Gastrointestinal Issues in the Assessment and Management of the Obese Patient

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Address correspondence to: Dr. Eamonn M. M. Quigley, Department of Medicine, Clinical Sciences Building, Cork, Ireland; E-mail: e.quigley@ucc.ie Abstract: As the obesity epidemic spreads across the world, physicians of all specialties will be called on to participate in the management of this condition. Gastroenterologists are no exception and can expect, in the future, to play a major role in all aspects of the care of the obese patient. Thus, gastroenterologists must learn to recognize, prevent, and treat gastrointestinal disorders related to obesity, and they must have an understanding of the risks and bene-fits of various management strategies. Gastroenterologists may also be called upon to assist in the evaluation and management of liver and gastrointestinal problems that have developed following bariatric surgery. When treating these problems, a thorough understanding of the anatomic and physiologic perturbations associated with a given procedure is essential, as is the knowledge of which complications are linked to weight loss and which are linked to a specific surgical approach.

besity is a chronic and stigmatizing condition that has become a major health problem in most industrialized countries because of its prevalence, serious health consequences, and economic impact. It is estimated that illnesses related to obesity are responsible for 300,000 deaths in the United States alone each year.¹

Obesity has particular relevance for the gastroenterologist for several reasons: first, because of its association with a variety of gastrointestinal disorders; second, as the gastroenterologist may be called upon to evaluate obese patients prior to surgery or to assess and treat postoperative complications in patients who have undergone obesity surgery; and, third, because the gastrointestinal tract may be an important target for future obesity therapies.^{2,3}

From a clinical perspective, obesity can be defined as excess body fat that leads to increased morbidity and mortality.² The differentiation of a healthy fat mass from an unhealthy fat mass is not absolute, as it is influenced by factors such as genotype, age, gender, fat distribution, and physical fitness.²

To facilitate comparisons between individuals of different heights, body weight status may be conveniently described in terms of the body mass index (BMI), which is calculated as weight (in kg) divided by height (in m²) or as weight (in pounds) divided by height (in inches) times 703. A normal BMI ranges from 18.5 to 24.9, and

Keywords

obesity, bariatric surgery, gastrointestinal endoscopy, GERD, gastrointestinal cancer, gastrointestinal hemorrhage obesity is defined as a BMI in excess of 30. Additional risk factors for obesity-related illness and mortality include a waist circumference in excess of 102 cm in men and 88 cm in women, a weight gain of 5 kg or more between the ages of 18–20 years, poor aerobic fitness, and being of Southeast Asian descent.⁴

Before discussing the complications of bariatric procedures, it should be noted that obesity has been associated with a number of gastroenterologic disorders, in which natural history may be altered, either favorably or unfavorably, by interventions for weight loss. In the evaluation of postsurgical patients, it is essential to acknowledge the range of gastrointestinal disorders associated with obesity, lest a disorder attributed to the intervention was, in fact, preexistent.

Obesity and Gastrointestinal Disease

Although some gastroenterologic associations of obesity such as gastroesophageal reflux disease (GERD) and cholelithiasis have been recognized for decades, recent epidemiologic studies have revealed the broad spectrum of gastrointestinal symptoms and disorders that may occur in obese patients. In addition to confirming the association between obesity and GERD symptoms, these studies have also described an increased prevalence of symptoms such as dyspepsia, diarrhea, nausea, vomiting, abdominal pain, constipation, and bloating in obese subjects.⁵⁻¹⁰ Indeed, mindful of these preoperative associations, some surgical series have taken pains to document their resolution following surgical intervention.¹¹ For the most part, the physiologic basis of some of these symptoms remains unclear; some patients have reported association with Helicobacter pylori infection,12 others have documented delayed gastric emptying,13 and still others have noted accelerated emptying and reduced postprandial gastric volumes.14 The possible contribution of gastric physiology to altered satiation has also been the focus of some attention, although data to date have provided conflicting results.¹⁵ For now, it is sufficient to say that the evaluation of obese patients, be it preoperatively or postoperatively, should be mindful of the high prevalence of various gastrointestinal complaints in this patient population.

Gastroesophageal Reflux Disease

Most large epidemiologic studies have documented a higher prevalence of symptoms and/or pathology attributable to GERD among obese individuals than lean individuals.^{5-10,16-19} A recent meta-analysis confirmed this association; the odds ratio was 1.94 for GERD symptoms in obese subjects and 1.76 for esophagitis in overweight or obese individuals together.²⁰ Studies of GERD pathophysiology support these findings, documenting increased acid exposure,^{21,22} more frequent transient lower esopha-

geal sphincter relaxations,²³ and compromised integrity of the esophagogastric junction.²⁴ The relevance of weight changes to these conditions was nicely illustrated in a study conducted in lean individuals, in which even a modest degree of weight loss, such as 2–3 kg, resulted in a marked improvement in symptoms.²⁵ Of relevance to the occurrence of postoperative dysphagia, there is some evidence that obese patients with GERD are more likely to harbor impaired esophageal motility.²⁶

Obesity and Esophageal Adenocarcinoma

Although the relationship between obesity and Barrett esophagus remains to be defined,²⁰ a large volume of evidence indicates that there is a strong association between obesity and adenocarcinoma of the esophagus and gastric cardia.^{20,27-33} In a study based on a registry of the entire Swedish population, Lagergren and colleagues demonstrated a dose-dependent relationship between BMI and esophageal adenocarcinoma.²⁷ The adjusted odds ratio was 7.6 for individuals in the highest BMI quartile compared with those in the lowest quartile, whereas obese individuals (BMI >30 kg/m²) had an odds ratio of 16.2 compared with lean individuals (BMI <22 kg/m²). The odds ratio for adenocarcinoma of gastric cardia was 2.3 for individuals in the highest BMI quartile compared with those in the lowest quartile and 4.3 for obese individuals compared with lean individuals. These findings suggest a powerful association between obesity and adenocarcinoma of the esophagus and gastric cardia; the precise basis for this link and how obesity interacts with other risk factors such as GERD, gender, and social habits, however, remains to be clarified.³¹ Furthermore, the impact of weight loss and bariatric surgery on this association is unclear; whether or not such patients should be enrolled in a surveillance program remains an unresolved issue.

Gallbladder Disease

Obesity is an important risk factor for gallstones, particularly in women, among whom the risk of symptomatic gallstones increases linearly with BMI.³⁴ Data from the Nurses Health Study demonstrated that obese women $(BMI > 30 \text{ kg/m}^2)$ had a 2-fold excess risk and extremely obese women (BMI >45 kg/m²) had a 7-fold excess risk of symptomatic gallstones compared with lean women (BMI <24 kg/m²).³⁵ Most recently, Tsai and colleagues defined abdominal circumference and waist-to-hip ratio as further independent risk factors for cholecystectomy in women.³⁶ Here again, a plausible physiologic basis can be provided.³⁷ These associations have important clinical implications: in one series of 144 patients being evaluated for bariatric surgery, 30% had already undergone cholecystectomy and 22% of patients with an intact gallbladder were found to have gallstones on preoperative ultrasonography.³⁸

Pancreatitis

Not surprisingly, given the association described above with cholelithiasis, obesity has been identified as a risk factor for the development of pancreatitis.³⁹ Furthermore, obese patients who develop pancreatitis from any cause are at a greater risk for developing severe pancreatitis and related complications and often experience a worse outcome than do lean patients.⁴⁰⁻⁴² This latter observation tends to support the use of prophylactic cholecystectomy at the time of a bariatric surgical procedure.

Liver Disease

Obesity is associated with a distinct spectrum of liver abnormalities, now encompassed within the term nonalcoholic fatty liver disease (NAFLD), which range pathologically from macrovesicular steatosis and steatohepatitis to fibrosis and cirrhosis. These abnormalities manifest clinically as hepatomegaly, elevated liver enzymes, or abnormal imaging studies.⁴³ The critical issue here is recognition; failure to recognize significant liver disease preoperatively may lead to disaster in a patient with established cirrhosis and portal hypertension. A liver status assessment is, therefore, an important prerequisite for any patient being considered for obesity surgery, and severe liver disease may preclude some surgeries.

Gastrointestinal Cancer

Obesity is recognized as an important risk factor for cancer of the esophagus, colon, rectum, gallbladder, pancreas, and kidney in both genders, and for cancer of the breast, uterus, cervix, and ovary in women.³² Of particular importance to the gastroenterologist are the associations between obesity and colorectal cancer44-48 and obesity and colonic adenomas.^{49,50} It has been suggested that the presence of obesity may adversely influence the outcome of patients with colon cancer.⁵¹ These observations have important implications for the care of obese patients following bariatric surgery and beg the questions: does weight loss reduce or eliminate the risk of colon cancer, and should surveillance be initiated or continued? Given the magnitude of the risk associated with obesity (80% increased risk for men) and the lack of prospective data indicating otherwise, it would appear to be prudent to regard obese patients as belonging in a high-risk group for colon cancer, regardless of surgical intervention.

Assessment of Risk and Benefit

Nonsurgical treatments, whether dietary regimens or pharmacologic approaches, have a poor track record in the management of morbid obesity. Weight loss associated with pharmacologic therapy has been modest, and several agents have been withdrawn because of toxicity.⁵²

For these reasons, surgery has assumed the preeminent role in the management of obesity. Surgical approaches are based, primarily, on two main mechanisms: the restriction of caloric intake through the creation of a very small gastric reservoir or the induction of malabsorption by shortening the absorptive length of the small intestine. Before considering a patient as a surgical candidate, it is important to assess their risk for postoperative complications, which is greater in individuals with a history of obesity-related cardiovascular disease, impaired pulmonary function, thromboembolic disease, diabetes, liver disease, or prior surgery. The current overall mortality rate for obesity surgery is 1.5% but should be less, and approximately 75% of the deaths are caused by anastomotic leaks and peritonitis and 25% are caused by pulmonary embolism.²

Indications for Surgery

Although comparative randomized trials with other approaches have not been performed, gastrointestinal surgery is currently the most effective approach to achieving weight loss in severely obese patients.^{52,53} Indications for the surgical management of morbid obesity were developed by the National Institutes of Health Consensus Development Panel in 1991⁵⁴ and continue to be widely accepted.53 Eligible patients should have: a) either class III obesity (BMI >40 kg/m²) or class II obesity (BMI >35.0-39.9 kg/m²) accompanied by one or more severe obesity-related medical complications (eg, hypertension, type 2 diabetes mellitus, heart failure, or sleep apnea) and b) failed dietary attempts at weight control. The goal of surgery is to reduce the morbidity and mortality associated with obesity and to improve metabolic and end-organ function. Several studies have amply demonstrated that bariatric surgery is effective in achieving these objectives and that surgery improves quality of life.55-56

It is also important to recognize that the majority of individuals who are at an increased risk for obesity-related complications lie in the BMI range between 25 and 35. Numerically, this group greatly outnumbers the minority who suffer from morbid obesity (BMI >35); efforts to comprehensively address the toll of obesity-related illnesses will ultimately have to tackle the management, surgical or otherwise, of this group, as well.

The Role of Preoperative Endoscopy

It has been widely held that upper gastrointestinal endoscopy should be performed in all patients being actively considered for bariatric surgery. This approach was predicated not only on the need to document the preoperative state of the upper gastrointestinal tract to better interpret the significance of postoperative lesions, but also on the need to identify preexisting pathology. Several series have, indeed, documented the discovery of significant upper gastrointestinal pathology at endoscopy in patients selected for various bariatric procedures.⁵⁷⁻⁶² In reviewing these series, it is notable that the most prevalent findings, not surprisingly, are related to GERD or to pathologies associated with *H. pylori*, leading some clinicians to suggest that endoscopy should be restricted to those with GERD symptoms⁶⁰ or positive *H. pylori* serology.⁶² Other clinicians, reporting a high prevalence of both esophagitis and gastroduodenal lesions among asymptomatic patients, do not support the recommendation.⁵⁸ Pending definitive results from prospective studies, the place of preoperative endoscopy remains unclear.

Surgical Options

Bariatric surgical procedures can be divided into two main types: malabsorptive and restrictive (Table 1). Vertical banded gastroplasty (VBG) and laparoscopic adjustable gastric banding (LAGB) are purely restrictive procedures that result in similar anatomical changes. Both procedures limit solid food intake by restricting the size of the gastric reservoir and leave the absorptive function of the small intestine intact. In contrast, malabsorptive procedures are performed to decrease nutrient absorption by shortening the functional length of the small intestine. Jejunoileal bypass (JIB), biliopancreatic diversion (BPD), and BPD with duodenal switch operation are examples of malabsorptive procedures. Some procedures, such as the Roux-en-Y gastric bypass (RYGB), have both restrictive and malabsorptive impacts. In the United States, RYGB has been the most popular approach; in Europe and elsewhere, LAGB has acquired considerable support. In head-to-head comparisons, weight loss has been greater following RYGB than with restrictive procedures, with little difference among individual restrictive procedures in terms of long-term weight loss.⁵²

Efficacy of Bariatric Surgical Procedures

Gastric Bypass Initially developed as an open procedure, RYGB is now usually performed laparoscopically. The move to the laparoscopic approach is supported by a number of studies. In a prospective, randomized study conducted on 104 patients with morbid obesity comparing the laparoscopic approach with open surgery, operating time (mean 186.4 minutes vs 201.7 minutes) and duration of hospital stay (5.2 days vs 7.9 days) were shorter and late complications less common (11% vs 24%) following the laparoscopic approach, yet weight loss was similar to that achieved with open surgery and conversion rates were low at 8%.⁶³ Podnos and colleagues reviewed 10 laparoscopic studies covering 3,464 patients and 8 open gastric bypass studies involving 2,771 patients. Table 1. Types of Bariatric Surgical Procedures

Restrictive

- Vertical banded gastroplasty
- · Laparoscopic adjustable gastric band
- Intragastric balloon

Malabsorptive

- Jejunoileal bypass
- Biliopancreatic diversion
- · Biliopancreatic diversion with duodenal switch

Mixed

• Roux-en-Y gastric bypass

Laparoscopic gastric bypass was associated with a lower frequency of iatrogenic splenectomy, wound infection, incisional hernia, and mortality; however, there was an increase in the frequency of early and late bowel obstruction, gastrointestinal hemorrhage, and stomal stenosis. There were no significant differences in the frequencies of anastomotic leak, pulmonary embolism, or pneumonia.⁶⁴

Banded Gastroplasty VBG is a purely restrictive procedure in which the upper part of the stomach is partitioned by a vertical staple line, leaving a narrow outlet that is then wrapped by a prosthetic mesh or band to maintain the lumen. LAGB is a purely restrictive procedure that compartmentalizes the upper stomach by placing a tight, adjustable prosthetic band around the entrance of the remainder of the stomach. These two procedures have been compared in a number of studies. In one such study, 100 morbidly obese patients were randomized to either VBG or LAGB and followed for a minimum of 2 years.⁶⁵ There were no deaths or conversions in either group. Although operation time (65.4 minutes vs 94.2 minutes) and hospital stay (3.7 days vs 6.6 days) were, on average, shorter for LAGB, late complications were more prevalent in this group (32.7% vs 14%), with band slippage being the most frequent complication. Weight loss was greater in the VBG group; using Reinhold's classification, a residual excess weight of less than 50% was achieved at 2 years in 74% of the VBG group, in comparison to only 35% of the LAGB group.⁶⁵

An evaluation of 1,863 patients who had undergone LAGB revealed a mortality rate of 0.53% (mainly attributable to cardiovascular complications such as myocardial infarction and pulmonary embolism), a conversion rate of 3.1%, and a postoperative complication rate of 10.2%. The most common problems were tube port failure, gastric pouch dilation, and gastric erosion.⁶⁶

Intragastric Balloon The BioEnterics Intragastric Balloon (Inamed) consists of a soft, saline-filled balloon inserted endoscopically to promote a feeling of satiety, thereby restricting food intake. Mean excess weight loss has been reported to be 38% and 48% for 500 mL and 600 mL balloons, respectively.⁶⁷⁻⁷¹ Associated morbidity can be severe, however,^{68,69} and includes a high incidence of severe nausea and vomiting (as high as 76.8% in one study),⁶⁸ esophagitis, erosive gastritis, and even gastric perforation. This approach may be of greatest value in the short term and has been proposed as a valuable bridge to a more definitive anti-obesity surgical procedure.⁷¹ Currently not available in the United States, this procedure has been evaluated only in Europe and Brazil.

Sleeve Gastrectomy Sleeve gastrectomy is a relatively new technique that is most commonly offered to patients with supermorbid obesity as a first stage in surgical management. This technique allows a surgeon to first perform a less technically challenging partial gastrectomy and delay the more demanding laparoscopic RYGB or BPD until the patient has reached a lower body weight.⁷² Sleeve gastrectomy has also been used as an isolated initial bariatric procedure, particularly in highrisk patients.⁷³ In a head-to-head comparison, sleeve gastrectomy was associated with more rapid and greater weight loss and was also better tolerated and safer than the intragastric balloon.⁷⁴

Biliopancreatic Diversion BPD consists of a partial gastrectomy and the formation of a gastroileostomy with a long Roux limb and a short common channel, thus causing malabsorption. Weight loss has been impressive, and the main complication has been nutritional deficiencies. In one study, almost 80% of operated patients reported an improvement in self-esteem, physical activity level, work ability, and social life.⁷⁵

Biliopancreatic Diversion With Duodenal Switch In this variant of the BPD procedure described above, a sleeve gastrectomy involving the removal of approximately 60% of the greater curvature of the stomach is performed, the proximal duodenum is transected, the distal end of the duodenum is closed, and the proximal duodenum is anastomosed to the distal small intestine 250 cm proximal to the ileocecal valve. The sleeve procedure causes gastric restriction but also allows better preservation of gastric physiologic function than distal gastrectomy. This procedure has been advocated for patients with supermorbid obesity (BMI >50 kg/m²). In one study, the average excess weight loss was 46% after 6 months and 58% after 9 months.⁷⁶

Jejunoileal Bypass First reported in 1969, this procedure was designed to bypass most of the small intestine and induce weight loss through malabsorption. The JIB procedure involved the division of the jejunum close to the ligament of Treitz and the connection of its proximal end to the ileum a short distance proximal to the ileocecal valve, thereby diverting nutrients from the majority of the small bowel and resulting in profound malabsorption. Although the degree of excess weight loss achieved was impressive, JIB was associated with multiple complications including severe diarrhea, electrolyte imbalance, oxalate renal stones, vitamin deficiencies, malnutrition, arthritis, liver failure, and death. This operation is, therefore, no longer performed.

Gastrointestinal Complications of Surgical and Endoscopic Procedures For Obesity

Gastrointestinal complications following surgical and endoscopic procedures for obesity may be conveniently divided into two main categories: complications that are common to any anti-obesity procedure and those that result from a specific procedure.⁷⁷⁻⁷⁹

Consequences of All Surgical Procedures

Gallstones Gallstone formation is associated with rapid weight loss. The prevalence of gallstones 6 months following obesity surgery has been reported to be as high as 22% and may be even higher among patients who have undergone malabsorption-inducing procedures.⁸⁰ A reduction in the gallbladder-emptying rate, an increase in gallbladder residual volume, and a decrease in the gallbladder-refilling rate all contribute to gallbladder stasis and, thereby, the formation of gallstones.⁸¹ For these reasons, as well as the aforementioned morbidity associated with pancreatitis in obese patients, some clinicians advocate performing prophylactic cholecystectomy in all patients at the time of bariatric surgical procedures; most clinicians, however, resort to removing the gallbladder only in those who have gallstones at the time of surgery.⁸² Alternatively, a dose of ursodiol 600 mg daily may provide prophylaxis against gallstone formation after GBP-induced rapid weight loss.

Vomiting The most common complication of bariatric surgery is prolonged vomiting. Recording a dietary history of both intake and portion sizes is important, as the most common reason for vomiting after gastric bypass is stomal stenosis, which occurs in 5–15% of patients⁸³ and is usually treated successfully by endoscopic balloon dilatation.⁸⁴ If left untreated, stomal stenosis can result in serious nutrient deficiencies and can even lead to the development of Wernicke-Korsakoff encephalopathy and peripheral neuropathy.⁸⁵

Pulmonary Embolism Pulmonary embolism (PE) remains one of the leading causes of early mortality following bariatric surgery.⁸⁶ In one series, the risk of fatal embolism was 0.2% and increased to 4% among those with preexisting severe venous stasis.⁸⁷ The diagnosis of PE can be problematic because standard diagnostic modalities (eg, ventilation-perfusion radionuclide scans, computed tomography [CT] angiography, pulmonary angiography, lower extremity duplex scanning) may be technically challenging in extremely obese patients. Primary prevention is the key to reducing morbidity and mortality. Most bariatric surgeons employ prophylaxis with compression devices in conjunction with subcutaneous heparin.88 Immediate anticoagulation is prescribed for patients for whom there is a high level of clinical suspicion. In rare patients in whom anticoagulation is contraindicated, a mechanical filter can be placed in the inferior vena cava to lower the risk of continued embolization.

Wound Infection Rates of wound infection are significantly greater with open procedures (10–15%) compared with laparoscopic procedures. The incidence of wound infections can be decreased by the preoperative administration of antibiotics.⁸⁹

Rhabdomyolysis Postoperative rhabdomyolysis has been reported among morbidly obese patients following bariatric procedures and, especially, laparoscopic duodenal switch procedures, which have an associated incidence of 1.4%.⁹⁰ Affected patients present in the early postoperative period with muscle pain in the buttock, hip, or shoulder regions. Routine measurement of creatine kinase and serum creatinine levels, both prior to and following surgery, can aid detection.

Hemorrhage Gastrointestinal bleeding is an uncommon complication of bariatric surgery. Upper gastrointestinal bleeding in patients who have undergone gastric bypass surgery may occur in the esophagus, gastric pouch, or Roux limb just distal to the anastomosis.91 Instances of hemorrhage from a Mallory-Weiss tear have also been reported.⁹² These areas are readily accessible for diagnosis and therapy using a standard upper gastrointestinal endoscope. Although present in up to 20% of patients following gastric bypass procedures, anastomotic ulceration is a rare cause of bleeding and when it occurs is typically associated with concomitant nonsteroidal anti-inflammatory drug (NSAID) use. As a result, NSAIDs should be avoided in this patient population. Whether H. pylori infection is an important risk factor for anastomotic ulceration is somewhat controversial.

The distal stomach and proximal duodenum may not be accessible with a conventional endoscopic approach. If bleeding from the distal stomach or proximal small bowel is suspected, a pediatric colonoscope or enteroscope may be used to evaluate the area. In addition, interventional angiography may be able to both locate and treat bleeding lesions in these areas.

Weight Gain Although failure to lose weight following bariatric surgery is rare, some patients may subsequently regain a significant percentage of the weight that they initially lost. Apart from technical failures, this occurrence is most commonly associated with maladaptive eating patterns during the early postoperative period, progressive noncompliant eating, and behavioral problems.

Cancer Given the associations between obesity and cancer previously discussed and the lack of sufficient longitudinal data to indicate the elimination of the increased risk of cancer through bariatric surgery, postoperative surveillance for cancer, including gastrointestinal, colon, and, in particular, esophageal cancer remains a concern. No consensus exists on this issue; one researcher recommended commencing surveillance for patients with symptomatic GERD for 15 or more years after surgery.⁹³ As we learn more regarding the associations between obesity treatments and cancer,⁹⁴ these issues will, hopefully, be resolved.

Consequences of Specific Surgical Procedures

Roux-en-Y Gastric Bypass Overall mortality rates for RYGB are very low, ranging from 0% to 1% for both open and laparoscopic approaches, in various reports.⁹⁵ Preoperative predictors include a BMI greater than 50 kg/m², a forced expiratory volume of less than 80%, an abnormal electrocardiogram, and a history of prior abdominal surgery.⁹⁶

Complications associated with gastric bypass include stomal stenosis, anastomotic leak leading to peritonitis, splenic injury, hemorrhage, marginal ulceration, staple line disruption, dilatation of the bypassed stomach, and internal hernia formation, as well as the development of dumping syndrome and nutrient deficiencies. Complications requiring invasive therapeutic intervention are relatively common, occurring in up to a quarter of all patients.^{97,98} Predictors of complications include lack of surgical experience and sleep apnea and hypertension prior to surgery.⁹⁸

The most common complication is a stricture at the gastrojejunal anastomosis and has been reported in 3–17% of patients in various case series.⁹⁷⁻¹⁰³ The vast majority can be treated successfully by endoscopic dilatation without compromising weight loss.^{99-101,103} In one series of 14 patients, a 15-mm hydrostatic balloon was used in 12 patients and an 18-mm balloon was used in 2 patients. In the first group, 7 of the 12 patients (58%) had a treatment response, making further dilation unnecessary; 1 patient had a response to a further 15-mm balloon dilation; 3 patients had a response to a subsequent 18-mm balloon.

loon dilation; and 1 patient required 18-mm and 25-mm balloon dilations. The 2 patients initially treated with an 18-mm balloon required no further dilations. There were no complications with any of the 23 dilations performed.¹⁰³

In one series, intestinal obstruction was the second most common complication, occurring in 7.3% of patients. Causes included adhesions, an internal hernia at the transverse mesocolon level, jejunojejunostomy stricture, and cicatrix formation around the Roux limb at the transverse mesocolon level.⁹⁸ In another series of 711 RYGB patients, 13 (1.8%) developed an obstruction that required surgical intervention. The etiology of obstruction was an internal hernia in 6 cases, adhesive bands in 5 cases (only 2 were related to prior open surgery), scarring of the mesocolonic window in 1 case, and an incarcerated ventral hernia in 1 case.¹⁰⁴

Although less common, an anastomotic leak is a potentially life-threatening complication of RYGB¹⁰⁵⁻¹¹⁰ and has a mortality rate of 6-10% in some circumstances.¹⁰⁶⁻ ¹⁰⁸ This complication usually develops soon after surgery; a series of over 2,000 patients reported an overall leak rate of 2.1% and a median interval to leak presentation of 3 days.¹⁰⁸ Early diagnosis, before the development of peritonitis, is critical. Hamilton and colleagues attempted, therefore, to define early signs and symptoms of anastomotic leaks. In a retrospective review of 210 consecutive patients who underwent RYGB, 9 leaks were documented, at an incidence rate of 4.3%. Evidence of respiratory distress and a heart rate exceeding 120 bpm were the two most sensitive leak indicators.¹¹⁰ Various studies have advocated the use of upper gastrointestinal contrast studies^{106,109} or CT.¹⁰⁷ Although surgical exploration is indicated in most cases, a small series very recently reported the successful management of chronic leaks by a variety of endoscopic techniques.105

Marginal ulceration, noted quite frequently at endoscopy following RYGB,¹⁰² may result in hemorrhage, pain, or perforation. In one series of over 1,000 patients, the median interval to diagnosis was 2 months.¹⁰² This complication is most likely to occur in smokers or patients using NSAIDs¹⁰² and will, of course, be exacerbated by anticoagulant use. Marginal ulcers may be satisfactorily treated by proton-pump inhibitor therapy.¹¹¹ A gastrogastric fistula may also develop at the site of a marginal ulcer; this late complication (mean interval to diagnosis 80 days in one large series.¹¹²) was described in 1.2% of 1,292 patients in one series,¹¹² can be diagnosed by upper gastrointestinal radiology or endoscopy,^{112,113} and can usually be managed conservatively, especially if weight gain does not occur.¹¹³

Hemorrhage may occur from other sites. Hemorrhage from bleeding that originates at the staple line has even been reported to present as small-bowel obstruction resulting from the impaction of an intraluminal clot at the jejunojejunostomy.¹¹⁴ NSAID users are especially susceptible to bleeding because of the bypassed stomach, an area that may be challenging to access.¹¹⁵

Less common complications such as weight gain resulting from dilatation of the gastrojejunal anastomosis,¹¹⁶ dilatation of the excluded stomach,¹¹⁷ and biliary disease¹¹⁸ may pose considerable challenges to the endoscopist.

Because RYGB should ameliorate gastrointestinal symptoms that are commonly experienced by morbidly obese patients,¹¹⁹ the clinician needs to be alert to the new onset of gastrointestinal symptoms following this procedure. As symptoms related to the aforementioned complications may be nonspecific and difficult to assess postoperatively, additional investigations are commonly necessary. The value of conventional contrast radiology and CT in the detection of leaks, an early problem, has already been alluded to. Endoscopy plays an important role in the detection and management of later complications, such as stenosis, marginal ulceration, and fistulization. At this stage, compared to the early postoperative period, endoscopy can be performed safely and with a high degree of diagnostic accuracy, provided that the endoscopist is familiar with the anatomic features of the procedure.102,120

Vertical Banded Gastroplasty Complications related to VBG include stomal stenosis, staple-line disruption, band erosion, and GERD.

Stomal stenosis occurs in 20–33% of cases¹²¹ and leads to food intolerance, GERD, and weight regain, due to a dietary shift to calorically dense liquids and softer foods. Stomal stenosis following VBG can be managed initially by endoscopic dilatation, with surgery reserved for failures. A success rate of 68% for endoscopic dilatation of stomal stenosis was described in one series.¹²²

Staple-line disruption, which can occur in up to 35% of patients,¹²³ typically leads to weight regain due to increased food consumption. Surgical revision may be indicated if significant weight regain occurs or an intractable stomal ulcer and pain develop.

Band erosion occurs in 0–3% of patients and is thought to develop as a result of either gastric wall ischemia from an excessively tight band or from direct mechanical trauma related to the band buckle. Band erosion can be diagnosed endoscopically and treated by endoscopic removal of the band.^{124,125} If the band cannot be removed endoscopically, a laparoscopic approach is often successful.¹²⁵

If successful and uncomplicated, VBG should not promote reflux.¹²⁶ In one study, 43 morbidly obese patients approved for either VBG or LAGB were evaluated for GERD before and after surgery at regular intervals.¹²⁷ Although the prevalence of heartburn and acid regurgitation among patients treated with LAGB increased from 14% and 13% to 63% and 69%, respectively, the prevalence of these same symptoms, present before surgery in 32% and 23% of patients, respectively, were unchanged following VBG. Similarly, esophageal acid exposure, as expressed by the total time that pH was less than 4, increased from 6.4% to 30.9% in patients treated with LAGB but was unchanged in patients treated with VBG. The prevalence rates for esophagitis after LAGB and VBG were 75% and 20%, respectively. Acid suppression was required by 81% of patients after LAGB and 29% of patients after VBG.127 These results notwithstanding, severe reflux, requiring band removal or conversion to another procedure such as RYGB, may occasionally prove necessary in VBG patients. Instances of the late development of achalasia following VBG have also been reported.128

Gastric Banding Major complications following LAGB include acute stomal obstruction, band erosion, band slippage/prolapse, pouch/esophageal dilatation, GERD, esophagitis, and infection.

Acute stomal obstruction is an early and infrequent (approximately 2%) postoperative complication and is usually caused by perigastric fat. Patients usually present with persistent nausea, vomiting, and inability to tolerate oral intake or accommodate their own secretions. The diagnosis may be confirmed by an upper gastrointestinal barium study, which demonstrates no passage of contrast beyond the band. Persistent obstruction requires surgical revision or removal of the band.

Band slippage occurs when the posterior wall of the stomach herniates through the band, leading to gastric obstruction. In initial series, the frequency of slippage was as high as 24%, but a more recent series revealed the much lower incidence rate of 2–14%.^{129,130} Diagnosis can be confirmed with a barium contrast swallow study and also requires surgical revision.

Abnormal dilation of the esophagus (pseudoachalasia) proximal to the band device has been observed in as many as 10% of patients.¹³¹ Deflation of the band alone is usually successful in reversing esophageal dilation. However, persistent dilation may require replacement of the band in a new location on the stomach or conversion to a different procedure.

In a study of 171 patients who underwent a laparoscopic adjustable band procedure for morbid obesity, 40 patients underwent a classic gastric banding procedure, and 131 patients underwent esophagogastric banding. Pouch dilation developed in 6 patients (15%) following the classic procedure and in 12 patients following esophagogastric banding, and this complication was noted more frequently in patients with presurgical hiatal hernias.¹³¹ Pouch formation may also be a factor in the development of GERD, which has been reported in up 30% of patients on long-term follow-up.^{132,133} Reflux and dysphagia are especially likely among patients who exhibited defective esophageal motility preoperatively.¹³⁴ This should not come as a surprise, as the procedure has been shown to impair esophageal motility, increase lower esophageal sphincter and length, and cause incomplete relaxation of the sphincter.¹³³⁻¹³⁶

Intragastric Balloon The major complications related to this procedure include vomiting, balloon displacement, GERD, gastric erosion, balloon leakage, and gastric perforation. In one study of 126 patients, 76.8% complained of severe nausea and vomiting lasting an average of 1 week, which resulted in early balloon removal in 3 patients.⁶⁸ Two patients suffered a gastric perforation that presented as acute peritonitis at 3 and 4 months after placement. Esophagitis was evident in 11 patients, and 1 patient developed diffuse gastric erosions.

A prior fundoplication, or indeed any prior gastric surgical procedure, represents an absolute contraindication to positioning a balloon.¹³⁷

Balloon migration into the small intestine can occur and lead to small-bowel obstruction. Plain radiographs and abdominal CT scans can confirm migration. When this occurs, the device can be removed laparoscopically.¹³⁸

Jejunoileal Bypass This procedure was associated with serious complications including arthritis, cirrhosis, hepatic failure, nephrolithiasis, protein malnutrition, vitamin deficiencies, and erythema nodosum, many of which were associated with the development of small intestinal bacterial overgrowth.¹³⁹ Complications related to bacterial overgrowth can be treated successfully in the short term with oral metronidazole.¹⁴⁰ Patients with serious complications such as liver disease required surgical revision. Because of overall rates of morbidity and mortality in the region of 50% and 10%, respectively, this procedure has been abandoned.¹⁴¹

Biliopancreatic Diversion and Duodenal Switch This procedure causes more nutritional abnormalities than any of the other currently popular anti-obesity techniques and may result in deficiencies in protein, fat-soluble vitamins, vitamin B₁₂, iron, and calcium. Steatorrhea may also result.¹⁴² In rare instances, this procedure has resulted in some of the serious complications associated with JIB.

New Horizons

Apart from the intragastric balloon, which has recently been the subject of a randomized sham-controlled trial,¹⁴³

other endoscopic approaches to the management of obesity are under development, including endoscopic transoral gastric partitioning¹⁴⁴ and endoscopic VBG.¹⁴⁵ Based on the experimental observation that gastric and intestinal electrical stimulation can increase gastric volume and decrease food intake,¹⁴⁶ implantable gastric stimulation has been introduced into the therapeutic armamentarium of clinicians managing obesity.^{147,148} The ultimate role, if any, of these therapies in the management of obesity remains to be determined; it is possible that the therapies may ultimately serve, as the intragastric balloon has, as a bridge to a more definitive surgical procedure or as the sole approach for those who are not surgical candidates.¹⁴⁹

Summary

Obesity is increasing in prevalence worldwide and has become a significant healthcare issue. The management strategy for an obese subject should be based upon a detailed risk assessment. Gastroenterologists may be consulted following surgery to assist in the evaluation and management of a number of gastrointestinal problems. In their approach to these patients, it is vital that they understand the anatomic and physiologic perturbations associated with each procedure and the likely complications. Gastroenterologists should be mindful of problems that may result from weight loss, problems that are generic to anti-obesity procedures in general, and problems that are specific to each procedure. Upper gastrointestinal endoscopy is an all-important tool in the assessment and therapy of these complications.

References

1. Allison DB, Fontaine KR, Manson JE, et al. Annual deaths attributable to obesity in the United States. *JAMA*. 1999;282:1530-1538.

2. Klein S, Wadden T, Sugerman HJ. AGA technical review on obesity. *Gastroenterology*. 2002;123:882-932.

3. Ley RE, Turnbaugh PJ, Klein S, Gordon JI. Microbial ecology: human gut microbes associated with obesity. *Nature*. 2006;444:1022-1023.

 Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. *Obes Res.* 1998;6:(suppl 2)51S-209S.

5. Cremonini F, Locke GR 3rd, Schleck CD, et al. Relationship between upper gastrointestinal symptoms and changes in body weight in a population-based cohort. *Neurogastroenterol Motil.* 2006;18:987-994.

6. Ronkainen J, Aro P, Storskrubb T, et al. High prevalence of gastroesophageal reflux symptoms and esophagitis with or without symptoms in the general adult Swedish population: a Kalixanda study report. *Scand J Gastroenterol.* 2005;40: 275-285.

7. Aro P, Ronkainen J, Talley NJ, et al. Body mass index and chronic unexplained gastrointestinal symptoms: an adult endoscopic population based study. *Gut.* 2005;54:1377-1383.

 Talley NJ, Howell S, Poulton R. Obesity and chronic gastrointestinal tract symptoms in young adults: a birth cohort study. *Am J Gastroenterol.* 2004;99: 1807-1814.

9. Delgado-Aros S, Locke GR 3rd, Camilleri M, et al. Obesity is associated with increased risk of gastrointestinal symptoms: a population-based study. *Am J Gastroenterol.* 2004;99:1801-1806.

10. Talley NJ, Quan C, Jones MP, Horowitz M. Association of upper and lower gastrointestinal tract symptoms with body mass index in an Australian cohort. *Neurogastroenterol Motil.* 2004;16:413-419.

11. Foster A, Laws HL, Gonzalez QH, Clements RH. Gastrointestinal symptomatic outcome after laparoscopic Roux-en-Y gastric bypass. *J Gastrointest Surg.* 2003;7:750-753.

12. Ramaswamy A, Lin E, Ramshaw BJ, Smith CD. Early effects of *Helicobacter pylori* infection in patients undergoing bariatric surgery. *Arch Surg.* 2004;139: 1094-1096.

13. Jackson SJ, Leahy FE, McGowan AA, et al. Delayed gastric emptying in the obese: an assessment using the non-invasive (13)C-octanoic acid breath test. *Diabetes Obes Metab.* 2004;6:264-270.

14. Vazquez Roque MI, Camilleri M, Stephens DA, et al. Gastric sensorimotor functions and hormone profile in normal weight, overweight, and obese people. *Gastroenterology*. 2006;131:1717-1724.

15. Park MI, Camilleri M. Gastric motor and sensory functions in obesity. *Obes Res.* 2005;13:491-500.

16. Corley DA, Kubo A, Zhao W. Abdominal obesity, ethnicity, and gastroesophageal reflux disease. *Gut.* 2007;56:756-762.

17. Corley DA, Kubo A. Body mass index and gastroesophageal reflux disease: a systematic review and meta-analysis. *Am J Gastroenterol*. 2006;101:2619-2628.

18. Lagergren J, Bergstrom R, Nyren O. No relation between body mass and gastro-oesophageal reflux symptoms in a Swedish population based study. *Gut.* 2000;47:26-29.

19. El-Serag HB, Graham DY, Satia JA, Rabeneck L. Obesity is an independent risk factor for GERD symptoms and erosive esophagitis. *Am J Gastroenterol.* 2005;100:1243-1250.

20. Hampel H, Abraham NS, El-Serag HB. Meta-analysis: obesity and the risk for gastroesophageal reflux disease and its complications. *Ann Intern Med.* 2005;143:199-211.

21. Fisher BL, Pennathur A, Mutnick JL, Little AG. Obesity correlates with gastroesophageal reflux. *Dig Dis Sci.* 1999;44:2290-2294.

22. El-Serag HB, Ergun GA, Pandolfino J, et al. Obesity increases esophageal acid exposure. *Gut.* 2007;56:749-755.

23. Wu JC, Mui LM, Cheung CM, et al. Obesity is associated with increased transient lower esophageal sphincter relaxation. *Gastroenterology*. 2007;132: 883-889.

24. Pandolfino JE, El-Serag HB, Zhang Q, et al. Obesity: a challenge to esophagogastric junction integrity. *Gastroenterology.* 2006;130:639-649.

25. Fraser-Moodie CA, Norton B, Gornall C, et al. Weight loss has an independent beneficial effect on symptoms of gastro-oesophageal reflux in patients who are overweight. *Scand J Gastroenterol.* 1999;34:337-340.

26. Quiroga E, Cuenca-Abente F, Flum D, et al. Impaired esophageal function in morbidly obese patients with gastroesophageal reflux disease: evaluation with multichannel intraluminal impedance. *Surg Endosc.* 2006;20:739-743.

27. Lagergren J, Bergstrom R, Nyren O. Association between body mass and adenocarcinoma of the esophagus and gastric cardia. *Ann Intern Med.* 1999;130:883-890.

 Kubo A, Corley DA. Body mass index and adenocarcinomas of the esophagus or gastric cardia: a systematic review and meta-analysis. *Cancer Epidemiol Biomarkers Prev.* 2006;15:872-878.

29. Samanic C, Chow WH, Gridley G, et al. Relation of body mass index to cancer risk in 362,552 Swedish men. *Cancer Causes Control*, 2006;17:901-909.

30. Ryan AM, Rowley SP, Fitzgerald AP, et al. Adenocarcinoma of the esophagus and gastric cardia: male preponderance in association with obesity. *Eur J Cancer.* 2006;42:1151-1158.

31. Lagergren J. Controversies surrounding body mass, reflux, and risk of oesophageal adenocarcinoma. *Lancet Oncol.* 2006;7:347-349.

32. McMillan DC, Sattar N, McArdle CS. ABC of obesity. Obesity and cancer. *BMJ*. 2006;333:1109-1111.

33. El-Serag HB. Obesity and disease of the esophagus and colon. *Gastroenterol Clin North Am.* 2005;34:63-82.

34. Maclure KM, Hayes KC, Colditz GA, et al. Weight, diet, and the risk of symptomatic gallstones in middle-aged women. *N Engl J Med.* 1989;321: 563-569.

35. Stampfer MJ, Maclure KM, Colditz GA, et al. Risk of symptomatic gallstones in women with severe obesity. *Am J Clin Nutr.* 1992;55:652-658.

36. Tsai C-J, Leitzmann MF, Willett WC, Giovannucci EL. Central adiposity, regional fat distribution, and the risk of cholecystectomy in women. *Gut.* 2006;55:708-714.

37. Petroni ML. Review article: gall bladder motor function in obesity. *Aliment Pharmacol Ther.* 2000;14(suppl 2):48-50.

38. Vanek VW, Catania M, Triveri K, Woodruff RW Jr. Retrospective review of the preoperative biliary and gastrointestinal evaluation for gastric bypass surgery. *Surg Obes Relat Dis.* 2006;2:17-22.

39. Torgerson JS, Lindroos AK, Naslund I, Peltonen M. Gallstones, gallbladder disease, and pancreatitis: cross-sectional and 2-year data from the Swedish Obese Subjects (SOS) and SOS reference studies. *Am J Gastroenterol.* 2003;98: 1032-1041.

40. Martinez J, Sanchez-Paya J, Palazon JM, et al. Is obesity a risk factor in acute pancreatitis? A meta-analysis. *Pancreatology*. 2004;4:42-48.

41. Martinez J, Johnson CD, Sanchez-Paya J, et al. Obesity is a definitive risk factor of severity and mortality in acute pancreatitis: an updated meta-analysis. *Pancreatology.* 2006;6:206-209.

42. Papachristou GI, Papachristou DJ, Avula H, et al. Obesity increases the severity of acute pancreatitis: performance of APACHE-O score and correlation with the inflammatory response. *Pancreatology*. 2006;6:279-285.

43. Diehl AM. Hepatic complications of obesity. *Gastroenterol Clin North Am.* 2005;34:45-61.

44. Frezza EE, Wachtel MS, Chiriva-Internati M. The influence of obesity on the risk of developing colon cancer. *Gut.* 2006;55:285-291.

45. Pischon T, Lahmann PH, Boeing H, et al. Body size and risk of colon and rectal cancer in the European Prospective Investigation Into Cancer and Nutrition (EPIC). *J Natl Cancer Inst.* 2006;98:920-931.

46. Gunter MJ, Leitzmann MF. Obesity and colorectal cancer: epidemiology, mechanisms and candidate genes. J Nutr Biochem. 2006;17:145-156.

 Rapp K, Schroeder J, Klenk J, et al. Obesity and incidence of cancer: a large cohort study of over 145,000 adults in Austria. *Br J Cancer.* 2005;93:1062-1067.
Lukanova A, Bjor O, Kaaks R, et al. Body mass index and cancer: results

from the Northern Sweden Health and Disease Cohort. *Int J Cancer*. 2006;118: 458-466.

49. Almendingen K, Hofstad B, Vatn MH. Does high body fatness increase the risk of presence and growth of colorectal adenomas followed up in situ for 3 years? *Am J Gastroenterol*. 2001;96:2238-2246.

50. Morita T, Tabata S, Mineshita M, et al. The metabolic syndrome is associated with increased risk of colorectal adenoma development: the Self-Defense Forces health study. *Asian Pac J Cancer Prev.* 2005;6:485-489.

51. Meyerhardt JA, Catalano PJ, Haller DG, et al. Influence of body mass index on outcomes and treatment-related toxicity in patients with colon carcinoma. *Cancer.* 2003;98:484-495.

52. Snow V, Barry P, Fitterman N, et al. Pharmacologic and surgical management of obesity in primary care: a clinical practice guideline from the American College of Physicians. *Ann Intern Med.* 2005;142:525-531.

53. Saltzman E, Anderson W, Apovian CM, et al. Criteria for patient selection and multidisciplinary evaluation and treatment of the weight loss surgery patient. *Obes Res.* 2005;13:234-243.

54. NIH conference. Gastrointestinal surgery for severe obesity. Consensus Development Conference Panel. *Ann Intern Med.* 1991;115:956-961.

55. Christou NV, Sampalis JS, Liberman M, et al. Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. *Ann Surg.* 2004; 240:416-423.

56. Ballantyne GH. Measuring outcomes following bariatric surgery: weight loss parameters, improvement in co-morbid conditions, change in quality of life and patient satisfaction. *Obes Surg.* 2003;13:954-964.

57. Schirmer B, Erenoglu C, Miller A. Flexible endoscopy in the management of patients undergoing Roux-en-Y gastric bypass. *Obes Surg.* 2002;12:634-638.

58. Verset D, Houben JJ, Gay F, et al. The place of upper gastrointestinal tract endoscopy before and after vertical banded gastroplasty for morbid obesity. *Dig Dis Sci.* 1997;42:2333-2337.

 Frigg A, Peterli R, Zynamon A, et al. Radiologic and endoscopic evaluation for laparoscopic adjustable gastric banding: preoperative and follow-up. *Obes Surg.* 2001;11:594-599.

 Korenkov M, Sauerland S, Shah S, Junginger T. Is routine preoperative upper endoscopy in gastric banding patients really necessary? *Obes Surg.* 2006;16:45-47.
Sharaf RN, Weinshel EH, Bini EJ, et al. Endoscopy plays an important preoperative role in bariatric surgery. *Obes Surg.* 2004;14:1367-1372.

62. Azagury D, Dumonceau JM, Morel P, et al. Preoperative work-up in asymptomatic patients undergoing Roux-en-Y gastric bypass: is endoscopy mandatory? *Obes Surg.* 2006;16:1304-1311.

63. Lujan JA, Frutos MD, Hernandez Q, et al. Laparoscopic versus open gastric bypass in the treatment of morbid obesity: a randomized prospective study. *Ann Surg.* 2004;239:433-437.

64. Podnos YD, Jimenez JC, Wilson SE, et al. Complications after laparoscopic gastric bypass: a review of 3,464 cases. *Arch Surg.* 2003;138:957-961.

65. Morino M, Toppino M, Bonnet G, del Genio G. Laparoscopic adjustable silicone gastric banding versus vertical banded gastroplasty in morbidly obese patients: a prospective randomized controlled clinical trial. *Ann Surg*, 2003;238:835-841. 66. Angrisani L, Furbetta F, Doldi SB, et al. Lap Band adjustable gastric banding system: the Italian experience with 1863 patients operated on 6 years. *Surg Endosc.* 2003;17:409-412.

67. Roman S, Napoleon B, Mion F, et al. Intragastric balloon for "non-morbid" obesity: a retrospective evaluation of tolerance and efficacy. *Obes Surg.* 2004; 14:539-544.

68. Totte E, Hendrickx L, Pauwels M, Van Hee R. Weight reduction by means of intragastric device: experience with the bioenterics intragastric balloon. *Obes Surg.* 2001;11:519-523.

69. Sallet JA, Marchesini JB, Paiva DS, et al. Brazilian multicenter study of the intragastric balloon. *Obes Surg.* 2004;14:991-998.

70. Doldi SB, Micheletto G, Perrini MN, et al. Treatment of morbid obesity with intragastric balloon in association with diet. *Obes Surg.* 2002;12:583-587.

71. Evans JD, Scott MH. Intragastric balloon in the treatment of patients with morbid obesity. *Br J Surg.* 2001;88:1245-1248.

72. Regan JP, Inabnet WB, Gagner M, Pomp A. Early experience with two-stage laparoscopic Roux-en-Y gastric bypass as an alternative in the super-super obese patient. *Obes Surg.* 2003;13:861-864.

73. Mognol P, Chosidow D, Marmuse JP. Laparoscopic sleeve gastrectomy as an initial bariatric operation for high-risk patients: initial results in 10 patients. *Obes Surg.* 2005;15:1030-1033.

74. Milone L, Strong V, Gagner M. Laparoscopic sleeve gastrectomy is superior to endoscopic intragastric balloon as a first stage procedure for super-obese patients. *Obes Surg.* 2005;15:612-617.

75. Nanni G, Balduzzi G, Botta C, et al. Biliopancreatic diversion. Clinical experience. *Minerva Gastroenterol Dietol.* 2005;51:209-212.

 Ren CJ, Patterson E, Gagner M. Early results of laparoscopic biliopancreatic diversion with duodenal switch: a case series of 40 consecutive patients. *Obes Surg.* 2000;10:514-523.

77. Ovsiowitz M, Kanagarajan N, Ahmad AS. Endoscopic issues in the post-gastric bypass patient. *Gastrointest Endosc Clin N Am*. 2006;16:121-132.

78. Martin JA, Pandolfino JE. Gastrointestinal complications of bariatric surgery. *Curr Gastroenterol Rep.* 2005;7:321-328.

79. Kaplan LM. Gastrointestinal management of the bariatric surgery patient. *Gastroenterol Clin North Am.* 2005;34:105-125.

80. Weinsier RL, Wilson LJ, Lee J. Medically safe rate of weight loss for the treatment of obesity: a guideline based on risk of gallstone formation. *Am J Med.* 1995; 98:115-117.

81. Al-Jiffry BO, Shaffer EA, Saccone GT, et al. Changes in gallbladder motility and gallstone formation following laparoscopic gastric banding for morbid obesity. *Can J Gastroenterol.* 2003;17:169-174.

82. Villegas L, Schneider B, Provost D, et al. Is routine cholecystectomy required during laparoscopic gastric bypass? *Obes Surg.* 2004;14:60-66.

83. Sanyal AJ, Sugerman HJ, Kellum JM, et al. Stomal complications of gastric bypass: incidence and outcome of therapy. *Am J Gastroenterol.* 1992;87:1165-1169.

84. Barba CA, Butensky MS, Lorenzo M, Newman R. Endoscopic dilation of gastroesophageal anastomosis stricture after gastric bypass. *Surg Endosc.* 2003; 17:416-420.

85. Oczkowski WJ, Kertesz A. Wernicke's encephalopathy after gastroplasty for morbid obesity. *Neurology*. 1985;35:99-101.

86. Melinek J, Livingston E, Cortina G, Fishbein MC. Autopsy findings following gastric bypass surgery for morbid obesity. *Arch Pathol Lab Med.* 2002;126: 1091-1095.

87. Sugerman HJ, Sugerman EL, Wolfe L, et al. Risks and benefits of gastric bypass in morbidly obese patients with severe venous stasis disease. *Ann Surg.* 2001;234:41-46.

88. Wu EC, Barba CA. Current practices in the prophylaxis of venous thromboembolism in bariatric surgery. *Obes Surg.* 2000;10:7-13.

89. Pories WJ, van Rij AM, Burlingham BT, et al. Prophylactic cefazolin in gastric bypass surgery. *Surgery*. 1981;90:426-432.

90. Khurana RN, Baudendistel TE, Morgan EF, et al. Postoperative rhabdomyolysis following laparoscopic gastric bypass in the morbidly obese. *Arch Surg.* 2004;139:73-76.

91. Nguyen NT, Longoria M, Chalifoux S, Wilson SE. Gastrointestinal hemorrhage after laparoscopic gastric bypass. *Obes Surg*. 2004;14:1308-1312.

92. Madan AK, Kuykendall SJ 4th, Ternovits CA, Tichansky DS. Mallory-Weiss tear after laparoscopic Roux-en-Y gastric bypass. *Surg Obes Relat Dis.* 2005;1: 500-502.

93. De Roover A, Detry O, Desaive C, et al. Risk of upper gastrointestinal cancer after bariatric operations. *Obes Surg.* 2006;16:1656-1661.

94. Freeman HJ. Risk of gastrointestinal malignancies and mechanisms of cancer development with obesity and its treatment. *Best Pract Res Clin Gastroenterol.* 2004;18:1167-1175.

95. Schneider BE, Villegas L, Blackburn GL, et al. Laparoscopic gastric bypass surgery: outcomes. *J Laparoendosc Adv Surg Tech A*. 2003;13:247-255.

96. Gonzalez R, Bowers SP, Venkatesh KR, et al. Preoperative factors predictive of complicated postoperative management after Roux-en-Y gastric bypass for morbid obesity. *Surg Endosc.* 2003;17:1900-1904.

97. Perugini RA, Mason R, Czerniach DR, et al. Predictors of complication and suboptimal weight loss after laparoscopic Roux-en-Y gastric bypass: a series of 188 patients. *Arch Surg.* 2003;138:541-545.

98. Papasavas PK, Caushaj PF, McCormick JT, et al. Laparoscopic management of complications following laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Surg Endosc.* 2003;17:610-614.

99. Rossi TR, Dynda DI, Estes NC, Marshall JS. Stricture dilation after laparoscopic Roux-en-Y gastric bypass. *Am J Surg.* 2005;189:357-360.

100. Goitein D, Papasavas PK, Gagne D, et al. Gastrojejunal strictures following laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Surg Endosc.* 2005;19:628-632.

101. Escalona A, Devaud N, Boza C, et al. Gastrojejunal anastomotic stricture after Roux-en-Y gastric bypass: ambulatory management with the Savary-Gilliard dilator. *Surg Endosc.* 2007;21:765-768.

102. Wilson JA, Romagnuolo J, Byrne TK, et al. Predictors of endoscopic findings after Roux-en-Y gastric bypass. *Am J Gastroenterol.* 2006;101:2194-2199.

103. Ahmad J, Martin J, Ikramuddin S, et al. Endoscopic balloon dilation of gastroenteric anastomotic stricture after laparoscopic gastric bypass. *Endoscopy*. 2003;35:725-728.

104. Champion JK, Williams M. Small bowel obstruction and internal hernias after laparoscopic Roux-en-Y gastric bypass. *Obes Surg.* 2003;13:596-600.

105. Merrifield BF, Lautz D, Thompson CC. Endoscopic repair of gastric leaks after Roux-en-Y gastric bypass: a less invasive approach. *Gastrointest Endosc.* 2006;63:710-714.

106. Carucci LR, Turner MA, Conklin RC, et al. Roux-en-Y gastric bypass surgery for morbid obesity: evaluation of postoperative extraluminal leaks with upper gastrointestinal series. *Radiology*. 2006;238:119-127.

107. Lyass S, Khalili TM, Cunneen S, et al. Radiological studies after laparoscopic Roux-en-Y gastric bypass: routine or selective? *Am Surg.* 2004;70:918-921.

108. Gonzalez R, Sarr MG, Smith CD, et al. Diagnosis and contemporary management of anastomotic leaks after gastric bypass for obesity. *J Am Coll Surg.* 2007;204:47-55.

109. Madan AK, Stoecklein HH, Ternovits CA, et al. Predictive value of upper gastrointestinal studies versus clinical signs for gastrointestinal leaks after laparoscopic gastric bypass. *Surg Endosc.* 2007;21:194-196.

110. Hamilton EC, Sims TL, Hamilton TT, et al. Clinical predictors of leak after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Surg Endosc.* 2003;17:679-684.

111. Gumbs AA, Duffy AJ, Bell RL. Incidence and management of marginal ulceration after laparoscopic Roux-Y gastric bypass. *Surg Obes Relat Dis.* 2006;2:460-463.

112. Gumbs AA, Duffy AJ, Bell RL. Management of gastrogastric fistula after laparoscopic Roux-en-Y gastric bypass. *Surg Obes Relat Dis.* 2006;2:117-121.

113. Carrodeguas L, Szomstein S, Soto F, et al. Management of gastrogastric fistulas after divided Roux-en-Y gastric bypass surgery for morbid obesity: analysis of 1,292 consecutive patients and review of literature. *Surg Obes Relat Dis.* 2005;1:467-474.

114. Awais O, Raftopoulos I, Luketich JD, Courcoulas A. Acute, complete proximal small bowel obstruction after laparoscopic gastric bypass due to intraluminal blood clot formation. *Surg Obes Relat Dis.* 2005;1:418-422.

115. Sakai P, Kuga R, Safatle-Ribeiro AV, et al. Is it feasible to reach the bypassed stomach after Roux-en-Y gastric bypass for morbid obesity? The use of the double-balloon enteroscope. *Endoscopy*. 2005;37:566-569.

116. Thompson CC, Slattery J, Bundga ME, Lautz DB. Peroral endoscopic reduction of dilated gastrojejunal anastomosis after Roux-en-Y gastric bypass: a possible new option for patients with weight regain. *Surg Endosc.* 2006;20:1744-1748.

117. Nosher JL, Bodner LJ, Girgis WS, et al. Percutaneous gastrostomy for treating dilatation of the bypassed stomach after bariatric surgery for morbid obesity. *AJR Am J Roentgenol.* 2004;183:1431-1435.

118. Martinez J, Guerrero L, Byers P, et al. Endoscopic retrograde cholangiopancreatography and gastroduodenoscopy after Roux-en-Y gastric bypass. *Surg Endosc.* 2006;20:1548-1550.

119. Foster A, Laws HL, Gonzalez QH, Clements RH. Gastrointestinal symptomatic outcome after laparoscopic Roux-en-Y gastric bypass. *J Gastrointest Surg.* 2003;7:750-753.

120. Huang CS, Forse RA, Jacobson BC, Farraye FA. Endoscopic findings and their clinical correlations in patients with symptoms after gastric bypass surgery. *Gastrointest Endosc.* 2003;58:859-866.

121. Suter M, Jayet C, Jayet A. Vertical banded gastroplasty: long-term results comparing three different techniques. *Obes Surg.* 2000;10:41-46.

122. Sataloff DM, Lieber CP, Seinige UL. Strictures following gastric stapling for morbid obesity. Results of endoscopic dilatation. *Am Surg.* 1990;56:167-174.

123. MacLean LD, Rhode BM, Sampalis J. Results of the surgical treatment of obesity. *Am J Surg*, 1993;165:155-162.

124. Evans JA, Williams NN, Chan EP, Kochman ML. Endoscopic removal of eroded bands in vertical banded gastroplasty: a novel use of endoscopic scissors (with video). *Gastrointest Endosc.* 2006;64:801-804.

125. Regusci L, Groebli Y, Meyer JL, et al. Gastroscopic removal of an adjustable gastric band after partial intragastric migration. *Obes Surg.* 2003;13:281-284.

126. Di Francesco V, Baggio E, Mastromauro M, et al. Obesity and gastroesophageal acid reflux: physiopathological mechanisms and role of gastric bariatric surgery. *Obes Surg*, 2004;14:1095-1102.

127. Ovrebo KK, Hatlebakk JG, Viste A, et al. Gastroesophageal reflux in morbidly obese patients treated with gastric banding or vertical banded gastroplasty. *Ann Surg.* 1998;228:51-58.

128. Ren CJ, Horgan S, Ponce J. US experience with the LAP-BAND system. *Am J Surg.* 2002;184:46S-50S.

129. Rubenstein RB. Laparoscopic adjustable gastric banding at a U.S. center with up to 3-year follow-up. *Obes Surg*, 2002;12:380-384.

130. DeMaria EJ. Laparoscopic adjustable silicone gastric banding: complications. J Laparoendosc Adv Surg Tech A. 2003;13:271-277.

131. Niville E, Dams A. Late pouch dilation after laparoscopic adjustable gastric and esophagogastric banding: incidence, treatment, and outcome. *Obes Surg.* 1999;9:381-384.

132. Gutschow CA, Collet P, Prenzel K, et al. Long-term results and gastroesophageal reflux in a series of laparoscopic adjustable gastric banding. *J Gastrointest Surg.* 2005;9:941-948.

133. Tolonen P, Victorzon M, Niemi R, Makela J. Does gastric banding for morbid obesity reduce or increase gastroesophageal reflux? *Obes Surg.* 2006;16: 1469-1474.

134. Klaus A, Gruber I, Wetscher G, et al. Prevalent esophageal body motility disorders underlie aggravation of GERD symptoms in morbidly obese patients following adjustable gastric banding. *Arch Surg.* 2006;141:247-251.

135. de Jong JR, van Ramshorst B, Timmer R, et al. Effect of laparoscopic gastric banding on esophageal motility. *Obes Surg.* 2006;16:52-58.

136. Suter M, Dorta G, Giusti V, Calmes JM. Gastric banding interferes with esophageal motility and gastroesophageal reflux. *Arch Surg.* 2005;140:639-643.

137. Genco A, Bruni T, Doldi SB, et al. BioEnterics intragastric balloon: the Italian experience with 2,515 patients. *Obes Surg.* 2005;15:1161-1164.

138. Eynden FV, Urbain P. Small intestine gastric balloon impaction treated by laparoscopic surgery. *Obes Surg.* 2001;11:646-648.

139. Deitel M, Shahi B, Anand PK, et al. Long-term outcome in a series of jejunoileal bypass patients. *Obes Surg*, 1993;3:247-252.

140. Drenick EJ, Fisler J, Johnson D. Hepatic steatosis after intestinal bypass-prevention and reversal with metronidazole, irrespective of protein-calorie malnutrition. *Gastroenterology*. 1982;82:535-548.

141. Griffen WO, Bivins BA, Bell RM. The decline and fall of the jejunoileal bypass. *Surg Gynecol Obstet.* 1983;157:301-308.

142. Scopinaro N, Adami GF, Marinari GM. Biliopancreatic diversion. World J Surg. 1998;22:936-946.

143. Genco A, Cipriano M, Bacci V, et al. BioEnterics Intragastric Balloon (BIB): a short-term, double-blind, randomised, controlled, crossover study on weight reduction in morbidly obese patients. *Int J Obes.* 2006;30:129-133.

144. Hu B, Chung SC, Sun LC, et al. Transoral obesity surgery: endoluminal gastroplasty with an endoscopic suture device. *Endoscopy*. 2005;37:411-414.

145. Awan AN, Swain CP. Endoscopic vertical band gastroplasty with an endoscopic sewing machine. *Gastrointest Endosc.* 2002;55:254-256.

146. Ouyang H, Yin J, Chen JD. Gastric or intestinal electrical stimulationinduced increase in gastric volume is correlated with reduced food intake. *Scand J Gastroenterol*. 2006;41:1261-1266.

147. Miller K, Hoeller E, Aigner F. The implantable gastric stimulator for obesity: an update of the European experience in the LOSS (Laparoscopic Obesity Stimulation Survey) study. *Treat Endocrinol.* 2006;5:53-58.

148. Cigaina V. Long-term follow-up of gastric stimulation for obesity: the Mestre 8-year experience. *Obes Surg.* 2004;14(suppl 1):S14-22.

149. Gostout CJ, Rajan E. Endoscopic treatments for obesity: past, present and future. *Gastroenterol Clin North Am.* 2005;34:143-150.