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