Metabolic Consequences of Bariatric Surgery

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Abstract: Obesity has gained prominence as a main cause of preventable illness and death in the developed world. Surgical therapy for obesity is extremely effective in terms of weight reduction and amelioration of comorbidities. Bariatric procedures are not simply cosmetic operations, however, and involve considerable manipulation of the gastrointestinal tract to induce weight loss. The metabolic consequences of these procedures can be severe if not preempted with relatively simple postoperative precautions on the part of the patient and surgeon. Modern bariatric procedures are much safer than their predecessors, but nutritional and metabolic changes must be anticipated and compensated to fully realize the benefits of surgery. The metabolic consequences of the now outdated jejunoileal bypass, and the more modern Roux-Y gastric bypass, gastroplasty, and biliopancreatic diversion, are presented here, along with specific considerations of patient populations.

Key Words: bariatric surgery, metabolism, weight loss

(J Clin Gastroenterol 2006;40:659–668)

Obesity and its comorbidities dominate American health care concerns. Data from the National Health and Nutrition Examination Survey (NHANES) shows that 31% of Americans are obese, with a body mass index (BMI) of 30 kg/m² or greater, and that another 30% are overweight (BMI 25 to 29 kg/m²) with risk for developing obesity. These percentages represent a disturbing trend in excess weight in the American population since systematic data reporting began. More troubling is the massive rise in obesity in children and adolescents, with 15% of these individuals overweight for age in 1999 to 2000, as compared with 4% to 5% from the 1960s to 1980. The sequelae of such weight excess are seen in every organ system and medical specialty. Obesity is closely tied to the development of type 2 diabetes, cardiovascular disease, a number of types of cancer, and respiratory disorders such as asthma and obstructive sleep apnea. Obesity contributes to 600,000 deaths per year, up from 300,000 less than a decade ago. The price of caring for persons with obesity-related conditions is estimated at 5.7% of total medical expenditures in the United States. Overweight and obese individuals cost US business $2.6 billion and $10.1 billion, respectively, per year.

Diets and increased physical activity represent the first line of therapy for obesity, but are largely unsuccessful in achieving durable weight loss. Low-calorie or low-carbohydrate diets can produce up to 10% weight loss at 6 months, but attrition is high, and weight losses are not well-preserved 12 months after beginning diets or new physical regimens. Pharmacologic therapy can be combined with diet regimens to moderately boost their effect. Modern prescription medications include sibutramine and orlistat. Sibutramine is a mixed noradrenergic-serotonergic reuptake inhibitor with appetite suppressant effects, whereas orlistat inhibits intestinal lipases and prevents the hydrolysis and subsequent absorption of dietary fat. Both medications have been shown to give approximately 4 kg greater weight loss than placebo, and may be used for weight maintenance. Sibutramine’s limiting side effect is a dose-dependent increase in blood pressure and pulse, whereas orlistat is associated with steatorrhea, fecal incontinence, and risk of fat-soluble vitamin deficiency.

Surgical options for weight reduction provide larger and more durable results. Gastric bypass, the most commonly performed bariatric procedure in the United States, typically gives more than 30% weight loss at 12 months, that is maintained up to 10 years. Large studies have shown that glucose tolerance can be returned to normal in 83% of type 2 diabetics who have gastric bypass surgery, and that the procedure is associated with reduction in blood pressure, decreased low-density lipoprotein, and increased high-density lipoprotein. Decreased medication requirements and improvements in comorbidities, in addition to durable weight loss, make bariatric procedures a cost-effective answer to the obesity epidemic.

Bariatric surgery involves an abdominal operation in an otherwise high-risk patient population, and as such, is not without perioperative risk. Perioperative morbidity and mortality for laparoscopic gastric bypass is 6% and 0.5% to 1.5%, respectively. Nutritional deficiency and metabolic perturbations are potential long-term complications from the operation.

Bariatric procedures are categorized as restrictive, where food intake is limited by a small gastric “pouch,” as malabsorptive, where the length of intestine available for nutrient absorption is reduced, or as a combination of both. Pure restrictive procedures, such as gastric banding, produce less weight loss than procedures with...
a malabsorptive component, but are generally associated
with fewer postoperative metabolic problems.17 The
metabolic consequences of bariatric surgery will be
considered in this review, and are summarized in
Tables 1 and 2.

MICRONUTRIENT AND MACRONUTRIENT
ABSORPTION IN THE NORMAL GUT

Once food is ingested, carbohydrates, fats, and
proteins are subjected to enzymatic hydrolysis. After
hydrolysis, their components are absorbed in the small
intestine. Micronutrients and vitamins in food are
preferentially absorbed in certain parts of the small
intestine. The sequential process of liberation from food,
protection from enzymatic lysis, and transport across gut
epithelium can be critical for the efficient absorption of
micronutrients. Therefore, anatomic or functional
changes in the proximal gastrointestinal (GI) tract can
impair digestion and absorption at more distal segments.

Carbohydrate digestion begins with the limited
action of salivary amylase in the oropharynx. In the
proximal small intestine, pancreatic amylase hydrolyzes
the bulk of ingested carbohydrates to short chains of
simple sugars. Oligosaccharidases and specialized trans-
porters in the apical enterocyte membranes complete the
digestion and absorption of carbohydrates in the prox-
imal jejunum.

Protein digestion begins in the stomach. Gastric
acid and pepsin denature and hydrolyze dietary protein,
before passing the semidigested chyme to the duodenum,
where proteolytic enzymes reduce polypeptides to amino
acids, which are then transported as mono- or oligopep-
tides across the intestinal epithelium.

Ingested fat is also broken down and taken-up in a
multistep and multiorgan process. Initially, lipid is
released from food by gastric acid and digested in the
proximal duodenum by enzymes released by the pancreas.

At the same time bile, that has been secreted by the
liver and stored and concentrated in the gallbladder, is
secreted into the duodenum in response to dietary fat
ingestion. Bile aids in the emulsification of dietary lipid,
whereas pancreatic lipase cleaves triglycerides into
their component fatty acids. These in turn form into
more hydrophilic micelles. Peristaltic motion circulates
the micelles over the gut epithelium for terminal
processing and transport of dietary lipid across the
small bowel mucosa. In contrast to the relatively free
uptake of sugars and peptides throughout the small
intestine, the cholesterol-based bile salts are dependant on
specialized transporters within the terminal ileum for
absorption.

Specific micronutrients are preferentially absorbed
at specific sites along the GI tract. Cobalamin (vitamin
B₁₂) is released from protein in the acidic milieu of the
stomach, and is protected from hydrolysis by binding to
R-proteins secreted from the gastric mucosa. During
passage through the small intestine, the bound R-protein
is degraded and replaced by intrinsic factor, itself a
product of the gastric parietal cell. Cobalamin is finally
absorbed in the terminal ileum as a cobalamin-intrinsic
factor complex. Folate, iron, and to a lesser extent,
calcium, are taken up preferentially by the duodenum and
proximal jejunum. Thiamine uptake is less restricted, but
mainly occurs in the proximal small bowel. The large
bowel is functionally important for the bacterial fermen-
tation that generates vitamin K precursors, and for its
role in reclaiming water from the GI tract.

Fortunately, there is plasticity and redundancy
within the GI tract, allowing for a degree of compensa-
tion after surgical resection or bypass of certain bowel
segments. This adaptability is not perfect, however, and
surgical procedures designed to limit macronutrient
(carbohydrates, fats, and proteins) intake or absorption
in order to affect weight loss will also affect micronutrient
(vitamins and minerals) absorption.

<table>
<thead>
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<th>TABLE 1. Comparison of Average Weight Loss and Other Metabolic Aspects of Medication, JIB, RYGB, Gastroplasty, and BPD</th>
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<tr>
<td><strong>Pharmacologic</strong></td>
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<td>Weight loss at 1 y</td>
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<td>Weight loss at 5 y</td>
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<tr>
<td>Meal size restriction</td>
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<td>Anemia</td>
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<td>Calcium deficiency</td>
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<td>Bone demineralization</td>
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<td>Fat-soluble vitamin deficiency</td>
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*Depends on diet/exercise regimen followed with pharmacologic theraphy.
EBW indicates excess body weight; TBW, total body weight.
In addition to digestion and absorption, the gut also has endocrine functions, some still incompletely understood. Ghrelin is a small peptide hormone produced in the foregut, with stimulatory effects on appetite and fat synthesis. Incretins, such as gastric inhibitory peptide and glucagonlike polypeptide-1, are secreted by specialized cells within the small intestine in response to ingested carbohydrates. Incretins are thought to play a role in insulin sensitivity, and abnormal incretin physiology has been implicated in the pathogenesis of type II diabetes. Overall, incretin secretion and its effects on type II diabetes may be favorably altered by foregut surgery. The resolution of type II diabetes after bariatric surgery may not be due to mere weight loss. Bypassing the foregut, with stimulatory effects on appetite and fat synthesis, produce rapid and lasting weight loss. In the classic version of JIB, the jejunum was transected 14 inches from the ligament of Treitz, and the proximal jejunal stump connected to the terminal 4 inches of ileum. The distal jejunum and proximal ileum were then drained to the colon. Another commonly performed method of JIB was to create an end-to-side anastomosis between the proximal jejunum and the ileum 4 inches from the ileocecal valve (Fig. 1A), creating the so-called fourteen-inch operation. Weight loss after JIB was dramatic and durable, but the metabolic sequelae of the procedure proved to be equally dramatic, and worsened over time.

Initially, metabolic consequences of JIB were thought to be limited to the development of malabsorptive diarrhea and steatorrhea. However, potassium and calcium losses requiring hospitalization were found in 29% of patients over 15 years of follow-up. Other lasting problems include protein and fat-soluble vitamin malabsorption from decreased intestinal absorption, leading to peripheral edema, hair loss, and night blindness. A significant source of morbidity was the development of oxalate urinary stones, which occurred in 37% of patients. Renal stones form after JIB because of poor reabsorption of bile salts by the shortened ileum. The bile salts preferentially bind calcium in the lumen, and leave excess oxalic acid to be absorbed and secreted in the kidney tubules, resulting in oxalate stones. Prevention of stone disease centers on a diet that is high in protein and calcium, and low in fat and citrate, to minimize oxalate absorption. Increased dietary calcium, along with fat-soluble vitamin supplementation, is also necessary after JIB to prevent accelerated bone loss. From patients surviving the resection of long segments of bowel indicated that a shortened intestinal tract could produce rapid and lasting weight loss. In the classic version of JIB, the jejunum was transected 14 inches from the ligament of Treitz, and the proximal jejunal stump connected to the terminal 4 inches of ileum. The distal jejunum and proximal ileum were then drained to the colon. Another commonly performed method of JIB was to create an end-to-side anastomosis between the proximal jejunum and the ileum 4 inches from the ileocecal valve (Fig. 1A), creating the so-called fourteen-inch operation. Weight loss after JIB was dramatic and durable, but the metabolic sequelae of the procedure proved to be equally dramatic, and worsened over time.

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The presence of a long blind loop of bowel after JIB creates additional problems. Up to half of postoperative patients develop skin rashes and polyarticular arthritis, along with cryoprecipitable immune complexes in the circulation. This arthritis-dermatitis syndrome likely results from mucosal shedding in the intestinal blind loop and antigen absorption in the gut circulation. This has been found to respond to resection of the blind loop. The most significant cause of early and late mortality after JIB is liver failure. Hepatic steatosis and fibrosis are well-described histologic features of obese patients with fatty liver disease. Unfortunately, patients who underwent JIB showed a dramatic worsening and paradoxical progression of fibrosis on needle biopsy. Clinical studies suggest that up to 10% of patients develop acute liver failure after JIB, with continued risk even in long-term survivors. Animal studies suggest that this liver damage from JIB may be due to exotoxin liberated from bacterial overgrowth in the blind loop. The high rate of late complications after JIB led to surgical reversal in up to 25% of patients, and the procedure has since fallen out of favor.

Gastric Bypass

Gastric bypass, as surgical treatment of morbid obesity, is associated with fewer long-term morbidity than JIB. Roux-Y gastric bypass (RYGB) affects weight loss via restrictive and hormonal mechanisms, with only minimal malabsorptive consequences. The stomach is divided across the cardia, so that the esophagus empties into a gastric pouch with a volume between 15 and 30 mL. The jejunum is then transected approximately 50 to 100 cm distal to the ligament of Treitz, and the distal end brought up to the pouch to create a gastrojejunostomy (the Roux limb). The operation is completed by making an end-to-side anastomosis between the previously transected biliopancreatic (enzymatic) limb and the Roux limb of jejunum 50 to 100 cm distal to the gastric outlet. In this manner, the distal stomach, duodenum, and proximal jejunum are effectively bypassed, while still allowing biliary and pancreatic drainage into the food stream. Consequently, micronutrient absorption is bypassed from the proximal small bowel. Metabolic complications of RYGB are largely due to this exclusion of the duodenum and the proximal jejunal from nutrient absorption.

A prospective study in RYGB patients found that 36% of women and 6% of men developed laboratory evidence of postoperative anemia without a GI source of blood loss. Low total body iron content was found in 50% of the female and 20% of male patients, and 18% overall were found to have low folate stores. Retrospective studies found a similar range of iron and folate deficiency, with markedly worse levels in menstruating women. There are 3 separate mechanisms to explain iron deficiency after gastric bypass. First, and most obvious, is reduced dietary intake. Second, some studies show diminished gastric acid exposure to dietary iron. In the normal stomach, gastric acid oxidizes dietary iron into a more easily absorbed state. After gastric bypass, iron absorption is purportedly decreased secondary to inadequate oxidation. Finally, the duodenum is the preferred site of dietary iron absorption. The exclusion of the duodenum from the stream of ingested food is probably the major factor in impaired iron absorption after RYGB. The duodenum is also the primary site of folate and calcium absorption, deficiencies of which are discussed below.

Vitamin B<sub>12</sub> depletion is also reported in up to 70% of patients postoperatively. Although initially believed to be due to a lack of intrinsic factor, this deficiency is now known to result from diminished gastric acidity and poor release of cobalamin from dietary protein. A less common cause of B<sub>12</sub> deficiency in postoperative patients is consumption of dietary B<sub>12</sub> secondary to bacterial overgrowth in the blind loop, facilitated by decreased gastric sterilization. A short course of antibiotics may resolve small bowel bacterial overgrowth and relieve vitamin...
malabsorption, but in recurrent cases, surgical revision of the loop may be necessary to reduce stasis.30

Fortunately, bioavailability of crystalline cobalamin given in multivitamin tablets is not reduced by hypochlorhydria, so that oral supplements provide sufficient means of preventing B12 deficiency postoperatively.36,44 In patients who are incapable of tolerating a diet secondary to other postoperative issues, or in those with acute deficiencies, intramuscular and nasal spray B12 may be used to replete stores. In any patient requiring more than maintenance B12 dosing, however, it is critical to first investigate folate levels to exclude masked folate deficiency as a cause of anemia, and copper deficiency as a cause of neuropathy.41

The clinical significance of iron, folate, and cobalamin deficiency is controversial. Obesity has been associated with low preoperative serum folate concentrations, so that postoperative deficiencies are difficult to interpret.42 Although laboratory evidence of anemia exists in a large percentage of postoperative patients, significant symptoms are rare. In the previously cited prospective study, only 3.5% of patients required transfusion, and other large series do not describe any incidence of transfusion-requiring anemia. The reason for this is the ease with which metabolic causes of anemia can be corrected postoperatively. Retrospective studies have shown that daily multivitamin supplements containing 400 mcg of folate and 6 mcg of vitamin B12 can prevent deficiencies and correct existing deficits in more than 80% of patients.43,44 Iron stores may remain below preoperative levels despite standard oral supplementation in more than 50% of patients, however. A prospective study supports the administration of extra iron supplements, at 320 mg twice daily, to premenopausal women to prevent microcytosis and decreased ferritin.45 Long-term data from the majority of prospective and retrospective studies suggest that micronutrient deficiencies arise quickly and are most severe within the first year after RYGB, but plateau in their effect on hematopoiesis thereafter. On the basis of this trend, patient follow-up should include more frequent laboratory screening in the early postoperative period, with a greater interval between laboratory follow-up after 12 to 18 months.

In addition to hematologic manifestations of nutritional deficiencies, bone demineralization is also an area of concern after RYGB. Postoperative patients may have metabolic and radiographic evidence of increased bone resorption despite normal serum calcium, vitamin D, and parathyroid hormone levels.46 Postmenopausal women who have undergone gastric bypass have been found to be at increased risk of developing secondary hyperparathyroidism and accelerated bone loss over nonoperative controls.47 In these recent studies, the postoperative patients had increased their dietary calcium intake, indicating that changes in bone homeostasis may be more insidious and resistant to oral supplementation of calcium and vitamin D than previously expected.48 Preliminary data suggests that bisphosphonates may be of benefit in preserving bone mass in postmenopausal women after gastric bypass.49 When followed over 2 years, patients in the treatment group had improved biometric and clinic measures of bone density, including decreased incidence of fractures, as compared with calcium/vitamin D only.

Another metabolic consequence of gastric bypass is the development of the dumping syndrome, a postprandial sensation of abdominal pain and distension, with vasomotor changes including flushing and dizziness. Nausea, vomiting, and diarrhea are sometimes present. Symptoms seem to be the result of rapid entry of hyperosmolar material into the small intestine, which in turn leads to intravascular fluid shifts and a peptide hormone surge.50 Up to 70% of patients have at least some symptoms of dumping syndrome after RYGB, which may also contribute to postoperative weight loss.51,52 Patients with dumping syndrome are typically able to control their symptoms with dietary modification, including avoidance of simple sugars and increased dietary fiber and protein consumption.50 Severe symptoms are rare, and can be managed with octreotide53 or acarbose54 therapy. Clinical data for these drugs is primarily from general surgical and nonsurgical dumping syndrome, however, so that their utility has not yet been proven in bariatric patients. Although dumping syndrome may lead to changes in patient behavior that support weight loss, the presence of the syndrome is not conclusively linked to better results after RYGB.55

The metabolic complication profile of RYGB is manageable by routine laboratory monitoring and prophylaxis with dietary supplements. Beyond the previously discussed problems of anemia and bone demineralization, gastric bypass has few long-term risks. Protein malnutrition, which was common and occasionally severe in JIB, is a rare complication of gastric bypass, and is amenable to short-term parenteral correction.56 Problems associated with the restrictive nature of RYGB are rare, and will be discussed with restrictive procedures below. The low overall risk and excellent weight loss characteristics of RYGB have made it the standard bariatric procedure in current clinical practice and the baseline for comparison with the development of newer techniques. A recent meta-analysis that reviewed 9413 gastric bypass operations showed a 6% total incidence of postoperative nutritional deficiencies and anemias, with an overall mortality for RYGB of only 0.98% over 10 years.57

**Gastric Restriction**

Gastric restriction procedures aid in weight loss by producing early satiety and dramatically reducing the total caloric intake. The continuity of the small bowel is not altered, and there is no malabsorption element to the operation. Gastroplasty was introduced in the early 1970s as a safe alternative to JIB, and has evolved considerably as a surgical technique. Initially, an incomplete line of staples was placed horizontally across the stomach to create a small pouch with an outlet along the greater curvature of the stomach. Although weight loss with this technique was initially promising, dilatation of the stomach led to decreasing efficacy of gastric restriction
and poor long-term results. Several variations in technique were used to try to overcome late dilatation, and in 1982, results with vertical banded gastroplasty (VBG) were first published. This technique creates a vertically-oriented pouch with a volume of 15 to 45 mL, and uses synthetic material to prevent dilatation of the outlet (Fig. 1C). Weight loss with VBG has been reported to be as high as 28% at 10 years. However, comparative studies have consistently shown RYGB to produce more significant and durable weight loss than VBG, as well as a greater excess weight loss in superobese individuals (BMI > 50 kg/m²).59,60

In general, gastric restrictive surgery has a low incidence of metabolic sequelae. Digestion and absorption are unaffected by the procedure, and late metabolic perturbations are generally limited to those induced by chronic vomiting. Emesis is a poorly reported consequence of gastric restriction, but may occur once or more per week in up to 40% of RYGB and 64% of VBG patients.61 The differential diagnosis of vomiting after RYGB and VBG includes intolerance of certain foods, noncompliance with diet, and outlet obstruction or stenosis. Meats, eggs, and breads can all cause vomiting if not thoroughly chewed and eaten slowly. Such episodic vomiting usually has a clear history correlating with food intake and responds well to changes in eating habits, posing little metabolic risk. Foods with indigestible components, such as popcorn and fruits, can also cause vomiting by a “ball valve” intermittent blockage of the outlet. Vomiting in these cases occurs long after the causative meal, and can even lead to intolerance of liquids.62 In these cases, the offending object may require endoscopic removal. General noncompliance with diet can lead to chronic vomiting or, conversely, weight gain.

Chronic vomiting may also be caused by stomal stenosis, which is usually associated with accelerated weight loss. Stomal stenosis occurs in 2% to 5% of restrictive procedures, and must be suspected as a cause of emesis especially within the first 3 months after surgery.67 Stomal stenosis is rarely overcome with behavioral modification and usually requires endoscopic dilatation to achieve adequate symptom relief.68

The metabolic risks associated with chronic vomiting have been noted for over a century, beginning with Wernicke’s 1881 description of the neurologic changes that now bear his name. Thiamine deficiency, or beriberi, affects cardiac muscle, the vasculature, and both the peripheral and central nervous system. Short-term total thiamine depletion from the diet causes resting tachycardia and a peripheral vasodilatory state, and may progress to shoshin beriberi, or fulminant cardiovascular collapse. Such acute and dramatic manifestations are rarely seen, however. Instead, peripheral neuropathy and encephalopathy may develop insidiously, initially presenting as Wernicke syndrome. This constellation of nystagmus, ataxia, and tremors often coexists with Korsakoff psychosis, a retrograde amnesia with confabulation. Severe thiamine deficiencies such as these are classically found in alcoholics, and may present as a mixed syndrome depending on caloric intake and the degree and chronicity of vitamin deficiency.64 Thiamine deficiency from chronic emesis can cause symptoms after 1 to 4 months, and early recognition of signs and symptoms is necessary to limit long-term damage.55,66 The incidence of neurologic changes with vomiting after restrictive operations, including RYGB, is 0.06%, and approximately half the cases resolve with medical treatment.67

Another risk of chronic vomiting is protein malnutrition, which may occur while the patient still has an elevated BMI. With significant vomiting and reduced serum albumin concentrations, edema at the gastric stoma may compound the initial cause of vomiting by causing or worsening mechanical obstruction. Prevention of vitamin and protein malnutrition after restrictive procedures involves early identification and correction of the cause of vomiting. Vitamin supplementation and routine laboratory measurement are not necessary after VBG as long as the patient takes in a reduced-portion, but healthy, diet. In the case of the patient with chronic emesis, vitamin supplementation may be best accomplished parenterally, as oral forms may be regurgitated before being absorbed.68

Laparoscopic adjustable gastric banding (LAGB) is a newer gastric restrictive technique with reduced surgical complications and possibly, even better metabolic characteristics than VBG.17,69 LAGB involves placement of an inflatable cuff around a 15 to 30 mL pouch of proximal stomach. This cuff is connected to a subcutaneous reservoir, which may be filled or emptied to change the caliber of the gastric outlet (Fig. 1D). LAGB may have an advantage over VBG in reducing postoperative emesis, as the reservoir can be deflated to increase outlet diameter. No long-term studies yet exist comparing morbidity between the 2 techniques, although the LAGB seems to induce stable long-term weight loss.17

Biliopancreatic Diversion and Duodenal Switch

Although the 1970s saw development of less-invasive bariatric procedures, perhaps in response to the lessons of JIB, the early 1980s witnessed the rise in popularity of the more radical biliopancreatic diversion (BPD) and a derivative procedure, the BPD with duodenal switch (BPD/DS).70,71 BPD creates much more significant malabsorption than traditional gastric restriction. BPD is performed by partitioning the stomach horizontally to create a proximal 200 to 500 mL pouch. The ileum is then transected 250 cm from the ileocecal valve, and the distal end brought up to the gastric pouch to create a Roux limb. The biliopancreatic limb is reconnected to Roux limb by an end-to-side anastomosis 50 to 100 cm from the ileocecal valve (Fig. 1E). In this configuration, nutrients and biliopancreatic secretions are combined in a “common channel,” with a length of 50 to 100 cm, for nutrient absorption. Some additional absorptive area is created by backwash of secretions proximally into the Roux limb. This is a compromise between the minimally malabsorptive RYGB, where all but 100 cm (of the approximately
7 m of total small bowel length) is available for nutrient absorption, and JIB, where only 35 cm of small bowel is exposed to the combined flow of nutrients and digestive secretions.

BPD/DS is a technical variant of BPD where a vertical gastric pouch is created by resection of the greater curvature of the stomach, thereby sparing the vagus nerves and pylorus. The total pouch volume is the same as in BPD, and the outlet is constructed by anastomosis of the Roux limb to the proximal 5 cm of duodenum rather than to the stomach (Fig. 1F). The lengths of the Roux, biliopancreatic, and common limbs are similar to BPD, with a standard common channel of 100 cm. Advantages of the DS are believed to be preservation of normal gastric emptying, decreased incidence of stomal ulcer, and improved iron and calcium homeostasis. Recent studies of DS procedures show a 73% excess weight loss, sustained up to 10 years.

The biliopancreatic bypass procedures produce the greatest weight loss among modern surgical techniques. BPD has been shown to result in 77% excess body weight loss out to 12 years when a 50 cm common limb is used. Direct comparison between BPD and BPD/DS using a 100 cm common limb in both has shown comparable (63% vs. 70%) excess weight loss over 6 or more years.

Weight loss with BPD and BPD/DS is not without metabolic consequences, however. Virtually all patients have steatorrhea and flatulence, and 60% have occasional diarrhea. Although electrolyte disturbances are rare, protein malnutrition occurs in up to 12% of BPD patients with 50 cm common limb, and in up to 3% of BPD/DS patients. In addition, anemia is found in up to 33% of 50 cm BPD patients. With a 100 cm BPD, 20% of patients develop iron-deficiency anemia, compared with 9% of BPD/DS patients. Even with aggressive replenishment, 6% of BPD patients have intractable anemia.

Disruption of calcium absorption is expected with BPD, and to a lesser extent, the DS. Early comparative data found hypocalcemia in 16% of BPD patients, compared with 8% after BPD/DS. A more recent BPD series finds hypocalcemia in 29% of patients after nearly 3 years of follow-up despite 83% compliance with oral multivitamins. The clinical significance of hypocalcemia in these patients is uncertain. A prospective study of DS showed that 15% of patients may develop bone demineralization sufficient to double their risk of fracture. A mixed BPD and BPD/DS group showed low-corrected calcium levels in 48% of patients after 4 years, but only 3% had clinical evidence of increased bone resorption.

Malabsorptive procedures of the BPD type are also associated with fat-soluble vitamin deficiencies, such as vitamin A, vitamin K, and vitamin D. Correction of low vitamin A and K levels reverses night blindness and prolonged prothrombin time, but when vitamin replenishment becomes a chronic issue, surgical revision to a normal or less-malabsorptive operation may be necessary.

The literature on malabsorptive procedures clearly shows that both weight loss and adverse metabolic consequences vary with the length of the alimentary and common intestinal limbs. Variations on the RYGB to create distal or “very-long” gastric bypasses have few advantages over a BPD/DS operation, as malabsorption is a limiting step in weight loss. Although BPD/DS should have advantages in calcium and iron absorption, there is as yet no clear data to back up this hypothesis, and more intense supplementation may be required to adequately protect bone homeostasis.

A significant benefit of RYGB or BPD/DS over BPD alone is the decreased risk of stomal ulceration. Gastric bypass leaves little parietal tissue in the pouch, and BPD/DS has its stoma in the duodenum, so ulcerative breakdown of the outlet is less likely in these procedures than in traditional BPD, where the anastomosis is bathed in acid chime. On the whole, however, the current generation of malabsorptive procedures is clearly safer than JIB, and though cases of hepatic failure after BPD exist, they are rare.

A comparison of the different weight loss modalities is outlined in Table 1.

### SPECIAL CONSIDERATIONS IN BARIATRIC SURGERY

With the increasing rate of obesity in America, bariatric procedures are being increasingly performed. Consequently, the patient profile is continually expanding. Metabolic considerations in some of the emerging patient groups merit consideration.

#### Patient Age

Although early series of bariatric procedures included mostly middle-aged adults, adolescents now account for an increasing percentage of surgical patients. The rationale for offering bariatric surgery to adolescents is that an early and decisive intervention in obesity will lead to decreased morbidity and a longer lifespan. Gastric bypass surgery is effective in this population, but behavioral noncompliance after surgery is more common than in adults. In one series, only 13% of RYGB patients regularly took all vitamin supplements postoperatively. Despite this poor compliance, growth parameters were within normal limits for adolescent patients after surgery, and metabolic complications did not seem to differ from those in adults.

The metabolic complications of bariatric surgery in the elderly will merit study as the population ages and obesity continues to become more prevalent. Studies have shown VBG, RYGB, and BPD to produce weight loss in patients over age 55 comparable with that seen in standard series. Other data indicate that when compliance with vitamin supplementation is high, there is no increased incidence of metabolic complications after RYGB. Although promising, these results must be weighed against the previously described risk of osteomalacia in postmenopausal women, a standardized approach to osteomalacia risk in the elderly population has not yet been formulated, but may encompass...
preoperative radiologic testing and aggressive calcium and vitamin D supplementation.

**Menstruation and Pregnancy**

As described earlier, menstruating women who have undergone gastric bypass require increased iron supplementation. Maternal and fetal complications of pregnancy are reduced by weight loss from VBG, RYGB, and BPD. Pregnancy after bariatric procedures, especially those with a malabsorptive component, also places a special burden on the patient to maintain adequate nutrition. A devastating consequence of nutrient deficiency is the development of neural tube defects, but this is a rarely reported event, and must be separated from the increased incidence of these defects also seen in the obese population. Prudence dictates an aggressive approach to birth control education and folate supplementation for premenopausal women after bariatric surgery, as decreased BMI greatly increases fertility and the chances of conception.

**Gallbladder Disease**

Cholelithiasis is a recognized risk of both obesity and rapid weight loss. Gallstones are found in 28% of patients presenting for bariatric surgery. Increased rates of gallstone formation after bariatric surgery are seen in patients who have weight loss at a rate of more than 1.5 kg/wk, and among those who have very low levels of fat in their diet. Postoperative development of gallstones occurs in 40% of RYGB and 20% of LAGB patients over 3 years. In patients who develop stones, 17% will go on to develop symptoms over 50 years. Medical prevention of stone disease uses ursodiol to increase cholesterol solubility. A randomized trial showed a drop in stone incidence 6 months after RYGB from 32% to 2% with daily ursodiol treatment, whereas other studies are less conclusive. Some authors suggest preoperative ultrasound screening of bariatric surgery patients, and prophylactic cholecystectomy in those with gallstones, but most centers currently use an expectant approach to gallstone disease.

**SUMMARY**

Bariatric surgery is the emerging gold standard for operative treatment of severe obesity. Although this review has focused on the metabolic sequelae of gastric bypass, gastroplasty, and BPD, it must be remembered that these procedures are only offered to a select group of obese patients. When behavioral and medical alternatives cannot supply weight loss sufficient to reduce comorbidity, surgery is appropriate. Specifically, bariatric surgery is approved for patients with a BMI > 40 kg/m², or those with a BMI > 35 kg/m² who also have related comorbidities.

The RYGB is the best surgical option for most patients, combining excellent weight loss characteristics with a minimal need for vitamin supplementation and postoperative laboratory follow-up. Malabsorptive procedures such as the biliopancreatic bypass and DS are currently less popular in the United States. These procedures involve a significantly greater risk of micro and macronutrient deficiencies, and are mostly used in the treatment of the superobese. One additional problem with BPD and BPD/DS is that they have less of a restrictive component, and may minimize the patient’s perceived role in weight reduction by not forcing a change in his or her eating habits. Psychologic investment and active patient involvement in a postoperative lifestyle change is not only the key to optimum weight loss, but is also crucial to avoiding metabolic complications of any bariatric procedure.

**ACKNOWLEDGMENTS**

Illustrations by Diana Daly.

**REFERENCES**