



Surgical Approaches in the Treatment of Obesity

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Phong Ching Lee and John B. Dixon

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P. C. Lee (✉)

Obesity and Metabolism Unit, Department of Endocrinology, Singapore General Hospital, Singapore, Singapore

e-mail: lee.phong.ching@singhealth.com.sg

J. B. Dixon

Clinical Obesity Research, Baker Heart and Diabetes Institute, Melbourne, VIC, Australia

e-mail: john.dixon@baker.edu.au

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Abstract

Bariatric-metabolic surgery is the most effective treatment option for clinically severe obesity that offers significant and durable weight loss. In this chapter, we examine the evidence for the common bariatric surgical procedures. We explore the mechanisms of action of surgery on weight loss and glycemic control, including changes in key hormones related to energy balance and glucose homeostasis. We discuss the role of the endocrinologist in helping decide the appropriate patients for bariatric-metabolic surgery and addressing non-surgical aspects of perioperative care, most notably nutritional and metabolic support. The acute post-surgical and longer-term nutritional and metabolic complications of surgery as well as recommendations for post-operative lifestyle, nutritional and comorbidity follow-up are also reviewed.

Keywords

Bariatric surgery · Metabolic surgery · Weight loss · Obesity

Chapter Objectives

At the end of the chapter, the reader should be able to:

1. Discuss the common bariatric surgical procedures and understand the mechanisms of action.
2. Discuss the evidence for the use of bariatric-metabolic surgery.
3. Understand the mechanisms of weight loss and improvement in obesity-related complications following bariatric-metabolic surgery.
4. Identify and select appropriate patients for bariatric-metabolic surgery.
5. Discuss the appropriate preoperative work-up prior to surgery.
6. Recognize acute and long-term complications after surgery.
7. Appreciate the long-term lifestyle, nutritional and comorbidity follow-up required after bariatric-metabolic surgery.
8. Discuss future directions in surgical approaches to treat obesity, including the use of medical devices.

Introduction

Obesity is a serious chronic disease that is associated with significant morbidity and mortality. Obesity leads to metabolic and mechanical complications including type 2 diabetes (T2DM), hypertension, hyperlipidemia, nonalcoholic fatty liver disease (NAFLD), cardiovascular disease, obstructive sleep apnea, osteoarthritis, and certain cancers. The social, psychological, and economic impact of obesity is also well recognized.

Lifestyle interventions focusing on healthy diet and exercise remain the foundation of obesity management, as they are for other chronic conditions such as T2DM, hypertension, and coronary artery disease. Improvement in obesity-related medical conditions is observed with moderate sustained total body weight loss (TBWL) of 5–10%, which may be achieved with lifestyle interventions (Jensen et al. 2014). However, for the majority, such modest weight loss achieved is usually difficult to sustain in the long-term. This is due to physiological neuro-hormonal changes following weight loss that drive food-seeking behavior to increase energy intake while simultaneously reducing energy expenditure and together these physiological adaptations provide ideal conditions for weight regain and obesity relapse (Sumithran et al. 2011).

Bariatric-metabolic surgery is the most effective treatment option for clinically severe obesity that offers significant and durable weight loss. Surgical alteration of the gastrointestinal (GI) anatomy leads to sustained change in central regulation of energy balance and metabolism. The resulting weight loss provides broad ranging benefits including reduced mortality (especially from cardiovascular disease, diabetes, and cancer), improvement in obesity-related health outcomes, and better quality of life (Mechanick et al. 2013).

Common Types of Bariatric-Metabolic Surgical Procedures

The three most common bariatric-metabolic procedures performed worldwide are sleeve gastrectomy (SG), Roux-en-Y gastric bypass (RYGB), and adjustable gastric band (AGB) (Fig. 1).

Adjustable Gastric Banding

The AGB involves laparoscopic placement of an adjustable silicone band around the proximal stomach just below the gastro-esophageal junction. The band is filled with an isotonic solution (e.g., 0.9% sodium chloride), and the amount of fluid can be adjusted via a subcutaneous reservoir fixed to the anterior rectus sheath. In recent years, the use of AGB has decreased compared with other bariatric surgical procedures such as RYGB or SG. Optimal band adjustment is vital for long-term success with the AGB and can be achieved by aiming for the “green zone” (Fig. 2) in adjusting the band (Dixon et al. 2012).

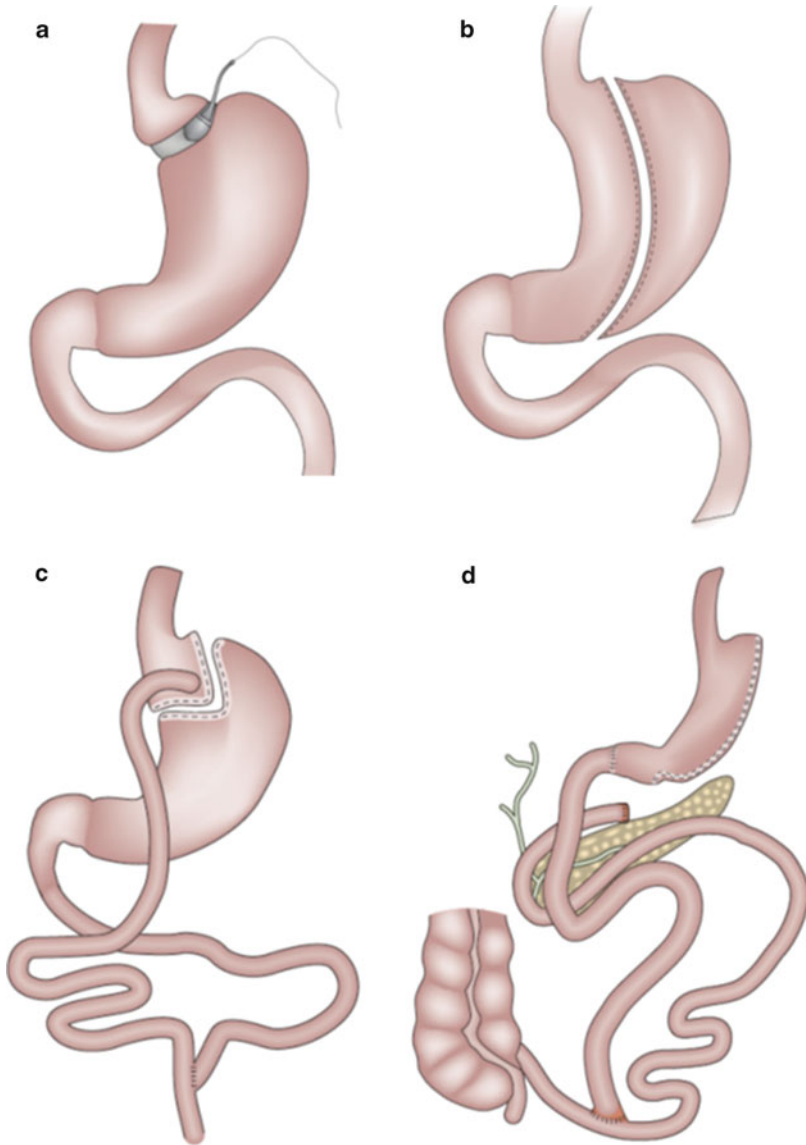


Fig. 1 The four established bariatric-metabolic procedures (Dixon et al. 2011a). (a) Adjustable gastric band, (b) Sleeve gastrectomy, (c) Roux-en-Y gastric bypass, and (d) Biliopancreatic diversion with duodenal switch

Sleeve Gastrectomy

The SG was initially devised as the first part of a two-stage procedure involving biliopancreatic diversion with duodenal switch (BPD-DS). The rationale for the two-step approach was that the weight loss due to SG would reduce morbidity and mortality

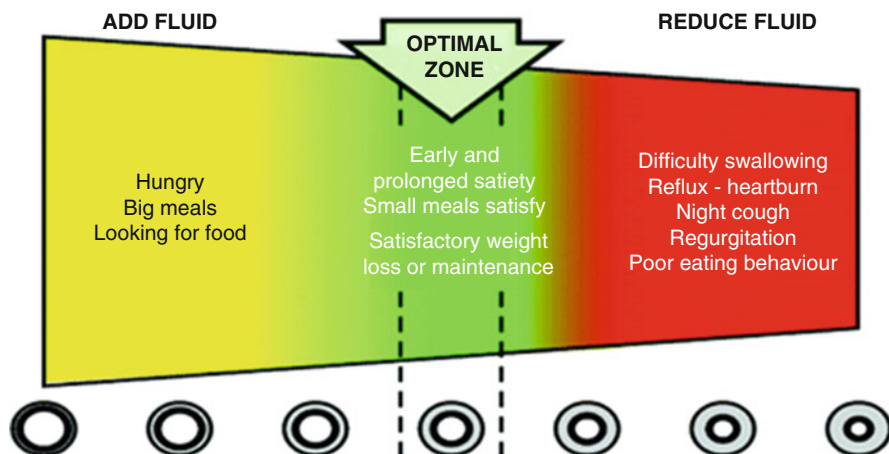


Fig. 2 Green zone for optimal band adjustment

of the more technically demanding BPD-DS procedure, especially in the super-super obese individuals ($\text{BMI} > 60 \text{ kg/m}^2$). However, it was observed that significant weight loss was achieved following SG, which has now cemented its position as a stand-alone procedure to treat clinically severe obesity. Indeed, the SG has now become the most commonly performed bariatric procedure in the US and worldwide.

The SG procedure involves removing the greater portion of the fundus and body of the stomach, creating a long, tubular gastric pouch, or sleeve. Gastric volume is reduced by 80% following the procedure, which leads to changes in various gut hormones and helps to reduce food intake.

Roux-en-Y Gastric Bypass

RYGB is still considered the “standard” bariatric surgical procedure that consists of two parts: gastric volume reduction and proximal alimentary diversion. First, a small gastric pouch of about 15 ml is created, thereby reducing gastric volume. This pouch is then anastomosed to the jejunum, hence diverting nutrients from the distal stomach, duodenum, and proximal jejunum.

Biliopancreatic Diversion – Duodenal Switch (BPD-DS)

As mentioned, the BPD-DS has two components. The first part is similar to the SG, whereby a vertical gastrectomy is performed to create a tubular gastric pouch, with reduction in gastric volume. The distal ileum is then divided and anastomosed to the duodenum, in a Roux-en-Y configuration. A second anastomosis of the ileoileostomy is created approximately 75–150 cm proximal to the ileocecal valve. These anastomoses cause ingested nutrients to effectively bypass about 50% of the

small intestine, leading to significant malabsorption of protein and fat, as well as certain micronutrients and vitamins.

The BPD-DS provides the largest amount of weight loss and improved glycemic control in patients with severe obesity and T2DM, compared with RYGB, SG, or AGB. However, the downsides include increased acute postsurgical complications such as leaks and obstruction and long-term complications such as nutritional deficiencies. Hence, its use is limited and BPD-DS comprises <1% of all bariatric operations in the US (Ponce et al. 2016).

Evidence for the Use of Bariatric-Metabolic Surgery

Bariatric-metabolic surgery leads to significant long-term weight loss which translates to numerous health benefits including reduced mortality especially from cardiovascular disease and cancer, improvement in obesity-related diseases, and improved physical functioning and emotional well-being (Mechanick et al. 2013).

Weight Loss

The Swedish Obese Subjects (SOS) study is the longest and largest prospective study on bariatric-metabolic surgery, which indicated that weight loss of 20–32% maximally achieved at 1 to 2 years after surgery, was sustained at 18% even after 20 years (Sjostrom 2013). Several randomized controlled trials (RCTs) comparing bariatric-metabolic surgery versus intensive medical therapy for T2DM over a period of 12–60 months have shown that the weight loss achieved varies with the different procedures; BPD has the highest total body percentage weight loss (TBWL) (31%), followed by RYGB (25–28%), SG (21%), and AGB (15%) (Nguyen and Varela 2017). Weight loss seen after AGB is slow and gradual, whereas rapid weight loss can be expected with SG, RYGB, and BPD. Weight loss observed in the medical therapy arms range from 4–8%, which was similar to the results from the LOOK AHEAD trial examining intensive lifestyle intervention in individuals with overweight/obesity and T2DM (Look 2014).

Mortality Benefit

The sustained weight loss over the long-term following bariatric-metabolic surgery has led to improved overall survival, with cardiovascular and cancer mortality approximately halved after surgery, when compared to matched nonsurgical cohort (Pontiroli and Morabito 2011; Kwok et al. 2014).

Data for Improvement in Diabetes and Comorbidities

Marked weight loss from bariatric-metabolic surgery leads to corresponding improvements in insulin sensitivity and glycemia, obesity-related dyslipidemia,

inflammatory markers, NAFLD, obstructive sleep apnea (OSA), and ovulatory function and fertility in women with polycystic ovary syndrome. In patients with T2DM, significant improvements in glycemic control are observed in the majority of cases, even to the point of remission of diabetes (stable nondiabetic glycemia of all diabetes medications) (Rubino et al. 2016). RCTs have shown that treatment of obesity and T2DM with bariatric-metabolic surgery is superior to medical therapy in controlling hyperglycemia and cardiovascular risk factors over the medium term (Mingrone et al. 2015; Schauer et al. 2017).

Similar to the weight loss data, the rate of T2DM improvement or remission varies depending on the type of procedure, with BPD-DS being the most effective, followed by RYGB, SG, and AGB. A recent report from the SOS study showed that bariatric-metabolic surgery was associated with reduced risk of microvascular complications over a median follow-up period of 19 years (Carlsson et al. 2017) implicating that improvement in glycemia and other cardiovascular risk markers translate to reduced hard end-points over the longer term.

Apart from T2DM, other obesity-related complications also improve following bariatric-metabolic surgery. A systematic review and meta-analysis have shown that cardiovascular risks factors such as hyperlipidemia, hypertension, and sleep apnea improved in $\geq 70\%$, 78.5%, and 83.6% of patients who had surgery, respectively (Buchwald et al. 2004). These improvements were maintained with fewer medications required to treat T2DM, hypertension, and hyperlipidemia, compared to nonsurgical controls.

Improvement in Quality of Life

It has been estimated that obesity has greater negative impact on quality of life than 20 years of aging, an impact that persists even after accounting for demographics, health habits, medical conditions, and depression (Dixon 2010). Quality of life is an important outcome measure for evaluating the efficacy of an intervention for obesity. Numerous studies using quality-of-life questionnaires have consistently shown substantial improvement in health-related quality of life following weight loss from bariatric-metabolic surgery (Dixon 2010; Schauer et al. 2017).

Mechanisms of Weight Loss Following Bariatric-Metabolic Surgery

The major reason for success with bariatric-metabolic surgery is its ability to alter energy balance and circumvent the body's compensatory physiological responses to weight loss. The sustained weight loss following bariatric-metabolic surgery provides an opportunity to better understand the role of integrated GI physiology in the regulation of energy balance. The body weight regulates at a lower "set-point" following surgery, and a feeling of satiety is created following a small intake of

food. This allows continued weight loss, which is maintained once the body reaches a new set-point.

How surgery modifies these homeostatic mechanisms is still incompletely understood, but there have been several postulations put forth. Earlier constructs based on anatomic “restrictive” and “malabsorptive” concepts do not fit well with more recent clinical observations. There is no significant delay in transit of food within the foregut nor any significant malabsorption of macronutrients observed with major surgical procedures performed such as AGB, SG, or RYGB. Similarly, varying the gastric pouch in RYGB from 10 ml to 30 ml or varying the length of Roux limb from 75 cm to 150 cm does not increase long-term weight loss or improve outcomes.

Bariatric-metabolic surgery leads to changes in key hormones, especially gut hormones, which are related to energy balance and weight loss. Changes in these hormones following the common surgical procedures are shown in Table 1. Ghrelin is the only gut hormone that stimulates appetite. Circulating levels of ghrelin surge before meals and are suppressed by food ingestion, thus implicating the important role of ghrelin in meal initiation and short-term feeding control. Ghrelin levels increase following AGB, as expected for the degree of weight loss but are reduced in SG, due to removal of the gastric fundus, and is variable following RYGB. The role of ghrelin in long-term energy homeostasis and in the action of bariatric-metabolic surgery is still uncertain.

Apart from ghrelin, several other gut hormones have anorexic effects and provide lasting satiety, limiting food intake, and promoting weight loss. One hormone that has a potential important role in the action of bariatric-metabolic surgery is glucagon-like peptide 1 (GLP-1). GLP-1 is secreted from the neuroendocrine L cells in the intestinal mucosa, in response to nutrient stimulation after a meal. GLP-1 has numerous modes of action including stimulation of beta cells to produce insulin, delay of gastric emptying, and suppression of appetite. Indeed, the GLP-1 receptor

Table 1 Summary of changes in key hormones related to energy balance and weight for the established surgical procedures and for intentional dietary weight loss (Sweeney and Morton 2014; Dixon et al. 2015)

	AGB	SG	RYGB	BPD	Diet
Leptin	↓	↓	↓	↓	↓
Insulin	↓	↓	↓	↓	↓
Adiponectin	↑	↑	↑	↑	↑
Glucagon	↔	?	↑	?	↓
Ghrelin	↑↔	↓	↓↔↑	↓	↑
GLP-1	↔	↑	↑	?	↔
PYY	?	↑	↑	↑	↔

↑ and ↓ indicate a substantial number of studies indicate an increase or decrease, respectively

↔ indicates a substantial number of studies find no change

? indicates that there are too few data to provide reliable trends

For some procedures, there are a number of quality studies that show different results for the change in ghrelin following surgery.

There was insufficient data on GIP, CCK, amylin, and PPP to include these in the table.

agonist, liraglutide, has been approved by the USFDA to treat obesity. GLP-1 levels rise following SG, RYGB, and BPD, probably as a result of expedited transit of nutrients to the distal intestine.

Peptide YY (PYY) is another peptide hormone that is produced by the L cells of the gut. PYY levels rise in proportion to calories ingested following a meal and likely affect central appetite control and gut motility. As with GLP-1, PYY levels are also elevated after RYGB and, to a lesser extent, SG. The diminished acute weight loss effects of gastric bypass in PYY knock-out mice suggests that PYY has a key role in mediating early weight loss in bariatric-metabolic surgery (Chandarana et al. 2011).

Vagal afferent receptors in the upper GI tract send signals to the brain in response to gastric distension and nutrient type. All effective bariatric procedures have a gastric component, which affects the gastric mechanosensitive stretch and motility receptors. The role of these afferents in energy homeostasis is exemplified by studies of the AGB. It is thought that food moving through the gastric band area activates vagal sensory afferents embedded in the gastric muscle of the cardia and generates satiation. Given that the AGB does not directly influence GI hormone concentrations or gastric emptying, it is clear that these mechanical changes to the stomach have a significant impact on long-term energy balance.

Bariatric procedures such as SG and RYGB, but not AGB, lead to changes in plasma bile acid (BA) levels. BA are synthesized in the liver, stored in the gallbladder, and then secreted into the duodenum after ingestion of a meal. The BA are then mostly reabsorbed in the terminal ileum, with a small amount excreted in the feces. These BA act on farnesoid X receptor (FXR) and G protein-coupled bile acid receptor 5 (TGR5) and affect glucose, lipid, and energy metabolism. FXR, a bile acid regulator, may influence hepatic glucose handling by inhibiting gluconeogenesis, improving insulin secretion and sensitivity and stimulating glycogen synthesis. BA activation of TGR5 may stimulate GLP-1 secretion from the L cells, leading to improved weight loss. However, as yet, there is no direct evidence for a causal relationship between BA and improved metabolic control after RYGB.

The human gut microbiome performs important functions including nutrient extraction, prevention of pathogenic colonization, and immunomodulation. In obesity and T2DM, gut microbiome composition is altered and diversity is reduced. This diversity is restored after weight loss following bariatric-metabolic surgery. The potential causal relationship between gut microbiome and metabolic control is supported by evidence that fecal transplantation from obese mice to gnotobiotic mice can transmit the abnormal phenotype (Ridaura et al. 2013). In addition, the transfer of fecal microbiodata from patients who had RYGB or vertical banded gastroplasty to germ-free mice resulted in reduced body fat accumulation in recipient mice (Tremaroli et al. 2015). Put together, the evidence suggests that bariatric-metabolic surgery causes specific changes to gut microbiome, which affect weight and metabolic control. At present, it is still unclear whether these changes are specific to the surgical procedure or due to diet-induced weight loss, and whether there are weight-independent therapeutic effects of gut microbiome changes on glycemic control.

Mechanisms of Improved Glycemic Control: Is It All Due to Weight Loss?

In recent years, there has been intense focus on the potential weight-independent benefits of bariatric-metabolic surgery, particularly in the treatment of T2DM. The notion that T2DM can be treated successfully with surgery with little or no weight loss is indeed attractive, but the evidence supporting this is conflicting. One key feature that supports the notion of therapeutic weight-independent metabolic effects of surgery is the observation in two RCTs that different surgical procedures yielded different T2DM remission rates, at relatively similar amounts of weight loss. RYGB leads to higher DM remission as compared with SG (Schauer et al. 2014), and BPD produces better DM remission rates than RYGB (Mingrone et al. 2015), an observation that is disproportionate to the differences in weight loss seen.

There are a number of hypotheses on the potential mechanisms to explain the beneficial weight loss-independent effects on glycemic control that are seen with some bariatric-metabolic procedures. The main focus is on the improvement in insulin sensitivity after surgery and the potential role of GLP-1 as a mediator of diabetes improvement.

Insulin resistance is the hallmark feature of T2DM, and hyperglycemia ensues when the body is unable to produce enough insulin to overcome the insulin resistance. The improvement in insulin resistance seen after bariatric-metabolic surgery is due to two major factors. Acute caloric restriction following RYGB leads to decreased hepatic glycogen stores and glucose production rate and improvement in hepatic insulin sensitivity. This probably accounts for the early improvement in insulin sensitivity seen after surgery, before any significant weight loss has occurred. Subsequently, postoperative weight loss further improves insulin sensitivity in liver, adipose tissue, and skeletal muscle. There are no differences in improvements in insulin sensitivity with RYGB, AGB, or even control subjects, as long as they are matched for calorie intake and weight loss (Chondronikola et al. 2016). On the other hand, BPD appears to have unique effects on insulin sensitivity, with rapid near-normalization of insulin sensitivity early after surgery (Chondronikola et al. 2016; Mingrone and Cummings 2016).

The “incretin” effect is a well-known phenomenon whereby oral glucose elicits a greater insulin secretion response compared with intravenous glucose, at identical plasma glucose levels. The hormones responsible for this effect are GLP-1 and glucose-dependent insulinotropic polypeptide (GIP). Bariatric procedures such as RYGB and SG cause five- to tenfold increases in postprandial GLP-1 secretion, leading to speculation that GLP-1 underlies the metabolic improvement following surgery. However, the role of GLP-1 has been recently challenged. Animal knock-out studies have shown that mice lacking GLP-1 receptors responded similarly to controls after SG (Wilson-Perez et al. 2013). Likewise, studies using GLP-1 receptor antagonist exendin (9–39) demonstrated that by blocking GLP-1 action after SG or RYGB, there is diminished insulin secretion but the impact on glucose tolerance is limited (Jimenez et al. 2013, 2014). In one study, GLP-1 responses to meal stimuli were also almost identical regardless of glycemic status (DM remission,

nonremission, or relapse) at 2 years after SG (Jimenez et al. 2014), suggesting that GLP-1 is not the key determinant in glycemic improvement in T2DM patients after bariatric-metabolic surgery. This was in contrast to other studies, which have suggested that raised GLP-1 remains a major factor for the marked improvement of glucose tolerance after surgery (Jorgensen et al. 2013; Shah et al. 2014). Hence, the contribution of GLP-1 to the glycemic benefits seen after bariatric-metabolic surgery is still not entirely certain. It may be that elevated GLP-1 levels are important in the early glycemic improvement seen after surgery but of diminishing relevance over time as other factors such as beta-cell function and peripheral insulin sensitivity come into play (Madsbad and Holst 2014).

There are other factors that contribute to improved glucose homeostasis, though it is controversial whether these are beyond weight-loss effects. Intestinal insulin resistance seen in obesity is ameliorated after RYGB, with normalization in insulin-stimulated jejunal glucose uptake after surgery (Makinen et al. 2015). This observation correlated with improvement in whole body insulin sensitivity, though it is unclear if the effect is due to weight loss or the UGI tract bypass per se. Brown adipose tissue (BAT) also has a potential role in glucose homeostasis as BAT activation increases glucose uptake and improves whole-body insulin sensitivity (Chondronikola et al. 2016). BAT activity is increased following weight loss, regardless of whether it is induced by caloric restriction alone, AGB, or RYGB. It remains unclear whether RYGB stimulates BAT activity in addition to that observed in diet restriction alone, given that RYGB also leads to increased BA and GLP-1, which in turn, can increase BAT metabolic activity and cause browning of white adipose tissue (Chondronikola et al. 2016).

Bariatric-metabolic surgery may well offer some weight-independent metabolic benefits, in particular with improvement in glycemic control. However, given the strong association between post-operative weight loss and diabetes remission/improvement, and that the most important factor for diabetes relapse is weight regain, weight loss remains the major contributor to the metabolic benefits observed after surgery. The effects of weight loss and acute caloric restriction on glucose and energy physiology are profound and far-reaching, and it would be challenging to discern the exact contribution of bariatric-metabolic surgery over and above these factors.

Indication for Bariatric-Metabolic Surgery

The most widely referenced indications for bariatric-metabolic surgery are now historic and date back to the NIH Consensus Statement in 1991 (NIH Conference 1991). Primarily based on BMI and presence of obesity-related complications, individuals that could benefit from surgery include those with BMI > 40 kg/m² or BMI > 35 kg/m² with one or more obesity-related complications.

Given the mounting evidence of improvement in glycemic control in patients with severe obesity and T2DM who have had bariatric-metabolic surgery, major international diabetes organizations have now proposed bariatric-metabolic surgery

to be an established treatment option in the algorithm to manage T2DM (Rubino et al. 2016). Surgery is **recommended** for all individuals with T2DM and BMI ≥ 40 kg/m², or those with BMI 35–40 kg/m² with inadequate glycemic control despite lifestyle and optimal medical therapy. In addition, surgery may be **considered** in those with BMI between 30–35 kg/m² and uncontrolled hyperglycemia despite optimal medical therapy (Rubino et al. 2016). However, the evidence in this lower-BMI cohort is limited, and long-term data demonstrating net benefit is still lacking.

There are few absolute contraindications to surgery; they include contraindications to general anesthesia, serious blood or autoimmune disorders, active drug or alcohol abuse, and severe, untreated psychiatric illness. Patients with limited life expectancy due to cardiopulmonary or other end-organ failure or metastatic/inoperable malignancy are also not suitable for surgery.

As with any operation, the potential benefits of surgery must outweigh the perioperative and long-term risks of surgery. Bariatric-metabolic surgery is strongly recommended and should be prioritized for individuals who suffer from super obesity (BMI >50 kg/m²) or class III obesity (BMI >40 kg/m²) with serious complications that would respond to weight loss. Surgery is also suitable for younger patients, who are likely to develop complications of obesity and subsequent reduced quality of life over time without active intervention. On the other hand, increasing age is a risk factor for postoperative complications and mortality. Caution is advised if the patient is over 65 years of age.

Procedure Selection: Choice of Procedure

The choice of surgical procedure is guided by the individual's characteristics, aims of therapy, available surgical expertise, and informed patient choice. RYGB provides more weight loss than SG or AGB and may be appropriate for individuals with very high BMI. AGB has lower perioperative risk and reduced early complications compared to RYGB, but higher rate of reoperation for inadequate weight loss (Tice et al. 2008), and thus may be suitable for older individuals where the extent of weight loss and nutritional risks can be controlled, or for those at lower BMI ranges. Apart from the BPD (which is less commonly performed due to concerns surrounding malabsorption), RYGB provides the greatest rate of diabetes remission, a consideration for T2DM patients. The presence of gastroesophageal reflux disease (GERD) may also be another determining factor: RYGB and AGB usually improve GERD symptoms whereas SG may exacerbate GERD. Finally, good quality postoperative care is crucial to the success of the AGB, as regular adjustments to the band are often necessary to maintain optimal function. The decision for AGB should take into account the availability of an appropriate after-care program. A large English RCT (the By-Band-Sleeve study) is ongoing to evaluate the comparative effectiveness and cost-effectiveness of the three most commonly performed bariatric surgical procedures (AGB, RYGB, and SG) in

severe and complex obesity (Rogers et al. 2014), and results of the study, when available, could further guide procedure selection.

Predictors of Weight Loss and Metabolic Benefits

One of the holy grails of bariatric medicine is the preoperative identification of individuals that would do well (or for that matter badly) following bariatric-metabolic surgery. Successful outcomes following surgery seem largely dependent on inherent physiological-biological factors, rather than psychological and environmental factors. The performance of bariatric-metabolic surgery may not be as strongly influenced by patient compliance but rather driven by the physiological and hormonal changes that occur following surgery. Hence, it is damaging and unfair to blame the patient for lack of effort with regard to diet and exercise when weight loss is less than expected.

The search continues in isolating the factors that can predict weight loss outcomes following surgery. Preoperative weight loss has been touted as an indicator of “intrinsic motivation” that may guide postoperative weight loss. However, many of the studies were retrospective; uncontrolled studies and a systematic review has shown that the relationship between preoperative weight loss with weight change postoperatively is tenuous at best (Livhits et al. 2012).

Given that biology appears to play the central role in determining successful weight loss following surgery, there is substantial interest in the investigation of genetic factors that may predict individual responses to bariatric-metabolic surgery. However, the correlation between genotype and treatment outcome is still unclear, made more complex with the influence of epigenetics and the environment. At present, potential genetic markers or biomarkers of weight loss following bariatric-metabolic surgery have been limited.

As with any form of weight-loss intervention (or indeed most medical interventions), the distribution of outcomes following bariatric-metabolic surgery follow a normal distribution with a broad standard deviation, and variance is poorly explained. Therefore, there is a wide range of weight loss results that can be classed as “normal” and yet disappointing. This is common in managing a chronic disease.

Meanwhile, the striking metabolic benefits that bariatric-metabolic surgery could offer has spurred intense interest in the potential factors that can influence diabetes outcomes after surgery, and if surgery could potentially be employed primarily as a “metabolic” procedure in nonobese individuals with T2DM. It is worthwhile to note that the pathophysiology of diabetes is the integral factor that determines the degree of metabolic improvement after surgery. The two main determinants of glucose homeostasis are insulin resistance and insulin secretion. In individuals with obesity, insulin resistance usually predominates whereas those with normal weight may have a greater element of insulin secretory defect. Given that insulin resistance generally decreases proportionately to the amount of weight loss (Nannipieri et al. 2011; Ikramuddin et al. 2014), the efficacy of surgery in generating both weight loss and

glycemic control appears attenuated in normal to overweight individuals with T2DM compared with those who are obese (Dixon et al. 2013; Lee et al. 2015).

On the other hand, the improvement in insulin sensitivity and reduced demand on islet cells following weight loss may be insufficient if pancreatic beta-cell dysfunction is the main pathophysiologic driver for hyperglycemia. Approximately 50% of beta-cell function is already lost even at diagnosis of T2DM, with continuing decline in beta-cell function over time (Holman 1998). Hence, individuals with shorter duration of T2DM would be expected to have higher insulin secretory capacity and higher probability of achieving diabetes remission after surgery. Similarly, other factors that reflect better beta-cell function such as better baseline glycemic control, non-insulin requiring, higher c-peptide levels are positively correlated with likelihood of diabetes remission. Given the above, there are compelling reasons for consideration of bariatric-metabolic surgery, early in the diagnosis of T2DM, before the onset of significant diabetic complications or beta cell exhaustion. Indeed, the UK NICE guidelines have recommended that surgery should be prioritized and expedited for those with recent-onset T2DM (within 10 years of diagnosis) (Welbourn et al. 2016).

Preoperative Assessment and Preparation

Once a decision has been made for bariatric-metabolic surgery, a series of detailed assessments should be organized. Pre-operative assessment involves identification and optimization of obesity-related complications, with the aim to improve perioperative safety and outcomes after surgery.

T2DM is a well-recognized complication of obesity and a major focus of bariatric-metabolic surgery. In the absence of history of T2DM, routine screening for DM using established methods (fasting glucose, 75 g oral glucose tolerance tests or glycosylated hemoglobin) is recommended and would detect presence of undiagnosed DM. For those with preexisting T2DM, serum c-peptide levels may be useful as a surrogate for beta-cell reserve and aid in assessment of the likelihood of diabetes remission following surgery.

Perhaps surprisingly, nutritional deficiencies are often observed in clinically severe obesity, which is masked by ample energy excess. Up to 80% of bariatric-metabolic surgery candidates have micronutrient deficiencies preoperatively, with common deficiencies being iron, vitamin B₁₂, folate, and vitamin D. Appropriate nutritional assessment allows deficiencies to be detected and corrected prior to surgery.

An essential element of preoperative assessment of any surgical patient involves evaluation of the patient's cardiorespiratory status and cardiac risk. After a focused cardiac history and physical examination, an electrocardiogram (ECG) is often obtained routinely. Referral to a cardiologist for more extensive evaluation would be appropriate for patients with preexisting cardiac disease, high cardiovascular risk, or abnormal ECG. OSA is extremely common in the cohort of patients with clinically severe obesity, with some estimates indicating prevalence of OSA as high as 88%. Untreated OSA may develop pulmonary hypertension and are at risk

of cardiac arrhythmias as a result of chronic nocturnal hypoxemia. Patients should be screened for symptomatic OSA using the STOP-BANG questionnaire and if at risk, an overnight polysomnography arranged. These can now be readily performed at home. Continuous positive airway pressure (CPAP) therapy is the mainstay of treatment for moderate to severe OSA, and if used, a period of stabilization is recommended before surgery to allow for adaptation to the device.

Prior to surgery, thorough assessment of the patients' psychosocial situation and their ability to incorporate nutritional and lifestyle changes should be conducted. Unrealistic expectations or incorrect beliefs on what the procedure can achieve must be rectified. Depression, anxiety, binge eating disorder, and other psychiatric disorders are prevalent in individuals considering bariatric-metabolic surgery, and further evaluation by psychologist or psychiatrist recommended if psychiatric illness is suspected.

As part of the preoperative preparation, it is common to institute very low energy diets (VLED) with the aim of achieving weight loss prior to surgery. Preoperative weight loss can preferentially reduce liver volume and visceral adiposity (Colles et al. 2006), which may ease technical aspects of surgery and lead to improved short-term outcomes (Tarnoff et al. 2008). In T2DM, preoperative weight loss with medical nutrition therapy can also improve glycemic control. The VLED, which consists of meal replacements providing ≤ 800 kcal/day, is usually started at least 2 weeks prior to surgery. During VLED, patients with T2DM are recommended to self-monitor their capillary blood glucose regularly, especially if they are on insulin or insulin secretagogues. Reductions in insulin doses are often necessary whilst on VLED, in order to prevent hypoglycemia.

Risks and Complications of Bariatric-Metabolic Surgery

Bariatric-metabolic surgery is generally regarded as safe, with low morbidity and mortality that is comparable to elective laparoscopic cholecystectomy. Meta-analysis of observational studies has shown that the AGB had the lowest perioperative and postoperative mortality rates (0.07% and 0.21%), followed by SG (0.29% and 0.34%), and then RYGB (0.38% and 0.72%) (Chang et al. 2014). The converse is true for reoperation rates. RCT data have suggested that RYGB has the lowest reoperation rate (3%), followed by SG (9%), and AGB (12%) (Chang et al. 2014). However, the complexity and risks of reoperations are greater with the more complex procedures.

Surgical and GI Complications

Surgical complications can be general, such as can occur after any surgical procedure, or specific to the type of procedure performed. The procedure-specific complications and "red flag" symptoms that should prompt referral to the bariatric surgical team are shown in Table 2.

Table 2 “Red flag” symptoms and procedure-specific complications of AGB, SG, and RYGB

Type of procedure	“Red flag” symptoms	Potential causes to rule out
AGB	Inability to achieve lasting satiety/ inadequate weight loss despite optimal band adjustment	Erosion, leaks in the band system (usually tubing or port), unbuckled band, and proximal gastric pouch dilatation
	Intractable reflux symptoms Persistent cough, pneumonia Dysphagia with solids and liquids	Over-filled band Proximal gastric pouch enlargement
	Abdominal pain, low-grade fever, port site infections	Band erosion (early or late)
SG	Fever, tachypnea, tachycardia, abdominal pain	Staple-line leak (early)
	Intractable reflux symptoms	GERD and hiatal hernia
	Dysphagia, vomiting	Stricture/stenosis (early or late)
RYGB	Dysphagia, nausea, vomiting	Gastrojejunal strictures (early or late)
	Abdominal pain	Leak over anastomotic junction (early) Internal hernias (late), marginal ulcer
	Iron-deficiency anemia	Inadequate supplementation, marginal ulcers
	“Dumping syndrome” early and late	Hypoglycemia syndromes

Hemorrhage is a general early complication that occurs in up to 4% of cases, though rates vary depending on operative experience and complexity of cases. Enteric leaks are another early complication that can be difficult to manage and can potentially cause severe peritonitis, sepsis, and multiorgan failure. Leakage can occur at any of the anastomotic junctions or staple lines in RYGB, SG, or BPD, most commonly at the gastrojejunostomy junction with RYGB, and high on the staple line with SG. There is no anastomosis with the AGB, and perforation of the gastroesophageal junction is an uncommon complication with incidence of <0.5% (Neff et al. 2013).

Regurgitation is common after surgery and is often due to eating too much or too quickly. However, more serious complications such as stricture, stoma stenosis, or herniation must be excluded with relevant clinical and radiological evaluations. Gastrojejunal strictures can occur after RYGB in about 10% of individuals, with typical symptoms of dysphagia and vomiting (Neff et al. 2013). Diarrhea is reported in up to 40% after RYGB with an unclear etiology (Neff et al. 2013). Treatment involves dietary modification and antidiarrheal medications.

Late complications (occurring >30 days after surgery) include marginal ulcers, internal hernias, strictures, small bowel obstruction, and gastrogastric fistula. Internal hernias with the reported frequency of 0.4–5.5% in RYGB and up to 38% in BPD have the potential to cause bowel obstruction. Late complications that can arise from AGB include proximal gastric enlargement, erosion, or migration of band and leaks of the band system. High rates of these complications that require reoperation, as well as variability in weight loss and lack of appropriate after-care program has contributed to the decline in popularity of AGB in Europe and US.

The rapid emptying of gastric contents after RYGB, or less commonly SG, can lead to postprandial symptoms, a phenomenon known as dumping syndrome. Symptoms of dumping syndrome are varied and can include diarrhea, nausea, bloating, facial flushing, palpitations, hypotension, and syncope after meals rich in simple carbohydrates. These vasomotor symptoms typically occur within 1 h after eating and are classified as “early” dumping symptoms. “Late” dumping symptoms can include sweating, palpitations, hunger, tremors, confusion, and syncope and are thought to be usually due to exaggerated incretin response leading to hypoglycemia, though the definitive cause is yet to be identified. Treatment of dumping syndrome involves dietary modification by avoiding concentrated simple sugars and aiming for small regular meals consisting of protein and complex carbohydrates with low glycemic index. Occasionally, pharmacotherapy with acarbose may be needed. Rarely, endogenous hyperinsulinism due to nesidioblastosis or insulinoma have been reported after surgery (Service et al. 2005), so if symptoms are unresponsive to dietary modification or if there are atypical symptoms, then full workup for hypoglycemia to evaluate for endogenous hyperinsulinism is warranted.

Long-Term Nutritional Complications

Another major concern after bariatric-metabolic surgery relate to nutritional deficiencies, which depend on the type of surgery performed (common nutritional concerns for each procedure shown in Table 3). More aggressive surgical procedures which promote greater weight loss would also incur higher risk of nutritional deficiencies, with their attendant long-term complications. Short common channels in procedures such as BPD and duodenal switch lead to more malabsorption, due to reduced opportunity for mixture of bile and pancreatic secretions with small intestinal chyme before nutrient absorption (Bal et al. 2012). The concern surrounding malabsorption and malnutrition is the major reason BPD is not widely performed.

Table 3 Common nutritional concerns for each procedure (Dixon et al. 2011a)

	AGB	SG	RYGB	BPD / BPD-DS
Iron	+	+++	+++	+++
Thiamine	+	++	+	+
Vitamin B₁₂	+	++	+++	++
Folate	++	++	++	++
Calcium	+	++	++	+++
Vitamin D	+	+	++	+++
Protein	+	+	+	++
Fat soluble vitamins and essential fatty acids	+	+	+	+++

+ Recommended daily intake (allowance) or standard multivitamin preparation likely sufficient

++ Significant risk of deficiency or increased requirements. Specific supplementation is appropriate especially in higher risk groups

+++ High risk of deficiency. Careful monitoring is recommended. Supplementation well in excess of daily requirements may be necessary to prevent deficiency

Although the standard RYGB does not produce significant malabsorption of macronutrients, long-term vitamin or mineral deficiencies can occur due to gastro-duodenal exclusion, major sites of micronutrient preparation and absorption.

The recommended daily protein intake postoperatively is 60–120 g, (Heber et al. 2010; Mechanick et al. 2013) to enhance healing, maintain adequate protein stores, and stem the loss of lean body mass. Protein malnutrition remains a concern after bariatric-metabolic surgery, especially BPD and some forms of gastric bypass, with patients often requiring high-quality protein foods and protein supplements to achieve adequate protein intake. Signs of protein deficiency include edema, loss of lean muscle mass, and hair loss, as well as biochemical findings of anemia and hypoalbuminemia. In severe protein malnutrition, which is not responsive to oral protein supplementation, parenteral nutrition or naso-jejunal feeding may be considered.

Multiple studies have reported on the prevalence of nutritional deficiencies after bariatric-metabolic surgery. However, these results have to be taken in context with the preoperative nutritional status as well as nutritional stores of the individual nutrients. A large study which examined 318 patients at 1 year after RYGB showed various micronutrient deficiencies; vitamin A (11%), vitamin C (34.6%), vitamin D (7%), thiamine (18.3%), riboflavin (13.6%), vitamin B6 (17.6%), and vitamin B₁₂ (3.6%) (Clements et al. 2006). Chronic micronutrient deficiencies have detrimental long-term consequences, including nutritional anemias, metabolic bone disease, and neurological complications.

Nutritional Anemias

A meta-analysis in 2015 have shown that prevalence of anemia nearly doubled in the 12 months after RYGB (Weng et al. 2015). Serum ferritin levels, an indicator of iron stores were lower at 6 months after surgery and continued to decline at 24 and 36 months. Reasons for iron deficiency include changes in dietary composition with reduced meat and dairy intake; hypochlorhydria, which decreases bioavailability of dietary iron; and bypass of the stomach, duodenum and proximal jejunum, where physiological iron absorption takes place. Iron deficiency anemia presents as microcytic, hypochromic anemia and symptoms include fatigue, weakness, pallor, anorexia, depression, light-headedness, hair loss, and koilonychia. Oral iron supplementation (ferrous sulfate, fumarate, or gluconate) may be needed to prevent iron deficiency after surgery, especially in menstruating women (Mechanick et al. 2013). Vitamin C can increase iron absorption and help with resistant iron deficiency (Mechanick et al. 2013). Intravenous iron infusions may be preferable to oral administration in severe iron deficiency, as it replenishes iron stores quicker and is usually better tolerated than large doses of oral iron replacement.

Vitamin B₁₂ deficiency is common after RYGB and can occur following SG, due to impaired stomach acidity and intrinsic factor secretion, which facilitate B₁₂ absorption. B₁₂ deficiency is usually not seen in the short-term due to hepatic and renal stores that can last for up to 3 years. The prevalence of B₁₂ deficiency is 3.6% at 12 months after RYGB but rises to 61.8% at ≥ 5 years after RYGB (Bal et al. 2012). Manifestations of B₁₂ deficiency include macrocytic anemia, leucopenia, glossitis, thrombocytopenia, paresthesia, and irreversible neuropathies. Both B₁₂ and folate are required for

maturation of the erythrocyte and deficiencies in either nutrient can lead to macrocytic anemia. Folate deficiency has also been associated with neural tube defects and cardiovascular disease. Treatment of B₁₂-deficiency involves B₁₂ replacement, orally or intranasally, with parenteral (intramuscular or subcutaneous) supplementation if adequate B₁₂ levels cannot be maintained (Mechanick et al. 2013). Folate deficiency can be treated with 1–5 mg of oral folate daily (Bal et al. 2012). Women who are planning pregnancy should also take 1 mg of folic acid daily as a routine supplement to reduce risks of neural tube defects in the fetus (Bal et al. 2012).

Metabolic Bone Disease

Clinically severe obesity is associated with impaired bone health due to abnormalities in mineral metabolism such as vitamin D deficiency and secondary hyperparathyroidism (Rousseau et al. 2016). T2DM (which closely tracks obesity) may also adversely impact bone health due to factors including effects of hyperglycemia, adipokines, and antidiabetic medications.

The detrimental effect on bone is compounded following bariatric-metabolic surgery, due to various factors including decreased calcium and vitamin D intake and absorption as a result of surgically induced anatomical changes, change in hormonal and metabolic milieu, and weight loss with consequent skeletal unloading and loss of lean body mass (Yu 2014). Initial concerns about skeletal health following bariatric-metabolic surgery involve observations of sequelae of severe calcium and vitamin D deficiencies such as osteomalacia, osteoporosis, and brown tumors that were seen after earlier bariatric-metabolic procedures. These complications have declined with more aggressive vitamin and mineral supplementation and a shift towards less malabsorptive procedures. However, despite calcium and vitamin D supplementation, longitudinal studies have still shown marked bone loss and increase in bone turnover markers after bariatric metabolic surgery (Yu 2014).

There are challenges in obtaining accurate bone mineral density (BMD) using dual-energy X-ray absorptiometry (DXA) scans in severely obese patients and during weight loss. Technical issues include unpredictable impact of soft tissue artifact on DXA, increasing precision errors with increasing BMI and changing fat-lean tissue ratios in the region of interest. Despite these limitations, markedly increased bone resorption markers and quantitative computed tomography measurements of BMD support the data on bone loss after bariatric-metabolic surgery (Yu 2014).

Several population-based cohort studies have examined the effect of bariatric-metabolic surgery on fracture rates (Nakamura et al. 2014; Rousseau et al. 2016; Yu et al. 2017). Most studies showed higher fracture risk with RYGB, though one study showed no increased fracture risk over 2.2 years in a cohort predominantly consisted of AGB patients (Lalmohamed et al. 2012). A recent study suggested a positive relationship between risk of fracture and degree of obesity; highest in the group undergoing bariatric-metabolic surgery, followed by obese controls, and lowest in nonobese controls (Rousseau et al. 2016). In the study, fracture incidence increased after surgery and was site specific, changing from a pattern associated with obesity to a pattern typical of osteoporosis (Rousseau et al. 2016). Another recent study

estimated that RYGB is associated with a 43% increased risk of nonvertebral fracture compared with AGB, with risk increasing >2 years after surgery (Yu et al. 2017).

Given the adverse effect of bariatric-metabolic surgery on bone health, vigilance against nutritional deficiencies and ongoing bone loss is imperative. Despite limitations of DXA scans, they remain the most practical and accessible method of measuring BMD and are recommended at baseline and subsequently every 1–2 years after surgery until BMD stabilizes (Mechanick et al. 2013). Adequate calcium and vitamin D supplementation is also important to maintain sufficiency and avoidance of secondary hyperparathyroidism (Mechanick et al. 2013).

Neurologic Complications

The estimated incidence of neurologic complications following bariatric-metabolic surgery can be as high as 5%, with the majority being consequences of vitamin (most commonly thiamine and vitamin B₁₂) or mineral (most commonly copper) deficiency. Risk factors for development of neuropathy include rate and amount of weight loss, prolonged gastrointestinal symptoms such as vomiting or diarrhea, lack of vitamin or mineral supplementation, low serum albumin and transferrin (as a marker of poor nutritional status), postoperative surgical complications requiring hospitalization, and nonattendance at nutrition clinic after surgery (Ba and Siddiqi 2010).

Perhaps the most feared neurologic complication is Wernicke's encephalopathy, a manifestation of thiamine deficiency that can lead to permanent neurologic disability. Being a water-soluble vitamin, the body has limited thiamine stores and deficiencies can occur within days to weeks after surgery. The classical triad of Wernicke's encephalopathy includes ataxia, ophthalmoplegia, and confusion, though not all features are seen in most patients. Intractable vomiting, though, is a common theme amongst patients who develop this complication, and prophylactic thiamine replacement should be considered in these patients. In post bariatric-surgery patients, polyradiculopathy mimicking Guillain-Barre syndrome can also be seen in thiamine deficiency (Becker et al. 2012). The diagnosis of thiamine deficiency can be confirmed by measuring red blood cell transketolase activity, a thiamine-dependent enzyme, but if suspected treatment should not be delayed pending the result. Treatment includes at least 250–500 mg of intravenous thiamine daily for 3–5 days and supportive care while avoiding intravenous dextrose (Mechanick et al. 2013). Refractory thiamine deficiency should raise the suspicion of small intestinal bacterial overgrowth.

Neurologic presentations of B₁₂ deficiency include peripheral neuropathies, depression, paresthesia, spastic paralysis, decreased reflexes, and loss of proprioception. All patients should receive B₁₂ supplementation after bariatric-metabolic surgery, although complications can take months to years to develop, by which time both patients and clinicians may be lax about supplementation.

Copper deficiency can occur more than 10 years after RYGB, and is likely due to reduced absorption due to bypass of the stomach and duodenum. It can also result from excessive zinc supplementation, which can interfere with copper absorption. The clinical features and neuroimaging findings closely resemble B₁₂ deficiency, with neurologic syndrome of myeloneuropathy-like disorder with spastic gait and

sensory ataxia. Treatment of copper deficiency involves intravenous copper 2–4 mg daily for 6 days (Mechanick et al. 2013), but neurologic complications may not be fully reversible even after replacement.

Sarcopenia

Lean mass is integral to long-term maintenance of metabolic rate, core body temperature, skeletal integrity, muscle strength, functional capacities, and loss of lean mass is a major factor that contributes to disability in obesity. The combination of low lean mass and high fat mass, known as sarcopenic obesity, works synergistically to adversely impact on numerous health outcomes including hypertension, arterial stiffness, NAFLD, insulin resistance, functional capacity, and activities of daily living.

Presence of sarcopenia does not seem to adversely influence bariatric surgical outcomes after RYGB or SG (Mastino et al. 2016). Sarcopenic patients had similar operative risks, complication rates, and improvement in comorbidities, compared to their nonsarcopenic counterparts. However, management goals for sarcopenic obesity have to focus on maintenance or accretion of lean mass during the period of weight loss, in order to maintain muscle strength and function. This can be achieved with regular aerobic and resistance exercises following surgery. Indeed, several studies incorporating exercise program in postbariatric surgery patients have shown numerous benefits of exercise including improvement in muscle strength, lean mass, aerobic fitness, mobility, coordination, and postprandial blood glucose levels (Shah et al. 2011; Campanha-Versiani et al. 2017; Coleman et al. 2017).

Prevention of Long-Term Complications

Adherence to post-operative clinical follow-up and nutritional monitoring and supplementation is key to reduce the risks of long-term complications after surgery. Nutritional screening allows for early detection and treatment of deficiencies, although costs do add up over the long-term. Algorithms have been developed to optimize cost-effectiveness of the nutrient panel by reducing the extensiveness of testing without sacrificing detection of clinically relevant deficiencies (Bazuin et al. 2017).

Patients are usually aware of the need for supplementation in the early post-operative period. However, they may believe that it is no longer necessary when their eating habits have stabilized or healthy weight achieved. Some may find the frequency or costs of taking pills in the long-term hard to sustain. An ongoing educational process is needed to remind patients that the nutritional complications of bariatric-metabolic surgery can occur even years to decades after the surgery and on the importance of long-term supplementation and follow-up.

The level of nutritional supplementation should depend on the type of surgical procedure performed, though literature is scant on the micronutrient requirements after bariatric-metabolic surgery. The Endocrine Society recommends that individuals who have had bariatric-metabolic surgery should receive one to two

multivitamins with minerals daily (including at least 1200 mg of elemental calcium and 1000 U of vitamin D3 per day) (Heber et al. 2010). However, procedures with significant malabsorptive component such as extended gastric bypass surgery or BPD-DS would likely require more supplementation.

Psychological Impact of Bariatric-Metabolic Surgery

Bariatric-metabolic surgery can have profound psychological impact on patients due to variety of reasons including large amounts of weight loss achieved, changes in gut-brain hormone signaling pathways, changes in alcohol absorption and metabolism and psychosocial adaptation to post-surgical lifestyle. Consistent evidence have shown overall improvement in psychopathology, self-esteem, body image, and mental quality of life after bariatric-metabolic surgery, with decrease in depressive symptoms and psychiatric medication use (Yen et al. 2014). There is also a tendency for better cognitive function, with improvement in memory, attention, and executive function (Yen et al. 2014).

Despite overall benefit postoperatively, there remains a sizeable minority of patients who either report temporary mental health benefits only or no psychological improvement at all (Kubik et al. 2013). One potential explanation could be that pre-operative patient beliefs that life will dramatically change after bariatric-metabolic surgery may have detrimental effect on psychological health, if these expectations are not met. Presurgical problems that were previously attributed to obesity, but subsequently persist after surgery, may also disappoint.

Similarly, risks of self-harm among bariatric-metabolic surgery patients warrant attention. A recent large cohort study suggests that the risks of self-harm emergencies are higher after surgery than before surgery, with those with a history of mental health issues and living in rural areas particularly vulnerable (Bhatti et al. 2016). Unlike other psychopathologies that improve after bariatric-metabolic surgery, suicide risk remains high and requires long-term monitoring. The reasons for this are still unclear but it is possible that alteration of ghrelin signaling pathways may have a role (Dixon 2016). Evidence suggests that ghrelin may have important central effects beyond energy homeostasis, including learning, memory, reward, motivation, stress responses, anxiety, and depression (Andrews 2011).

Maladaptive and disordered eating behaviors are increasingly recognized after bariatric-metabolic surgery. Some examples include routinely choosing foods with liquid consistencies such as soups and shakes over solid foods, which allow for more volume to be ingested, intentional vomiting or regurgitation after meals, and grazing on food throughout the day. A detailed food diary or dietary history may highlight the presence of an abnormal eating pattern. If persistent, maladaptive eating patterns could lead to weight regain and increase risks of nutritional deficiencies and dental disease. These concerns highlight the importance of adequate support for patients' mental health and psychosocial needs and for access to a clinical psychologist when appropriate (Welbourn et al. 2016).

Future Directions

Medical Devices

The success of bariatric-metabolic surgery in delivering significant and durable weight loss indicates that manipulation of the GI tract holds the key in modifying the central regulation of energy balance. There is great interest in the development and use of medical devices targeting the GI tract as a form of less-invasive therapy that could bridge the safety-efficacy gap between lifestyle interventions and bariatric-metabolic surgery (Lee and Dixon 2017). Apart from the AGB, three other types of devices have been approved by USFDA over the past 2 years to treat obesity, including intragastric balloons, vagal nerve neuromodulation, and gastric emptying systems.

There has been decades-long experience with the use of intragastric balloons to treat obesity. It is indicated in individuals with BMI 30–40 kg/m² for 6-month period of therapy, with TBWL ranging from 6.6–10.2%. The vagal nerve blocking system involves surgical placement of neuromodulator device with electrodes connected to infra diaphragmatic vagal nerve trunks that electrical stimuli to intermittently block vagal nerve signals, with TBWL of 9.2% reported at 1 year. The gastric emptying system involves endoscopic placement of a gastrostomy tube, which allows patients to aspirate gastric contents and ingested food, with reported TBWL of 12.1% at 1 year.

Endoluminal and Novel Surgical Techniques

The quest for better surgical procedures continues to evolve and aims to address certain limitations and complexities of current surgical techniques. These include single anastomosis gastric bypass, single anastomosis duodenal switch, and gastric plication. The adoption of laparoscopic techniques in performing the various bariatric surgical procedures today has also greatly reduced the mortality and complication risks of surgery. There is a drive towards even less invasive approaches, with the use of endoscopic platforms to pursue the goal of incisionless surgery. Endoluminal gastric plication can be performed with suturing and stapling devices. These devices allow gastric partitioning to reduce the size of the gastric pouch, mimicking bariatric surgical plication. Early data involving small numbers of patients showed promising TBWL of 15–19% over 6–12 month period (Dargent 2016). These techniques remain investigational in nature and require sufficient good quality short- and long-term data through vigorous studies before they can be accepted as established therapies.

Summary

In patients with severe obesity, bariatric-metabolic surgery provides large and sustained weight loss, which is otherwise difficult to achieve due to the homeostatic feedback control of energy balance. Manipulation of the gut has provided effective solutions, although our understanding of the mechanisms involved is still

incomplete. With increasing knowledge of the gut-brain interactions concerning weight and energy homeostasis, we have tremendous opportunity to develop more targeted and less invasive therapies.

The long-term weight loss following bariatric-metabolic surgery also translates to improvement and remission of obesity-related complications (especially T2DM), better quality of life and survival. Guidelines pertaining bariatric-metabolic surgery are evolving with the emerging evidence. Indications for surgery may broaden in the future, and BMI cut-offs may be lowered such as for those with BMI < 35 kg/m² with T2DM or other metabolic disease. However, surgery is clearly not appropriate for all individuals with severe obesity, and careful multidisciplinary assessments are needed to ensure suitability prior to recommending surgery. As with any other procedure, bariatric-metabolic surgery is not without its complications and long-term follow-up and nutritional supplementation is crucial for safe and successful outcomes.

Cross-References

- ▶ [Roles of Gut Hormones in the Regulation of Food Intake and Body Weight](#)
- ▶ [The Microbiota and Energy Balance](#)

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