thomson, J A, *Clinical Science*, 1964, **26**, 55.

Complications of transvenous (transjugular) liver biopsy

SIR,—We were interested to read the account by Dr I T Gilmore and his colleagues of their experience of transjugular liver biopsy (9 July, p 100). At the present time we have performed 335 transvenous (transjugular) liver biopsies; our early experience, based on our first 100 transvenous liver biopsies, has been reported in a paper to be published shortly in the *American Journal of Digestive Diseases*.

The only serious complication directly related to the procedure that we observed in our series was a fatal intraperitoneal haemorrhage due to perforation of the liver capsule; perforation was due to excessive forward rotation applied to the needle. The reason for this manoeuvre was that the needle advancing parallel to the lumen of the vessel; therefore forward rotation, by placing the tip of the catheter against the venous wall, placed the needle obliquely, which favoured penetration into the liver parenchyma. This manoeuvre must be avoided or limited to only a slight forward rotation applied to the needle. In three other patients we have indirect evidence that the liver capsule was perforated (aspiration of ascitic fluid, aspiration of bile from the gall bladder, presence of renal tissue in the biopsy specimen), but no clinical complication resulted from the perforation; in these three patients the size of the liver was small.

These observations suggest that in patients with a small liver transvenous liver biopsy must be avoided or employed with caution—namely,

by advancing the needle into the liver parenchyma by only 1 cm instead of the usual 2 cm.

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SI units and acidity

SIR,—Dr P J Tomlin's letter (24 September, p 833) illustrates very well the difficulties which arise when doctors are unaware of the real nature of the data they use and apply inappropriate arithmetical procedures to them. He correctly points out the difficulty in using means and standard deviations to indicate the normal range when the true mean is unknown and the frequency distribution is not Gaussian. But in such circumstances it is surely impossible to make any rational decision on the basis of such data. To put it bluntly, how can one decide what is abnormal when one does not know what is normal?

To ask clinicians to remember all the details of every test they use is to impose an absurd and unnecessary burden on them. For them to use constantly a book of tables is little better. The same information is available perfectly simply by establishing the means and the nature of the frequency distribution, changing the latter to Gaussian form so that the standard deviation now has some real meaning, and then giving the results in standard units of T-score form. This would be additional to the actual values of the measurements. I have advocated this procedure for many years on the grounds that such a single unit, which would apply to all tests, present and future, would make for a tremendous simplification of the clinician's task.

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Modified jejunoileal bypass for obesity

SIR,—We agree with the findings of the report by Dr J D Maxwell and others (17 September, p 726) on the modified jejunoileal bypass for morbid obesity and would like to make the following comments.

There is still considerable confusion regarding the extent of small-bowel resection or bypass which will result in serious intestinal decompensation. Estimates of the mean length of small intestine removed from its mesentery at necropsy have varied from 300 to 990 cm and have often been used in formulating statements on the effects of small-bowel excision in the human. These post-mortem estimates do not reflect the length of the small intestine in vivo, and as most clinical observations should relate to the length of the small intestine measured at operation it is surprising that this information is so scanty and confused. Indeed, accurate measurements of small-intestine length at operation are difficult to obtain because of the changing state of the coils of small bowel consequent on peristaltic activity, handling, and exposure. In 30 adult patients requiring elective abdominal surgery we have measured the small-bowel length from the duodenojejunal flexure to the ileocaecal valve after a preliminary exploratory laparotomy and

obtained a mean length of 298 cm with a standard deviation of 30 cm. If this is a reliable estimate of small-bowel length, then the average length of jejunum and ileum in the adult human should average 120 cm and 180 cm respectively as measured at the time of surgery.

These observations stress the real need to avoid, whenever possible, ileal excisions or bypass in excess of 150-160 cm. There must indeed be a critical length of residual ileum below which gross decompensation occurs, and a review of the literature clearly shows that the majority of patients who have died after intestinal bypass for morbid obesity have been those patients in whom less than 20 cm of terminal ileum was retained for absorptive purposes.

The procedure recommended by Dr Maxwell and his colleagues of increasing the amount of residual ileum at the expense of the jejunum in bypass procedures for morbid obesity has also been advocated by Scott¹ and is the most sensible contribution that has been made on the subject. It is certainly corroborated by our own experience.

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¹ Scott, H W. *Journal of the Royal College of Surgeons of Edinburgh*, 1977, **22**, 241.

Oxytocin induction and neonatal hyperbilirubinaemia

SIR,—The report by Mr W C Chew (10 September, p 679) has prompted us to communicate our preliminary observations regarding the pathogenetic role of oxytocin infusion, given for induction or augmentation of labour, in neonatal hyperbilirubinaemia.

The association between maternal infusion of oxytocin during labour and neonatal hyperbilirubinaemia is generally accepted.¹⁻³ However, the exact mechanism of production of hyperbilirubinaemia by oxytocin is still a matter of speculation. It is conceivable that the oxytocin may cause water retention owing to its antidiuretic-hormone-like effect, resulting in hyponatremia and hyposmolality in the maternal blood. These alterations would be reflected in the fetal blood.⁴ The hyposmolality in the newborn would result in swelling of the red blood cells, thus making them more fragile and vulnerable to haemolysis. The jaundice may therefore appear earlier and serum bilirubin levels are likely to exceed physiological limits in these neonates.

We have attempted to test the above hypothesis by undertaking a prospective evaluation of 49 full-term, vertex-presenting, vaginally delivered newborn babies with feto-maternal blood group compatibility. Seventeen infants were delivered after oxytocin infusion and 32 were born spontaneously. Cord blood was collected for estimation of serum osmolality (by Fiske osmometer) and sodium concentration. The age at onset of jaundice and serum bilirubin at the age of 4 days $\pm$ 12 h was assessed. Serum osmolality and sodium concentration were significantly lower in the babies delivered after oxytocin infusion than in the control group (see table). Jaundice appeared within 24 h of birth in four neonates belonging to the oxytocin group, the serum bilirubin concentration exceeding 170 μ mol/l (10 mg/100 ml) in two of these. None of the

Serum osmolality and sodium concentration in infants born spontaneously and after oxytocin infusion

	Serum osmolality (mmol/l)	Sodium (mmol/l)
Control group (32)	306.14 $\pm$ 5.16	136.46 $\pm$ 5.08
Oxytocin group (17)	290.10 $\pm$ 3.60	127.95 $\pm$ 5.98

* $t = 12.70$, $P < 0.001$.

$\dagger t = 5.00$, $P < 0.001$.

babies in the control group manifested early jaundice or hyperbilirubinaemia.

These initial observations suggest that the proposed hypothesis is tenable, though it is desirable to enlarge the series and include evaluation of the osmotic fragility of the red blood cells to lend additional support.

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¹ Chalmers, I, Campbell, H, and Turnbull, A L. *British Medical Journal*, 1975, **2**, 116.

² Chew, W C, and Swann, L L. *British Medical Journal*, 1977, **1**, 72.

³ Oski, F A. *American Journal of Diseases of Children*, 1975, **129**, 130.

⁴ Oh, W. *Pediatric Clinics of North America*, 1976, **23**, 601.

Acceptable and unacceptable risks

SIR,—Drs J M Evans and J A M Keogh (17 September, p 735) rightly conclude that an intravenous anaesthetic induction agent which had some advantages over others was nevertheless unacceptable because the incidence of adverse reactions was 1 in 1000.

In the same issue (p 738) Dr Barbara Bannister and others report a case of severe hypokalaemia and cardiac arrest after the consumption of about 1.8 kg of liquorice sweets per week, corresponding to a daily intake of about 250 g. Most people who appreciate sweets of all sorts would agree that 60-120 g (about 2-4 ounces) is a reasonable and not too greedy feast. Thus about twice the higher normal dose had caused very severe ill effects after an unspecified time. This was predictable. Life-threatening toxicity has been reported before¹ and there has been a warning about serious adverse reactions after eating modest amounts for only one week.² Liquorice is no longer recommended in pharmaceutical preparations.¹ It is doubtful whether in Britain any pharmacist would sell or any pharmaceutical manufacturer would make anything so toxic as liquorice sweets that is so good to eat.

The same issue also contains a report (p 726) by Dr J D Maxwell and others on the results of 120 jejunoileal bypass operations for obesity which caused substantial weight loss but were followed by five deaths as well as clinically and statistically significant liver damage lasting 6-12 months in all the 35 patients whose liver function was studied. The authors state that "most patients . . . were satisfied with the outcome," though "at the expense of a certain morbidity and mortality." One is not told how satisfaction was assessed, how many is "most," or what were the views of the three patients who died slowly of liver failure.

It seems probable that any trial of a new drug in a chronic and not too severe condition such as obesity would include control data from comparable untreated or differently treated patients and that liver damage over