distension undoubtedly allowed a clear demarcation between normal and dilated colon.

The presence of tenderness in the left lumbar region followed by haematuria on the evening of admission suggests that the left kidney was ruptured by the original injury.

The intact posterior parietal peritoneum provided good splintage for the injured kidney until the nineteenth day when this barrier had to be disrupted when the colon was excised. This was probably the reason why a urinary fistula appeared after the second operation. Earlier laparotomy and disruption of the peritoneum overlying the kidney may well have resulted in a more permanent urinary fistula by interfering with the healing process at a more crucial stage.

It is also felt worthwhile to stress the value of unabsorbable sutures in the repair of the diaphragm.

There is little doubt that, had the diaphragmatic repair given way during the height of the tension pneumoperitoneum, the child would not have survived. The ability of chromic catgut to withstand such tension for such a prolonged period late in the postoperative phase is highly problematical.

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Hypoglycaemic coma after partial gastrectomy

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THE SYMPTOM complex of faintness, sweating, palpitations and weariness occurring in post-gastrectomy patients some hours after a meal, especially of carbohydrates, constitutes the 'late' dumping syndrome. It is probably related to hypoglycaemia (Barnes, 1947; Gilbert & Dunlop, 1947) in contrast to 'early' dumping which occurs immediately after meals and is related to jejunal distension and changes in plasma volume (Le Quesne, Hobsley & Hand, 1960). Dumping is a fairly common complication of the early postoperative period but the symptoms, although troublesome to the patient, are not serious and usually clear up spontaneously. In a minority they persist in a more intractable form or may develop for the first time at a long interval after the operation, but even in this group the blood sugar does not often fall to dangerous levels.

Marks & Rose (1965), in an extensive review, assert that coma in essential reactive hypoglycaemia is rare, and do not mention it at all in post-gastrectomy states. Randall (1966) states that coma never occurs in post-gastrectomy hypoglycaemia, although Stammers & Williams (1963) quote one case which occurred 10 min after eating. In view of this we

report the following two cases of post-gastrectomy hypoglycaemia, both of whom had repeated episodes of loss of consciousness.

Case reports

Case 1

Male aet 55. In 1950 (aet 39) he had a gastroenterostomy for duodenal ulcer, followed in 1954 by Polya partial gastrectomy and in 1958 by vagotomy. After the third operation he was free of pain but started to have attacks of sweating during meals. He developed mild diarrhoea, passing pale bulky stools two-four times daily and lost 4 stones in weight over the next 8 years. In March 1963 he suddenly lost consciousness about 2 hr after a meal associated with profuse sweating, making a complete recovery. He had five or six similar episodes after that. In April 1966 he was admitted in coma to the West London Hospital. His blood sugar was estimated by 'Dextrostix' as less than 40 mg/100 ml and he recovered rapidly following intravenous dextrose. Neurological examination after recovery was entirely normal. Lumbar puncture showed clear sterile CSF under normal pressure with 3 cells/mm³, protein 30 mg/100 ml, WR negative. Skull X-rays and an EEG were normal. He was

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Fig. 1. Glucose tolerance test (GTT) showing initial overshoot and later fall to hypoglycaemia levels in both cases. The tolbutamide tolerance tests are normal. Case 1: ●, GTT with 50 g CHO; ■, GTT with 75 g CHO; △, 1 g tolbutamide i.v. Case 2: ●, 1957 GTT; △, 1 g tolbutamide i.v.

found to have steatorrhoea, faecal fat excretion 10.5 g/day. Other investigations: Hb 14 g/100 ml, ESR 2 mm/hr, plasma calcium 8.6 mg/100 ml, alkaline phosphatase 13 K.A. units/100 ml, albumin 2.4 g/100 ml, globulin 2.4 g/100 ml, xylose absorption test 1.1 g excreted 5 hr after 5 g oral dose (22%). Fasting blood glucose 75 mg/100 ml. A standard glucose tolerance test showed a flat curve with blood sugar rising to 90 mg at $\frac{1}{2}$ hr and falling to 45 mg at $1\frac{1}{2}$ hr. A larger (75 g) glucose load produced a very different curve with an initial overshoot, followed by a profound fall to 22 mg at 3 hr (see Fig. 1). An intravenous tolbutamide test showed a normal curve falling to 38 mg after $1\frac{1}{2}$ hr and rising slowly thereafter. He was treated with a high protein, low fat diet and warned to avoid large carbohydrate meals. He responded well, lost his sweating attacks and gained weight. In particular he has had no further episodes of coma over the subsequent 2 years.

Case 2

R.B. Male aet 47. In 1949 vagotomy and gastroenterostomy was performed for duodenal ulcer after which he was symptom-free for 7 years. In 1957 he started to have 'dizzy turns' occurring about four times per year, associated with sweating and relieved by eating sweets. A standard glucose tolerance test showed a fall to hypoglycaemic levels at 2 hr (see Fig. 1). In 1961 he complained of feeling tired all the time unless he continually ate sweets. His symptoms

were worse after a meal and he felt better if his stomach was empty. He had three episodes of coma in 1 year, one just before lunch and two late in the afternoon. He was found to have steatorrhoea, faecal fat excretion 28.5 g/day. Other investigations were as follows: Hb 14.6 g/100 ml, plasma calcium 9.8 mg/100 ml, alkaline phosphatase 12 K.A. units/100 ml, serum proteins 4.7 g/100 ml, fasting blood glucose 60 mg/100 ml, glucose tolerance test (see Fig. 1) now showed an initial overshoot with later fall to low levels (42 mg/100 ml at 2 hr). A 24-hr fast failed to precipitate hypoglycaemia. A tolbutamide test using 1 g Na tolbutamide i.v. showed a normal blood sugar curve falling to 49 mg at 1 hr and then returning to basal levels. Neurological examination was normal and subsequent tests, including skull X-rays, EEG and ultrasonic brain scan were all normal. He was treated surgically by conversion to a Bilroth gastrectomy which relieved his symptoms and was followed by a gain in weight.

Discussion

The possibility that the episodes of unconsciousness in these two patients were unrelated to the previous gastric surgery is considered very unlikely. An insulinoma is virtually ruled out by the tolbutamide test which is very reliable as a provocative test for this tumour (Marks & Rose, 1965). The good response to further gastric surgery in Case 2 and to simple dietary measures in Case 1 is also very much

against it. In view of the normal neurological investigations in both cases, the possibility of separate central nervous lesions is slight.

Both patients showed aspects of the early and late dumping syndrome which commonly occur together. Late dumping is due to hypoglycaemia but the mechanism by which it is produced is poorly understood. It has been postulated that such cases are examples of essential reactive hypoglycaemia with symptoms before operation as well as after. Smith *et al.* (1953) claimed that there was an increased incidence of hypoglycaemia in peptic ulcer patients before operation and that the symptoms were related to excessive nervous reactivity. However, neither of our cases had symptoms preoperatively and both appeared well-balanced personalities.

The probable mechanism for the late hypoglycaemia is suggested by the glucose tolerance curves in post-gastrectomy patients. The lag-type curve with rapid rise in blood glucose leads to excessive production of insulin by the pancreas with a consequent late fall in blood glucose. Sullivan & Boshell (1964) found impaired tolbutamide tolerance in twelve of eighteen post-gastrectomy patients, suggesting that over-stimulation of the pancreas occurs. As peptic ulcer is not itself associated causally with diabetes it is likely that the pancreatic cell exhaustion is secondary to repeated stimulation, as has been postulated in other diabetogenic conditions (Ingle, 1956).

The results of a glucose tolerance test are not always exactly reproducible nor do they necessarily imitate what happens after a normal meal. These difficulties are illustrated by Case 1 who showed a flattened curve with no late fall after a standard 50 g test meal but with a 75 g meal showed a rapid rising diabetic type of curve with a marked fall to hypoglycaemic levels at 3 hr, which fits well with the insulin overshoot theory.

The individual response to a lag-type glucose tolerance test may be expected to vary according to the patient's previous metabolic state. It may be

significant that both these patients also had steatorrhoea. Malnutrition after gastrectomy with reduced liver glycogen stores could impair the normal response to hypoglycaemia and exaggerate the late fall in blood sugar after a meal. This would explain the severity of the hypoglycaemia in these patients and also the long interval between the gastric operation and the appearance of symptoms. It seems likely that the hypoglycaemic coma in these two cases, occurring hours after a meal and years after the operation, has a different aetiology from that of Stammers' case, whose coma developed 10 min after a meal and soon after operation.

Repeated attacks of hypoglycaemia can produce permanent brain damage and the importance of treating this easily remediable cause of coma is obvious. The recognition of hypoglycaemia as a cause of coma after gastrectomy may save such patients being subjected to fruitless extensive neurological investigations.

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