bypass operation than after resection,[2 3] the rates after five years following the first operation are similar.[3] The bypassed bowel is being resected at a second operation, converting the bypassed person into a resected person who then carries the recurrence "risk" of a resected patient. The "bypassed" recurrences thus roughly equal the "resected" recurrences plus one for each patient.

Moreover, it is only after the second operation that bypassed patients develop "recurrent" disease proximal to the site of the initial disease.[3] This, together with Oberhelman's observations[4] in relation to colonic disease, suggests that as long as a fertile soil remains, even in disconnected bowel, the disease does not macroscopically "spread" proximally. These observations must have a bearing on at least the perpetuation of the disease if not on its pathogenesis.

I am in no way advocating a return to a primary bypass procedure and its attendant risks but rather drawing attention to the greater information that may be derived from the available data.

J F FIELDING

Jervis Street Hospital,
Dublin

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[2] Homan, W R, and Dineen, P, *Annals of Surgery*, 1978, **187**, 530.
[3] Alexander-Williams, J, Fielding, J F, and Cooke, W T, *Gut*, 1972, **13**, 973.
[4] Oberhelman, H A, in *The Management of Crohn's Disease*, ed I T Weterman, A S Pena, and C C Booth. Amsterdam, Excerpta Medica, 1976.

Antenatal diagnosis of fetal duodenal atresia by ultrasonic scan

SIR,—Fetal ultrasound examination has been of substantial benefit, but the short report by Dr H Gee and Mr U Abdulla (4 November, p 1265) leaves me puzzled. They conclude, "Our case illustrates the importance of ultrasonic examination of the fetal abdomen." The diagnosis was confirmed only 12 hours after birth by an *x*-ray and no treatment was given. Also baffling is the suggestion that amniocentesis to look for Down's syndrome should be considered after ultrasonography has suggested the possibility of duodenal atresia. What action is to be recommended on the results at that late stage of pregnancy?

Would it not be better for energies and resources to be directed to expert neonatal care? Duodenal atresia would be diagnosed 12 hours after birth (as in this case) in the infant born after a pregnancy complicated by hydramnios, and many other anomalies would be diagnosed earlier and practical benefit would accrue.

D G YOUNG

University Department of Paediatric Surgery,
Royal Hospital for Sick Children,
Glasgow

Thyrotoxic Graves's disease after primary hypothyroidism

SIR,—We were interested to see the report of spontaneous change from primary hypothyroidism to thyrotoxic Graves's disease described by Dr S Olczak and others (2 September, p 666). We have been particularly interested in spontaneous change in thyroid function seen in patients with dysthyroid eye disease and have described five such cases.[1] Two of these were initially hypothyroid and subsequently became thyrotoxic.

Case 1—A 62-year-old man presented in January 1974 with an 18-month history of right proptosis. Clinically he was marginally hypothyroid with a low plasma thyroxine (T4) concentration of 54·0 nmol/l (4·2 μg/100 ml) (normal range 51·5-108 nmol/l (4·0-8·4 μg/100 ml)) and a low radioiodine neck uptake (9·4% at 4 h (normal range 15-35%)). By September 1974 he was nervous and irritable and the plasma triiodothyronine (T3) assays were 3·5 nmol/l (2·3 ng/ml) and 2·98 nmol/l (1·94 ng/ml) (normal range 12·3-24·6 nmol/l (8-16 ng/ml)), with a plasma T4 concentration of 95·2 nmol/l (7·4 μg 1/100 ml).

Case 2—A 61-year-old man developed proptosis in 1972, affecting the right eye more than the left. In August 1974 he was first seen and had oedema of the right disc. At this time he was clinically hypothyroid with a plasma T4 concentration of 30·9 nmol/l (2·4 μg/100 ml) and a free thyroxine index of 1·9 (normal range 3·6-8·9). Two months later he was clinically thyrotoxic; the plasma T4 concentration was 123 nmol/l (9·6 μg/100 ml), the free thyroxine index 10·1, and ^{99m}Tc neck uptake at 20 min 4·5% (normal range 0·7-3·0%). He was treated with ^{131}I.

Neither of the above patients was given thyroxine as medication before developing thyrotoxicosis.

A J COAKLEY

Addenbrooke's Hospital,
Cambridge

D N CROFT

St Thomas's Hospital,
London

[1] Croft, D N, *et al*, in *Scientific Foundations of Ophthalmology*, p 135. London, Heinemann, 1977.

Dexamethasone in acute stroke

SIR,—We read with interest the article on acute stroke and dexamethasone by Dr Graham Mulley and others (7 October, p 994). We have carried out a similar but smaller, previously unreported study. We looked at the effect of dexamethasone and mannitol upon the mortality of acute stroke. Thirty-six patients with an acute stroke were randomly allocated to a treatment or a placebo group. The active treatment group received dexamethasone 4 mg intravenously 8-hourly and 200 ml of 10% mannitol intravenously once a day for five days after admission. The exclusions from the trial and investigations were similar to those of Dr Mulley and his colleagues.

The overall mortality at three months was 45%. There was no significant difference in the mortality of the two groups (χ^2, $P > 0.05$), which lends support to Dr Mulley's results.

We have analysed our data further as we wished to ascertain whether the use of oedema-reducing agents ought to be more selective. We therefore divided each group into those who were hypertensive before or on admission ($n = 18$) and those who were normotensive (diastolic blood pressure < 110 mg Hg) ($n = 18$). Patients who are normotensive are more liable to have had a large infarct, whereas those who are hypertensive are more liable to have had a haemorrhage or a small, deep infarct associated with the Charcot Bouchard aneurysm. We found no significant difference between the effects of treatment and placebo on the mortality of the normotensive group ($P > 0.05$). However, we found that the hypertensives who received active treatment had significantly more deaths than those in the placebo group ($0.05 > P > 0.01$). Thus it would appear that dexamethasone and mannitol have little effect and perhaps even a detrimental effect on mortality in the hypertensive group.

As the study was carried out within a district general hospital with no immediate access to computerised tomography or echoencephalography to aid distinction between haemorrhage and infarct we feel that our findings endorse Dr Mulley's statement that there is no indication for the routine use of dexamethasone or mannitol in acute stroke.

JAN FREEMAN
J TAPPIN
A B A KARAT
JOHN MEECHAM

Birkenhead General Hospital,
Birkenhead, Merseyside

Iodine and acetone-containing plastic spray dressings

SIR,—The report by Dr J O Morgan-Hughes and Mr R A Bray (26 August, p 639) of severe erythema and blistering resulting from the use of Op-Site and Sleek dressings on skin treated with iodine tincture was very puzzling. The only compounds that might be expected to react together are iodine and acetone to form iodoform. This, however, requires the presence of an alkali and would not be expected to produce the severe effect experienced.

Investigation in this laboratory showed that an intermediate is formed when acetone and iodine are warmed without alkali. This compound, triiodoacetone, was not isolated but would certainly be extremely irritant and lacrymatory since related compounds are used as chemical warfare agents. Formation of this compound, even in minute quantities, on the skin and its retention under an impervious layer would without doubt produce the results observed.

Only two clear plastic dressing sprays are currently available for human use: these are Op-site and Nobecutane. They both use the same acrylic resin, but the latter does not contain acetone. Our investigation indicates that it is the reaction of acetone with iodine that produces erythema and blistering, and the use of sprays containing acetone on skin sites previously treated with iodine tincture is not to be recommended.

E POWELL

Pharmaceutical Department,
Sefton General Hospital,
Liverpool

Communication with Asian diabetics

SIR,—I would like to take issue with Dr B A Leatherdale and his colleagues (28 October, p 1198), who conclude that some Asian patients—in fact 77% in their survey—lay false claim to literacy. The conclusion is based on a novel and hitherto untested way of proving literacy—that of observing multilingual hospital notices and signs.

I must now confess to public embarrassment that, although an occasional visitor to Dudley Road Hospital, I cannot recall the subject of even one sign in any Asian language. But there lies a cautionary tale. Most of those Asian diabetic patients accused of falsely claiming literacy may indeed be highly literate in reading and interpreting ancient and difficult religious scripts—the only familiar way they have learnt to use their literacy—and yet fail to comprehend the use