Metabolic surgery: a concise overview and understanding of potential complications

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Abstract

Bariatric surgery, despite a relatively short history, has had a tremendous impact on the discipline of surgery. In addition, it has had a profound influence on the development of laparoscopic surgery and the treatment of the worldwide epidemic of obesity. Contrary to the expectations of the 1990s, that obesity surgery would converge into a single procedure, there has been a trend favouring the safest and least invasive surgery on the one hand, and a diligence towards the most effective and complicated surgery on the other hand. The latter, being the biliopancreatic diversion with duodenal switch, requires advanced laparoscopic skills and an experienced medical team. This review will focus predominantly on the laparoscopic Roux-en-Y bypass, a recognised procedure which is performed in 80% of patients going to theatre for bariatric surgery.

Rationale for the surgical treatment of morbid obesity

Morbid obesity correlates with a body mass index (BMI) of 40 kg/m^2.[1] Being overweight is associated with numerous significant physical problems that are now well recognised. It is clear that overweight people of both sexes, especially young overweight people, tend to die sooner than their lean contemporaries.[1,-3] While obesity is a risk factor, additional mortality and morbidity is associated with co-morbid conditions.[4] This applies to non-operated, as well as perioperative, mortality and morbidity. These conditions were described at the 1985 National Institutes of Health Consensus conference, and include diseases such as hypertension, hypertrophic cardiomyopathy, hyperlipidaemia with ischaemic heart disease (IHD), diabetes, cholelithiasis, nonalcoholic steatohepatitis (NASH), obstructive sleep apnoea, hypoventilation, degenerative arthritis, cancer, polycystic ovarian syndrome (PCOS), and psychosocial impairments.[4,5]

Surgical treatment goals

The only proven method of achieving long-term weight control for the morbidly obese is surgical treatment. Metabolic surgery involves reducing the size of the gastric reservoir, with, or without, a degree of associated malabsorption. Eating behaviour improves dramatically.[6]

The surgery reduces kilojoule intake, and ensures that the patient practises behaviour modification by eating small amounts slowly, and experiences enhanced satiety through peptide modification. Success of surgical treatment must begin with realistic goals and progress through the best possible use of well-designed and tested operations. These have been determined over the last 30 years, and are now standardised, clearly defined procedures, with well-recognised and documented outcome results.

Prevention of secondary complications of morbid obesity is an important management goal. The option of surgical treatment is a rationale supported by the accepted principle that diseases that harm call for therapeutic intervention. The biological basis for morbid obesity is complex. Recent work has demonstrated a genetic component of 25-50%.[7] Many studies confirm the influence of genetically determined proteins produced by the fat cell to be among the many mechanisms that have a place in the control of satiety.[8-10] It has also been determined that morbid obesity is a disease with a physiological, biochemical, and genetic basis. It is not a disorder of willpower, as is sometimes implied. Additional contributing causes include environmental, cultural, socio-economic, and psychological factors.

The long-term mortality rate of gastric bypass procedure (GBP) patients is reduced by up to 40%.[10,11] Currently, the general death rate from bariatric surgery seems to

vary between 0.2-1%. The rate is lowest for the lap-band procedure (0-0.2%) and highest for the biliopancreatic diversion (BPD) procedure (0.2-0.8%), with GBP at 0.5%.

A recent Canadian study revealed that severely obese patients who chose to undergo bypass surgery reduced their risk of premature death by up to 89%, compared to equally overweight patients who did not receive surgical treatment. Mortality for cardiac disease was reduced from 5% in the control group to 0.45% in the study group. In another study of 66 000 obese patients, about 3% of GBP patients (under the age of 40) died within 13.6 years after the surgery, compared with 14% of obese patients who were not treated surgically. Mortality for this study was 0% in 401 laparoscopic cases, and 0.6% in 955 open procedures. The largest bariatric surgery patient population ever analysed (57 918 consenting patients) demonstrated a total mortality rate across all procedures of 0.135%, which is equivalent to approximately one death per 1 000 patients. Rates for 90- and 30-day all-cause mortality were 0.112% and 0.089%, respectively.[12]

Surgical procedures

Various types of procedures are listed below.

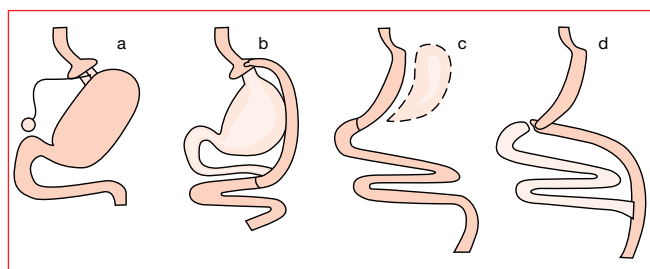**Procedures currently accepted and endorsed by international obesity societies (Figure 1)**

Figure 1: Accepted and endorsed procedures
(Reproduced with permission from Prof W Pories)

- Adjustable gastric band (a)
- Roux-en-Y gastric bypass (b)
- Vertical sleeve gastrectomy (c)
- Biliopancreatic diversion with a duodenal switch (d).

Non-accredited procedures
- Mini laparoscopic sleeve gastric bypass (Bilroth II reconstruction)
- Intragastric balloon.

Future and experimental procedures[13]
- EndoBarrier gastrointestinal liner
- Implantable Maestro System
- Transoral gastroplasty
- Transoral gastric volume reduction.

Banned procedures
- Jejunoileal bypass.

Gastric bypass procedures

Laparoscopic gastric bypass

The GBP is constructed by creating a small (15-30 ml) pouch from the upper stomach, accompanied by bypass of the remaining stomach (400-1 000 ml). This restricts the volume of food that can be eaten. The stomach may be divided into two parts, with staples. A total division is usually advocated, to reduce the possibility that the two parts of the stomach will fuse together ("fistulise"), thereby negating the operation.

Reconstruction of the gastrointestinal tract is carried out to enable drainage of both stomach segments. This reconstruction technique produces several variants of the operation, which differs in the lengths of small bowel used, the degree to which food absorption is affected, and the likelihood of adverse nutritional effects.

Gastric bypass [Roux-en-Y (proximal)]

The Roux-en-Y laparoscopic GBP was first performed in 1993. It is regarded as one of the most difficult limited access procedures to perform. However, use of this method has greatly popularised the operation, with benefits including a shortened hospital stay, reduced discomfort, shorter recovery time, less scarring, and minimal risk of an incisional hernia.

This procedure is the operation that is least likely to result in nutritional difficulties. The small bowel is divided approximately 45 cm from the pylorus, and is rearranged into a Y configuration, which enables outflow of food from the small upper stomach pouch, via a "Roux limb." In the proximal version, the Y intersection is formed near the upper (proximal) end of the small bowel. The Roux limb is constructed to a length of 80-150 cm, preserving most of the small bowel for nutrient absorption. The patient experiences a very rapid onset of a sense of stomach fullness, followed increasing satiety or lack of interest in food shortly after the start of a meal.

Gastric bypass [Roux-en-Y (proximal)]: long loop laparoscopic Roux-en-Y gastric bypass

A normal small bowel is 600-1 000 cm in length. As the Y connection is moved further down the gastrointestinal tract, the amount of bowel that is capable of fully absorbing nutrients is progressively reduced, in pursuit of greater operation efficacy. The Y connection is formed much closer to the lower (distal) end of the small bowel, usually 100-150 cm from the lower end of the bowel, causing reduced

absorption (malabsorption) of food, primarily of fats and starches, but also of various minerals and fat-soluble vitamins. The unabsorbed fats and starches pass into the large intestine, where bacterial action produces irritants and malodorous gases (steatorrhoea). These increasing nutritional effects are associated with a relatively modest additional weight loss, and have a limited application.

Loop gastric bypass ("mini gastric bypass")

At its inception in 1967, the mini GBP was performed using a loop of small bowel for reconstruction, rather than a Y construction, as is prevalent today. Although this process is simpler, it can cause bile and pancreatic enzymes from the small bowel to enter the oesophagus, sometimes causing severe inflammation and ulceration of either the stomach, or the lower oesophagus. If a leak into the abdomen occurs, this corrosive fluid can have severe consequences. Numerous studies show that the loop reconstruction (Billroth II gastrojejunostomy) increases safety when placed low on the stomach, but can be disastrous when placed adjacent to the oesophagus. Thus, even today, thousands of "loops" are used for general surgical procedures, such as ulcer surgery, stomach cancer, and injury to the stomach. However, bariatric surgeons abandoned use of the construction in the 1970s, when it was recognised that the risk was not associated with weight management. In addition, nutritional abnormalities are very prevalent and difficult to manage.

Physiology of the gastric bypass procedure

The GBP reduces the size of the stomach by more than 90%. A normal stomach can stretch, sometimes to a size of > 1 500 ml, while the pouch of the GBP may be 30 ml in size. The GBP pouch is usually formed from a part of the stomach that is least susceptible to stretching. This, in conjunction with its small original size, prevents any significant long-term change in pouch volume. What does change over time is the size of the connection between stomach and bowel, and the ability of the small bowel to hold a greater volume of food. When the patient ingests only a small amount of food, the first response is a stretching of the wall of the stomach pouch, stimulating nerves that signal to the brain that the stomach is full.

Food is first churned in the stomach before passing into the small bowel. When the lumen of the small bowel comes into contact with nutrients, a number of hormones are released, including cholecystokinin from the duodenum, and total peptide YY and glucagon-like peptide 1 from the ileum. These hormones inhibit further food intake, and have been dubbed "satiety factors". Ghrelin, a hormone that is released in the stomach, stimulates hunger, as well as food intake. Changes in circulating hormone levels after GBP have been hypothesised to produce reductions in food intake and body weight in obese patients. To gain the maximum benefit from this physiology, it is important that the patient ingests food only at mealtimes (three to four small and equally sized meals daily), and avoids snacks between meals, which can effectively "bypass the bypass". This requires a change in eating behaviour, and alteration of long-acquired eating habits. In almost every case where weight gain occurs late after surgery, it is not the capacity for a meal that has greatly increased, but rather the pattern of constant eating has become fixed. Prospective patients need extensive nutritional and psychological counselling in order to avail themselves of the merits of how to use their newly acquired "tool".

Complications of obesity surgery

Intraoperative complications

Complications which occur during bariatric operations can be divided into three categories:

- Bleeding
- Inadvertent injury to the gastrointestinal tract
- Stapling misadventures.

The incidence of intraoperative complications ranges between 0-1.4%, at best.

- Intraoperative complications can generally be avoided by unabated concentration, and careful technique
- Injuries can be successfully corrected, or repaired, if recognised intraoperatively
- The splenectomy rate is about 0.3-0.5%
- Intraoperative complications should be rare in experienced hands (< 1%).

Early postoperative complications (morbidity)

Complication rates in the early postoperative period, such as infection, dehiscence, leaks from staple breakdown, stomal stenosis, ulcers, and deep vein thrombosis (DVT) with pulmonary embolism, are approximately 4-8%. However, the combined risk of the most serious complications (gastrointestinal leak and DVT) is < 3%.

Pulmonary embolism

Pulmonary embolism is the leading cause of perioperative death in bariatric surgical patients. The incidence of pulmonary embolism is reported to be 0.5% in most large series of bariatric operations. Nearly one-third of bariatric patients who suffer severe pulmonary embolism die. This incidence is lower than for other large surgeries. The incidence of severe pulmonary embolism does not

seem to be altered much by the routine use of methods of perioperative DVT prophylaxis and of anticoagulant therapy, but careful preoperative screening, treating contributing risk factors, and early mobilisation will further diminish risk.

Gastrointestinal leaks

The percentage of gastrointestinal leaks associated with primary GBP operations is 1-2%. Leaks are sometimes difficult to recognise after GBP, because fever and abdominal tenderness are frequently absent. The leukocyte count and C-reactive protein are often elevated, but may be within normal limits. Left-shoulder pain and anxiety are early symptoms. Persistent tachycardia and progressive tachypnoea are the most common early signs. Hence, it is common to initially suspect pulmonary embolism in a patient with a gastric leak. Most surgeons attempt to identify leaks using radiographic gastrointestinal contrast studies. However, a normal contrast study by no means excludes a leak, since extravasation from the gastric staple line is often not identified by gastrointestinal contrast studies. An isolated left-sided pleural effusion is a common finding on a plain chest radiograph.

Because failure to recognise a leak can result in the patient's death, exploratory laparoscopy or laparotomy should be empirically performed in patients with progressive tachypnoea and tachycardia in whom pulmonary embolism has been ruled out. In patients who are rapidly deteriorating, exploratory surgery should be undertaken without gastrointestinal radiographs.

The incidence of leaks following revision procedures is five to 10 times higher than after primary operations, presumably because of ischaemic damage of the stomach.

Major wound infection

The incidence of major wound infection after GBP is reported to be 1-3%. Conversely, seromas in the subcutaneous fatty layer are common, with an incidence approaching 40%. The potential for wound dehiscence after GBP is greatly increased, because of the tension placed on the closure by massive overweight. Hence, the abdominal fascia should be closed with heavy nonabsorbable sutures. The incidence of fascia dehiscence is about 1% in large published series.

Gastrointestinal bleeding within the 30-day perioperative interval

There are various causes of bleeding, including marginal ulceration, ulcers in the bypassed stomach or duodenum, gastritis, and bleeding from fresh staple lines. Ulceration of the anastomosis occurs in 1-16% of patients.[14]

Possible causes of such ulcers are restricted blood supply to the anastomosis (compared to the blood supply available

to the original stomach), in addition to:

- Anastomotic tension (technical).
- *Helicobacter pylori* (should be treated preoperatively if identified).
- Smoking (should not be allowed pre- or post-surgery).
- Use of nonsteroidal anti-inflammatory drugs. Avoidance of these, or prescription under strict supervision, can drastically reduce incidence.

This condition can be treated with:

- Proton-pump inhibitors (e.g. esomeprazole).
- Cytoprotection and an acid-buffering agent (e.g. sucralfate, very slow healing time).
- Temporary restriction of consumption of solid foods.

Small bowel obstruction

Within the first several weeks postoperatively, small bowel obstruction may occur in 1-2% of patients. Most cases of early obstruction can be treated successfully by tube decompression, which is best accomplished using fluoroscopy. Early obstruction may follow dehiscence of the primary trocar site with bowel entrapment, like a Richter hernia.

Cardiorespiratory complications

Cardiorespiratory complications are surprisingly uncommon after GBP. Sudden cardiac arrest is quite rare in the postoperative period, and death after cardiac arrest is often the result of the inability to intubate patients who develop acute respiratory distress. Intubation under these difficult circumstances may require flexible bronchoscope-assisted awake intubation, and may be lifesaving.

Mortality

Important risk factors for complications and premature death following GBP include:

- The physical condition of the patient, including weight and specific co-morbid conditions.
- The complexity of the bariatric procedure.
- The skill and experience of the surgeon.

Laparoscopic GBP typically leads to less pain and fewer incisional hernias (1-2%) than open surgery bypass. However, laparoscope-assisted surgery is more demanding, and inexperienced bariatric surgeons can expect an initial increase in peri- and postoperative complications. Mortality is affected by complications, which, in turn, are affected by pre-existing risk factors, such as degree of obesity, heart disease, obstructive sleep apnoea, diabetes mellitus, and a history of prior pulmonary embolism. It is also affected by the experience of the operating surgeon. The "learning curve" for laparoscopic bariatric surgery is estimated to be about 125 cases.

Intermediate postoperative complications

In the first three to six months after surgery, as the body reacts to rapid weight loss, the patient may experience one or more of the following rarely documented changes:

- Body aches
- Dumping syndrome
- Feeling tired or having malaise
- Feeling cold when others feel comfortable
- Dry skin
- Hair thinning and hair loss
- Changes in mood and relationship issues.

Some changes are due to a slowing of the body's metabolism from weight loss, and are usually resolved within three to six months.

Dumping syndrome

Normally, the pyloric valve at the lower end of the stomach regulates the release of food into the bowel. When the GBP patient eats sugary food, the sugar passes rapidly into the bowel as a result of the absence of a pylorus, giving rise to a physiological reaction called the dumping syndrome. The body will flood the intestines in an attempt to dilute the sugars. An affected person may feel his or her heart beating rapidly and forcefully, and break into a cold sweat. The patient could be very uncomfortable for about 30-45 minutes. Diarrhoea may then follow. Traditionally, there are two explanations, or two types of dumping, namely, mesenteric blood shunting and fluid shifts, or the vasomotor type and the hypoglycaemic type, due to excessive insulin release following ingestion of high-calorie sugary foods.

Late postoperative complications

Incisional hernia

Incisional hernia is the most common late complication after open GBP, with an incidence of 10-20% in most large series. The incidence for laparoscopic surgery is 1-2%.

Internal hernia with obstruction

This is called a Petersen space herniation. The small bowel herniates through the mesenteric defect between the two loops of the Y anastomosis. This space should be closed at the time of primary surgery. It accounts for only 7% of all hernias, but can be a potentially lethal complication due to late diagnosis.

Symptomatic gallbladder disease

This ranges from 3-30%. The practice of removing the gallbladder prophylactically at the time of surgery has been a controversial issue among bariatric surgeons. Several surgeons who have recommended prophylactic

cholecystectomy report histological evidence of gallbladder pathology in 90% of cases. It is acceptable practice to remove the gallbladder for asymptomatic gallstones at the time of bypass surgery, and it is regarded as a standard procedure with the BPD and duodenal switch, because of the more rapid weight loss.

Vomiting and dehydration

Although vomiting is a side-effect of GBP in the early postoperative period, severe intractable vomiting is rare. Most cases of severe vomiting are caused by stenosis of the outlet stoma. Patients with vomiting who cannot tolerate liquids should be hospitalised, and placed on intravenous fluids. In many cases, the oedema of the outlet stoma, which results from protracted vomiting, will resolve without further intervention. Patients who cannot tolerate liquids after several days of being nil by mouth and only having intravenous fluids should undergo upper endoscopy and stomal dilatation, using endoscopic balloons. Stomal dilatation is usually successful, except in patients with prosthetic stomal reinforcement. Many patients with prosthetic stomal reinforcement require re-operation for intractable stomal stenosis.

Late disruption of the stapled gastric partition

This breakdown is responsible for patients regaining lost weight after GBP. Despite conflicting data, most surgeons now routinely divide the stomach during GBP. The incidence of staple-line leaks and gastrogastric fistulae after tran-section is reported to be in the range 1-2%.

Marginal ulceration after Roux-en-Y gastric bypass

Marginal ulceration incidence ranges from 3-10%. These ulcers typically develop on the jejunal side of the gastroenterostomy, and are caused by excessive production of gastric acid. Marginal ulcers that are not associated with disruption of the stapled partition almost always respond to proton-pump inhibitors. Conversely, ulcers that occur in patients with staple-line breakdown are often intractable to medications, and require operative treatment.

Intestinal obstruction

Intestinal obstruction is relatively uncommon after GBP, but it may be life-threatening. The incidence of small bowel obstruction after Roux-en-Y gastric bypass and other malabsorptive procedures is 2-3%. Because gastric capacity is greatly reduced after Roux-en-Y gastric bypass, vomiting is often not a prominent symptom.

Although most cases of late small bowel obstruction are caused by adhesions, volvulus related to internal hernia is a recognised, and occasionally fatal, type of obstruction (Petersen hernia), and constitutes 7% of all hernias. Because

obstruction of the bypassed bowel may not be obvious on plain abdominal radiographs, computed tomography scanning should be promptly performed when abdominal films are non-diagnostic.

Aggressive operative treatment is warranted in patients whose symptoms do not quickly improve with tube decompression.

Nutritional effects of bariatric surgery

After surgery, patients feel full after ingesting only a small volume of food, followed soon thereafter by a sense of satiety and loss of appetite. Total food intake is markedly reduced. As a result of the reduced size of the newly created stomach pouch, and reduced food intake, adequate nutrition is essential. This includes the number of meals to be taken daily, adequate protein intake, and the use of vitamin and mineral supplements. Total food intake and the absorbance rate of food will rapidly decline after GBP surgery.

After GBP surgery, there may be an increase in the number of acid-producing cells in the lining of the stomach. Acid-reducing medications can be prescribed to counteract the high acidity levels in certain patients. As a result, some patients may experience achlorhydia.[15] Subsequent overgrowth of bacteria may cause the gut ecology to change, and can induce recurring nausea and vomiting. This can potentially change the absorbance rate of food, which contributes to the vitamin and nutrition deficiencies in postoperative GBP patients. A lifelong follow-up programme, managed by a bariatric clinic with endocrinologists, is indicated for all patients.

Protein nutrition

With reduced ability to eat a large volume of food, GPB patients must focus on eating their protein requirements first, and with each meal. In some cases, a liquid protein supplement may be prescribed (e.g. Optifast®).

Calorie nutrition

The significant weight loss that occurs after bariatric surgery is due to the patient now taking in much fewer calories than the body needs. As the body becomes smaller, its energy requirements decrease, while the patient simultaneously finds it possible to eat more food. When the energy consumed is equal to the calories eaten, weight loss will cease. Proximal GBP typically results in the loss of 60-80% excess body weight, and very rarely leads to excessive weight loss.

Vitamins

As the amount of food that will be eaten after GBP is reduced, the vitamin content is correspondingly reduced. Therefore,

supplements should be taken to provide the minimum daily requirements of all vitamins and minerals. Absorption of some vitamins is not seriously affected after proximal GBP, although vitamin B_{12}, calcuim and vitamin D are not well absorbed. Intramuscular preparations of B_{12} will provide adequate absorption. After the distal GBP, the fat-soluble vitamins A, D and E may not be well absorbed, particularly if fat intake is high. Water-dispersible forms of these vitamins may be indicated upon physician recommendation.

Minerals

The duodenum is the primary site of absorption of both iron and calcium. Supplementation of iron and calcium is therefore preferable in all patients. Ferrous sulfate is poorly tolerated. Alternative forms of iron (fumarate, gluconate, chelates) are less irritating, and probably better absorbed. Calcium carbonate preparations should also be avoided. Calcium as citrate or gluconate, 1 200 mg as elemental calcium, has greater bioavailability, independent of acid in the stomach, and is likely to be better absorbed. Vitamin C is a co-factor for the conversion of iron from the ferri to ferro form for absorption.

Alcohol metabolism

Because of the decreased levels of alcoholic dehydrogenase, patients who have undergone GBP surgery will have a lower tolerance for alcohol than people who have not undergone the surgery. It will also take a GBP patient longer to return to a sober level after drinking alcohol than a person who has consumed alcohol and who has not had the surgery.[16]

Pica

Pica is more likely to occur in GBP patients who have a history of the condition prior to the surgery.[17]

Iron

Since iron absorption occurs in the duodenum primarily, malabsorption of ingested iron is the primary cause of post-gastric-bypass iron deficiency. Ferrous sulphate, in normal doses, can cause considerable gastrointestinal distress. Alternatives include ferrous fumarate, or a chelated form of iron. Patients who are intolerant of oral iron, or who have inadequate ferritin levels, will require parenteral iron (e.g. Venofer®, Cosmofer®), six monthly.

Vitamin B_{12} deficiency

After GBP surgery, there is a failure to cleave food-bound vitamin B_{12} from its protein moiety in the upper gastric pouch. Conversely, crystalline B_{12} is absorbed normally in the distal ileum. Vitamin B12 requires intrinsic factor derived from the gastric mucosa to be absorbed. In patients with a small gastric pouch, it may not be absorbed, even if supplemented

orally, and deficiencies can result in pernicious anemia and neuropathies, including Wernicke encephalopathy. Sublingual B$_{12}$ may be adequately absorbed. Vitamin B$_{12}$ deficiency occurs in up to 39% of patients when supplementation is not followed. Intramuscular vitamin B$_{12}$ injections (1 000 μg once, and then once every fourth month), may be required in the majority of cases.

Folate

Although the aetiology of folate deficiency after GBP is unknown, inadequate dietary intake and absorption are probably the most common causes of deficiency. Deficiencies of this micronutrient can result in anaemia.

Thiamine

Thiamine deficiency (also known as beriberi) rarely occurs when its absorption site in the jejunum is bypassed. The deficiency can also result from inadequate nutritional supplements being taken postoperatively.

Fat malabsorption

Fat malabsorption is a result of distal long limb GBP. Patients who have these procedures are prone to develop diarrhoea, malodorous flatus, and deficiencies in fat-soluble vitamins. Fat malabsorption is commonly associated with the long loop or distal Roux-en-Y gastric bypass and mini bypass, but is rarely seen with standard laparascopic Roux-en-Y gastric bypass. Fat malabsorption may lead to a deficiency of the fat-soluble vitamins, including vitamins A, D, E and K, if not replaced. Night blindness, as well as deterioration of general eyesight due to deficiency in vitamin A levels, may occur in these patients after two years.

Hyperparathyroidism

Vitamin D deficiency is present in most obese patients prior to surgery, resulting from NASH and an inability to form 25-OH-vitamin D. Inadequate replacement of vitamin D can lead to hyperparathyroidism. Inadequate absorption can be corrected postoperatively with a dose of 50 000-100 000 U/week is sufficient to prevent hyperparathyroidism.

Trace elements

Deficiencies in copper, zinc and molybdenum may occur, but they are extremely rare.

Conclusion

It is becoming increasingly clear that Roux-en-Y GBP is particularly effective for weight loss as a result of neurohormonal changes. It is important to bear in mind that morbidity remains extremely low post-bariatric surgery, provided that the correct procedure is selected for the best outcome, and that the patient is well prepared and then cared for post-surgery by an expert team. Resolution of diabetes and co-morbidities of obesity range from 80-98%. An experienced and accredited surgeon and medical team are paramount to the success of the patient's treatment.

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