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Progress report

Surgical management of morbid obesity

Morbid obesity describes a condition of more than twice the 'ideal weight' for at least five years despite attempts at an effective and sustained weight reduction.[1–4] The surgical approach to morbid obesity lies in the dual premise that the condition is serious and of life-shortening severity and that long-term medical treatment by prolonged starvation,[5–8] hypnotherapy,[9] psychotherapy,[10] anorexic and anti-obesity drugs[11–13] usually fails.[1–3 14–16]

Jejunoileal bypass

Kremen's experimental[17] and Payne's clinical studies[1 18] have led to more than 400 publications on the subject reporting over 10 000 patients. Most articles appeared during the last six years and 50% deal with only 5% of the patients. Approximately half the patients have had a Payne procedure,[1] one-third a Scott,[19] and the remaining operations are modifications of these two procedures or are not stated.[20] Theoretical advantages are given for each procedure regarding weight loss and decrease in related morbidity.[3 14 16 21–30] The aim is to leave approximately 10% of the small bowel in continuity for absorption[1–3 16 31] and Maclean's review[32] concludes that both types are successful if the length of functioning bowel is between 45 and 75 cm.

OPERATION

In Payne and De Wind's operation, 35 cm of jejunum is anastomosed end-to-side to 10 cm of ileum.[1 33] With Scott's end-to-end operation, 30 cm of jejunum is anastomosed to either 30, 20, or 15 cm of ileum[2 19 34 35] and the 90% bypassed small bowel is drained into the colon.[2 3] Modifications include anastomosis of gallbladder to the proximal excluded loop to reduce diarrhoea,[36–38] automatic stapling,[39] plication of the ileum,[40] or formation of a valve.[41]

The overall mortality in 2500 patients was 3·1% with a range from 0 to 11·5%.[2 16 21 29 31–34 43 44] Immediate postoperative complications can be as high as 30% with wound problems being the commonest.[14 32 45] Death is due to thromboembolism, myocardial infarction, and sepsis.[14 16 22 24 33 34 46]

WEIGHT LOSS

Weight loss cannot be predicted[3] and comparisons between reports are difficult because of variations in operative technique and follow-up assessment.[16] Quaade[20] found only 45 publications (1711 patients) relating to weight loss at one year and only five reports referring to more than a five year follow-up. Data from six well-documented series indicate a 20% weight loss within six months, and about 33% by the first year.[47] Weight loss is due to both malabsorption and decreased dietary intake.[48 49] The only prospective randomised study comparing J-I bypass and medical treatment has been conducted by the Danish Obesity Project in 130 patients.[20 30 50] At 24 months,

median weight loss was 43 kg after bypass compared with 6 kg in the control ($P < 0{\cdot}001$).

Insufficient data are available to prove the effect of bypass on life expectancy,[14] but the answers may be provided in the future by the Danish study.[50] Serum cholesterol and triglycerides are decreased, and type II, IV, and V hyperlipoproteinaemic electrophoretic patterns and hyperglycaemia may return to normal.[51–53]

BOWEL ADAPTATION

Bowel adaptation occurs in the functioning remnant[54] and measurements taken at subsequent laparotomy[55 56] or by contrast radiology[57–59] show a hypertrophy, dilatation, and gradual elongation. Villi lengthen, especially in the jejunum,[60] and crypts deepen in the ileum.[61] The plateau in weight loss correlates with the villous hypertrophy.[62] Surgery reduces the total intestinal absorption surface area to 8·5%.[62] This increases by 300% at two years, giving a preoperative net surface area of 26%.[62] Increases occur in mucosal disaccharidases[56 63] alkaline phosphatase, thymidine kinase,[63] and fat transportation[60 62] with decreased B12 and glucose absorption.[14 60] The bypassed segment, especially the ileum, atrophies[59 61] and the villi are either reduced in size,[59 61] remain unchanged,[64 65] or even hypertrophy[55] at the site of colonic regurgitation. Basal enteroglucagon and pancreatic polypeptide increase after operation,[6 60] whereas gastrin rises only after a liquid meal.[60 66]

DIARRHOEA

All patients develop diarrhoea that ranges from eight to 20 times per day; this decreases after one year to two or three soft stools per day.[21 31] Causes include loss of absorptive surface, decreased transit time, disaccharidase deficiency, excessive colonic bile and fatty acids, and fat malabsorption.[67] At four years, the transit time remains at a mean of five minutes. Diarrhoea is controlled by restricting dietary fat and using diphenoxylate HCl, codeine phosphate, loperamide, cholestyramine, calcium carbonate,[21 69] tetracycline, or metronidazole.[70] Rarely, the severity of the diarrhoea and electrolyte disturbance requires either a bypass reversal or a proximal blind loop jejunostomy for feeding.[69]

LIVER FAILURE

Approximately 5% of patients develop some degree of liver failure postoperatively and 1% progress to frank cirrhosis.[71–77] Between 60–98% of patients, especially males, have fatty metamorphosis of the liver before bypass[74 76–78] which may[78] or may not[76] correlate with the extent of obesity. Centrolobular fibrosis or frank cirrhosis are contraindications to surgery.[77] Liver changes are most severe at six to 12 months postoperatively, occurring simultaneously with the maximum rate of weight loss and thereafter there is a resolution.[74 77] Death from liver failure or cirrhosis occurs in 0·5–2% of patients at five to 24 months after operation with 14 reported cases.[1 2 16 71 76 77] Fatty metamorphosis has many causes[77] and these include protein malnutrition[79 80]; increased chenodeoxycholic acid[81]; inactivation of dietary choline[82]; anaerobic bacterial overgrowth[72] especially bacteroides,[83] and production of endogenous alcohol[84] or hepatotoxins.[72] Liver histology varies from minimal to marked steatosis[73] with variable fibrosis[71 74 77] resembling either kwashiokor[80] or typical alcoholic hepatitis.[71 85] Nasogastric or jejun-

ostomy feeding of amino acids,[80 86] protein,[87] or intralipid[88] causes some improvements.[83 87] The effects of parenteral nutrition may be beneficial[86 89 90] or of no use,[89 91] and antibiotics including metronidazole or tetracycline are given.[2] Liver failure is reversible in more than half after reanastomosis and accounts for 36% of the operations for complications.[96]

BONE DISEASE

Bone disease occurs in 17 to 48% of patients one to 14 years after operation[92–94] with clinical symptoms, biochemical, and radiological investigations often being unreliable.[92 93] In histologically proven bone disease, plasma 25-OH vitamin D_3 is low[92–95] and plasma PTH raised.[94] Alterations in vitamin D metabolism are due to malabsorption, steatorrhoea, liver disease, and the blind loop syndrome.[95] Several characteristic histological changes in bone have been described, including osteopaenia with excessive osteoid.[95]

ARTHROMYALGIA

Arthromyalgia is a troublesome problem found in up to 20% of patients[96–98] within three years after both a jejunocolic[99–100] and J-I anastomosis.[98] The commonest is a non-specific self-limiting transient arthromyalgia,[100] although a polyarthritis may persist.[97] Extra-articular rheumatic manifestations include tenosynovitis,[100] erythema nodosum, pleural effusions,[43 97] and skin pustules.[101] The arthritis is usually acute with symmetrical involvement of hands, feet, wrists, and ankles with or without erosions.[97 100 102] Abnormal circulating immune complexes have been identified[99 102 103] including HLA B27[98 99] but not confirmed.[97] Together with circulating antibodies to *B. coli* and *B. fragilis* and associated sacroileitis, this suggests a blind loop syndrome with bacterial overgrowth.[97–100] If treatment with salicylates and other non-steroidal anti-inflammatory drugs fail,[101] tetracycline or metronidazole may give transient relief,[97 100 103] but occasionally a reversal operation is required.[104]

CALCULI

Urinary calculi and nephrocalcinosis occur in approximately 1%[105] of patients, but the figures given in reports vary up to 32%[2 3 14 16 22 46] within the first two years[105] and these disorders can occur up to 10 years after J-I bypass.[106] Hyperoxalaemia and hyperoxaluria is due to increased absorption of exogenous oxalates, especially by the colon,[107–109] but can be secondary to alterations in either the hepatic or enteric oxalate pathway.[105] Oxalate is deposited in the renal interstitium[108 110] and, as the incidence and severity of hyperoxaluria after operation is similar in both stone and non-stone formers,[108] additional causative factors are necessary.[105 107 111] Pyridoxine, folic acid, calcium, magnesium, and pyrophosphate are effective in decreasing the hyperoxalaemia.[110 112] A low fat diet, aluminium hydroxide, cholestyramine, and diethylamino-ethanolcellulose may prevent recurrent calcium oxalate stones.[105 107 112 113] Low oxalate diet (< 50 mg/day) decreases exogenous sources,[114] but the exclusion of many fruits and vegetables is unacceptable to most patients.[108] Progressive renal failure may require regular haemodialysis[108] or reversal of the bypass.[115]

Biliary calculi are found in 30% of morbidly obese patients before bypass surgery,[2 116] and after bypass increase by 5% per year.[33 116 119] Lithogenic factors include alterations in cholesterol and bile salt metabolism and bacterial infection.[116–121] The lithogenicity of gallbladder bile is unexpectedly increased

fourfold with an increased length of functioning ileum relative to jejunum (37·5 cm versus 12·5 cm),[121] but follow-up is required to determine if there is also an increased incidence of gallstones.[50 121] Prophylaxis and therapy with chenodeoxycholic or ursodeoxycholic acid need investigation.[119]

INTESTINAL COMPLICATIONS

A group of intestinal complications have been reported which include intussusception,[122–125] bypass enteropathy,[126–128] intestinal pneumatosis,[129–132] pseudo-obstruction,[133 134] transmural ileocolitis,[135] and the blind loop syndrome.[136] Intussusception of the oversewn proximal end of the bypassed segment is rare because of adequate fixation with only 26 reported cases.[122–125 137] Vomiting and air-fluid levels may be absent, but the separation of marker clips or ultrasound help in diagnosis.[124 125] Patients with colonic pseudo-obstruction[138 139] present with intermittent or acute abdominal distension 18 to 36 months after J-I bypass with air-fluid levels.[133 139] The pseudo-obstruction occurs distal to the drainage of bypassed intestine. Conservative treatment with nasogastric and rectal intubation produces rapid relief, and, as anaerobic bacteria may play a role, antibiotics are given.[133 139] In the 42 reported cases of intestinal pneumatosis, the symptoms were similar to irritable colon and bypass enteritis,[129–133 140 141] and may occur as a separate entity or with bypass enteritis[127 131] and colonic pseudo-obstruction.[133 139] Routine postoperative radiographs of the abdomen revealed pneumatosis intestinalis in 16% of patients who were all asymptomatic.[141] Treatment includes breathing of 70% oxygen, antibiotics,[127 129 131] and surgery only for bleeding or obstruction.[130] Bypass enteropathy (enteritis)[126 127 140] or the 'excluded loop syndrome'[134] probably represents a spectrum of diseases. An acute form of enteropathy was found in four of 28 patients (14%) in the early weeks postoperatively,[127] and can progress to gangrene with septicaemia or pneumatosis intestinalis.[127 134] The aetiology is unknown but may include bacterial overgrowth within the excluded bowel,[127] changes in intraluminal pH, loss of peristalsis,[140] an ileosigmoidostomy[131] and mechanical obstruction by a sigmoid volvulus.[142] Prostigmine, methadone, and other narcotics accentuate the condition[134 140] and antibiotics are often effective in treatment.[126 127]

TUBERCULOSIS

Tuberculosis, especially reactivation, appears to be higher after J-I bypass than in the general population[143] and, in the nine reported cases, six occurred at extrapulmonary sites.[143–146] These include tuberculous peritonitis, pleuritis, and cervical lymphadenitis which may not respond to therapy and may even cause death.[143] Protein malnutrition and immunoreactive changes may be the cause.[143 147]

REVISION SURGERY

Revision surgery is performed for inadequate weight loss (5–20%)[1 2 3 14 16 23 43] or for severe side-effects (1–23%).[104 147] Procedures include conversion, more proximal or distal reconstruction, resection, or reanastomosis.[148–153] Unless the patient is extremely ill, the tendency is for reversal of the J-I bypass with a concurrent gastric bypass.[104 151–153] In a short follow-up, these synchronous operations correct the metabolic abnormality and maintain weight loss.[104 152 153]

Gastric bypass (gastric partition, gastroplasty)

Gastric bypass, developed by Mason and Ito in 1966,[154 155] avoids the metabolic and nutritional complications of a J-I bypass. The operation requires the formation of a small gastric pouch (50–60 ml), a measured outlet of 10–12 mm, a secure partition, and a method of preventing dilatation of the stoma.[156–160] Mason estimates that 25 000 patients have had some form of gastric procedure for obesity in the USA,[152–183] of which 880 have been performed by his group (Mason, 1980 . Personal communication). The operative technique has undergone several periods of development and modification. Initially (1966–70), a subtotal gastrectomy with a small fundic pouch was created with a large stoma. Because it led to unsatisfactory weight loss and a high mortality, it was followed in 1971 by gastroplasty; this involved a partial transection of the stomach from the lesser curve, leaving a small channel along the greater curve. Similarly, patients undergoing this procedure lost weight only during the first six months. Between 1972–74, a gastric bypass was again performed with a small stoma and a loop of jejunum. However, the era of the large upper pouch led to a fourth period in 1975 when it was realised that, to achieve optimum weight loss, it was necessary to bypass 90% of the stomach, leaving a small fundic pouch and narrow gastro-enterostomy stoma.[158 168 169]

Since 1978 the gastroplasty or gastric partition has been gaining popularity, because of the ease of operation using autosuture stapling machines with the stoma along the greater curve or midbody[27 165 167 168 184] or using a Roux-en-Y anastomosis.[161] The upper pouch volume of 50–100 ml is determined by using saline,[168] a silicone balloon,[185] or measuring along the greater and lesser curves.[162] To prevent disruption, either a second application[161] or oversewing of the staple line is performed.[168 185]

The initial operative mortality of 4% has been reduced to 1%[150 159 170 183] but depends on the type of procedure. Deaths are due to anastomotic leaks; and gastric ischaemia and perforation, which initially occurred in 4·4%,[170] have now decreased to 0%.[158] Late complications occur in up to 17% of patients[170] and include severe and persistent nausea and vomiting, afferent or efferent loop obstruction, dumping syndrome, hair loss, and renal stones (1%).[170] The serum gastrin response to a meal is increased but gastric secretory responses fall.[154 157] The incidence of peptic ulceration has decreased to 1·5% using a smaller pouch[158] and cimetidine.[178] Duodenal perforation and death have been described in two patients, but, according to the registry figures, this is a rare complication.[158 159] There have been no reports of liver disease after operation,[161 163 170] and biopsies at one year showed improvement in 50–75% of patients.[157 163 169]

Weight loss is most rapid in the first six weeks.[158 170] The greater the initial weight, the greater the loss, with the final weight dependent on the balance between the reduced food intake and energy expenditure.[156 169 183] In an analysis of nearly 1600 patients, Griffen[170] found the mean weight loss at one year was 47·6 kg. In a 10 year follow-up by Mason of 67 patients, there was a 43% failure rate. Similarly, in the 58 gastroplasties operated on in 1971, there was a 53% failure rate, the patients requiring revision or further procedures because of inadequate weight loss (Mason, 1980. Personal communication).

There have been only three comparative studies of gastric versus J-I bypass.[161 163 169] Alden's study[161] of 200 patients was not randomised, and the weight loss in each group was just over 40 kg. The complications of liver

disease, calculi, arthritis, diarrhoea, and electrolyte depletion in J-I bypass patients were not found after gastric bypass. Alden[161] found that the gastric bypass was technically more difficult, but that stapling decreased both operating time and complications. The two prospective randomised studies reported by Griffen[169] and Buckwalter[163] indicate that the gastric bypass is superior to the J-I bypass; the loss of weight is much the same but there are fewer long-term sequelae. However, all three studies[161 163 169] retained longer lengths of small bowel in the J-I bypass and early complications after gastric bypass reached 62·5% because of technical difficulties.[169]

Dental splintage

Initially, two patients were reported who lost 40 kg in five months after interdental splinting[186] and another 37 patients have been studied.[16 187 188] The diet consisted of milk or soup amounting to 3·35 MJ with iron and vitamin supplements.[186 188] Dental caps were removed every three months for dental hygiene and to prevent trismus.[16 186] Early failures occurred in half the patients who could not tolerate the splints or removed them, and in only 10 of Baddeley's cases was there full cooperation, which allowed between 20 and 60 kg of weight to be lost in the first three months.[16] In Rogers' study[187] there were no major complications and all 17 patients lost weight in a way comparable to a J-I bypass, but only one patient achieved and maintained her ideal weight. Jaw wiring can be performed in outpatients and has less morbidity than abdominal surgery[16 187 188] and the major risk of aspiration can be minimised by correct posturing during vomiting.[187] Long-term results are unlikely to be much better than other conservative measures.

Vagotomy

Kral[189] reported three obese women who underwent bilateral truncal vagotomy and lost 15 kg weight in 20 weeks. A bilateral truncal vagotomy without drainage has now been carried out in 13 obese patients with a weight reduction of between 20–30 kg (range 2–64 kg) in the four to 24 month follow-up period.[196] Weight reduction may have been due to impaired gastric emptying creating a sensation of fullness, decreased acid production, and changes in hormonal or food preference.[189 190] Two patients have failed to lose weight and it is too early to consider this procedure for clinical use.[190]

Biliary-pancreatic bypass

After experimental work in dogs[191] a biliopancreatic bypass was carried out in 18 obese patients.[192] The operation consists of a partial gastrectomy and closure of duodenal stump. The jejunum is then transected 20 cm distal to the ligament of Treitz and a gastrojejunostomy is performed using the distal part of the Roux loop. The proximal part of the jejunum is then anastomosed to the distal ileum. This anatomical arrangement causes malabsorption of fat and carbohydrate leaving the enterohepatic bile circulation intact. In the 18 patients, a combination of four operations using different measurements of jejunum and ileum has been performed.[192] The mean weight loss was 24% at six months and 34% at 12 months, with no late complications in the 17 month follow-up. The exact length of ileum and jejunum required to achieve maximum weight reduction with minimal complications has not yet been determined.

Current status

The following is a summary of information obtained regarding the current status of the surgical treatment of morbid obesity from Bray, Buchwald, Payne, Phillips, and Scott in the USA, Salmon in Canada, Hallberg and Quaade in Scandinavia, and Baddeley and Gazet in the United Kingdom. All have published studies on J-I bypass but only six (60%) are still carrying out the procedure and two are using the bilio-intestinal anastomosis.[37 38] At present, three authors are not performing any surgery for morbid obesity. Bray is planning and Gazet is involved in a study of gastric versus J-I bypass. Three authors performed an end-to-side, and four an end-to-end type of J-I bypass and Buchwald has recently increased the length of jejunum to 65 cm. Gastric bypass or partitioning has been performed by six of the authors and is being routinely performed by two. Mason expects improved results with the gastric bypass now that the criteria for an adequate operation have been identified. However, with so many variations of the operation, an adequate period of follow-up is required for assessment.

Payne probably summarises the present situation regarding the surgical treatment of morbid obesity: 'from an ethical and moral aspect, all of these operations—gastric partitioning, gastric bypass and jejuno-ileal bypass, are being abused. Too many operations are being done on patients who are not valid candidates and by surgeons who are not qualified or have no efficient follow-up programme. The malignant abuse of these operations will result in discreditation of the surgical approach to morbid obesity. This could result in the abandonment of the only practical method, at this time, for the treatment of the morbidly obese patient'.

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