

AMERICAN ASSOCIATION OF CLINICAL ENDOCRINOLOGISTS, THE OBESITY SOCIETY, AND AMERICAN SOCIETY FOR METABOLIC & BARIATRIC SURGERY MEDICAL GUIDELINES FOR CLINICAL PRACTICE FOR THE PERIOPERATIVE NUTRITIONAL, METABOLIC, AND NONSURGICAL SUPPORT OF THE BARIATRIC SURGERY PATIENT

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American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery Medical Guidelines for Clinical Practice are systematically developed statements to assist health-care professionals in medical decision making for specific clinical conditions. Most of the content herein is based on literature reviews. In areas of uncertainty, professional judgment was applied.

These guidelines are a working document that reflects the state of the field at the time of publication. Because rapid changes in this area are expected, periodic revisions are inevitable. We encourage medical professionals to use this information in conjunction with their best clinical judgment. The presented recommendations may not be appropriate in all situations. Any decision by practitioners to apply these guidelines must be made in light of local resources and individual patient circumstances.

The American Society for Parenteral & Enteral Nutrition fully endorses sections of these guidelines that address the metabolic and nutritional management of the bariatric surgical patient.

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Abbreviations:

AACE = American Association of Clinical Endocrinologists; **ACS** = American College of Surgeons; **ASMBS** = American Society for Metabolic & Bariatric Surgery; **BEL** = “best evidence” rating level; **BMD** = bone mineral density; **BMI** = body mass index; **BPD** = biliopancreatic diversion; **BPD/DS** = biliopancreatic diversion with duodenal switch; **CAD** = coronary artery disease; **CK** = creatine kinase; **CPAP** = continuous positive airway pressure; **CPG** = clinical practice guidelines; **CSF** = cerebrospinal fluid; **CT** = computed tomography; **DVT** = deep venous thrombosis; **EBW** = excess body weight; **EFA** = essential fatty acids; **EL** = evidence level; **FA** = fatty acids; **GERD** = gastroesophageal reflux disease; **GLP-1** = glucagon-like peptide-1; **HDL** = high-density lipoprotein; **ICU** = intensive care unit; **LAGB** = laparoscopic adjustable gastric band; **LDL** = low-density lipoprotein; **MI** = myocardial infarction; **NAFLD** = nonalcoholic fatty liver disease; **NIH** = National Institutes of Health; **1,25-(OH)₂D** = 1,25-dihydroxyvitamin D; **25-OHD** = 25-hydroxyvitamin D; **OHS** = obesity-hypoventilation syndrome; **OSA** = obstructive sleep apnea; **PCOS** = polycystic ovary syndrome; **PE** = pulmonary embolus; **PN** = parenteral nutrition; **PTH** = parathyroid hormone; **R** = recommendation; **RDI** = respiratory disturbance index; **RYGB** = Roux-en-Y gastric bypass; **SOS** = Swedish Obese Subjects; **T1DM** = type 1 diabetes mellitus; **T2DM** = type 2 diabetes mellitus; **TOS** = The Obesity Society; **UGI** = upper gastrointestinal; **VBG** = vertical banded gastroplasty

1. PREFACE

Surgical therapy for obesity, or “bariatric surgery,” is indicated for certain high-risk patients, termed by the National Institutes of Health (NIH) as having “clinically severe obesity.” These clinical practice guidelines (CPG) are cosponsored by the American Association of Clinical Endocrinologists (AACE), The Obesity Society (TOS), and the American Society for Metabolic & Bariatric Surgery (ASMBS). These guidelines represent an extension of the AACE/American College of Endocrinology Obesity Task Force position statements (1 [evidence level or EL 4], 2 [EL 4]) and the National Heart, Lung, and Blood Institute and the North American Association for the Study of Obesity Practical Guide to the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults (3 [EL 4]). These CPG will focus on the nonsurgical aspects of perioperative management of the bariatric surgery patient, with special emphasis on nutritional and metabolic support. The organization of these CPG is as follows: (1) an *introduction* section to familiarize the reader with the principles of bariatric surgery, (2) a *Methods*

section to outline the a priori evidence-based system of recommendations, (3) an *Executive Summary* section of specific, practical evidence-based recommendations, (4) an *Appendix* section containing in-depth discussions and ratings of the clinical evidence referred to in the Executive Summary of Recommendations, and lastly (5) an extensive *Reference* section in which each clinical report or study is assigned an evidence level (for further details, see Section 7.3).

2. HISTORY OF BARIATRIC SURGERY

The original bariatric surgical procedure was the jejunocolic bypass, followed shortly thereafter by the jejunoileal bypass. This approach was introduced in 1954 and consisted of 14 inches (35.6 cm) of jejunum connected to 4 inches (10.2 cm) of ileum as either an end-to-end or an end-to-side anastomosis, which bypassed most of the small intestine (4 [EL 4], 5 [EL 3]). This procedure resulted in substantial weight loss but with an unacceptably high risk of unanticipated early and late complications, including life-threatening hepatic failure and cirrhosis, renal failure, oxalate nephropathy, immune complex disease, and multiple nutritional deficiencies (6 [EL 4]). Because of these complications, the jejunoileal bypass procedure is no longer performed. Nevertheless, because there may still be patients who have had this procedure who seek nutritional and metabolic management from their health-care providers, familiarity with this outdated procedure is justified.

In the late 1970s, the gastric bypass was developed on the basis of information gathered from gastrectomy procedures and then modified to a Roux-en-Y anastomosis. This procedure was found to have equivalent weight loss to the jejunoileal bypass but with a much lower risk of complications (7 [EL 3]). At present, there are 3 broad categories of bariatric procedures: (1) purely gastric restriction, (2) gastric restriction with some malabsorption, as represented by the Roux-en-Y gastric bypass (RYGB), and (3) gastric restriction with significant intestinal malabsorption. Estimates suggest that the number of bariatric procedures performed in the United States increased from 13,365 in 1998 to nearly 150,000 in 2005 (8 [EL 4]) and to approximately 200,000 procedures in 2007, according to the ASMBS. In 1998 in the United States, there were approximately 250 bariatric surgeons, which increased to approximately 700 in 2001 and expanded to nearly 1,100 by 2003 (9 [EL 4], 10 [EL 4]). Currently, RYGB procedures account for more than 80% of bariatric operations according to unpublished data from the ASMBS, although the proportion is changing with the advent of the laparoscopic adjustable gastric band (LAGB) procedure (11 [EL 2]). The majority of patients (80%) are female, from a higher socioeconomic class, privately insured, and between 40 and 64 years of age (12 [EL 3]). When one considers the prevalence of class 3 obesity among US adults of nearly 5% of the population—which equals approximately 10

million individuals (13 [EL 3])—there are almost 10,000 potential surgical candidates for every bariatric surgeon (9 [EL 4], 14 [EL 3]).

3. INDICATIONS FOR BARIATRIC SURGERY

Overweight and obesity are at epidemic proportions in the United States, affecting nearly 65% (or approximately 130 million) of the adult population (13 [EL 3]). Obesity is defined as a body mass index (BMI; weight in kg/[height in meters]²) ≥ 30 kg/m², in an overall classification in which the healthy range of weight is 18.5 to 24.9 kg/m², overweight is 25 to 29.9 kg/m², class 1 obesity is 30 to 34.9 kg/m², class 2 obesity is 35 to 39.9 kg/m², and class 3 obesity is ≥ 40 kg/m². Some groups further subcategorize this last entity into class 4 obesity (superobesity) as 50 to 59.9 kg/m² and class 5 obesity (super-superobesity) (15 [EL 3]) as >60 kg/m² (16 [EL 4]). The older terminology of *morbid obesity*, empirically defined as more than 100 lb (45.4 kg) or 100% over ideal body weight, has been replaced with newer descriptive terms, including class 3 obesity, extreme obesity, or clinically severe obesity. “Morbid obesity,” however, is still listed in the *International Classification of Diseases, Ninth Revision, Clinical Modification*. The term “morbid obesity” is used for coding and is also used by the National Library of Medicine and in medical journals and texts. In these guidelines, these terms are used interchangeably. Class 3 obesity was present in 3.1% of the 2005 American adult population (17 [EL 4]). Its prevalence had quadrupled between 1986 and 2000 (class 4 obesity increased 5-fold during the same period) and increased another 2-fold between 2000 and 2005 (class 4 obesity increased 3-fold during the same period) (17 [EL 4], 18 [EL 4]). Class 3 obesity has also been associated with a notable increase in mortality, especially for male subjects, in comparison with that for nonobese patients (19 [EL 4]). A BMI ≥ 45 kg/m² is associated with a decrease of 13 and 8 years of life expectancy for white male and female subjects, respectively, and a decrease of 20 years for the younger black male population (20 [EL 4]). Cardiovascular mortality is 50% greater in obese people and 90% greater in severely obese persons in comparison with that for people of average weight (21 [EL 3]). More than \$238 billion has been spent annually on obesity in the United States (9 [EL 4]). In sum, because of the dramatically increased risk of morbidity and mortality associated with extreme obesity, such patients who do not achieve a significant weight reduction with therapeutic lifestyle changes or pharmacotherapy (or both) would benefit from surgical treatment.

The 1991 NIH Consensus Development Conference Panel (22 [EL 4], 23 [EL 4]) established the following general criteria for eligibility for bariatric surgery: patients with BMI ≥ 40 kg/m² could be considered surgical candidates; patients with less severe obesity (BMI ≥ 35 kg/m²) could be considered if they had high-risk comorbid conditions such as life-threatening cardiopulmonary problems

(for example, severe sleep apnea, pickwickian syndrome, or obesity-related cardiomyopathy) or uncontrolled type 2 diabetes mellitus (T2DM). Other possible indications for patients with BMIs between 35 and 40 kg/m² include obesity-induced *physical* problems interfering with lifestyle (for example, joint disease treatable but for the obesity, or body size problems precluding or severely interfering with employment, family function, and ambulation) (22 [EL 4], 23 [EL 4]).

Since the 1991 NIH consensus conference, there have been at least 13 systematic reviews of the bariatric surgery literature (24-36 [EL 4]). Although there have been new procedures and techniques since 1991, these assessments deviate little from the NIH recommendations. One review, however, is substantially different. Medicare initiated an internal, evidence-based review of the bariatric surgical literature (30 [EL 4]). In November 2004, the National Coverage Advisory Committee received input from experts and the lay community such that they could evaluate the available evidence in the proper context of clinical needs in relationship to evidence (30 [EL 4]). After this extensive analysis, the panel concluded that bariatric surgery could be offered to Medicare beneficiaries with BMI ≥ 35 kg/m² who have at least one comorbidity associated with obesity and have been unsuccessful previously with medical treatment of obesity (<http://www.cms.hhs.gov/MLN MattersArticles/downloads/MM5013.pdf>) (30 [EL 4]). Initially, the panel concluded that the evidence did not support bariatric surgery for patients ≥ 65 years of age, and the initial decision did not support bariatric surgery in this age-group (30 [EL 4]). Older surgical patients most likely will have more complications and deaths (37-40 [EL 3]); however, some case series have reported excellent outcomes (41-45 [EL 2-3]). The National Coverage Advisory Committee panel carefully considered the surgical risks for older patients and could not conclude that these procedures should not be offered to older individuals (30 [EL 4]). Hence, the National Coverage Decision for bariatric surgery issued in February 2006 did not stipulate an age limit for such surgical procedures (46 [EL 4]).

In 2006, the US Department of Veterans Affairs and the Department of Defense published their evidence-based Clinical Practice Guideline for Management of Overweight and Obesity, in which bariatric surgery is reported to be associated with successful weight loss and improvement of comorbid conditions, quality of life, and long-term survival (>5 years) (47 [EL 4]).

Currently, a consensus does not exist on the possible contraindications to bariatric surgery. Suggested contraindications would include an extremely high operative risk (such as severe congestive heart failure or unstable angina), active substance abuse, or a major psychopathologic condition (48 [EL 4]). Patients who cannot comprehend the nature of the surgical intervention and the lifelong measures required to maintain an acceptable level of health should not be offered these procedures.

A controversial issue that is reflected by the divergent preoperative strategies among various bariatric programs in the United States is whether or not patients should lose weight (approximately 10%) *before* bariatric surgery. Two studies suggested that preoperative weight loss was associated with greater weight loss 1 year postoperatively (49 [EL 3], 50 [EL 2]). In a randomized study of 100 patients undergoing RYGB, Alami et al (51 [EL 2]) found that preoperative weight loss of 10% was associated with improved short-term (6 months) but not long-term weight loss. In contrast, another study found that insurance-mandated preoperative weight loss did not improve postoperative weight loss and was associated with increased dropout rates before gastric bypass surgery (52 [EL 3]). Individuals who seek bariatric surgery typically report an extensive dieting history, which further calls into question the utility of insurance-mandated weight loss preoperatively (53 [EL 3]). A recent study, however, suggested a more functional benefit of preoperative weight loss. In this prospective trial, at least 2 weeks of a very-low-calorie meal plan preoperatively significantly reduced liver volume and thereby potentially improved operative exposure (54 [EL 2]). On balance, consideration should be given to recommendation of preoperative weight loss, particularly in patients with hepatomegaly.

4. TYPES OF BARIATRIC SURGERY

Various bariatric procedures are available for management of high-risk obese patients (Table 1 and Fig. 1). Minimal scientific data exist for establishing which procedure should be performed for which patient. Currently, most bariatric procedures are being performed laparoscopically. This approach has the advantages of fewer wound complications, less postoperative pain, a briefer hospital stay, and more rapid postoperative recovery with comparable efficacy (55-59 [EL 2-4]). These advantages, however, may be offset by more frequent complications associated with techniques used for laparoscopic gastrojejunostomy creation, anastomotic strictures, and higher rates of postoperative bowel obstructions (60 [EL 4], 61 [EL 3], 62 [EL 2]).

4.1. Gastric Restriction

The purposes of a gastric restriction procedure are to produce early satiety, limit food intake, and thus induce weight loss. Gastric restriction can be performed by the vertical banded gastroplasty (VBG) by means of (1) limiting the volume of an upper gastric pouch, into which the esophagus empties, to 15 to 45 mL and (2) limiting the pouch outlet to the remaining stomach to 10 to 11 mm. Currently, the LAGB has almost completely replaced the VBG because it is less invasive, is adjustable, and is reversible, and it has better outcomes (8 [EL 4]).

The LAGB procedure is associated with not only substantially better maintenance of weight loss than lifestyle intervention alone (4 [EL 4], 63 [EL 2]) but also a very

low operative mortality rate (0.1%). In one randomized, prospective trial, however, it was associated with significantly less excess weight loss than RYGB at 5 years (11 [EL 2]), which is consistent with the findings of the 10-year Swedish Obese Subjects (SOS) Study that used a nonadjustable gastric band (64 [EL 3], 65 [EL 3]). In a systematic literature search and review, LAGB was associated with less loss of fat-free mass (lean tissue such as muscle) compared with RYGB and biliopancreatic diversion (BPD) (62 [EL 2]). Factors that are associated with greater weight loss after LAGB include an initial BMI <45 kg/m² and the presence of postprandial satiety postoperatively (66 [EL 2]). The LAGB procedure has also been demonstrated to be safe among patients >55 years of age (67 [EL 3]). Complications associated with the LAGB include band slippage, band erosion, balloon failure, port malposition, band and port infections, and esophageal dilatation. Some of these problems have been decreased by a different method of band insertion (the *pars flaccida* instead of the *perigastric* approach) and revision of the port connection (63 [EL 2], 68 [EL 2]). Overall, complication and mortality rates are much lower for LAGB than for RYGB.

Table 1
Types of Bariatric Surgical Procedures

Primary

- Vertical banded gastroplasty
- Gastric banding
- Silastic ring gastroplasty
- Laparoscopic adjustable gastric band (LAGB)
- Roux-en-Y gastric bypass
 - Standard
 - Long-limb
 - Distal
- Biliopancreatic diversion (BPD)
- BPD with duodenal switch (BPD/DS)
- Staged restrictive and malabsorptive procedure

Secondary

- Reversal of gastric restriction
- Revision of Roux-en-Y gastric bypass
- Revision of BPD
- Revision of BPD/DS
- Conversion of LAGB to Roux-en-Y gastric bypass
- Conversion of LAGB to BPD or BPD/DS

Investigational

- Gastric bypass with LAGB
- Robotic procedures
- Endoscopic (oral)-assisted techniques
- Gastric balloon
- Gastric pacer
- Vagus nerve pacing
- Vagus nerve block
- Sleeve gastrectomy

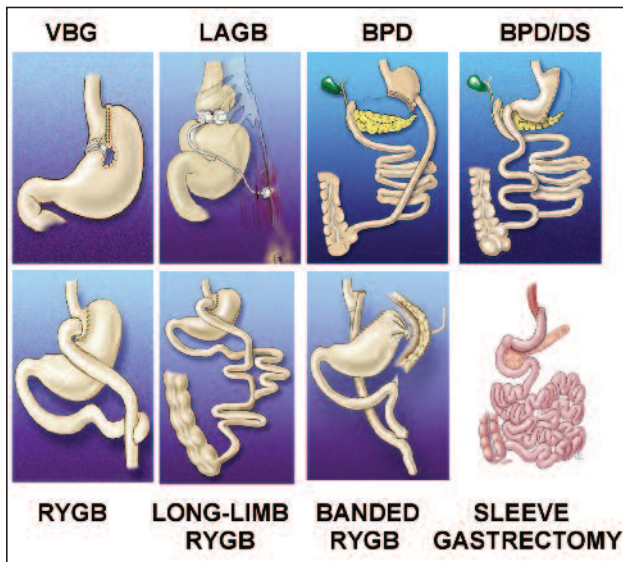


Fig. 1. Currently available bariatric surgical procedures. See text for descriptions of procedures. *BPD* = biliopancreatic diversion; *BPD/DS* = BPD with duodenal switch; *LAGB* = laparoscopic adjustable gastric band; *RYGB* = Roux-en-Y gastric bypass; *VBGB* = vertical banded gastroplasty. The first 7 graphics (*VBGB*, *LAGB*, *BPD*, *BPD/DS*, *RYGB*, Long-Limb *RYGB*, and Banded *RYGB*) are reprinted with permission of the American Society for Metabolic and Bariatric Surgery, copyright 2008, all rights reserved. The last graphic (Sleeve Gastrectomy) is reprinted with the permission of The Cleveland Clinic Center for Medical Art & Photography, copyright 2008. All Rights Reserved.

4.2. Roux-en-Y Gastric Bypass

Currently, in the United States, *RYGB* is the most commonly performed bariatric procedure (8 [EL 4], 69 [EL 3]). The weight loss achieved with *RYGB* is greater than that attained with pure gastric restrictive procedures (11 [EL 2], 64 [EL 3], 65 [EL 3], 70 [EL 2]). In *RYGB*, the upper part of the stomach is transected; thus, a very small proximal gastric pouch, measuring 10 to 30 mL, is created. The gastric pouch is anastomosed to a Roux-en-Y proximal jejunal segment, bypassing the remaining stomach, duodenum, and a small portion of jejunum. The standard Roux (alimentary) limb length is about 50 to 100 cm, and the biliopancreatic limb is 15 to 50 cm. As a result, the *RYGB* limits food intake and induces some nutrient malabsorption. In procedures that result in much longer Roux limbs (“distal gastric bypass”) and a short common channel, macronutrient malabsorption can be significant (71 [EL 3], 72 [EL 3]). Another modification that has been used involves combining the gastric band with the *RYGB* (“banded *RYGB*”) (73 [EL 3], 74 [EL 4], 75 [EL 3]). Studies have been published regarding conversion of *VBGB* and *LAGB* procedures to *RYGB* for band-related complications, staple-line disruptions, and inadequate weight loss (76-78 [EL 3]).

4.3. Biliopancreatic Diversion

The *BPD* was developed by Scopinaro et al (79 [EL 3]) as a hybrid bariatric surgical procedure incorporating

gastric restrictive and extensive malabsorptive components. In this procedure, a subtotal gastrectomy is performed, and a proximal gastric pouch of 200 to 500 mL is created. The distal 250-cm segment of small intestine is isolated from the proximal small intestinal segment. The proximal portion of this distal segment is anastomosed to the gastric remnant (alimentary limb). The distal portion of the proximal segment (biliopancreatic limb) is anastomosed to the distal part of the ileum 50 cm from the ileocecal valve. Consequently, digestion and absorption of macronutrients and micronutrients are largely limited to this 50-cm “common channel” where biliopancreatic enzymes have the opportunity to mix with food delivered by the alimentary limb. In a variant of this procedure with no gastric restriction, weight loss is less, but the lipid status and glycemic control are improved in patients with dyslipidemia and diabetes, respectively (80 [EL 3]). Postoperative weight loss is principally due to caloric and fat malabsorption after *BPD* (81 [EL 3]). The procedure may be associated with protein-calorie malabsorption, which necessitates surgical lengthening of the common channel. The steatorrhea after *BPD* may produce foul-smelling flatus and stools. The *BPD* may be associated with a variety of nutrient deficiencies and metabolic derangements, such as iron deficiency anemia, deficiencies in the fat-soluble vitamins (A, D, E, and K), and metabolic bone disease.

4.4. Biliopancreatic Diversion With Duodenal Switch

The *BPD* was modified by Hess and Hess (82 [EL 3]) with a vertical, subtotal, pylorus-preserving gastrectomy (parietal or sleeve gastrectomy) that channels orally ingested nutrients through a type of duodenal switch (*BPD/DS*), as described originally for bile reflux gastritis (83 [EL 3]). An additional modification of the original *BPD* procedure was to increase the length of the common channel from 50 cm to 100 cm, leaving the nutrient limb at 250 cm (84 [EL 3]). Recently, banded *BPD/DS* procedures have been developed, resulting in weight loss comparable to that for patients with *RYGB* (85 [EL 3]). Laparoscopic *BPD/DS* was first performed in 1999 by Ren et al (86 [EL 3]). Morbidity and mortality were increased in patients with a preoperative BMI >65 kg/m² (86 [EL 3]). Patients with *BPD/DS* have also been found to have various postoperative nutritional and metabolic complications.

4.5. Staged Bariatric Surgical Procedures

Although the aforementioned surgical procedures represent the generally accepted weight loss operations, some surgeons have used other surgical variations in high-risk patients. Staging the bariatric procedure has been suggested for patients at high risk for complications. In the first stage, a restrictive procedure such as a sleeve (longitudinal) gastrectomy is performed, which can be associated with a 33% to 45% loss of excess body weight (EBW) at 1 year (87-92 [EL 3]). Then, after a 6- to 12-month period

to allow significant weight loss and improvement in comorbidities, a more definitive procedure (RYGB or BPD/DS) is performed. In a prospective study of 126 patients undergoing sleeve gastrectomy with a mean BMI of >65 kg/m² and a median of 10 comorbid conditions per patient, Cottam et al (92 [EL 3]) achieved a mean 46% loss of EBW, in conjunction with no deaths and an 8% rate of major complications.

In cases in which there is increased volume of the left lobe of the liver, common in patients with a BMI >60 kg/m², the poor visualization of the gastroesophageal junction and angle of His makes construction of the sleeve gastrectomy difficult (93 [EL 4]). Thus, in another type of staged procedure, the surgeon performs an initial modified RYGB with a low gastrojejunal anastomosis and larger gastric pouch, and then 6 to 12 months later, a completion sleeve gastrectomy and revision of the gastrojejunostomy are performed. This approach is investigational.

In several recent studies, the sleeve gastrectomy was performed as a stand-alone procedure (87-92 [EL 3]). One randomized, prospective trial has shown better weight loss after sleeve gastrectomy in comparison with the LAGB at 3 years (94 [EL 2]) and with the intragastric balloon at 6 months (95 [EL 3]); however, strong and confirmatory long-term data are lacking. Overall, there are several EL 3 publications supporting a role for staged bariatric procedures involving an initial sleeve gastrectomy; nevertheless, these operations remain investigational at the current time.

5. MORTALITY FROM BARIATRIC PROCEDURES

Considerable concern has been raised regarding the mortality associated with bariatric surgical procedures. One study using statewide outcome data for bariatric surgery found a 1.9% risk of death in the state of Washington; however, procedures performed by more experienced surgeons were associated with a much lower risk of death (40 [EL 3]). Another study that used similar methods found that the risk of intraoperative death was 0.18% and the 30-day mortality was 0.33% for gastric bypass surgery in the state of California (96 [EL 3]). In a national inpatient sample for bariatric surgical procedures, Santry et al (8 [EL 4]) noted a 0.1% to 0.2% inpatient mortality nationwide. The Surgical Review Corporation noted a 0.14% in-hospital mortality, a 0.29% 30-day mortality, and a 0.35% 90-day mortality on the basis of 55,567 bariatric surgery patients (97 [EL 4]). The Agency for Healthcare Research and Quality identified a 0.19% in-hospital mortality for all bariatric discharges in the United States for 2004 (98 [EL 3]). In a meta-analysis, the operative mortality rates were 0.1% for LAGB, 0.5% for RYGB, and 1.1% for other malabsorptive procedures (99 [EL 1]). In a multi-institutional consecutive cohort study involving US academic medical centers by Nguyen et al

(100 [EL 3]), the following conclusions were offered as benchmark figures:

- For restrictive procedures (N = 94), 92% were performed laparoscopically with no conversions, an overall complication rate of 3.2%, a 30-day readmission rate of 4.3%, and a 30-day mortality rate of 0%.
- For gastric bypass procedures (N = 1,049), 76% were performed laparoscopically with a conversion rate of 2.2%, an overall complication rate of 16%, an anastomotic leak rate of 1.6%, a 30-day readmission rate of 6.6%, and a 30-day mortality rate of 0.4%.

DeMaria et al (101 [EL 4]) proposed an “obesity surgery mortality risk score” for RYGB based on BMI, male sex, hypertension, risk of pulmonary embolus (PE), and patient age. The mortality in low-risk patients (class A) was 0.31%, in intermediate-risk patients (class B) was 1.9%, and in high-risk patients (class C) was 7.56% (101 [EL 4]). Thus, it appears that bariatric surgery is not uniformly a “low-risk” procedure, and judicious patient selection and diligent perioperative care are imperative.

To define contemporary morbidity and mortality outcomes better, the NIH recently initiated the 3-year multicenter prospective Longitudinal Assessment of Bariatric Surgery study (102 [EL 4]). This observational study will assess the safety and clinical response of bariatric surgery by using standardized techniques and measurements.

6. BENEFITS OF BARIATRIC SURGERY

The purpose of bariatric surgery is to induce substantial, clinically important weight loss that is sufficient to reduce obesity-related medical complications to acceptable levels (103-107 [EL 3]) (Table 2). The loss of fat mass, particularly visceral fat, is associated with improved insulin sensitivity and glucose disposal, reduced flux of free fatty acids, increased adiponectin levels, and decreased interleukin-6, tumor necrosis factor- α , and highly sensitive C-reactive protein levels. Loss of visceral fat also reduces intra-abdominal pressure, and this change may result in improvements in urinary incontinence, gastroesophageal reflux, systemic hypertension, pseudotumor cerebri, venous stasis disease, and hypoventilation (108-114 [EL 2-4]). Foregut bypass leads to improvement in the physiologic responses of gut hormones involved in glucose regulation and appetite control, including ghrelin, glucagon-like peptide-1 (GLP-1), and peptide YY₃₋₃₆ (115 [EL 4], 116 [EL 4]). Mechanical improvements include less weight bearing on joints, enhanced lung compliance, and decreased fatty tissue around the neck, which relieves obstruction to breathing and sleep apnea.

Fluid and hemodynamic changes that lower the blood pressure after bariatric surgery include diuresis, natriuresis, and decreases in total body water, blood volume, and indices of sympathetic activity. Other clinical benefits

Table 2
Effects of Bariatric Surgery on Obesity-Related Comorbidities^a

Comorbidity	Preoperative incidence (%)	Remission >2 years postoperatively (%)	Reference
T2DM, IFG, or IGT	34	85	103
Hypertension	26	66	104
Hypertriglyceridemia and low HDL cholesterol	40	85	105
Sleep apnea	22 (in men) 1 (in women)	40	106
Obesity-hypoventilation syndrome	12	76	107

^a HDL = high-density lipoprotein; IFG = impaired fasting glucose; IGT = impaired glucose tolerance; T2DM = type 2 diabetes mellitus.
 Adapted from Greenway (4).

include improvements in T2DM, obesity-related cardiomyopathy, cardiac function, lipid profile, respiratory function, disordered sleep, degenerative joint disease, obesity-related infections, mobility, venous stasis, nonalcoholic fatty liver disease (NAFLD), asthma, polycystic ovary syndrome (PCOS), infertility, and complications of pregnancy (44 [EL 3]). Most bariatric surgery patients also experience considerable improvements in psychosocial status and quality of life postoperatively (48 [EL 4], 117-119 [EL 4]).

In an extensive meta-analysis of 22,000 bariatric surgery patients, Buchwald et al (99 [EL 1]) found that an average EBW loss of 61% was accompanied by improvements in T2DM, hypertension, sleep apnea, and dyslipidemia. In another meta-analysis, Maggard et al (120 [EL 1]) found that bariatric surgery resulted in a weight loss of 20 to 30 kg maintained up to 10 years in association with reduction of comorbidities and an overall mortality rate <1%. These benefits were conclusive for those patients with a BMI ≥ 40 kg/m² but not <40 kg/m². The nonrandomized, prospective, controlled SOS Study involved obese subjects who underwent gastric surgical procedures (mostly gastroplasties and nonadjustable bands) and contemporaneously matched, obese control subjects treated conventionally (64 [EL 3], 65 [EL 3]). Two- and 10-year improvement rates in T2DM, hypertriglyceridemia, low levels of high-density lipoprotein (HDL) cholesterol, and hyperuricemia were more favorable in the surgically treated group than in the control group. Recovery from hypercholesterolemia and hypertension did not differ between the groups at 10 years (64 [EL 3]). In contrast, at 8 years the 6% of patients who underwent RYGB had a significant decrease in both the systolic and the diastolic blood pressure (121 [EL 2]).

The beneficial effect of bariatric surgery on T2DM is one of the most important outcomes observed. Control rates for most procedures currently performed vary from 40% to 100%. Gastric bypass and malabsorptive procedures offer the highest rates of remission of T2DM (Table 3) (14 [EL 3], 84 [EL 3], 122-129 [EL 3-4]). A shorter duration of T2DM and greater weight loss are independent predictors of T2DM remission (130 [EL 3]). Improvements in fasting blood glucose levels occur before significant weight loss (131-135 [EL 3]). Insulin-treated patients experience substantial decreases in insulin requirements, with the majority of patients with T2DM able to discontinue insulin therapy by 6 weeks after bariatric surgery (136 [EL 3]). Euglycemia has been maintained up to 14 years after RYGB, a superior outcome when compared with solely gastric restrictive procedures (103 [EL 3], 137-139 [EL 3]). BPD and BPD/DS may be even more effective at improvement of the metabolic abnormalities of T2DM, leading to discontinuation of glucose-lowering therapy in most patients (84 [EL 3], 125 [EL 3], 140 [EL 4]). The LAGB procedure has also been shown to improve T2DM, albeit at a slower rate (64% to 71% remission rates within the first year) than RYGB, BPD, or BPD/DS (141-143 [EL 2]).

Prevention of the development of T2DM has also been reported with bariatric surgery. Significantly, in a longitudinal observational study of a nonrandomized cohort (144 [EL 2], 145 [EL 2]) and a randomized controlled study (63 [EL 2]), LAGB was associated with decreased insulin resistance and a dramatic reduction in fulfillment of the criteria for the metabolic syndrome. Long et al (146 [EL 2]) reported a 30-fold decrease in the risk for T2DM among patients with preexisting hyperglycemia who underwent RYGB. In a prospective, con-

Table 3
Rates for Remission of Type 2 Diabetes Mellitus Reported After Bariatric Surgery

Procedure	Remission rate (%)
Vertical banded gastroplasty	75-83
Laparoscopic adjustable silicone gastric banding	40-47
Roux-en-Y gastric bypass	83-92
Biliopancreatic diversion	95-100

Data from Greenway (4).

trolled study of 18 nondiabetic patients with a mean BMI of 54 ± 9 kg/m² undergoing RYGB, insulin sensitivity improved by 5 months, with continued improvement through 16 months postoperatively, although still not achieving normal levels (147 [EL 3]). The prevalence of the metabolic syndrome also decreases after RYGB (148 [EL 3]). In contrast, after BPD, insulin sensitivity normalized by 6 months and reached supranormal values by 24 months despite a BMI still exceeding 30 kg/m² (147 [EL 3]). Moreover, in patients with BPD, glucose-induced thermogenesis and insulin-glucose metabolism are normalized postoperatively (149 [EL 3]). Furthermore, in a retrospective review of 312 patients after BPD, all the major components of the metabolic syndrome were found to be reversed throughout a 10-year follow-up period: hyperglycemia decreased from 100% to 3%, hypertriglyceridemia declined from 38% to 1%, hypercholesterolemia diminished from 63% to 0%, and arterial hypertension abated from 86% to 26% (150 [EL 3]).

Eight studies have documented a decreased mortality in patients who have undergone bariatric surgery when compared with those who have not. Two of these studies were comparisons with patients who were evaluated for bariatric surgery but for some reason (for example, lack of insurance coverage or patient decision) did not undergo a surgical procedure (151 [EL 3], 152 [EL 3]). One study in the state of Washington found a decreased mortality among patients who underwent bariatric surgery in comparison with morbidly obese patients who had not, excluding the high operative mortality in that state (39 [EL 3]). Five studies were comparisons with a matched medical cohort (65 [EL 3], 141 [EL 2], 153 [EL 3], 154 [EL 2], 155 [EL 3]). The reduced mortality was due to decreases in occurrence of myocardial infarction (MI), diabetes, and cancer-related deaths (65 [EL 3], 153 [EL 3], 154 [EL 2]). Adams et al (154 [EL 2]) also found, however, that bariatric surgery was associated with a 58% increased rate of death not caused by disease, such as accidents and suicide (11.1 versus 6.4 per 10,000 person-years).

Weight loss after malabsorptive bariatric surgery reaches a nadir about 12 to 18 months postoperatively, with an approximate 10% regain of weight during the next decade (64 [EL 3], 65 [EL 3]) (Table 4). Weight loss is more gradual for the restrictive LAGB procedure but may continue for several years (63 [EL 2]). In purely restrictive procedures, failure to experience optimal weight loss has been associated with consumption of calorically dense liquids that can pass through the stoma without producing satiety (70 [EL 2], 187 [EL 2]), although this finding has not been confirmed in other studies (217 [EL 3], 218 [EL 3]).

7. METHODS FOR DEVELOPMENT OF AACE-TOS-ASMBS CPG

In 2004, the AACE Protocol for Standardized Production of Clinical Practice Guidelines was published in *Endocrine Practice* (219 [EL 4]). These CPG for perioperative nonsurgical management of the bariatric surgery patient are in strict accordance with the AACE Task Force CPG protocols and have been approved by TOS and ASMBS. Important production attributes unique to these CPG are described in the subsequent material.

7.1. Mandate, Review Process, Objectives, and Target Audience

AACE, TOS, and ASMBS task forces were assembled concurrently to produce these CPG, as mandated by their respective Board of Directors. Cochairmen and primary writing teams were assigned, and their initial draft was then reviewed by additional AACE, TOS, and ASMBS members before further review by various AACE, TOS, and ASMBS committees. Finally, the cochairmen performed a review prior to publication. These CPG will expire in 2011 and will be updated by AACE, TOS, and ASMBS at a time determined by the societies. At present, implementation and evaluation of these CPG are at the discretion of AACE, TOS, and ASMBS Board of Directors.

The objectives of these CPG are to provide the following:

1. An overview of the important principles of bariatric surgery as context for interpretation of subsequent evidence-based recommendations
2. An evidence-based resource for the perioperative nonsurgical management, especially nutritional and metabolic support, of the bariatric surgery patient
3. Specific recommendations regarding the selection of appropriate patients for bariatric surgery
4. Specific recommendations regarding the preoperative evaluation for the bariatric surgical patient
5. Specific recommendations regarding postoperative nonsurgical management of the bariatric surgery patient

Table 4
Reported Weight Loss as Percentage
of Excess Body Weight After Bariatric Surgery^a

Procedure	Follow-up period (y)		
	1-2	3-6	7-10
Vertical banded gastroplasty ^b	50-72	25-65	...
Gastric banding ^c	29-87	45-72	14-60
Sleeve gastrectomy ^d	33-58	66	...
Roux-en-Y gastric bypass ^e	48-85	53-77	25-68
Banded Roux-en-Y gastric bypass ^f	73-80	66-78	60-70
Long-limb Roux-en-Y gastric bypass ^g	53-74	55-74	...
Biliopancreatic diversion ± DS ^h	65-83	62-81	60-80

^a DS = duodenal switch.
^b References 156-160.
^c References 11, 55, 65, 94, 160-186.
^d References 87, 88, 90-92, 94, 95.
^e References 11, 70, 73, 165, 187-205.
^f References 73-75.
^g References 72, 199, 201, 206.
^h References 125, 194, 207-216.

6. Specific recommendations regarding the recognition and management of postoperative complications
7. Specific recommendations regarding selection of patients for a second (staged) bariatric surgical procedure or a revision or reversal of a previous bariatric surgical procedure

The target audiences for these CPG are as follows:

1. Endocrinologists
2. Specialists in metabolic and gastrointestinal disorders, obesity, clinical nutrition, nutrition support, or other disciplines that manage obese patients
3. General internists, primary care physicians, and physician-extenders who treat obese patients
4. Surgeons who encounter patients considering bariatric surgery or who have already had a bariatric surgical procedure

7.2. Guidelines for CPG

Current guidelines for CPG in clinical medicine emphasize an evidence-based approach rather than simply expert opinion (219 [EL 4], 220 [EL 4]). Even though a purely evidence-based approach lacks applicability to all actual clinical scenarios, its incorporation in these CPG provides objectivity.

7.3. Transparency: Levels of Scientific Substantiation and Recommendation Grades

All clinical data that are incorporated in these CPG have been evaluated in terms of levels of scientific substantiation (**evidence levels [EL]**; **Table 5**). This evidence rating system has one minor modification in comparison with the original AACE protocol (219 [EL 4]) in that level 2 (**[EL 2]**) prospective studies may be randomized or non-randomized to allow for well-designed cohort studies. This modification was incorporated because it is difficult to perform well-controlled, randomized clinical trials in surgery, unlike what physicians have been accustomed to in pharmaceutical trials. Another point worth mentioning is that when consensus statements are cited, even if based on a synthesis of evidence as in a published “evidence-based report,” then an evidence level 4 [EL 4] has been assigned. Every clinical reference was assigned an evidence rating, which has then been inserted in brackets at the end of the citation in both the text and the reference sections. The “best evidence” rating level [BEL] corresponds to the best conclusive evidence found. The BEL accompanies the recommendation **Grade** in the **Executive Summary** and maps to the text in the **Appendix** section, where transparency is paramount. In the **Executive Summary**, BEL 2 ratings have been designated as “randomized,” “nonrandomized,” or both for additional trans-

parency. Final recommendation **Grades** (Table 6) incorporate **EL** ratings, and in situations in which there was no clinical evidence, various subjective factors were considered: physician preferences, costs, risks, and regional availability of specific technologies and expertise. Hence, recommendation grades are generally based on strong **BEL (Grade A; BEL 1)**, intermediate **BEL (Grade B; BEL 2)**, weak **BEL (Grade C; BEL 3)**, or subjective factors when there is no clinical evidence, inconclusive clinical evidence, or contradictory clinical evidence (**Grade D; BEL 4**). All recommendations resulted from a consensus among the AAACE, TOS, and ASMBS primary writers and influenced by input from reviewers. If subjective factors take priority over the **BEL** on the basis of the expert opinion of the task force members, then this is described explicitly. Thus, some recommendations may be “upgraded” or “downgraded” according to explicitly stated subjective factors. Furthermore, the correctness of the recommendation **Grades** and **EL** was subject to review at several levels. Also, recommendation **Grades** were assigned only if a specific action is recommended. The action may be ordering a particular diagnostic test, using a particular drug, performing a particular procedure, or adhering to a particular algorithm.

Shortcomings of this evidence-based methodology in these CPG are (1) relative paucity of strong (level 1 and 2) scientific data, leaving the majority of recommendations based on weaker, extant **EL 3** data and **EL 4** consensus opinion; (2) subjectivity on the part of the primary writers when weighing positive and negative, or epidemiologic versus experimental, data to arrive at an evidence-based recommendation grade or consensus opinion; (3) subjectivity on the part of the primary writers when weighing subjective attributes, such as cost-effectiveness and risk-to-benefit ratios, to arrive at an evidence-based recommendation **Grade** or consensus opinion; (4) potentially incomplete review of the literature by the primary writers despite extensive diligence; and (5) bias in the available publications, which originate predominantly from experienced bariatric surgeons and surgery centers and may therefore not reflect the experience at large. These shortcomings have been addressed by the primary writers through an a priori methodology and multiple levels of review by a large number of experts from the 3 participating societies.

8. EXECUTIVE SUMMARY OF RECOMMENDATIONS

The following recommendations (labeled “**R**”) are evidence-based (Grades A, B, and C) or based on expert opinion because of a lack of conclusive clinical evidence (Grade D). The “best evidence” rating level (**BEL**), which corresponds to the best conclusive evidence found, accompanies the recommendation grade in this Executive Summary. Details regarding the mapping of clinical evidence ratings to these recommendation grades are provided

in the Appendix (Section 9, “Discussion of the Clinical Evidence”).

8.1. Which Patients Should Be Offered Bariatric Surgery?

The selection criteria and exclusion factors for bariatric surgery are outlined in Table 7.

- **R1.** Patients with a BMI ≥ 40 kg/m² for whom bariatric surgery would not be associated with excessive risk should be eligible for one of the procedures (**Grade A; BEL 1**).
- **R2.** Patients with a BMI ≥ 35 kg/m² and one or more severe comorbidities, including coronary artery disease (CAD), T2DM, obstructive sleep apnea (OSA), obesity-hypoventilation syndrome (OHS), pickwickian syndrome (a combination of OSA and OHS), NAFLD or nonalcoholic steatohepatitis, hypertension, dyslipidemia, pseudotumor cerebri, gastroesophageal reflux disease (GERD), asthma, venous stasis disease, severe urinary incontinence, debilitating arthritis, or considerably impaired quality of life, may also be offered a bariatric procedure if the surgical risks are not excessive (**Grade A; BEL 1**).
- **R3.** Currently, insufficient data are available to recommend bariatric surgery for patients with a BMI < 35 kg/m² (**Grade D**).
- **R4.** There is insufficient evidence for recommending bariatric surgery specifically for glycemic control independent of BMI criteria (**Grade D**).

8.2. Which Bariatric Surgical Procedure Should Be Offered?

- **R5.** The best choice for any bariatric procedure (type of procedure and type of approach) depends on the available local-regional expertise (surgeon and institution), patient preferences, risk stratification, and other idiosyncratic factors, with which the referring physician (or physicians) must become familiar (**Grade D**). At this time, there is insufficient conclusive evidence to recommend specific bariatric surgical procedures for the general severely obese population (**Grade D**). Specialists in bariatric medicine, however, must also familiarize themselves with the outcome data among the various bariatric surgical procedures (**Grade D**). Physicians should exercise caution when recommending BPD, BPD/DS, or related procedures because of greater associated risks reported in the literature (**Grade C; BEL 3**).
- **R6.** Although risks and benefits are associated with both approaches, laparoscopic bariatric procedures are preferred over open bariatric procedures if sufficient surgical expertise is available (**Grade B; BEL 2 [randomized and nonrandomized]**).

Table 5
Levels of Scientific Substantiation in Evidence-Based Medicine^a

Level	Description	Comments
1	Prospective, randomized, controlled trials—large	Data are derived from a substantial number of trials, with adequate statistical power involving a substantial number of outcome data subjects Large meta-analyses using raw or pooled data or incorporating quality ratings Well-controlled trial at one or more centers Consistent pattern of findings in the population for which the recommendation is made (generalizable data) Compelling nonexperimental, clinically obvious, evidence (for example, use of insulin in diabetic ketoacidosis); “all-or-none” indication
2	Prospective controlled trials with or without randomization—limited body of outcome data	Limited number of trials, small population sites in trials Well-conducted single-arm prospective cohort study Limited but well-conducted meta-analyses Inconsistent findings or results not representative for the target population Well-conducted case-controlled study
3	Other experimental outcome data and nonexperimental data	Nonrandomized, controlled trials Uncontrolled or poorly controlled trials Any randomized clinical trial with 1 or more major or 3 or more minor methodologic flaws Retrospective or observational data Case reports or case series Conflicting data with weight of evidence unable to support a final recommendation
4	Expert opinion	Inadequate data for inclusion in level 1, 2, or 3; necessitates an expert panel’s synthesis of the literature and a consensus Experience-based Theory-driven

^a Levels 1, 2, and 3 represent a given level of scientific substantiation or proof. Level 4 or Grade D represents unproven claims. It is the “best evidence” based on the individual ratings of clinical reports that contributes to a final grade recommendation (Table 6).

- **R7.** A first-stage sleeve gastrectomy may be performed in high-risk patients to induce an initial weight loss (25 to 45 kg), with the possibility of then performing a second-stage RYGB or BPD/DS after the patient’s operative risk has improved. This is currently an investigational procedure (**Grade C; BEL 3**).
- **R8.** All patients should undergo evaluation for causes and complications of obesity, with special attention directed to those factors that could affect a recommendation for bariatric surgery (Table 8) (**Grade A; BEL 1**).
- **R9.** The preoperative evaluation must include a comprehensive medical history, physical examination, and appropriate laboratory testing (**Grade A; BEL 1**).

8.3. How Should Potential Candidates for Bariatric Surgery Be Managed Preoperatively?

Table 6
Grade-Recommendation Protocol Adopted by
the American Association of Clinical Endocrinologists,
The Obesity Society, and American Society for Metabolic & Bariatric Surgery^a

Grade	Description	Recommendation
A	≥1 conclusive level 1 publications demonstrating benefit >> risk	Action recommended for indications reflected by the published reports Action based on strong evidence Action can be used with other conventional therapy or as “first-line” therapy
B	No conclusive level 1 publication ≥1 conclusive level 2 publications demonstrating benefit >> risk	Action recommended for indications reflected by the published reports <i>If</i> the patient refuses or fails to respond to conventional therapy; must monitor for adverse effects, if any Action based on intermediate evidence Can be recommended as “second-line” therapy
C	No conclusive level 1 or 2 publication ≥1 conclusive level 3 publications demonstrating benefit >> risk <i>or</i> No risk at all and no benefit at all	Action recommended for indications reflected by the published reports <i>If</i> the patient refuses or fails to respond to conventional therapy, provided there are no significant adverse effects; “no objection” to recommending their use <i>or</i> “No objection” to continuing their use Action based on weak evidence
D	No conclusive level 1, 2, or 3 publication demonstrating benefit >> risk Conclusive level 1, 2, or 3 publications demonstrating risk >> benefit	Not recommended Patient is advised to discontinue use Action not based on any evidence

^a The final recommendation grades were determined by the primary writers by consensus on the basis of (1) “best evidence” ratings (see Table 5) and (2) subjective factors (see Methods Section 7.3 on Transparency).

- **R10.** The medical necessity for bariatric surgery should be documented (**Grade D**).
- **R11.** There should be a thorough discussion with the patient regarding the risks and benefits, procedural options, and choices of surgeon and medical institution (**Grade D**).
- **R12.** Patients should be provided with educational materials and access to preoperative educational sessions at prospective bariatric surgery centers (**Grade D**).
- **R13.** Financial counseling should be provided, and the physician should be able to provide all necessary clinical material for documentation so that third-party payer criteria for reimbursement are met (**Grade D**).
- **R14.** Preoperative weight loss should be considered in patients in whom reduction of liver volume can improve the technical aspects of surgery (**Grade B; BEL 2 [nonrandomized]**).

8.4. System-Oriented Approach to Medical Clearance for Bariatric Surgery

8.4.1. Endocrine

8.4.1.1. Diabetes

- **R15.** Preoperative glycemic control should be optimized with use of medical nutrition therapy and physical activity; orally administered agents and insulin should be introduced as needed (**Grade D**).

Table 7
Selection Criteria for Bariatric Surgery^a

Factor	Criteria
Weight (adults)	BMI \geq 40 kg/m ² with no comorbidities BMI \geq 35 kg/m ² with obesity-associated comorbidity
Weight loss history	Failure of previous nonsurgical attempts at weight reduction, including nonprofessional programs (for example, Weight Watchers, Inc)
Commitment	Expectation that patient will adhere to postoperative care Follow-up visits with physician(s) and team members Recommended medical management, including the use of dietary supplements Instructions regarding any recommended procedures or tests
Exclusion	Reversible endocrine or other disorders that can cause obesity Current drug or alcohol abuse Uncontrolled, severe psychiatric illness Lack of comprehension of risks, benefits, expected outcomes, alternatives, and lifestyle changes required with bariatric surgery

^a BMI = body mass index.

- **R16.** Reasonable targets for preoperative glycemic control should be a hemoglobin A1c value of 7.0% or less, a fasting blood glucose level of 110 mg/dL or less, and a 2-hour postprandial blood glucose concentration of 140 mg/dL or less (see <http://www.aace.com/pub/pdf/guidelines/DMGuidelines2007.pdf>), but these variables are based on evidence related to long-term outcome and may not be applicable in this setting (**Grade D**).
 - **R17.** A protocol for perioperative glycemic control should be reviewed *before* the patient undergoes bariatric surgery (**Grade D**).
- 8.4.1.2. Thyroid**
- **R18.** Routine screening recommendations for hypothyroidism are conflicting. When thyroid disease is suspected, a sensitive serum thyroid-stimulating hormone level should be ordered (**Grade D**).
 - **R19.** In patients found to have thyroid dysfunction, treatment should be initiated before bariatric surgery (**Grade D**).
- 8.4.1.3. Lipids**
- **R20.** A fasting lipid panel should be obtained in all patients with obesity (**Grade A; BEL 1**).
 - **R21.** Treatment should be initiated according to the National Cholesterol Education Program Adult Treatment Panel III guidelines (see <http://www.nhlbi.nih.gov/guidelines/cholesterol/>) (**Grade D**).
- 8.4.1.4. Polycystic ovary syndrome and fertility**
- **R22.** Candidates for bariatric surgery should minimize the risk of pregnancy for at least 12 months perioperatively (**Grade C; BEL 3**).
 - **R23.** All women of reproductive age should be counseled on contraceptive choices (**Grade D**).
 - **R24.** Women with a LAGB should be closely monitored during pregnancy because band adjustment may be necessary (**Grade B; BEL 2 [nonrandomized]**).
 - **R25.** Estrogen therapy should be discontinued before bariatric surgery (1 cycle of oral contraceptives in premenopausal women; 3 weeks of hormone replacement therapy in postmenopausal women) to reduce the risks for postoperative thromboembolic phenomena (**Grade D**).
 - **R26.** Women with PCOS should be advised that their fertility status may be improved postoperatively (**Grade D**).

Table 8
Metabolic Complications of Bariatric Surgery^a

Complication	Clinical features	Management
Acid-base disorder	Metabolic acidosis, ketosis	Bicarbonate orally or intravenously; adjust acetate content in PN
	Metabolic alkalosis	Salt and volume loading (enteral or parenteral)
Bacterial overgrowth (primarily with BPD, BPD/DS)	Abdominal distention Pseudo-obstruction Nocturnal diarrhea Proctitis Acute arthralgia	Antibiotics (metronidazole) Probiotics
Electrolyte abnormalities (primarily with BPD, BPD/DS)	Low Ca, K, Mg, Na, P Arrhythmia, myopathy	Enteral or parenteral repletion
Fat-soluble vitamin deficiency	Vitamin A—night vision Vitamin D—osteomalacia Vitamin E—rash, neurologic Vitamin K—coagulopathy	Vitamin A, 5,000-10,000 U/d Vitamin D, 400-50,000 U/d Vitamin E, 400 U/d Vitamin K, 1 mg/d ADEK, 2 tablets twice a day (http://www.scandipharm.com)
Folic acid deficiency	Hyperhomocysteinemia Anemia Fetal neural tube defects	Folic acid supplementation
Iron deficiency	Anemia	Ferrous fumarate, sulfate, or gluconate Up to 150-300 mg elemental iron daily Add vitamin C and folic acid
Osteoporosis	Fractures	DXA, calcium, vitamin D, and consider bisphosphonates
Oxalosis	Kidney stones	Low oxalate diet Potassium citrate Probiotics
Secondary hyperparathyroidism	Vitamin D deficiency Negative calcium balance Osteoporosis	DXA Serum intact PTH level 25-Hydroxyvitamin D levels Calcium and vitamin D supplements
Thiamine deficiency (vitamin B ₁)	Wernicke-Korsakoff encephalopathy Peripheral neuropathy Beriberi	Thiamine intravenously followed by large-dose thiamine orally
Vitamin B ₁₂ deficiency	Anemia Neuropathy	Parenteral vitamin B ₁₂ Methylmalonic acid level

^a BPD = biliopancreatic diversion; BPD/DS = biliopancreatic diversion with duodenal switch; DXA = dual-energy x-ray absorptiometry; PN = parenteral nutrition; PTH = parathyroid hormone.

8.4.1.5. Exclusion of endocrine causes of obesity

- **R27.** Routine laboratory testing to screen for rare causes of obesity (for example, Cushing syndrome, hypothalamic obesity syndromes, melanocortin-4 mutations, and leptin deficiency obesity) is not cost-effective and not recommended (**Grade D**).
- **R28.** Case-by-case decisions to screen for rare causes of obesity should be based on specific historical and physical findings (**Grade D**).

8.4.2. Cardiology and Hypertension

- **R29.** Noninvasive testing beyond an electrocardiogram is determined on the basis of the individual risk factors and findings on history and physical examination (**Grade D**).
- **R30.** Patients with known cardiac disease should have a formal cardiology consultation before bariatric surgery (**Grade D**).
- **R31.** Patients at risk for heart disease should undergo evaluation for perioperative β -adrenergic blockade (**Grade A; BEL 1**).

8.4.3. Pulmonary and Sleep Apnea

- **R32.** All patients considered for bariatric surgery should have a chest radiograph preoperatively (**Grade D**).
- **R33.** Patients with intrinsic lung disease or disordered sleep patterns should have a formal pulmonary evaluation, including arterial blood gas measurement and polysomnography, when knowledge of the results would alter patient care (**Grade D**).
- **R34.** Patients should stop smoking at least 8 weeks before bariatric surgery and should plan to quit smoking or to participate in a smoking cessation program postoperatively (**Grade C; BEL 3**).

8.4.4. Venous Disease

- **R35.** Patients at risk for, or with a history of, deep venous thrombosis (DVT) or cor pulmonale should undergo an appropriate diagnostic evaluation for DVT (**Grade D**).
- **R36.** A prophylactic vena caval filter should be considered for patients with a history of prior PE, prior iliofemoral DVT, evidence of venostasis, known hypercoagulable state, or increased right-sided heart pressures (**Grade C; BEL 3**).

8.4.5. Gastrointestinal

- **R37.** All gastrointestinal symptoms should be evaluated and treated before bariatric surgery (**Grade D**).

- **R38.** All patients considered for bariatric surgery who have increased liver function test results (2 to 3 times the upper limit of normal) should undergo abdominal ultrasonography and a viral hepatitis screen (**Grade D**).
- **R39.** There is inconsistent evidence to recommend routine screening for the presence of *Helicobacter pylori* before bariatric surgery (**Grade D**).

8.4.6. Rheumatologic and Metabolic Bone Disease

- **R40.** There are no evidence-based, routine preoperative tests required for evaluation of rheumatologic problems (**Grade D**).
- **R41.** There are insufficient data to warrant routine preoperative assessment of bone mineral density with dual-energy x-ray absorptiometry (**Grade D**).

8.4.7. Psychiatric

- **R42.** A psychosocial-behavioral evaluation, which assesses environmental, familial, and behavioral factors, should be considered for all patients before bariatric surgery (**Grade D**).
- **R43.** Any patient considered for bariatric surgery with a known or suspected psychiatric illness should undergo a formal mental health evaluation before performance of the surgical procedure (**Grade C; BEL 3**).
- **R44.** All patients should undergo evaluation of their ability to incorporate nutritional and behavioral changes before and after bariatric surgery (**Grade D**).

8.4.8. Nutritional

- **R45.** All patients should undergo an appropriate nutritional evaluation, including selective micronutrient measurements (see Tables 13 and 17), before any bariatric surgical procedure (**Grade C; BEL 3**). In comparison with purely restrictive procedures, more extensive perioperative nutritional evaluations are required for malabsorptive procedures.

8.5. Early Postoperative Care (<5 Days)

8.5.1. Nutrition

- **R46.** A clear liquid meal program can usually be initiated within 24 hours after any of the bariatric procedures, but this schedule should be discussed with the surgeon (**Grade C; BEL 3**).
- **R47.** A consultation should be arranged with a registered dietitian who is a member of the bariatric surgery team (**Grade D**).
- **R48.** A protocol-derived staged meal progression, based on the type of surgical procedure, should be provided to the patient. Sample protocols are shown in Tables 9, 10, and 11 (**Grade D**).

Table 9
Suggested Meal Progression After Roux-en-Y Gastric Bypass

Diet stage ^a	Begin	Fluids/food	Guidelines
Stage I	Postop days 1 and 2	Clear liquids Noncarbonated; no calories No sugar; no caffeine	On postop day 1, patients undergo a Gastrografin swallow test for leaks; once tested, begin sips of clear liquids
Stage II Begin supplementation: Chewable multivitamin with minerals, × 2/d Chewable or liquid calcium citrate with vitamin D	Postop day 3 (discharge diet)	Clear liquids • Variety of no-sugar liquids or artificially sweetened liquids • Encourage patients to have salty fluids at home • Solid liquids: sugar-free ice pops PLUS full liquids • ≤15 g of sugar per serving • Protein-rich liquids (limit 20 g protein per serving of added powders)	Patients should consume a minimum of 48-64 fluid ounces of total fluids per day: 24-32 ounces or more of clear liquids plus 24-32 ounces of any combination of full liquids: • Nonfat milk mixed with whey or soy protein powder (limit 20 g protein per serving) • Lactaid milk or soy milk mixed with soy protein powder • Light yogurt, blended • Plain nonfat yogurt; Greek yogurt
Stage III	Postop days 10-14 ^a	Increase clear liquids (total liquids 48-64+ ounces per day) and replace full liquids with soft, moist, diced, ground or pureed protein sources as tolerated Stage III, week 1: eggs, ground meats, poultry, soft, moist fish, added gravy, bouillon, light mayonnaise to moisten, cooked bean, hearty bean soups, cottage cheese, low-fat cheese, yogurt	Protein food choices are encouraged for 4-6 small meals per day; patients may be able to tolerate only a couple of tablespoons at each meal or snack. Chew foods thoroughly prior to swallowing (consistency of applesauce). Encourage patients not to drink with meals and to wait ~30 minutes after each meal before resuming fluids. Eat from small plates and advise using small utensil to help control portions
Stage III	4 weeks postop	Advance diet as tolerated; if protein foods, add well-cooked, soft vegetables and soft and/or peeled fruit. Always eat protein first	Adequate hydration is essential and a priority for all patients during the rapid weight-loss phase
Stage III	5 weeks postop	Continue to consume protein with some fruit or vegetable at each meal; some people tolerate salads at 1 month postop	AVOID rice, bread, and pasta until patient is comfortably consuming 60 g protein per day plus fruits and vegetables
Stage IV Vitamin and mineral supplementation daily. ^b May switch to pill form if <11 mm in width and length after 2 months postop	As hunger increases and more food is tolerated	Healthy solid food diet	Healthy, balanced diet consisting of adequate protein, fruits, vegetables, and whole grains. Eat from small plates and advise using small utensil to help control portions. Calorie needs based on height, weight, and age

^a There is no standardization of diet stages; there are a wide variety of nutrition therapy protocols for how long patients stay on each stage and what types of fluids and foods are recommended.

^b Nutritional laboratory studies should be monitored (see Table 13); bone density test at baseline and about every 2 years. Reprinted with permission from Susan Cummings, MS, RD. MGH Weight Center, Boston, Massachusetts.

Table 10
Suggested Meal Progression After Laparoscopic Adjustable Gastric Band Procedure

Diet stage ^a	Begin	Fluids/food	Guidelines
Stage I	Postop days 1 and 2	Clear liquids Noncarbonated; no calories No sugar; no caffeine	On postop day 1, patients may begin sips of water and Crystal Light; avoid carbonation
Stage II Begin supplementation: Chewable multivitamin with minerals, × 2/d Chewable or liquid calcium citrate with vitamin D	Postop days 2-3 (discharge diet)	Clear liquids • Variety of no-sugar liquids or artificially sweetened liquids PLUS full liquids • ≤15 g of sugar per serving • Protein-rich liquids (≤3 g fat per serving)	Patients should consume a minimum of 48-64 ounces of total fluids per day: 24-32 ounces or more of clear liquids plus 24-32 ounces of any combination of full liquids: • 1% or skim milk mixed with whey or soy protein powder (limit 20 g protein per serving) • Lactaid milk or soy milk mixed with soy protein powder • Light yogurt, blended • Plain yogurt
Stage III	Postop days 10-14 ^a	Increase clear liquids (total liquids 48-64 fl oz or more per day) and replace full liquids with soft, moist, diced, ground or pureed protein sources as tolerated Stage III, week 1: eggs, ground meats, poultry, soft, moist fish, added fat-free gravy, bouillon, light mayonnaise to moisten, cooked bean, hearty bean soups, low-fat cottage cheese, low-fat cheese, yogurt	NOTE: Patients should be reassured that hunger is common and normal postop. Protein food (moist, ground) choices are encouraged for 3-6 small meals per day, to help with satiety, since hunger is common within ~1 week postop. Mindful, slow eating is essential. Encourage patients not to drink with meals and to wait ~30 minutes after each meal before resuming fluids. Eat from small plates and advise using small utensil to help control portions
Stage III	4 weeks postop	Advance diet as tolerated; if protein foods tolerated in week 1, add well-cooked, soft vegetables and soft and/or peeled fruit	Adequate hydration is essential and a priority for all patients during the rapid weight-loss phase. Consume protein at every meal and snack, especially if increased hunger noted before initial fill or adjustment. Very well-cooked vegetables may also help to increase satiety
Stage III	5 weeks postop	Continue to consume protein with some fruit or vegetable at each meal; some people tolerate salads at 1 month postop	If patient is tolerating soft, moist, ground, diced, and/or pureed proteins with small amounts of fruits and vegetables, may add crackers (use with protein) AVOID rice, bread, and pasta
Stage IV Vitamin and mineral supplementation daily ^b	As hunger increases and more food is tolerated	Healthy solid food diet	Healthy, balanced diet consisting of adequate protein, fruits, vegetables, and whole grains. Eat from small plates and advise using small utensil to help control portions. Calorie needs based on height, weight, and age
Fill/adjustment	~6 weeks postop and possibly every 6 weeks until satiety reached	Full liquids × 2-3 days post-fill, then advance to Stage III, week 1 guidelines above, as tolerated for another 2-3 days, then advance to the final stage and continue	Same as Stage II liquids above × 48-72 hours (and/or as otherwise advised by surgeon). NOTE: When diet advanced to soft solids, special attention to mindful eating and chewing until in liquid form, since more restriction may increase risk for obstruction above stoma of band if food not thoroughly chewed (consistency of applesauce)

^a There is no standardization of diet stages; there are a wide variety of nutrition therapy protocols for how long patients stay on each stage and what types of fluids and foods are recommended.

^b Nutritional laboratory studies should be monitored (see Table 13); bone density test at baseline and about every 2 years.
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Table 11
Suggested Meal Progression After Biliopancreatic Diversion (± Duodenal Switch)

Diet stage ^a	Begin	Fluids/food	Guidelines
Stage I	Postop days 1 and 2	Clear liquids ^b Noncarbonated; no calories No sugar; no caffeine	Clear liquids started after swallow test
Stage II Begin supplementation: Chewable multivitamin with minerals, × 2/d Iron supplement • Add vitamin C for absorption if not already included within the supplement Chewable or liquid calcium citrate containing vitamin D, 2,000 mg/d Vitamin B₁₂ ; at least 350-500 µg crystalline daily; might need vitamin B ₁₂ intramuscularly Fat-soluble vitamins: A, D, E, K • High risk for fat-soluble vitamin deficiencies • A: 5,000-10,000 IU/d • D: 600-50,000 IU/d • E: 400 IU/d • K: 1 mg/d Advise ADEK tablets × 2/d	Postop day 3	Clear liquids • Variety of no-sugar liquids or artificially sweetened liquids • Encourage patients to have salty fluids at home • Solid liquids: sugar-free ice pops PLUS full liquids^b • ≤15 g of sugar per serving • Protein-rich liquids	Protein malnutrition is the most severe macronutrient complication after BPD/DS; regular monitoring and assessment of protein intake and status are very important ~90 g of protein a day is recommended; since early postop this is difficult for most patients, set goal to consume ≥60 g of protein per day plus clear liquids, and increase as tolerated. Patients should consume a minimum of 64 ounces of total fluids per day; 24-32 ounces or more of clear liquids plus 4-5 eight-ounce servings a day of any combination of full liquids— 1% or skim milk, Lactaid nonfat milk, or nonfat soy milk fortified with calcium mixed with: • Whey or soy protein powder (20-25 g protein per serving of protein powder) • Light yogurt, blended • Plain yogurt; Greek yogurt
Stage III	Postop days 10-14 ^a	Increase clear liquids (total liquids, 75+ ounces per day), and replace full liquids with soft, moist, diced, ground or pureed protein sources as tolerated Stage III, week 1: eggs, ground meats, poultry, soft, moist fish, added nonfat gravy, bouillon, light mayonnaise to moisten, cooked bean, hearty bean soups, low-fat cottage cheese, low-fat cheese, light yogurt	Protein food choices are encouraged for 3-6 small meals per day; patients may be able to tolerate only a couple of tablespoons at each meal or snack. Encourage patients not to drink with meals and to wait ≥30 minutes after each meal before resuming fluids. Patients might need to continue with supplementation of protein drinks to meet protein needs (90 g of protein daily is the goal)
Stage III	6 weeks postop	Advance diet as tolerated; add well-cooked, soft vegetables and soft and/or peeled fruit. Always eat protein first	Patients should be counseled to focus on protein at every meal and snack and to avoid starches or concentrated carbohydrates; 10-12 ounces of lean meats, poultry, fish, or eggs or some combination of high biologic value protein and protein supplement powders. Adequate hydration is essential and a priority for all patients during the rapid weight-loss phase. Wait ≥30 minutes after meals before resuming liquids
Stage III	12 weeks postop	Continue to consume protein with some fruit or vegetable at each meal; some people tolerate salads at 1 month postop; starches should be limited to whole grain crackers with protein, potato, and/or dry low-sugar cereals moistened with milk. Protein continues to be a high priority	AVOID rice, bread, and pasta until patient is comfortably consuming 90 g of protein per day plus fruits and vegetables

^a There is no standardization of diet stages; there are a wide variety of nutrition therapy protocols for how long patients stay on each stage and what types of fluids and foods are recommended.

^b Clear and full liquids for biliopancreatic diversion with duodenal switch (BPD/DS) are the same as for Roux-en-Y gastric bypass (see Table 9). Reprinted with permission from Susan Cummings, MS, RD. MGH Weight Center, Boston, Massachusetts.

- **R49.** Nutrition and meal planning guidance should be provided to the patient and family before bariatric surgery and during the postoperative hospital course and reinforced during future outpatient visits (**Grade D**).
- **R50.** Patients should adhere to a plan of multiple small meals each day, chewing their food thoroughly without drinking beverages at the same time (more than 30 minutes apart) (**Grade D**).
- **R51.** Patients should be advised to adhere to a balanced meal plan that consists of more than 5 servings of fruits and vegetables daily for optimal fiber consumption, colonic function, and phytochemical consumption (**Grade D**).
- **R52.** Protein intake should average 60 to 120 g daily (**Grade D**).
- **R53.** Concentrated sweets should be avoided after RYGB to minimize symptoms of the dumping syndrome or after any bariatric procedure to reduce caloric intake (**Grade D**).
- **R54.** Minimal nutritional supplementation includes 1 to 2 adult multivitamin-mineral supplements containing iron, 1,200 to 1,500 mg/d of calcium, and a vitamin B-complex preparation (**Grade B; BEL 2 [nonrandomized]**).
- **R55.** Fluids should be consumed slowly and in sufficient amounts to maintain adequate hydration (more than 1.5 L daily) (**Grade D**).
- **R56.** Parenteral nutrition (PN) should be considered in high-risk patients, such as critically ill patients unable to tolerate sufficient enteral nutrition for more than 5 to 7 days or noncritically ill patients unable to tolerate sufficient enteral nutrition for more than 7 to 10 days (**Grade D**).

8.5.2. Diabetes

- **R57.** In patients with T2DM, periodic fasting blood glucose concentrations should be determined. Preprandial and bedtime reflectance meter glucose (“finger-stick”) determinations in the home setting should be encouraged, depending on the patient’s ability to test and the level of glycemic control. Finger-stick glucose determinations should also be performed if symptoms of hypoglycemia occur (**Grade A; BEL 1**).
- **R58.** Use of all insulin secretagogue drugs (sulfonylureas and meglitinides) should be discontinued (**Grade D**).

- **R59.** In non-intensive care unit (ICU) hospitalized patients, a rapid-acting insulin analogue should be administered before meals and at bedtime to maintain maximal postprandial values below 180 mg/dL (**Grade D**).
- **R60.** In non-ICU hospitalized patients, fasting blood glucose levels should be maintained between 80 and 110 mg/dL with the use of a long-acting insulin analogue, such as insulin glargine (Lantus) or detemir (Levemir) (**Grade D**).
- **R61.** In the ICU, all blood glucose levels should be maintained ideally within the range of 80 to 110 mg/dL by using an intravenous insulin infusion (**Grade A; BEL 1**).

8.5.3. Cardiology

- **R62.** Patients with known or presumed CAD and high perioperative risk should be managed in an ICU setting for the first 24 to 48 hours postoperatively (**Grade D**).
- **R63.** Therapy with β -adrenergic blocking agents should be considered perioperatively for cardioprotection (**Grade D**).

8.5.4. Pulmonary

- **R64.** Appropriate pulmonary management includes aggressive pulmonary toilet and incentive spirometry, oxygen supplementation to avoid hypoxemia, and early institution of continuous positive airway pressure (CPAP) when clinically indicated (**Grade D**).
- **R65.** Prophylaxis against DVT is recommended for all patients (**Grade B; BEL 2 [randomized]**) and may be continued until patients are ambulatory (**Grade D**). Early ambulation is encouraged (**Grade C; BEL 3**).
- **R66.** Currently recommended prophylactic regimens include sequential compression devices (**Grade C; BEL 3**), as well as subcutaneously administered unfractionated heparin or low-molecular-weight heparin for 3 days before and after bariatric surgery (**Grade B; BEL 2 [randomized]**), and inferior vena cava filter placement in patients at high risk for mortality after PE or DVT (**Grade C; BEL 3**), with known pulmonary artery pressure exceeding 40 mm Hg (**Grade D**), or with known hypercoagulable states (**Grade C; BEL 3**).
- **R67.** Respiratory distress or failure to wean from ventilatory support should raise suspicion and prompt an evaluation for an acute postoperative complication, such as PE or anastomotic leak (**Grade D**).

8.5.5. Monitoring for Surgical Complications

- **R68.** In the clinically stable patient, meglumine diatrizoate (Gastrografin) upper gastrointestinal (UGI) studies or computed tomography (CT) may identify anastomotic leaks (**Grade C; BEL 3**).
- **R69.** Exploratory laparotomy is recommended in the setting of high clinical suspicion for anastomotic leaks despite a negative study (**Grade C; BEL 3**).
- **R70.** The presence of a new sustained pulse rate of more than 120 beats/min for longer than 4 hours should raise suspicion for an anastomotic leak (**Grade D**).
- **R71.** A routine Gastrografin UGI study may be considered to identify any subclinical leaks before discharge of the patient from the hospital (**Grade C; BEL 3**).

8.5.6. Fluid Management

- **R72.** The goals of fluid management during the early postoperative period after bariatric surgery are maintaining a urine output of more than 40 mL/h, avoiding volume overload, maintaining normal serum electrolyte levels, and limiting dextrose-containing solutions to avoid hyperglycemia (**Grade D**).
- **R73.** Postoperative urine output must be monitored, with a target of more than 30 mL/h or 240 mL per 8-hour shift (**Grade D**).

8.5.7. Preventing Rhabdomyolysis

- **R74.** Patients should have adequate padding at all pressure points during bariatric surgery (**Grade D**).
- **R75.** When rhabdomyolysis is suspected, creatine kinase (CK) levels should be determined (**Grade C; BEL 3**).

8.5.8. Anemia

- **R76.** The indications for transfusions of blood products after bariatric surgery are the same as for other surgical procedures (**Grade D**).
- **R77.** Persistence of anemia without evidence of blood loss should be evaluated in terms of nutritional deficiencies during the late postoperative period (**Grade D**).

8.6. Late Postoperative Management (≥5 Days)**8.6.1. Follow-up**

- **R78.** The frequency of follow-up depends on the bariatric procedure performed and the severity of comorbidities (**Grade D**) (Table 12).

8.6.2. Weight Loss

- **R79.** Inadequate weight loss should prompt evaluation for (1) surgical failure with loss of integrity of the gas-

tric pouch in gastroplasty or RYGB procedures, (2) a poorly adjusted gastric band, and (3) development of maladaptive eating behaviors or psychologic complications (**Grade B; BEL 2 [randomized]**).

- **R80.** The assessment of inadequate weight loss after bariatric surgery should include imaging studies to determine the integrity of the gastric pouch, ascertainment of the patient's understanding of the meal plan and compliance, and psychologic evaluation (**Grade D**).
- **R81.** Inadequate weight loss after a bariatric procedure without resolution or a recurrence of a major comorbidity may necessitate a surgical revision, such as conversion of a LAGB to either a RYGB or a BPD/DS (**Grade D**).

8.6.3. Metabolic and Nutritional Management

- **R82.** In those patients without complete resolution of their T2DM, hyperlipidemia, or hypertension, continued surveillance and management should be guided by currently accepted practice guidelines for those conditions (**Grade D**).
- **R83.** In those patients in whom T2DM, hyperlipidemia, and hypertension have resolved, continued surveillance should be guided by recommended screening guidelines for the specific age-group (**Grade D**).
- **R84.** Patients who have undergone RYGB, BPD, or BPD/DS and who present with postprandial hypoglycemic symptoms that have not responded to nutritional manipulation should undergo evaluation for the possibility of endogenous hyperinsulinemic hypoglycemia (**Grade C; BEL 3**).
- **R85.** Routine metabolic and nutritional monitoring is recommended after all bariatric surgical procedures (**Grade A; BEL 1**).
- **R86.** Patients should be advised to increase their physical activity (aerobic and strength training) to a minimum of 30 minutes per day as well as increase physical activity throughout the day as tolerated (**Grade D**).
- **R87.** All patients should be encouraged to participate in ongoing support groups after discharge from the hospital (**Grade D**).

8.6.3.1. Association of malabsorptive surgical procedures with nutritional deficiencies

- **R88.** The frequency and recommended nutritional surveillance in patients who have had a malabsorptive bariatric procedure are outlined in Table 13 (**Grade C; BEL 3**).

Table 12
Consensus for Follow-up Nutrition and Metabolic Consultations After Bariatric Surgery, Stratified by Type of Procedure Performed and Presence of Comorbidities (Grade D)^{a,b}

Procedure	Nutritional or metabolic comorbidities	First 6 months ^c	Second 6 months	Next year	Thereafter
VGB	No	q 3-6 mo	Once	Annually	Annually
	Yes	q 1-2 mo	Twice	q 6 mo	Annually
LAGB	No	q month prn	Once	Annually	Annually
	Yes	q month prn	Twice	q 6 mo	Annually
RYGB	No	q 2-3 mo	Once	q 6 mo	Annually
	Yes	q 1-2 mo	q 3-6 mo	q 6 mo	Annually
BPD/DS	No	q 2-3 mo	Twice	q 3-6 mo	Annually
	Yes	q 1-2 mo	q 6-12 mo	q 6-12 mo	q 6-12 mo

^a BPD/DS = biliopancreatic diversion with duodenal switch; LAGB = laparoscopic adjustable gastric band; prn = as the circumstances require; q = every; RYGB = Roux-en-Y gastric bypass; VGB = vertical banded gastroplasty.

^b These consultations are to be performed by a physician with expertise in nutritional and metabolic medicine.

^c The first follow-up visit is within the first postoperative month. Subsequent visit frequency depends on the severity of any complications and behavioral issues. After years 1 to 3, intestinal adaptation occurs, and metabolic derangements and weight loss should stabilize.

- **R89.** The recommended empiric vitamin and mineral supplementation after malabsorptive bariatric surgery is outlined in Table 14 (**Grade B; BEL 2 [randomized and nonrandomized]**).

8.6.3.2. Protein depletion and supplementation

- **R90.** Protein intake should be quantified periodically (**Grade D**).
- **R91.** Ideally, protein intake with meals, including protein supplementation, should be in the range of 80 to 120 g/d for patients with a BPD or BPD/DS and 60 g/d or more for those with RYGB (**Grade D**).
- **R92.** In patients with severe protein malnutrition not responsive to oral protein supplementation, PN should be considered (**Grade D**).

8.6.3.3. Skeletal and mineral homeostasis, including nephrolithiasis

- **R93.** Recommended laboratory tests for the evaluation of calcium and vitamin D metabolism and metabolic bone disease in patients who have undergone RYGB, BPD, or BPD/DS are outlined in Table 15 (**Grade D**).
- **R94.** In patients who have undergone RYGB, BPD, or BPD/DS, treatment with orally administered calcium, ergocalciferol (vitamin D₂), or cholecalciferol (vitamin

D₃) is indicated to prevent or minimize secondary hyperparathyroidism without inducing frank hypercalciuria (**Grade C; BEL 3**).

- **R95.** In cases of severe vitamin D malabsorption, oral doses of vitamin D₂ or D₃ may need to be as high as 50,000 to 150,000 U daily, and more recalcitrant cases may require concurrent oral administration of calcitriol (1,25-dihydroxyvitamin D) (**Grade D**).
- **R96.** In patients with RYGB, BPD, or BPD/DS, bone density measurements with use of dual-energy x-ray absorptiometry may be indicated to monitor for the development or presence of osteoporosis at baseline, in addition to a follow-up study at about 2 years, in accordance with the recommendations from the International Society for Clinical Densitometry (<http://www.iscd.org/Visitors/positions/OfficialPositionsText.cfm?fromhome=1>) and the National Osteoporosis Foundation (<http://www.nof.org/osteoporosis/bonemass.htm>) (**Grade D**).
- **R97.** Bisphosphonates approved by the US Food and Drug Administration may be a consideration in bariatric surgery patients with osteoporosis (*T* score -2.5 or below for the hip or spine) only after adequate and appropriate evaluation and therapy for calcium and vitamin D insufficiency. This evaluation should include and confirm a normal parathyroid hormone (PTH)

Table 13
Recommended Biochemical Surveillance of Nutritional Status
After Malabsorptive Bariatric Surgical Procedures^a

Surveillance factor	Roux-en-Y gastric bypass	Biliopancreatic diversion (± duodenal switch)
<i>Time interval</i>		
1st year	Every 3-6 mo	Every 3 mo
Thereafter	Annually	Every 3-6 mo depending on symptoms
<i>Laboratory tests</i>	CBC, platelets Electrolytes Glucose Iron studies, ferritin Vitamin B ₁₂ (MMA, HCy optional) Liver function (GGT optional) Lipid profile 25-Hydroxyvitamin D Optional: Intact PTH Thiamine RBC folate	CBC, platelets Electrolytes Glucose Iron studies, ferritin Vitamin B ₁₂ (MMA, HCy optional) Liver function (GGT optional) Lipid profile Albumin and prealbumin RBC folate Fat-soluble vitamins (6-12 mo) Vitamin A 25-Hydroxyvitamin D Vitamin E Vitamin K ₁ and INR Metabolic bone evaluation ^b Intact PTH (6-12 mo) 24-Hour urine calcium (6-12 mo) Urine N-telopeptide (annually) Osteocalcin (as needed) Metabolic stone evaluation (annually) 24-Hour urine calcium, citrate, uric acid, and oxalate Trace elements (annually or as needed) Zinc Selenium Miscellaneous (as needed) Carnitine Essential fatty acid chromatography

^a CBC = complete blood cell count; GGT = γ -glutamyltransferase; HCy = homocysteine; INR = international normalized ratio; MMA = methylmalonic acid; PTH = parathyroid hormone; RBC = red blood cell. See references 221-224.

^b Dual-energy x-ray absorptiometry should be performed annually to monitor bone density (**Grade D**).

level, 25-hydroxyvitamin D level of 30 to 60 ng/mL, normal serum calcium level, normal phosphorus level, and 24-hour urine calcium excretion between about 70 and 250 mg/24 h. Therapy considerations should be based on the National Osteoporosis Foundation-World Health Organization 2008 Guidelines (http://www.nof.org/professionals/NOF_Clinicians%20_Guide.pdf).

If therapy is indicated, then intravenously administered bisphosphonates should be used if concerns exist about adequate oral absorption and potential anastomotic ulceration with use of orally administered bisphosphonates (**Grade C; BEL 3**).

Table 14
Routine Nutrient Supplementation After Bariatric Surgery^a

Supplement	Dosage
Multivitamin	1-2 daily
Calcium citrate with vitamin D	1,200-2,000 mg/d + 400-800 U/d
Folic acid	400 µg/d in multivitamin
Elemental iron with vitamin D ^b	40-65 mg/d
Vitamin B ₁₂	≥350 µg/d orally or 1,000 µg/mo intramuscularly or 3,000 µg every 6 mo intramuscularly or 500 µg every week intranasally

^a Patients with preoperative or postoperative biochemical deficiency states are treated beyond these recommendations.
^b For menstruating women.

- **R98.** Recommended dosages of orally administered bisphosphonates in bariatric surgery patients with osteoporosis include the following: alendronate, 70 mg/wk; risedronate, 35 mg/wk or two 75-mg tablets/mo; or ibandronate, 150 mg/mo. Recommended intravenous dosages of bisphosphonates are as follows: zoledronic acid, 5 mg once a year, or ibandronate, 3 mg every 3 months (**Grade D**).
- **R99.** There are insufficient data to recommend empiric supplementation of magnesium after bariatric surgery beyond what is included in a mineral-containing multivitamin that provides the daily recommended intake of magnesium (>300 mg in women; >400 mg in men) (**Grade D**).
- **R100.** Oral phosphate supplementation may be provided for mild to moderate hypophosphatemia (1.5 to 2.5 mg/dL), which is usually due to vitamin D deficiency (**Grade D**).
- **R101.** Management of oxalosis and calcium oxalate stones includes avoidance of dehydration, a low oxalate meal plan, and oral calcium and potassium citrate therapy (**Grade D**).
- **R102.** Probiotics containing *Oxalobacter formigenes* have been shown to improve renal oxalate excretion and improve supersaturation levels and may therefore be used as well (**Grade C; BEL 3**).
- **R103.** The routine use of serum fatty acid chromatography to detect essential fatty acid deficiency is not cost-effective and should not be performed because this deficiency has not been reported (**Grade D**).
- **R104.** Routine supplementation of vitamin A is usually not necessary after RYGB or purely restrictive procedures (**Grade C; BEL 3**).
- **R105.** In contrast, routine screening for vitamin A deficiency is recommended, and supplementation is often needed after malabsorptive bariatric procedures, such as BPD or BPD/DS (**Grade C; BEL 3**).
- **R106.** Supplementation may be provided with use of vitamin A alone or in combination with the other fat-soluble vitamins (D, E, and K) (**Grade C; BEL 3**).
- **R107.** The value of routine screening for vitamin E or K deficiencies has not been documented for any bariatric procedure, including BPD and BPD/DS (**Grade C; BEL 3**).
- **R108.** In the presence of an established fat-soluble vitamin deficiency with hepatopathy, coagulopathy, or osteoporosis, assessment of a vitamin K₁ level should be considered in an effort to detect a deficiency state (**Grade D**).
- **R109.** Iron status should be monitored in all bariatric surgery patients and then appropriately treated as in any medical or surgical patient (**Grade D**).
- **R110.** Orally administered ferrous sulfate, fumarate, or gluconate (320 mg twice a day) may be needed to prevent iron deficiency in patients who have undergone a malabsorptive bariatric surgical procedure, especially in menstruating women (**Grade A; BEL 1**).

8.6.3.5. Iron, vitamin B₁₂, folic acid, and selenium deficiencies; the nutritional anemias

Table 15
Diagnostic Testing and Management for Skeletal and Mineral Disorders
in Patients Who Have Undergone Roux-en-Y Gastric Bypass,
Biliopancreatic Diversion, or Biliopancreatic Diversion With Duodenal Switch^a

Condition	Diagnostic testing	Management
Metabolic bone disease	Serum calcium, phosphorus, magnesium 25-Hydroxyvitamin D Bone-specific alkaline phosphatase (or osteocalcin) Intact parathyroid hormone Spot urine or serum N-telopeptide 24-Hour urine calcium excretion 1,25-Dihydroxyvitamin D (if renal compromise) Vitamin A and K ₁ levels Albumin and prealbumin Dual-energy x-ray absorptiometry (at 3 sites) at baseline and 2-year follow-up per ISCD and NOF recommendations ^c	Calcium citrate or gluconate Vitamin D ₂ or D ₃ orally Calcitriol orally Vitamin D intramuscularly (if available) Alendronate, ibandronate, or risedronate orally Ibandronate, pamidronate, or zoledronate intravenously ^b Calcitonin intranasally Human recombinant parathyroid hormone where appropriate
Nephrolithiasis	Urinalysis 24-Hour urine specimen for calcium, oxalate, citrate Renal ultrasonography	Low oxalate diet Calcium orally Cholestyramine Potassium citrate Lithotripsy Urologic surgery

^a ISCD = International Society for Clinical Densitometry; NOF = National Osteoporosis Foundation; WHO = World Health Organization.

^b Intravenously administered bisphosphonates may cause hypocalcemia and hypophosphatemia and should be used cautiously—only after documenting calcium and vitamin D sufficiency and with aggressive calcium and vitamin D supplementation. With intravenous bisphosphonate use, serum calcium and phosphate levels should be monitored. Intravenously administered pamidronate is not approved by the US Food and Drug Administration for osteoporosis prevention or treatment. See NOF-WHO 2008 guidelines (http://www.nof.org/professionals/NOF_Clinicians%20_Guide.pdf).

^c ISCD (see <http://www.iscd.org/Visitors/positions/OfficialPositionsText.cfm?fromhome=1>); NOF (see <http://www.nof.org/osteoporosis/bonemass.htm>).

- **R111.** Vitamin C supplementation should be considered in patients with recalcitrant iron deficiency because vitamin C can increase iron absorption and ferritin levels (**Grade C; BEL 3**).
- **R112.** Intravenous iron infusion with iron dextran, ferric gluconate, or ferric sucrose may be needed if oral iron supplementation is ineffective at correcting the iron deficiency (**Grade D**).
- **R113.** Evaluation for vitamin B₁₂ deficiency is recommended in all bariatric surgery patients (**Grade B; BEL 2 [nonrandomized]**).
- **R114.** Oral supplementation with crystalline vitamin B₁₂ at a dosage of 350 µg daily or more or intranasally administered vitamin B₁₂, 500 µg weekly, may be used to maintain vitamin B₁₂ levels (**Grade B; BEL 2 [non-randomized]**).
- **R115.** Parenteral supplementation with either 1,000 µg of vitamin B₁₂ monthly or 1,000 to 3,000 µg every 6 to 12 months is necessary if vitamin B₁₂ sufficiency cannot be maintained by means of oral supplementation (**Grade C; BEL 3**).
- **R116.** Assessment of vitamin B₁₂ status should be done annually in patients who have undergone RYGB or BPD/DS (**Grade D**).

- **R117.** Folic acid supplementation (400 µg/d) is provided as part of a routine multivitamin preparation (**Grade B; BEL 2 [randomized and nonrandomized]**).
- **R118.** Folic acid supplementation should be provided in all women of childbearing age because of the risk of fetal neural tube defects with folic acid deficiency (**Grade A; BEL 1**).
- **R119.** Nutritional anemias resulting from malabsorptive bariatric surgical procedures might also involve deficiencies in protein, copper, and selenium, necessitating evaluation of these nutrients when routine screening for iron, vitamin B₁₂, and folic acid deficiencies is negative (**Grade C; BEL 3**).
- **R120.** There are insufficient data to support routine screening for selenium deficiency or empiric selenium supplementation in patients after a bariatric surgical procedure (**Grade D**).
- **R121.** In patients treated with BPD or BPD/DS who have unexplained anemia or fatigue, persistent diarrhea, cardiomyopathy, or metabolic bone disease, selenium levels should be checked (**Grade C; BEL 3**).

8.6.3.6. Zinc and thiamine

- **R122.** Because zinc deficiency has been described, physicians should routinely screen for it after BPD or BPD/DS, while bearing in mind that plasma zinc levels are unreliable in the presence of systemic inflammation (**Grade C; BEL 3**).
- **R123.** There is inadequate clinical evidence to recommend empiric zinc supplementation after bariatric surgery (**Grade D**).
- **R124.** All bariatric surgery patients should be provided with an oral multivitamin supplement that contains thiamine (**Grade D**).
- **R125.** Routine screening for thiamine deficiency or additional empiric thiamine treatment (or both) is not recommended in bariatric surgery patients who are already routinely receiving a multivitamin supplement that contains thiamine (**Grade C; BEL 3**).
- **R126.** Patients with protracted vomiting should be screened for thiamine deficiency (**Grade C; BEL 3**).
- **R127.** In patients with persistent vomiting after any bariatric procedure, aggressive supplementation with thiamine is imperative; intravenously administered glucose should be provided judiciously in this situation because it can aggravate thiamine deficiency (**Grade C; BEL 3**).
- **R128.** In patients presenting with neurologic symptoms suggestive of thiamine deficiency (that is, Wernicke encephalopathy and peripheral neuropathy), aggressive parenteral supplementation with thiamine (100 mg/d) should be administered for 7 to 14 days (**Grade C; BEL 3**).
- **R129.** Subsequent oral thiamine supplementation (100 mg/d) should be continued until neurologic symptoms resolve (**Grade C; BEL 3**).

8.6.4. Cardiology and Hypertension

- **R130.** Lipid levels and need for lipid-lowering medications should be periodically monitored and evaluated (**Grade D**).
- **R131.** Use of antihypertensive medications should be evaluated repeatedly and reduced or discontinued as indicated with the resolution of hypertension (**Grade D**).

8.6.5. Gastrointestinal Complications

8.6.5.1. Diarrhea

- **R132.** If diarrhea persists, an evaluation should be initiated (**Grade C; BEL 3**).
- **R133.** Upper endoscopy with small bowel biopsies and aspirates remains the “gold standard” in the evaluation of celiac sprue (**Grade C; BEL 3**) and bacterial overgrowth (**Grade C; BEL 3**).
- **R134.** Colonoscopy should be performed and a stool specimen should be obtained if the presence of *Clostridium difficile* colitis is suspected (**Grade C; BEL 3**).
- **R135.** Persistent steatorrhea after BPD or BPD/DS should prompt an evaluation for nutrient deficiencies (**Grade C; BEL 3**).

8.6.5.2. Stomal stenosis or ulceration after bariatric surgery

- **R136.** Nonsteroidal antiinflammatory drugs should be avoided after bariatric surgery because they have been implicated in the development of anastomotic ulcerations (**Grade C; BEL 3**).
- **R137.** Alternative pain medication should be identified before bariatric surgery (**Grade D**).
- **R138.** Persistent and severe gastrointestinal symptoms (such as nausea, vomiting, and abdominal pain) warrant additional evaluation (**Grade C; BEL 3**).

- **R139.** Upper intestinal endoscopy is the preferred diagnostic procedure because, in many circumstances, upper endoscopy can also incorporate a therapeutic intervention with transendoscopic dilation of a recognized stricture (**Grade C; BEL 3**).
- **R140.** Evaluation should include *H pylori* testing as a possible contributor to persistent gastrointestinal symptoms after bariatric surgery (**Grade C; BEL 3**).
- **R141.** Anastomotic ulcers should be treated with H₂ receptor blockers, proton pump inhibitors, sucralfate, antibiotics, and, if *H pylori* is identified, multiple antibiotics and bismuth (**Grade C; BEL 3**).
- **R142.** Patients who previously underwent a RYGB with a nonpartitioned stomach and develop a gastrogastric fistula should undergo revisional RYGB to separate the upper and lower gastric pouches (**Grade D**).
- **R143.** Persistent vomiting, regurgitation, and UGI obstruction after LAGB should be treated with immediate removal of all fluid from the adjustable band (**Grade D**).
- **R144.** Persistent symptoms of gastroesophageal reflux, regurgitation, chronic cough, or recurrent aspiration pneumonia after LAGB are all problems suggestive of the band being too tight or the development of an abnormally large gastric pouch above the band. These symptoms should prompt immediate referral back to the surgeon (**Grade D**).

8.6.5.3. Gallbladder disease

- **R145.** Oral administration of ursodiol (300 mg twice a day) for 6 months postoperatively may be considered in patients not undergoing a prophylactic cholecystectomy (**Grade A; BEL 1**).
- **R146.** There is a debate regarding performance of cholecystectomy for known gallstones at the time of RYGB, BPD, or BPD/DS procedures. There is no consensus regarding the need to perform cholecystectomy at the time of bariatric operations (**Grade C; BEL 3**).

8.6.5.4. Bacterial overgrowth

- **R147.** Although uncommon, suspected bacterial overgrowth in the biliopancreatic limb after BPD or BPD/DS should be treated empirically with metronidazole (**Grade C; BEL 3**).
- **R148.** For antibiotic-resistant cases of bacterial overgrowth, probiotic therapy with *Lactobacillus plantarum* 299v and *Lactobacillus* GG may be considered (**Grade D**).

8.6.6. Incisional Hernias

- **R149.** Repair of asymptomatic hernias should be deferred until weight loss has stabilized and nutritional status has improved, to allow for adequate healing (12 to 18 months after bariatric surgery) (**Grade D**).
- **R150.** Incarcerated incisional or umbilical hernias in conjunction with abdominal pain necessitates aggressive surgical correction because of the risk of bowel infarction (**Grade C; BEL 3**).

8.6.7. Bowel Obstruction From Adhesions or Internal Hernias

- **R151.** Patients with cramping periumbilical pain at any time after RYGB, BPD, or BPD/DS should be emergently evaluated with an abdominal and pelvic CT scan to exclude the potentially life-threatening complication of closed-loop bowel obstruction (**Grade D**).
- **R152.** Exploratory laparotomy or laparoscopy is indicated in patients who are suspected of having an internal hernia because this complication can be missed with UGI studies and CT scans (**Grade C; BEL 3**).

8.6.8. Body-Contouring Surgery

- **R153.** Body-contouring surgery may be performed after bariatric surgery to manage excess tissue that impairs hygiene, causes discomfort, and is disfiguring (**Grade C; BEL 3**).
- **R154.** Circumferential tioroplasty or abdominoplasty may be used to remove excess abdominal skin (**Grade D**).
- **R155.** Breast reduction or lift, arm lift, resection of redundant gluteal skin, and thigh lift can also be pursued (**Grade D**).
- **R156.** Such procedures are best pursued after weight loss has stabilized (12 to 18 months after bariatric surgery) (**Grade D**).
- **R157.** Tobacco use must be avoided and nutritional status maintained in bariatric surgery patients undergoing postoperative body-contouring procedures (**Grade A; BEL 1**).

8.7. Criteria for Hospital Admission After Bariatric Surgery

- **R158.** Severe malnutrition should prompt hospital admission for initiation of nutritional support (**Grade D**).
- **R159.** The initiation of enteral or parenteral nutrition should be guided by established published criteria (**Grade D**).

- **R160.** Hospital admission is required for the management of gastrointestinal complications after bariatric surgery in clinically unstable patients (**Grade D**).
- **R161.** Surgical management should be pursued for gastrointestinal complications not amenable or responsive to medical therapy (**Grade D**).
- **R162.** If not dehydrated, most patients can undergo endoscopic stomal dilation for stricture as an outpatient procedure (**Grade D**).
- **R163.** Revision of a bariatric surgical procedure is recommended in the following circumstances: (1) presence of medical complications clearly resulting from the surgical procedure and not amenable or responsive to medical therapy (for example, malnutrition) and (2) inadequate weight loss or weight regain in patients with persistent weight-related comorbidities who previously underwent a restrictive procedure (for example, VBG) (**Grade C; BEL 3**).
- **R164.** Reversal of a bariatric surgical procedure is recommended when serious complications related to previous bariatric surgery cannot be managed medically and are not amenable to surgical revision (**Grade D**).

9. APPENDIX: DISCUSSION OF THE CLINICAL EVIDENCE

9.1. Effectiveness of Bariatric Surgery for Obesity Comorbidities

The comorbidities of severe obesity affect all the major organ systems of the body. Surgically induced weight loss will substantially improve or reverse the vast majority of these adverse effects from severe obesity.

9.1.1. Type 2 Diabetes

Unlike most obesity-related morbidities, improvements in hyperglycemia are observed almost immediately after RYGB and BPD/DS, in part because of increased release of GLP-1 (115 [EL 4], 116 [EL 4], 225 [EL 3]) or possibly as a result of the release of an unknown insulin sensitizer. Fasting plasma glucose concentrations have been reported to return to normal before hospital dismissal and before substantial weight loss (103 [EL 3], 122 [EL 3], 123 [EL 3], 130-135 [EL 3], 137 [EL 3], 146 [EL 2], 147 [EL 3], 226 [EL 3], 227 [EL 3]). Insulin-treated patients have a notable decrease in insulin requirement, with the majority of patients able to discontinue insulin therapy by 6 weeks after bariatric surgery (136 [EL 3], 226 [EL 3]) and even possibly able to discontinue insulin treatment before hospital discharge after RYGB and BPD/DS. The longer T2DM has been present, the less likely it is to respond to surgically induced weight loss (103 [EL 3], 122 [EL 3], 130 [EL 3], 137 [EL 3]). LAGB

is also associated with remission of T2DM; however, the effects take longer to achieve than with RYGB, BPD, or BPD/DS and are totally dependent on weight loss (63 [EL 2], 141 [EL 2]).

One explanation for the salutary effects of RYGB and intestinal bypass on glucose metabolism focuses on the enteroinsular axis and the main incretins: glucose-dependent insulinotropic polypeptide and GLP-1. Exclusion of ingested carbohydrate from the duodenal and proximal jejunal mucosa modifies the secretion of glucose-dependent insulinotropic polypeptide. Reintroduction of ingested carbohydrate into the distal ileum and colon increases the secretion of GLP-1 (138 [EL 3], 228 [EL 4]). Bypass of the duodenum without gastric bypass and ileal interposition have both been found to improve diabetes in animal models as well as in patients (225 [EL 3], 226 [EL 3], 229 [EL 3]). Furthermore, LAGB (230 [EL 2]) and RYGB (123 [EL 3], 231 [EL 3]) result in decreased leptin, whereas VBG (232 [EL 3]) and RYGB (233 [EL 3], 234 [EL 2]) eventuate in increased adiponectin in association with weight loss and improved insulin sensitivity.

In a literature review, Rubino and Gagner (235 [EL 4]) found that RYGB and BPD achieved durable primary beneficial effects on glycemic control in 80% to 100% of patients with T2DM, independent of effects on body weight. These conclusions were supported by studies in rats in which gastrojejunal bypass controlled T2DM independent of weight loss (236 [EL 4]). In a subsequent study of 10 obese patients undergoing RYGB, a potential mechanism was elucidated (226 [EL 3]). Bypass of the proximal small bowel was associated with a statistically significant decrease in GLP-1 and hyperinsulinemia. Moreover, early presentation of undigested food to the distal small bowel was associated with a trend toward greater levels of GLP-1 and restoration of normal glucose-stimulated insulin secretion (226 [EL 3]). These and other intestinal factors may also restore meal-induced suppression of ghrelin release from the stomach, resulting in decreased food intake (237 [EL 4]).

9.1.2. Pulmonary

9.1.2.1. Sleep apnea

Weight reduction after LAGB, RYGB, BPD, or BPD/DS bariatric procedures has been shown to improve sleep apnea (99 [EL 1], 106 [EL 3], 107 [EL 3], 238-245 [EL 2-4]). Charuzi et al (106 [EL 3]) reported that diminished symptoms seem to be dictated by percentage loss of EBW. Sleep apnea symptoms are likely to persist in patients who have a substantial amount of EBW despite weight loss or to recur in those who experience weight regain (106 [EL 3]). In multiple studies, sleep apnea has been shown either to resolve completely (for patients with a respiratory disturbance index [RDI] <40) or to improve appreciably for those with an RDI \geq 40 (107 [EL 3]); thus, the patient can discontinue the use of nasal CPAP or allow a tracheostomy to close.

9.1.2.2. Obesity-hypoventilation syndrome

A major comorbidity of severe obesity is OHS, in conjunction with chronic hypoxemia and hypercarbia when the patient is awake. When seen in conjunction with sleep apnea, it is often called the pickwickian syndrome. In patients with central obesity, OHS arises primarily from the increased intra-abdominal pressure, which leads to a high-riding diaphragm (109-111 [EL 2], 114 [EL 2], 240 [EL 3]). As a result, the lungs are squeezed, and a restrictive pulmonary defect is produced. A heavy, obese thoracic cage may also contribute to the pathophysiologic condition, attributable to decreased chest wall compliance. These patients have a considerably decreased expiratory reserve volume, leading to alveolar collapse and arteriovenous shunting at end-expiration (240 [EL 3]). They also have smaller reductions in all other lung volumes. Chronic hypoxemia leads to pulmonary artery vasoconstriction and hypertension. Frequently, however, patients with OHS have not only notably elevated pulmonary artery pressures but also increased pulmonary capillary wedge pressures, suggesting both right and left ventricular failure, probably as a result of increased intra-abdominal and intrathoracic pressures (240 [EL 3]). Surgically induced weight loss is associated with resolution of OHS, increasing oxygenation and normalizing hypercarbia, lung volumes, and cardiac filling pressures (109-111 [EL 2], 240 [EL 3]).

9.1.2.3. Asthma

Two studies have documented improvement in asthma after surgically induced weight loss (43 [EL 3], 246 [EL 3]). This outcome may be due to the resolution of GERD and acid-induced bronchospasm.

9.1.3. Dyslipidemia, Hypertension, and Cardiac Function

9.1.3.1. Dyslipidemia

Several studies have shown a substantial improvement in lipid abnormalities and risk for CAD, which persists for at least 5 to 10 years after bariatric surgery. In several reports, triglyceride and low-density lipoprotein (LDL) cholesterol levels decreased and the HDL cholesterol value increased after LAGB, RYGB, BPD, or BPD/DS surgery (64 [EL 3], 105 [EL 3], 247-259 [EL 1-3]). Nevertheless, conventional lipid measurements of total and LDL cholesterol levels may not be reflective of dyslipidemic risks or insulin resistance in obese people, as suggested by a cross-sectional study of 572 obese patients (260 [EL 2]). The improvement in dyslipidemia appears to be related not only to the percentage loss of EBW (256 [EL 3]) but also to the decrease in insulin resistance (247 [EL 3]). These changes should lead to a pronounced decrease in risk for CAD, stroke, and peripheral vascular disease. Recent studies have shown decreased cardiovascular and MI-related mortality in bariatric surgery patients (65 [EL 3], 154 [EL 2]).

9.1.3.2. Hypertension

LAGB, RYGB, BPD, or BPD/DS surgery is associated with clinically important and long-lasting improvement in systemic hypertension, with elimination of blood pressure medications or a distinct decrease in their use in two-thirds to three-quarters of the patients with hypertension (44 [EL 3], 104 [EL 3], 139 [EL 3], 142 [EL 2], 253 [EL 1], 261-263 [EL 3]). In the SOS Study, recovery from hypertension was notable at 2 years after bariatric surgery (253 [EL 1]); however, the difference between the surgical and nonsurgical groups was no longer present at 8 years postoperatively (264 [EL 2]). Nonetheless, at 8 years in the 6% of patients who underwent RYGB and had lost significantly more weight than the patients who had purely restrictive procedures (VGB and gastric banding), there was a significant decrease in both systolic and diastolic blood pressure (121 [EL 2]). Although no difference was observed between the bariatric surgical and nonsurgical cohorts at 10 years, only 8 patients had undergone RYGB at that time, too small a number for statistical analysis (64 [EL 3]).

9.1.3.3. Cardiac function

Severe obesity may be associated with cardiomegaly, increased left ventricular wall thickness, and impaired left, right, or bilateral ventricular function. In addition, severe obesity may be associated with high cardiac output and low systemic vascular resistance, leading to left ventricular hypertrophy. Obesity is also associated with hypertension, which leads to concentric left ventricular hypertrophy. This combination of obesity and hypertension with left ventricular eccentric and concentric hypertrophy may result in left ventricular failure (265 [EL 3], 266 [EL 2]). Correction of severe obesity decreases left ventricular wall thickness, increases left ventricular ejection fraction, and improves overall cardiac function in these patients (241 [EL 3], 266-271 [EL 2-3]). Bariatric surgery has been shown to improve cardiac function in patients with idiopathic cardiomyopathy (272 [EL 3]). Morbid obesity is also associated with an accelerated rate of coronary atherosclerosis and deaths due to MI (273 [EL 2], 274 [EL 2]). Surgically induced weight loss is associated with a substantial decrease in factors that pertain to obesity-related cardiac mortality (including the aforementioned conditions), significant and long-standing improvements in dyslipidemia, hypertension, and sleep apnea-induced cardiac arrhythmias, and a decrease in frequency of MI (275 [EL 2]) as well as the rate of deaths due to MI (65 [EL 3], 154 [EL 2]).

9.1.4. Gastrointestinal

9.1.4.1. Gastroesophageal reflux disease

After RYGB, GERD has been found to improve considerably because little acid or bile is available to reflux into the esophagus (188 [EL 3], 276-278 [EL 3-4]).

Moreover, studies have found complete regression of Barrett esophagus after RYGB (279 [EL 3]). Improvement in GERD has also been seen after VBG (280 [EL 3]); this improvement in reflux after either RYGB or purely restrictive procedures also may be a result of the decrease in intra-abdominal pressure seen after surgically induced weight loss (109 [EL 2], 111 [EL 2], 114 [EL 2]). Because a Nissen fundoplication relieves only one of the comorbidities of severe obesity and because there is an increased rate of failure of fundoplication in severely obese patients, RYGB should be the preferred treatment of morbidly obese patients with GERD.

9.1.4.2. Nonalcoholic fatty liver disease and nonalcoholic steatohepatitis

Many obese patients will have asymptomatic increases in serum alanine aminotransferase and aspartate aminotransferase levels. These changes are most commonly associated with NAFLD or, in its more advanced form, nonalcoholic steatohepatitis and cirrhosis. At the time of bariatric surgery, 84% of morbidly obese subjects have steatosis on liver biopsy specimens, and 20% and 8% have inflammation and fibrosis, respectively (281 [EL 3]). Weight loss after LAGB, RYGB, BPD, or BPD/DS leads to regression of steatosis and inflammation, including decreased bridging fibrosis in some patients (148 [EL 3], 282-294 [EL 2-4]).

9.1.5. Endocrine

9.1.5.1. Polycystic ovary syndrome

Women accounted for 82% of all bariatric procedures in the United States in 2004 (98 [EL 3]). Polycystic ovary syndrome is characterized by the presence of chronic anovulation, menstrual irregularity, and hyperandrogenism typically with a pubertal onset (295 [EL 2]) and can be associated with insulin resistance and T2DM. In many cases, the hyperandrogenic and anovulatory symptoms of PCOS are ameliorated with metformin treatment (296 [EL 1], 297 [EL 2]). Surgically induced weight loss can also result in decreased androgen levels, increasing fertility and restoring menstrual regularity (298-303 [EL 2-4]).

9.1.5.2. Male sex hormone dysfunction

Two recent studies have documented obesity-related abnormality of the pituitary-gonadal axis and hypoandrogenism, presumably due to peripheral aromatization of testosterone to estrogen in adipose tissue, which resolved after bariatric surgery (303 [EL 3], 304 [EL 4]).

9.1.6. Pregnancy

Increased weight also increases the risk of complications of pregnancy. Surgically induced weight loss is associated with decreased pregnancy-related complications, including preeclampsia, cephalopelvic disproportion, macrosomia, gestational diabetes, and the need for cesarean delivery (305-311 [EL 2-3]). Children born to mothers

after weight loss surgery weigh less at birth and maintain a lower weight than do siblings who were born before bariatric surgery (312 [EL 3]). Resolution of severe obesity should also lead to a decreased risk of venous thromboembolism in pregnant women, but thus far no data have been published.

9.1.7. Venous Disease

Severely obese patients often have problems with chronic edema of the lower extremities, which can lead to bronze discoloration and chronic ulceration, as well as an increased risk of thrombophlebitis and PE. This comorbidity is probably a result of increased intra-abdominal pressure, leading to an increased inferior vena caval pressure and decreased venous return (109-111 [EL 2], 114 [EL 2], 313 [EL 3]). Surgically induced weight loss considerably improves venous stasis disease, including resolution of venous stasis ulcers (113 [EL 4]).

9.1.8. Central Nervous System

9.1.8.1. Pseudotumor cerebri

Pseudotumor cerebri, also known as idiopathic intracranial hypertension, may be associated with extreme obesity. This problem occurs almost exclusively in women. Symptoms include severe headache that is usually worse in the morning, bilateral pulsatile auditory tinnitus, and visual field cuts. Severely increased intracranial pressure can lead to permanent blindness. Cranial nerves that may be involved include V (tic douloureux), VI (oculomotor nerve paralysis), and VII (Bell palsy). Studies suggest that pseudotumor cerebri is attributable to increased intra-abdominal pressure, leading to increased pleural pressure and decreased venous drainage from the brain, with consequent cerebral venous engorgement and increased intracranial pressure. Increased intracranial pressure has been demonstrated in an acute porcine model of increased intra-abdominal pressure, which was prevented by median sternotomy (314 [EL 4], 315 [EL 4]). In the past, pseudotumor cerebri was treated with ventriculoperitoneal or lumboperitoneal cerebrospinal fluid (CSF) shunts. The incidence of shunt occlusion is high (316 [EL 3]), and in some cases, patients can have continued headache and auditory tinnitus despite a patent shunt. These failures are probably related to shunting from one high-pressure system to another high-pressure system. Major neurologic complications may also develop after insertion of a ventriculoperitoneal or lumboperitoneal shunt. Because surgically induced weight loss decreases CSF pressure and relieves headache and tinnitus (112 [EL 3], 317 [EL 4], 318 [EL 3]), bariatric surgery is the intervention of choice over CSF-peritoneal shunting in severely obese patients.

9.1.8.2. Stroke

With improvement in hypertension and atherosclerosis, there should be a decrease in the rate of cerebrovascular accidents. One cohort study supports this prediction,

finding a decrease in stroke mortality in a cohort of patients who underwent bariatric surgery (154 [EL 2]).

9.1.9. Urologic

Severe obesity is associated with a very high frequency of urinary incontinence in women, which resolves almost uniformly after bariatric surgery. This problem is attributable to increased intra-abdominal and bladder pressures, which decrease substantially after surgically induced weight loss (111 [EL 2], 319-321 [EL 3]).

9.1.10. Musculoskeletal

The excessive weight in severe obesity leads to early degenerative arthritic changes of the weight-bearing joints, including the knees, hips, and spine (322 [EL 4]). Many orthopedic surgeons refuse to insert total hip or knee prostheses in patients weighing 250 lb (113.5 kg) or more because of an unacceptable incidence of prosthetic loosening (323 [EL 3]). There is a high risk of complications in obese patients after intramedullary nailing of femoral fractures (324 [EL 3]). Severe obesity is a common problem in patients requiring an intervertebral disk surgical procedure (325 [EL 3]). Weight reduction after gastric surgery for obesity allows subsequent successful joint replacement (326 [EL 3]) and is associated with decreased musculoskeletal and lower back pain (275 [EL 2], 327 [EL 3]). In some instances, the decrease in pain after weight loss eliminates the need for a joint operation (328 [EL 2], 329 [EL 3]) or intervertebral disk operation (330 [EL 3]). Bariatric surgery improves mobility and postural stability (331 [EL 2], 332 [EL 3], 333 [EL 3]).

9.1.11. Cancer

Severely obese patients are at an increased risk for cancer, including involvement of the breast, uterus, prostate, colon, liver, and esophagus. One study found a decrease in treatment for cancer in patients from Quebec Province who had undergone bariatric surgery in comparison with a cohort of patients who had not (153 [EL 3]). Two recent studies have found a decrease in cancer-related mortality among patients who had undergone bariatric surgery when compared with a nonsurgical cohort (65 [EL 3], 154 [EL 2]).

9.1.12. Psychosocial Issues

Extreme obesity is associated with considerable psychosocial distress (48 [EL 4], 118 [EL 4], 119 [EL 4]). Between 20% and 60% of persons seeking bariatric surgery meet the criteria for a major psychiatric disorder—most commonly, mood disorders (334-337 [EL 3]). Disordered eating behaviors seem to be more common among bariatric surgery patients than in the general population (338-348 [EL 3-4]). In comparison with persons of average weight, those with extreme obesity often experience increased symptoms of depression and anxiety, impaired quality of life, body image dissatisfaction, and problems with marital and sexual functioning (349-352

[EL 3-4]). The experience of weight-related prejudice and discrimination, which have been found in social, educational, occupational, and health-care settings, may be particularly common in the extremely obese population (349 [EL 3], 353 [EL 4], 354 [EL 3], 355 [EL 3]).

The majority of bariatric surgery patients report improvements in psychosocial functioning postoperatively (48 [EL 4], 117-119 [EL 4], 356 [EL 4]). Several studies have documented an improved quality of life after surgically induced weight loss (57 [EL 3], 189 [EL 2], 357-362 [EL 3]). For some patients, however, the psychosocial benefits of surgical treatment seem to wane over time, and a minority appear to experience untoward psychosocial outcomes. Several investigators have documented problems with substance abuse, alcoholism, and suicide postoperatively (122 [EL 3], 336 [EL 3], 341 [EL 3], 363 [EL 3]). There has been recent concern regarding the possibility of substitutive addictive behavior after bariatric surgery, but little supporting scientific evidence is available. At least one study found an increased divorce rate after bariatric surgery (364 [EL 3]). Careful examination of the data, however, revealed that the divorce rate was the result of the dissolution of very poor prior relationships and not the disintegration of healthy ones.

9.1.13. Mortality Reduction as a Result of Bariatric Surgery

Several reports have noted a significant decrease in mortality among patients who have undergone bariatric surgery in comparison with matched nonsurgical cohorts. MacDonald et al (151 [EL 3]) found that the mortality among patients who underwent bariatric surgery was 9% (N = 154) as compared with 28% (N = 78) among those who did not. Reasons for not undergoing surgical treatment were inability to obtain insurance coverage or choosing not to proceed with surgery (151 [EL 3]). In a similar study, Sowemimo et al (152 [EL 3]) found an 81% reduction in mortality among bariatric surgery patients versus those who did not have surgery. Christou et al (153 [EL 3]) also noted an 89% reduction in mortality in Quebec Province for patients who underwent surgery for obesity when compared with a nonsurgical cohort of patients with a diagnosis of morbid obesity. In addition, this study noted a significant decrease in treatment for cancer in the surgical group. Flum and Dellinger (39 [EL 3]) found a significant decrease in mortality at 1 and 15 years after gastric bypass; however, there was a 1.9% 30-day mortality, in part attributable to surgeon inexperience with the procedure. Busetto et al (155 [EL 3]) found that patients with LAGB treatment have a lower risk of death in comparison with matched cohorts who did not have surgical treatment. Similar findings were observed by Peeters et al (365 [EL 3]) in a study in which patients with LAGB had a 72% lower hazard of death than did an obese population-based cohort. The SOS Study reported a 25% decrease in mortality in bariatric surgery patients at 10 years postoperatively in comparison with a well-matched control

population (65 [EL 3]). Adams et al (154 [EL 2]) found a 40% decrease in mortality after RYGB in Salt Lake City, Utah, when compared with a matched nonsurgical cohort, with significant decreases in death associated with cancer, diabetes, and MI. None of these observations was based on randomized studies, and most involved experienced bariatric surgery centers. Therefore, conclusions concerning mortality may not be generalizable to all surgeons and patients.

9.2. Selection of Patients for Bariatric Surgery

All patients with a BMI of ≥ 40 kg/m², regardless of the presence of comorbidities, are potential candidates for bariatric surgery. Those patients with a BMI of 35 to 39 kg/m² are candidates, if they have an obesity-related comorbidity. One randomized, prospective trial supports the LAGB procedure for persons with a BMI between 30 and 35 kg/m² (63 [EL 2]). The only contraindications to bariatric surgery are persistent alcohol and drug dependence, uncontrolled severe psychiatric illness such as depression or schizophrenia, or cardiopulmonary disease that would make the risk prohibitive. Although the last-mentioned patients have a significantly increased risk of mortality, they should expect profound improvements in their weight-related pathologic condition if they can survive the bariatric procedure. Better risk-to-benefit stratification is needed for this group of patients.

9.3. Preoperative Evaluation

The preoperative evaluation of the patient seeking bariatric surgery involves multiple disciplines. Among clinical practices, the specialty of the physician guiding the evaluation varies from the general internist, to the endocrinologist or specialist in bariatric medicine, to the bariatric surgeon who will ultimately perform the operation. Regardless of the discipline of the professional guiding the initial evaluation, it is paramount for patients to be well informed and appropriately screened before these procedures. This educational process can be accomplished through the use of support groups and counseling sessions with members of the bariatric surgery team (Table 16). Proper screening allows for diagnosis of relevant comorbidities, which can then be managed preoperatively to improve surgical outcomes (Table 17).

Preexisting medical conditions should be optimally controlled before bariatric surgery. This optimization may necessitate the input of various medical specialists, including cardiologists, pulmonary specialists, and gastroenterologists. The registered dietitian skilled in preoperative and postoperative bariatric care should interact with the patient preoperatively for their evaluation and initiate a continuing nutrition education experience. The psychological assessment should be performed by a licensed psychologist, psychiatrist, or other mental health professional with experience in obesity and bariatric surgery. The psychological evaluation is a requirement for most insurance carriers and for the ASMBS Centers of Excellence and the

Table 16
Potential Members of a Bariatric Surgery Team

Bariatric surgeon
Bariatric coordinator (advanced practice nurse or well-educated registered nurse)
Internist with nutrition or bariatric medicine experience
Registered dietitian
Medical consultants ^a
Psychologist or psychiatrist
Endocrinologist
Physician nutrition specialist ^b
Certified nutrition support clinician ^c
Sleep medicine specialist
Cardiologist
Gastroenterologist
Physiatrist
Office support personnel

^a Consultants to be utilized as needed.

^b Designation by the American Board of Physician Nutrition Specialists.

^c Designation by the National Board of Nutrition Support Certification.

American College of Surgeons (ACS) Bariatric Surgery Centers. At the time of the surgical consultation, the surgeon should discuss the procedure that is recommended, explain the potential risks and benefits, and decide whether surgical treatment will be offered on the basis of this multidisciplinary approach.

For all patients seeking bariatric surgery, a comprehensive preoperative evaluation should be performed. This assessment includes an obesity-focused history, physical examination, and pertinent laboratory and diagnostic testing (366 [EL 4]). A detailed weight history should be documented, including a description of the onset and duration of obesity, the severity, and recent trends in weight. Causative factors to note include a family history of obesity, use of weight-gaining medications, and dietary and physical activity patterns. One need not document all previous weight loss attempts in detail, but a brief summary of personal attempts, commercial plans, and physician-supervised programs should be reviewed, along with the greatest duration of weight loss and maintenance. This information is useful in substantiating that the patient has made reasonable attempts to control weight before considering obesity surgery (53 [EL 3]). These issues also may be reviewed in greater detail by the program registered dietitian.

The patient's personal history should include current smoking, alcohol or substance abuse, and the stability of the home and work environments. An accurate medication list that includes over-the-counter supplements must be

Table 17
Screening and Management
of Comorbidities Before Bariatric Surgery^a

Routine chemistry studies (with fasting blood glucose, liver profile, and lipid profile), urinalysis, prothrombin time (INR), blood type, complete blood cell count, iron studies
Vitamin B ₁ (optional), vitamin B ₁₂ -folic acid assessment (RBC folate, homocysteine, methylmalonic acid) (optional)
Vitamins A and D (E and K optional) (if malabsorptive procedure planned), iPTH
<i>Helicobacter pylori</i> screening (optional) (if positive and epigastric symptoms present, then treatment with antibiotics and proton pump inhibitor)
Thyroid-stimulating hormone (thyrotropin) (optional)
Total or bioavailable testosterone, DHEAS, Δ_4 -androstenedione (if polycystic ovary syndrome suspected) (optional)
Overnight dexamethasone suppression, 24-hour urinary cortisol, 11 PM serum or salivary cortisol level screening tests (if Cushing syndrome suspected)
Cardiovascular evaluation (chest radiography, electrocardiography, and echocardiography if pulmonary hypertension or cardiac disease is known or suspected)
Gastrointestinal evaluation (gallbladder evaluation optional in asymptomatic persons or at the discretion of the surgeon, upper endoscopy if epigastric discomfort)
Sleep apnea evaluation if suspected; arterial blood gases if obesity-hypoventilation syndrome suspected or in superobese patients
Psychologic-psychiatric consultation

^a DHEAS = dehydroepiandrosterone sulfate; INR = international normalized ratio; iPTH = intact parathyroid hormone; RBC = red blood cell.

reviewed carefully. A small but growing list of psychiatric and neurologic medications (Table 18) may stimulate appetite, and such drugs have been associated with weight gain (367 [EL 4]). Considerable care must be exercised when discontinuation of such medications is being considered, inasmuch as decompensation of a known psychiatric condition may increase morbidity and threaten the success of bariatric surgery (368 [EL 4]). Typically, the preoperative mental health evaluation provides a more detailed assessment of psychiatric status and history.

In addition to elicitation of the past medical history, an inquiry of the review of systems is helpful for identification of undiagnosed symptoms and conditions associated with obesity. During the review of systems section of the history and the development of a problem list of obesity-related comorbidities, Table 19 can be used as a convenient checklist. During the recording of the preoperative history is also an opportune time to review and update screening recommendations from the standpoint of preventive medicine. Use of a printed questionnaire allows the interview to stay organized, leaves more time to focus on pertinent positive and negative factors, and provides a useful future reference (369 [EL 4]).

Finally, a summary of the patient's interest in and knowledge about the proposed surgical procedure, includ-

ing whether he or she has spoken to other patients, researched the procedure on the Internet, or attended support group meetings, should be completed (370 [EL 4], 371 [EL 4]). It is also important to assess the patient's expectations about postoperative weight loss. Many patients present for bariatric surgery with unrealistic expectations regarding the anticipated weight loss. Foster et al (372 [EL 3]) found that bariatric surgical candidates expect, on average, a 44% loss of preoperative weight. In contrast, a loss of only 27.3% was considered "disappointing," although losses of that magnitude are typically judged as successful by bariatric surgeons. Before bariatric surgery is scheduled, it is helpful for patients to read the program's information packet carefully, attend an orientation session, and speak to other patients who have undergone bariatric surgery at the hospital.

9.3.1. Mental Health Evaluation

The psychosocial evaluation serves 2 major purposes: (1) identification of potential contraindications to surgical intervention, such as substance abuse, poorly controlled depression, or other major psychiatric illness, and (2) identification of potential postoperative challenges and facilitation of behavioral changes that can enhance long-term weight management (370 [EL 4], 371 [EL 4]).

Table 18
Medications Associated With Body Fat Weight Gain^a

Class and subclass	Drug
Psychiatric or neurologic agents	
Antipsychotic agents	Phenothiazines, olanzapine, clozapine, risperidone
Mood stabilizers	Lithium
Antidepressants	Tricyclics, MAOIs, SSRIs, mirtazapine
Antiepileptic drugs	Gabapentin, valproate, carbamazepine
Steroid hormones	
Corticosteroids	...
Progestational steroids	...
Antidiabetes agents	Insulin, sulfonylureas, thiazolidinediones
Antihypertensive agents	β -Adrenergic and α_1 -adrenergic receptor blockers
Antihistamines	Cyproheptadine
HIV protease inhibitors	...

^a HIV = human immunodeficiency virus; MAOIs = monoamine oxidase inhibitors; SSRIs = selective serotonin reuptake inhibitors.

Although there are published recommendations regarding the structure and content of mental health evaluations (371 [EL 4], 373 [EL 4]), consensus guidelines have yet to be established. Typically, such evaluations are performed by psychologists, psychiatrists, or other mental health professionals who, ideally, have an appropriate working knowledge of the psychosocial issues involved in obesity and bariatric surgery. Almost all evaluations rely on clinical interviews with the patients; approximately two-thirds also include instrument or questionnaire measures of psychiatric symptoms or objective tests of personality or psychopathologic conditions (or both assessments) (374 [EL 3]). More comprehensive evaluations assess the patient's knowledge of bariatric surgery, weight and dieting history, eating and activity habits, and both potential obstacles and resources that may influence postoperative outcomes (370 [EL 4], 371 [EL 4]). Approximately 90% of bariatric surgery programs require their surgical candidates to undergo a mental health evaluation preoperatively (375 [EL 3]).

Assessment of the psychiatric status and history is the cornerstone of these mental health evaluations. Psychosocial distress is common among patients who present for bariatric surgery (48 [EL 4], 117-119 [EL 4]). Studies of clinical populations have found that up to 60% of persons who seek bariatric surgery fulfill the criteria for at least one Axis I psychiatric disorder (334-337 [EL 3]). Mood disorders were the most common diagnoses, although sizable minorities have been diagnosed as having eating, anxiety, and substance abuse disorders. Bariatric surgery patients also report severe impairment in quality of life, as

well as heightened dissatisfaction with their body image, marital relationship, and sexual functioning (349 [EL 3], 358 [EL 3], 364 [EL 3], 376-378 [EL 3]). In addition, many bariatric surgery patients report experiences with weight-related prejudice and discrimination.

Eating behaviors and habits should also be reviewed during the mental health evaluation, with specific attention to where and when the patient eats, who shops and cooks, snacking, portion sizes, intake of sweet beverages, and overall knowledge of nutrition (370 [EL 4], 371 [EL 4]). Specific inquiry concerning binge eating disorder should be undertaken. Early studies suggested that up to 50% of bariatric surgery candidates had this disorder (338 [EL 3], 339 [EL 3], 343 [EL 3], 344 [EL 3], 346 [EL 3]). More recent studies have suggested that the disorder may be far less common than thought initially, involving perhaps as few as 5% of patients (342 [EL 4], 347 [EL 3], 348 [EL 3]). Nevertheless, the diagnosis of preoperative binge eating disorder has been found to be associated with less weight loss or with weight regain within the first 2 postoperative years (340 [EL 4], 345 [EL 3], 379 [EL 1]).

At present, the relationship between preoperative psychological status and postoperative outcomes is unclear (48 [EL 4], 117-119 [EL 4]). Several studies have suggested that preoperative psychopathologic conditions and eating behavior are unrelated to postoperative weight loss; others have suggested that preoperative psychopathologic disorders may be associated with untoward psychosocial outcomes, but not with poorer weight loss. Unfortunately, the complex relationship between obesity and psychiatric illness, as well as a number of methodologic issues within

Table 19
Obesity-Related Review of Organ Systems

<i>Cardiovascular</i>	<i>Respiratory</i>
Hypertension	Dyspnea
Congestive heart failure	Obstructive sleep apnea
Cor pulmonale	Hypoventilation syndrome
Varicose veins	Pickwickian syndrome
Pulmonary embolism	Asthma
Coronary artery disease	<i>Gastrointestinal</i>
<i>Endocrine</i>	Gastroesophageal reflux disease
Metabolic syndrome	Nonalcoholic fatty liver disease
Type 2 diabetes mellitus	Cholelithiasis
Dyslipidemia	Hernias
Polycystic ovary syndrome, androgenicity	Colon cancer
Amenorrhea, infertility, menstrual disorders	<i>Genitourinary</i>
<i>Musculoskeletal</i>	Urinary stress incontinence
Hyperuricemia and gout	Obesity-related glomerulopathy
Immobility	End-stage renal disease
Osteoarthritis (knees and hips)	Hypogonadism (male)
Low back pain	Breast and uterine cancer
Carpal tunnel syndrome	Pregnancy complications
<i>Integument</i>	<i>Neurologic</i>
Striae distensae (stretch marks)	Stroke
Stasis pigmentation of legs	Idiopathic intracranial hypertension
Lymphedema	Meralgia paresthetica
Cellulitis	Dementia
Intertrigo, carbuncles	<i>Psychologic</i>
Acanthosis nigricans	Depression and low self-esteem
Acrochordon (skin tags)	Body image disturbance
Hidradenitis suppurativa	Social stigmatization

this literature, make drawing definitive conclusions difficult if not impossible. Perhaps psychiatric symptoms that are primarily attributable to weight, such as depressive symptoms and impaired quality of life, may be associated with more positive outcomes, whereas those symptoms representative of psychiatric illness—that is, independent of obesity—are associated with less positive outcomes (119 [EL 4]).

Studies have suggested that mental health professionals unconditionally recommend approximately 75% of bariatric surgery candidates for surgery (337 [EL 3], 374 [EL 3], 380 [EL 3]). In the remaining patients, the recommendation typically is to delay bariatric surgery until specific psychosocial or nutritional issues (or both) have been addressed with additional assessment or treatment. The benefits of recommending such a delay, however, should be weighed against the risk of patients not eventually returning for potential surgical treatment.

9.3.2. Physical Examination

For optimal comfort, the physician's office should be equipped properly with armless chairs, extra-large and reinforced examination tables, a suitable scale and stadiometer for measuring weight and height, large gowns, and appropriately sized blood pressure cuffs. The BMI should be computed and categorized by class. A comprehensive examination should be performed, with particular attention paid to signs of metabolic and cardiopulmonary disease. For example, a large neck circumference and a crowded posterior pharynx may be clues to the presence of OSA. Fungal infection in skinfolds may be a sign of undiagnosed diabetes. Observation of gait and breathing effort with modest exertion (for example, walking to the examination room or getting on and off the examination table) may provide clues to poor functional capacity or musculoskeletal disability.

9.3.3. Laboratory Studies and Procedures

The specific preoperative evaluation of the bariatric surgery patient should be directed toward symptoms, risk factors, and index of suspicion for secondary causes of obesity. Thus, Table 20 has been developed with use of an evidence-based approach for assessing comorbid conditions in obese patients.

When symptoms of OSA or hypercapnia (elevated PCO₂) are identified, polysomnography should be performed. Other treatable causes of hypercapnia, including OHS, other restrictive lung diseases, chronic obstructive pulmonary disease, left ventricular failure, and hypothyroidism, may also need to be considered (381 [EL 4]). Definitive diagnosis of patients suspected of having Cushing syndrome may be particularly difficult, inasmuch as weight gain, moon facies, posterior cervical fat pads, cutaneous stretch marks, hypertension, and glucose intolerance are relatively common among severely obese patients. If Cushing syndrome is suspected, measurement of a bedtime salivary cortisol level, which is often the earliest and most sensitive marker of the disease, has been recommended as a reasonable screening test (382 [EL 4]). In patients with equivocal results, repeated measures over time may be needed for a definitive diagnosis. Additional testing options include a 24-hour collection of urine for assessment of free cortisol excretion and the 1-mg overnight dexamethasone suppression test.

Women with a history of oligomenorrhea and androgenicity should be evaluated for PCOS. Numerous studies have demonstrated that women with PCOS are at a much higher risk for developing T2DM and cardiovascular disease than those without PCOS (383 [EL 4]). NAFLD is being increasingly recognized as an important cause of liver-related morbidity and mortality (384 [EL 4]) and is thought by many clinicians to be the most common cause of cryptogenic cirrhosis in the obese patient (385 [EL 3]).

Selection and timing of preoperative laboratory tests should be based on the patient's specific clinical indications and the evaluation by anesthesiology; obesity alone is not a risk factor for postoperative complications (386 [EL 3]). The current literature is not sufficiently rigorous to recommend ordering routine preoperative tests (387 [EL 4]). Nonetheless, a fasting blood glucose level and lipid profile, chemistry panel, and complete blood cell count are generally considered reasonable for the bariatric surgical patient. A pregnancy test should be obtained for all female patients of childbearing age. In patients at very low risk for heart and lung disease, routine chest radiography and electrocardiography add little information. On the basis of the high risk for development of micronutrient deficiencies after malabsorptive procedures, preoperative evaluation of iron status (iron, total iron-binding capacity, ferritin, serum transferrin receptor), vitamin B₁₂, 25-hydroxyvitamin D (25-OHD), and PTH should also be obtained. Preoperative micronutrient deficiencies have been described in bariatric surgery patients—14% to 43.9% have iron deficiency, 5% to 29% have vitamin B₁₂

deficiency, and 40% to 68.1% have vitamin D deficiency (388 [EL 3], 389 [EL 3]). Treatment for clinically significant deficiencies, such as iron deficiency anemia, should be initiated preoperatively. Although it seems prudent to screen all patients for metabolic bone disease after substantial weight loss, data are limited regarding preoperative screening. As with any patient, those patients at increased risk for osteoporosis should be screened with dual-energy x-ray absorptiometry.

Some physicians evaluate patients preoperatively with an esophagogastroduodenoscopy or UGI study to detect peptic ulcer disease, hiatal hernias, esophageal mucosal abnormalities related to gastroesophageal reflux, and the presence of *H pylori* infection (390 [EL 3]). The benefits with use of this approach have been described (391 [EL 3]). Some physicians recommend testing for *H pylori* antibody and treat patients with abnormal values (392 [EL 4]) because marginal ulceration is a late complication of RYGB. Whether prophylactic treatment lowers the incidence of bleeding and of marginal ulceration at the gastrojejunostomy after RYGB is not known. Routine UGI study and gallbladder ultrasonography are not recommended universally and are at the discretion of the surgeon. There is neither consensus nor data to guide the performance of cholecystectomy concomitantly with bariatric surgery, regardless of the technique (open versus laparoscopically). In practice, concurrent cholecystectomy is performed in about 28% of cases (38 [EL 3]).

In their evidence-based report, the European Association for Endoscopic Surgery recommended the following preoperative studies: standard laboratory testing, chest radiography, electrocardiography, spirometry, abdominal ultrasonography, and UGI endoscopy or a barium study (393 [EL 4]). In contrast, many surgeons consider most of these tests unnecessary in the asymptomatic patient, especially if a cholecystectomy will not be performed even if asymptomatic gallstones are seen. Obtaining polysomnography on all patients regardless of symptoms is currently controversial. Some physicians think that testing is indicated only if relevant symptoms are discovered during screening (393 [EL 4]).

9.3.4. Clinical Impression

The final impression and plan serve not only to inform the requesting practitioner but also to document the medical necessity and to provide a reference for other members of the bariatric surgery team. The following should be documented in the medical record: (1) the severity of obesity, (2) the duration of severe obesity, (3) whether or not the patient meets the accepted criteria for surgery, and (4) prior unsuccessful attempts at weight loss. The patient must understand the potential metabolic complications, such as anemia, metabolic bone disease, and electrolyte imbalance. Surgery does not guarantee a successful outcome, and the patient should be enrolled preoperatively in a comprehensive program for nutrition and lifestyle management.

Table 20
Laboratory and Diagnostic Evaluation of the Obese Patient
Based on Presentation of Symptoms, Risk Factors, and Index of Suspicion^a

Suspected condition	Studies to consider and interpretation
Obstructive sleep apnea (daytime sleepiness, loud snoring, gasping or choking episodes during sleep, and awakening headaches)	<ul style="list-style-type: none"> • Polysomnography for oxygen desaturation, apneic and hypopneic events • Measurement of neck circumference (>17 inches [>43.2 cm] in men, >16 inches [>40.6 cm] in women) • Otorhinolaryngologic examination for upper airway obstruction (optional)
Alveolar hypoventilation (pickwickian) syndrome (hypersomnolence, possible right-sided heart failure including elevated jugular venous pressure, hepatomegaly, and pedal edema)	<ul style="list-style-type: none"> • Polysomnography (to rule out obstructive sleep apnea) • Complete blood cell count (to rule out polycythemia) • Blood gases (PaO₂ decreased, PaCO₂ elevated) • Chest radiography (enlarged heart and elevated hemidiaphragms) • Electrocardiography (right atrial and right ventricular enlargement) • Pulmonary function tests (reduced vital capacity and expiratory reserve volume) (optional) • Right heart pressure measurement (optional)
Cushing syndrome (moon facies, thin skin that bruises easily, severe fatigue, violaceous striae)	<ul style="list-style-type: none"> • Elevated late-night salivary cortisol level (>7.0 nmol/L diagnostic, 3.0 to 7.0 nmol/L equivocal) • Repeatedly elevated measurements of cortisol secretion (urine free cortisol [upper normal, 110 to 138 nmol/d] or late-night salivary cortisol levels) may be needed
Diabetes mellitus	<ul style="list-style-type: none"> • Fasting blood glucose (\geq126 mg/dL on 2 occasions), random blood glucose (\geq200 mg/dL with symptoms of diabetes), or 120 minutes post-glucose challenge (\geq200 mg/dL) • Glycosylated hemoglobin (hemoglobin A1c) \geq7.1% • Microalbuminuria (>30 mg/d) at baseline • BP measurement and fasting lipid profile
Hypothyroidism	<ul style="list-style-type: none"> • Supersensitive TSH (> assay upper limit of normal range)
Metabolic syndrome	<p>3 of 5 criteria needed for diagnosis:</p> <ul style="list-style-type: none"> • Triglycerides >150 mg/dL • HDL cholesterol <40 mg/dL (men) or <50 mg/dL (women) • BP >130/>85 mm Hg • Fasting glucose >110 mg/dL • 120 minutes post-glucose challenge 140 to 200 mg/dL
Polycystic ovary syndrome (oligomenorrhea, hirsutism, probable obesity, enlarged ovaries may be palpable, hypercholesterolemia, impaired glucose tolerance, persistent acne, and androgenic alopecia)	<ul style="list-style-type: none"> • Morning blood specimen for total, free, and weak testosterone, DHEAS, prolactin, thyrotropin, and early-morning 17-hydroxyprogesterone level (normal values vary according to laboratory). Testing should be done OFF oral contraceptives (optional) • Lipid profile
Hypertension	<ul style="list-style-type: none"> • Mean of 2 or more properly measured seated BP readings on each of 2 or more office visits with use of a large BP cuff (prehypertension 120-139/80-89 mm Hg; hypertension 140-159/90-99 mm Hg) • Electrocardiography, urinalysis, complete blood cell count, blood chemistry, and fasting lipid profile
Liver abnormality, gallstones	<ul style="list-style-type: none"> • Liver function tests (serum bilirubin and alkaline phosphatase elevated) • Gallbladder ultrasonography (optional)
Hepatomegaly, nonalcoholic fatty liver disease	<ul style="list-style-type: none"> • Liver function tests elevated 1 to 4 times normal (ALT usually > AST, serum bilirubin, prothrombin time, decreased albumin) • Imaging study (ultrasonography or computed tomography) (optional) • Minimal or no alcohol intake with negative testing for viral hepatitis, autoimmune disease, and congenital liver disease • Definitive diagnosis with liver biopsy • Upper endoscopy to rule out esophageal varices if cirrhosis suspected

^a ALT = alanine aminotransferase; AST = aspartate aminotransferase; BP = blood pressure; DHEAS = dehydroepiandrosterone sulfate; HDL = high-density lipoprotein; TSH = thyroid-stimulating hormone.

Obesity-related medical conditions are summarized in the medical record, with particular emphasis on those comorbidities that are difficult to treat medically and have been shown to improve after bariatric surgery. Specific recommendations are then made concerning management of medical conditions preoperatively and postoperatively, as well as the disposition of the patient's current medications.

9.4. Choice of Bariatric Procedure

Pure gastric restriction procedures (LAGB, sleeve gastrectomy) are associated with fewer nutritional deficiencies postoperatively. Thus, there is less need for nutritional supplementation for pure gastric restriction procedures in comparison with procedures involving a malabsorptive component (161 [EL 1], 162 [EL 4], 394 [EL 2]). The BPD/DS may be associated with greater loss of excess weight than the RYGB (78 [EL 3], 117 [EL 4], 133 [EL 3], 386 [EL 3]). In comparison with RYGB, however, the BPD/DS is associated with (1) more nutritional deficiencies and therefore need for nutritional supplements, (2) more metabolic bone and stone disease and therefore need for closer monitoring, preventive medications, and procedures, (3) more nutritional anemia, and (4) higher surgical mortality (84 [EL 3], 125 [EL 3], 140 [EL 4], 207 [EL 3]). LAGB and laparoscopic RYGB, in comparison with open procedures, are associated with a shorter hospital stay, earlier resolution of pain, and improvement in quality of life without any additional morbidity or mortality (161 [EL 1], 162 [EL 4], 394 [EL 2]). Laparoscopic bariatric surgery is also associated with significantly fewer wound-related complications (wound infections, dehiscence, incisional hernias) in comparison with open procedures (162 [EL 4]). In contrast, however, laparoscopic operations are associated with a greater number of anastomotic strictures, internal hernias, and subsequent cholecystectomies than are open procedures (60 [EL 4], 61 [EL 3], 62 [EL 2], 163 [EL 2]). Weight loss and improvement in quality of life are equivalent between the approaches in long-term outcomes (100 [EL 3], 189 [EL 2]).

In their evidence-based evaluation, the European Association for Endoscopic Surgery concluded that the choice of bariatric procedure depends ultimately on individual factors, including BMI, perioperative risk, metabolic variables, comorbidities, surgeon competence, and other physician-patient preferences (393 [EL 4]). A similar algorithm was devised by Buchwald (395 [EL 4]) on the basis of a number of case series. For example, even a LAGB can induce significant weight loss with less risk in patients with a BMI of 50 to 100 kg/m² (208 [EL 2], 396 [EL 3], 397 [EL 3]); therefore, if risks of RYGB, BPD, or BPD/DS are excessive for an individual patient, the LAGB can be considered a good choice. T2DM appears to resolve more quickly, and is independent of weight loss, with RYGB, BPD, or BPD/DS than with LAGB. Other factors in the decision analyses are local experience by the

surgeon and institution with a specific procedure and insurance coverage.

9.4.1. Evidence Comparing RYGB With LAGB

The weight loss associated with RYGB is intermediate between a purely restrictive procedure and the BPD or BPD/DS (11 [EL 2], 64 [EL 3], 99 [EL 1]). According to a systematic review and meta-analysis of data from various bariatric procedures, BPD and banded RYGB procedures were associated with greater weight loss than were RYGB and LAGB; the latter 2 procedures were comparable at 3 to 7 years postoperatively (161 [EL 1]). The data demonstrating greater weight loss with RYGB over VBG are exemplified by the randomized, prospective trials of Sugeran et al (70 [EL 2]), Hall et al (398 [EL 3]), Howard et al (399 [EL 2]), MacLean et al (400 [EL 2]), and Sjöström et al (64 [EL 3]). These findings were supported by the matched-pair comparisons from a prospective collected database of 678 bariatric procedures, in which laparoscopic RYGB was associated with greater weight loss and fewer complications than LAGB (401 [EL 2]). A randomized, prospective trial showed that RYGB yielded a significantly greater loss of excess weight at 5 and 10 years postoperatively than did the LAGB (66.6% versus 47.5%, respectively; $P < .001$) (11 [EL 2]). The SOS Study, a prospective, nonrandomized but matched investigation, demonstrated greater weight loss for gastric bypass compared with gastric banding (nonadjustable and adjustable Swedish band) at 15 years postoperatively with 99.9% retention (27% versus 13% of initial body weight, respectively) (64 [EL 3], 65 [EL 3]). Specifically, follow-up of the prospective SOS Study found that at 1 to 2, 10, and 15 years postoperatively, weight losses stabilized at 32%, 25%, and 27% of initial weight for RYGB (N = 265), 25%, 16%, and 18% for VBG (N = 1,369), and 20%, 14%, and 13% for gastric banding (N = 376) (65 [EL 3]). This study was not sufficiently powered statistically to determine differences in mortality among the 3 surgical procedures. In contrast, in a retrospective study of 332 patients with BMI >50 kg/m², laparoscopic RYGB was associated with weight loss comparable to that with LAGB but at a price of greater morbidity (397 [EL 3]). In another retrospective study of 290 patients with BMI >50 kg/m², laparoscopic RYGB was associated with a significantly greater percentage loss of EBW but with increased early and late complication rates compared with LAGB (89 [EL 3]). In the retrospective studies by Jan et al (164 [EL 2], 402 [EL 2]), there was a greater percentage loss of EBW at 3 years and morbidity with the RYGB and a greater reoperation rate with the LAGB procedure. As procedural techniques evolved and incorporated more stapling and anastomoses, it is not surprising that the risk for postoperative complications increased.

On the basis of the clinical evidence, Sauerland et al (393 [EL 4]) concluded that the balance between complications and weight loss favored a LAGB in those patients with a BMI <40 kg/m², whereas RYGB was recommend-

ed in those patients with a BMI of 40 to 50 kg/m². This report, however, did not factor in the cost and increased risk of converting failed LAGB procedures to RYGB or the probable long-term effects of better control of diabetes achieved with RYGB. Another potential advantage for RYGB over LAGB would be in those patients with a greater number of obesity-related comorbidities, such as T2DM.

9.4.2. Evidence Regarding Risks and Benefits of BPD or BPD/DS

BPD and BPD/DS are complex hybrid surgical procedures with multiple suture lines and a mortality rate ranging from 0.4% to 2.0% attributable to PE, respiratory failure, and anastomotic leaks (82 [EL 3], 84 [EL 3], 209 [EL 3], 403 [EL 3]). In an “ad hoc stomach” type of BPD, with a 200-cm alimentary limb, a 50-cm common limb, and a 200- to 500-mL gastric volume (in which the stomach volume is adjusted according to the patient’s initial EBW, sex, age, eating habits, and anticipated adherence with postoperative instructions), the operative mortality was 0.4%, the early complication rate (wound dehiscence and infection) was 1.2%, and the late complication rate was 8.7% for incisional hernia and 1.2% for intestinal obstruction (207 [EL 3]). Closing mesenteric defects can reduce the incidence of internal hernias (393 [EL 4]). Other rates of complications associated with BPD include anemia in <5%, stomal ulcer in 3%, and protein malnutrition in 7%, with 2% requiring surgical revision by elongation of the length of the common limb or by restoration of normal gastrointestinal continuity (207 [EL 3]). Higher rates of complications after BPD were reported by Michielson et al (404 [EL 3]) and included diarrhea due to bacterial overgrowth (27%), wound infection (15%), incisional hernias (15%), peptic ulcers (15%), dumping syndrome (6%), and acute cholecystitis (6%). Liver function abnormalities may occur after BPD within the first few postoperative months as a result of malabsorption and can be treated with metronidazole and pancreatic enzymes (405 [EL 3], 406 [EL 3]). If these abnormalities persist, PN or surgical elongation of the common channel (or both)—or even reversal—may be necessary (209 [EL 3]). One study demonstrated that some patients with severe hepatopathy had improved liver histologic features, although others developed mild fibrosis after the BPD/DS (407 [EL 2]). Restriction of dietary fat may lessen the frequency of malodorous stools. Overall, quality of life is improved with BPD/DS, with rare occurrence of vomiting, >90% of patients eating whatever they desire, and 81.3% experiencing normal gastric emptying (209 [EL 3], 408 [EL 3]). Hypocalcemia and hypoalbuminemia occur less frequently after BPD/DS than after BPD (409 [EL 4]).

In one study, the mean operating time for laparoscopic hand-assisted BPD/DS was 201 minutes in conjunction with a median hospital stay of 3 days (range, 2 to 22), no deaths, but 7 conversions to open procedures, 14 reoperations, 21 readmissions, 3 PE, 2 DVT, and 4 perioperative

proximal anastomotic strictures (410 [EL 3]). Of note, no prospective randomized trials have compared BPD or BPD/DS with RYGB to date.

Brolin et al (190 [EL 2]) found that, compared with conventional RYGB, a long-limb RYGB (150-cm alimentary tract) yielded more weight loss in patients who were 200 lb (90.7 kg) or more overweight without additional metabolic complications or diarrhea. In the United States (411 [EL 4]), the BPD has been found to be associated with a much greater risk of severe protein-calorie malnutrition than in the series from Italy, which may be explained by a greater fat intake in American patients than in those from northern Italy. The BPD/DS has a lower risk of this complication in Canadian patients (84 [EL 3]). Overall, BPD procedures have been relegated to a less commonly used intervention, primarily attributable to reported risks in the literature.

9.4.3. Laparoscopic Versus Open Bariatric Surgery

Whenever possible—that is, when there is appropriate surgical and institutional expertise available—laparoscopic procedures should be selected over open procedures because of decreased postoperative complications (primarily wound-related), less postoperative pain, better cosmesis, and potentially shorter duration of hospital stay. This approach applies for VBG (56 [EL 2], 412 [EL 2]), LAGB (413 [EL 2]), RYGB (78 [EL 3], 189 [EL 2], 414-421 [EL 2-4]), and BPD/DS (422 [EL 3]). From 1999 to 2004, the percentage of laparoscopic bariatric procedures increased in one center from 10% to 90% (423 [EL 4]) owing to an increased use of bariatric surgery overall, improved technical skills and training, and the aforementioned positive clinical evidence.

9.5. Selection of Surgeon and Institution

In order to adhere to these guidelines, physicians faced with an appropriate candidate for a bariatric surgical procedure ought to be diligent in locating and communicating directly with an expert bariatric surgeon. In bariatric surgery, the complication rates associated with these procedures are linked to the experience of the surgeon; the critical threshold for minimizing complications occurs at approximately 100 to 250 operations (40 [EL 3], 424-426 [EL 3]). Moreover, the bariatric surgeon must be part of a comprehensive team that provides preoperative and postoperative care. In addition, the facility where the surgeon practices must have experience with bariatric patients and a familiarity with routine postoperative care. The Centers of Excellence initiative of the ASMBS and the ACS Bariatric Surgery Centers program offer prospective patients lists of programs that have met the foregoing criteria. Once a surgeon who meets these criteria has been identified, referrals should be made to that surgeon to improve a coordinated, perioperative care plan for future patients. Referring physicians should request specific experience and performance data from the bariatric surgeon regarding the procedure being considered. There are

various resources available to locate a suitable bariatric physician on the Internet or by contacting the Surgical Review Corporation, ASMBS, ACS, or TOS.

9.6. Preoperative Management

9.6.1. Endocrine

9.6.1.1. Type 2 diabetes mellitus

The bariatric specialist can expect to see many patients with T2DM, both diagnosed and undiagnosed. Although T2DM has been found to resolve in the overwhelming majority of patients after RYGB (67 [EL 3], 99 [EL 1], 129 [EL 3], 130 [EL 3], 139 [EL 3]), surgical stress can be associated with exacerbation of hyperglycemia in T2DM and “stress hyperglycemia” in nondiabetic patients. Moreover, after bariatric surgery, patients typically receive large volumes of dextrose-containing intravenous fluids and subsequently receive sucrose-containing liquid feedings. In general, achievement of preoperative glycemic control—hemoglobin A1c $\leq 7\%$, fasting blood glucose ≤ 110 mg/dL, and postprandial blood glucose ≤ 180 mg/dL—represents a realistic “best care” outcome (427 [EL 4], 428 [EL 3], 429 [EL 3]).

Preoperatively, diabetes control may be achieved by numerous measures. Medical nutrition therapy remains a cornerstone in the management of the patient with T2DM. Goals for glycemic control should follow the guidelines outlined by AACE (427 [EL 4]) and the American Diabetes Association (430 [EL 4]). Preoperative glycemic control represented by a hemoglobin A1c value $\leq 7\%$ has been associated with decreased perioperative infectious complications (427 [EL 4], 431 [EL 3]). Patients with poor glycemic control with use of orally administered medications or who require high doses of insulin preoperatively may require insulin for several days after bariatric surgery.

9.6.1.2. Thyroid

Although functional thyroid disorders are frequently associated with weight fluctuations, they are rarely the sole cause of severe obesity. Routine screening for abnormalities of thyroid function in all obese patients has not been supported by strong evidence. An increased incidence of clinical and subclinical hypothyroidism has been found among obese patients; thus, when thyroid disease is suspected, appropriate laboratory testing is indicated (432-434 [EL 3]). The best test for screening for thyroid dysfunction is an ultrasensitive thyroid-stimulating hormone assay (435 [EL 4]).

9.6.1.3. Lipids

Previously unrecognized lipid abnormalities may be identified and can strengthen the case for medical necessity for bariatric surgery. The only lipid abnormality that may necessitate immediate preoperative intervention is severe hypertriglyceridemia because serum triglyceride concentrations greater than 600 mg/dL are often associated with acute pancreatitis and the chylomicronemia syn-

drome. Lipid abnormalities should be treated according to the National Cholesterol Education Program Adult Treatment Panel III guidelines (436) [EL 4] (see <http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.htm>). Lipid-lowering therapy for LDL cholesterol and triglyceride values that remain above desired goals postoperatively should be continued. BPD and BPD/DS procedures have been associated with lower triglyceride and LDL values (99 [EL 1]). If target levels are reached postoperatively, doses of lipid-lowering agents can be reduced and even discontinued if target levels are maintained.

9.6.2. Cardiology and Hypertension

Current practice guidelines for perioperative cardiovascular evaluation for noncardiac surgical procedures should be used to guide preoperative assessment and management (437 [EL 4], 438 [EL 4]). As previously noted, obesity alone is not a risk factor for postoperative complications (386 [EL 3]); therefore, patients need not routinely undergo preoperative cardiac diagnostic testing. The challenge for the clinician before bariatric surgery is to identify the patient who is at increased perioperative cardiovascular risk, judiciously perform supplemental preoperative evaluations, and manage the perioperative risk. Several indices of risk and algorithms can be used as a guideline (437 [EL 4], 439 [EL 2]).

The patient with poor functional capacity, expressed as unable to meet 4-MET (metabolic equivalent) demand during most normal daily activities (such as climbing a flight of stairs, walking on level ground at 4 mph, or doing heavy work around the house), presents a particular challenge because it is important to distinguish between deconditioning with some expected dyspnea and underlying cardiac disease. Exercise capacity and cardiac risk factor analysis will determine whether formal testing beyond electrocardiography is required. An abdominal operation is an intermediate-risk procedure, and diabetes is an intermediate clinical predictor of cardiac risk. Poor exercise capacity may determine whether patients with intermediate predictors require pharmacologic stress testing. Testing considerations specific to patients with class 3 obesity include electrocardiographic changes related to chest wall thickness and lead placement, inability to increase physical activity to target (440 [EL 4]), and a body weight too heavy for the equipment. Similarly, both dual isotope scanning and dobutamine stress echocardiography may be challenging. In this population of patients with symptomatic angina, dobutamine stress echocardiography is a particularly useful diagnostic test because of its high sensitivity and specificity (441 [EL 1]), no need for treadmill running, and ability to image heart size and valves. Patients with known cardiac disease should have a cardiology consultation before bariatric surgery. Those patients who do not have active disease but are nonetheless at higher risk should be considered for prophylactic β -adrenergic blockade (442 [EL 1]). If CAD is documented with dual isotope scanning, these patients are often con-

sidered too obese to undergo either coronary artery bypass grafting or stent placement. Obese patients with clinically significant CAD should undergo aggressive medical weight loss with a very-low-calorie diet until they achieve a weight at which they can receive appropriate cardiac intervention.

Uncontrolled hypertension may increase the risk for perioperative ischemic events. Blood pressure levels >180/110 mm Hg should be controlled before bariatric surgery is performed. Because bariatric surgery is considered an elective operation, control should be achieved during a period of several days to weeks of outpatient treatment (443 [EL 4]).

9.6.3. Pulmonary and Sleep Apnea

Risk factors for postoperative pulmonary complications include chronic obstructive pulmonary disease, age greater than 60 years, functional dependence, OHS, congestive heart failure, and American Society of Anesthesiologists class II or greater (444 [EL 4]). Surgical risk factors pertinent to the bariatric patient include an abdominal surgical procedure and duration of operation >3 hours. Laparoscopic techniques may decrease the risk by causing less pain and disruption of diaphragmatic muscle activity and were found to be associated with improved postoperative pulmonary function (445 [EL 3], 446 [EL 4]). Although obesity is associated with abnormal respiratory function (for example, decreased lung volumes and reduced compliance), obesity alone has not been identified as a risk for increased postoperative pulmonary complications (447 [EL 1]). Available data are mixed regarding cigarette smoking, but patients should be advised to stop smoking at least 8 weeks before the elective operation in order to decrease the risk of pulmonary complications (448 [EL 3], 449 [EL 3]).

Even though preoperative chest radiographs and spirometry should not be used routinely for predicting risk, the extent of preoperative pulmonary evaluation varies by institution. Chest radiographs are often recommended for all patients, but the yield in patients without pulmonary signs or symptoms is very small. Routine preoperative chest radiographs are reasonable in all obese patients because of the increased risk of obesity-related pulmonary complications (393 [EL 4]). In patients in whom intrinsic lung disease is not suspected, routine arterial blood gas measurement and pulmonary function testing are not indicated (446 [EL 4]). Preoperative education in lung expansion maneuvers reduces pulmonary complications.

Obstructive sleep apnea may be present in as many as 50% of men with class 3 obesity. In general, women tend to develop OSA at a higher BMI than men. Loud snoring is suggestive, but symptoms generally are poor predictors of the apnea-hypopnea index. A presumptive diagnosis of OSA may be made on the basis of consideration of the following criteria: increased BMI, increased neck circumference, snoring, daytime hypersomnolence, and tonsillar

hypertrophy (387 [EL 4]). Because OSA is associated with airway characteristics that may predispose to difficulties in perioperative airway management, these patients should be referred for diagnostic polysomnography preoperatively and treated with nasal CPAP. In the absence of OSA or the OHS, routine performance of a sleep study may not be necessary because this will not alter care. For patients in whom OSA is diagnosed or suspected, postoperative cardiac and pulmonary monitoring, including continuous digital oximetry and use of CPAP postoperatively, is prudent. If prolonged apneas and hypoxemia are noted in patients without evidence of OSA preoperatively, such patients should be treated with nasal CPAP in the perioperative period.

9.6.4. Venous Thromboembolism

Obesity and general surgery are risk factors for venous thromboembolism. Thus, patients undergoing bariatric surgery are considered generally to be at moderate risk for lower extremity DVT and PE (450 [EL 4]). PE may be the first manifestation of venous thromboembolism and is the leading cause of mortality in experienced bariatric surgery centers (451 [EL 4]). Unfractionated heparin, 5,000 IU subcutaneously, or low-molecular-weight heparin therapy should be initiated shortly (within 30 to 120 minutes) before bariatric surgery and repeated every 8 to 12 hours postoperatively until the patient is fully mobile (452 [EL 4]). Alternatively, administration of heparin shortly after the operation as opposed to preoperatively may be associated with a lower risk of perioperative bleeding. Whether such patients benefit from a higher dose of low-molecular-weight heparin has not been determined. Most centers combine anticoagulant prophylaxis with mechanical methods of prophylaxis (for example, intermittent pneumatic lower extremity compression devices) to increase venous outflow or reduce stasis (or both) within the leg veins. Preoperative placement of a vena cava filter should also be considered for patients with a history of prior PE or DVT, although randomized trials to support this action are lacking (451 [EL 4], 453 [EL 4], 454 [EL 3], 455 [EL 3]).

9.6.5. Gastrointestinal

Undiagnosed gastrointestinal symptoms must be evaluated before bariatric surgery. It is commonplace for surgeons to perform a routine UGI study or endoscopy to screen for peptic ulcer disease before many other types of surgical procedures; however, this practice has been questioned for bariatric surgery (456 [EL 3]). Some programs now use an *H pylori* antibody as a screening procedure (392 [EL 4]). The incidence of *H pylori* seropositivity preoperatively ranges from 11% to 41% (389 [EL 3], 391 [EL 3], 457 [EL 2], 458 [EL 2], 459 [EL 3]), which supports a recommendation for preoperative screening, although Yang et al (457 [EL 2]) asserted that gastric ulcers after VBG or RYGB are due to the surgical procedure itself and not the *H pylori* infection. Patients with

positive results are empirically treated with proton pump inhibitors and antibiotics. Whether this treatment reduces the incidence of postoperative ulceration or is cost-effective has not been determined.

Many obese patients will have asymptomatic increases in serum alanine aminotransferase and aspartate aminotransferase levels. These changes are most commonly associated with NAFLD. At the time of bariatric surgery, 84% of morbidly obese subjects have steatosis on liver biopsy specimens (281 [EL 3]), whereas 20% and 8% have inflammation and fibrosis, respectively. Weight loss after LAGB, RYGB, BPD, or BPD/DS leads to regression of steatosis and inflammation, including decreased bridging fibrosis in some patients (148 [EL 3], 283 [EL 4], 285-293 [EL 2-3]). The clinical challenge is to determine which patients require additional evaluation before bariatric surgery. Gallstones, chronic hepatitis B or C, alcohol use, and potential side effects of medications (such as acetaminophen, nonsteroidal antiinflammatory drugs, and clopidogrel) are among some of the more common offenders. Patients with substantial increases in liver function test results (generally, 2 to 3 times the upper limit of normal) should be considered for additional testing by hepatobiliary ultrasonography or CT and a hepatitis screen preceding bariatric surgery (460 [EL 4]). Patients with advanced cirrhosis and increased portal pressures face major perioperative risks. Patients with mild to moderate cirrhosis may benefit from bariatric surgery and have an acceptable risk of complications (461 [EL 3]). If cirrhosis is suspected, preoperative endoscopy should be undertaken to rule out esophageal or gastric varices and portal hypertension gastropathy. Surgery-induced weight loss allows subsequent liver transplantation (288 [EL 3]). Alternatively, liver transplant patients may undergo successful bariatric surgery (462 [EL 3]).

9.6.6. Rheumatologic and Metabolic Bone Disease

Obese patients with a BMI >40 kg/m² are at greater risk for osteoarthritis, progression of arthritis, and gout, which can decrease with weight loss (322 [EL 4]). After bariatric surgery, hip and knee pain may diminish in conjunction with an increased exercise capacity (327 [EL 3], 329 [EL 3], 331 [EL 2], 463 [EL 3]). Moreover, serum uric acid levels decrease (464 [EL 3]). Nevertheless, if nonsteroidal antiinflammatory drugs are needed, cyclooxygenase-2 inhibitors should be used, although this recommendation has not been tested in bariatric surgery patients. Gout was found to be precipitated during weight loss after intestinal bypass (465 [EL 3]), just as a surgical procedure itself is a risk factor for an acute gout attack. Therefore, patients with frequent attacks of gout should have prophylactic therapy started well in advance of bariatric surgery to lessen the chance of acute gout immediately postoperatively.

Obese persons have higher bone mass despite the common presence of secondary hyperparathyroidism due to vitamin D deficiency (466 [EL 3], 467 [EL 3]). The

increased PTH levels are positively correlated with BMI in obese patients owing to (1) decreased exposure to sunlight with a more sedentary lifestyle (468 [EL 3]) or (2) PTH resistance of bone due to increased skeletal mass (467 [EL 3]) or both of these factors. The frequency of secondary hyperparathyroidism preoperatively is approximately 25% (467 [EL 3]). Typically, there are decreased serum levels of 25-OHD in conjunction with normal or increased 1,25-dihydroxyvitamin D [1,25-(OH)₂D] because of the compensatory stimulatory effect of PTH on renal 1 α -hydroxylase activity (467 [EL 3]).

Preoperative dual-energy x-ray absorptiometry of the lumbar spine and hip should be performed in all estrogen-deficient women as well as in premenopausal women and men with conditions associated with low bone mass or bone loss (469 [EL 4]). The incidence of low bone mass in *obese* men and *obese* premenopausal women without risk factors, however, may be sufficiently low as to militate against baseline bone densitometry in these persons. A thorough work-up for secondary causes of low bone mass is imperative for such patients before bariatric surgery. Thus, preoperative biochemical screening with intact PTH, vitamin D metabolites, and markers of bone metabolism may be helpful in patients at increased risk for metabolic bone disease.

9.7. Final Clearance

Final clearance to proceed with bariatric surgery is usually provided by the surgeon who will perform the surgical procedure. At this time, medical need has been established, no medical or psychologic contraindications have been identified, medical comorbidities are well controlled, and the patient has expressed good understanding and commitment to the intervention planned.

9.8. Financial Evaluation

It is generally recommended that patients learn about their insurance coverage for bariatric procedures by contacting their third-party payers before pursuing bariatric surgery. This important factor is to ensure that they are well informed regarding what services are covered and what requirements exist for approval that may be unique to their provider. Most bariatric programs are well informed regarding specific requirements of most major providers; however, this is a fast changing field. For most third-party payers, prior authorization is required. Medicare is the only provider that will not grant prior authorization for bariatric surgery but will base its decision to cover the services on medical necessity at institutions designated as Centers of Excellence by the Surgical Review Corporation or the ACS. Medicare has decided in a National Coverage Decision that bariatric surgery is appropriate for beneficiaries who have a BMI of ≥ 35 kg/m² and a comorbidity, without an age limit, if previous attempts at nutritional management have failed. This information is available online at <http://www.cms.hhs.gov/mcd/viewdecisionmemo.asp?id=160>.

Requesting prior written authorization for bariatric surgery necessitates the fulfillment of several criteria. Most third-party payers require the following:

- A letter stating current height, weight, and BMI
- Documentation of medical necessity, outlining weight-related comorbidities present
- Clinical documentation from a registered dietitian, primary care provider, or medical subspecialist, psychologist or psychiatrist, and surgeon indicating the absence of contraindications to bariatric surgery, and signed informed consent regarding the risks and benefits of the procedure planned
- Documentation of previous weight-loss attempts

In comparison with previously, many third-party payers are currently requiring more detailed documentation of previous weight-loss efforts with required medical supervision and detailed weigh-ins. Others require participation in a 6- to 12-month medically supervised weight-loss program before consideration. There are no published studies supporting the value of this approach, and one study noted an increased dropout rate among patients in whom a 6-month period of physician-directed nutritional management was required by a health insurance carrier (52 [EL 3]). It is important to encourage patients who are seeking bariatric surgery to become well informed regarding the requirements of their individual insurance policy to avoid misunderstandings or unnecessary delays.

9.9. Early Postoperative Management

The management of the obese patient after bariatric surgery can present numerous challenges. It involves the prevention of and monitoring for postoperative complications, management of preexisting medical conditions, and guidance of patients through the transition of life after bariatric surgery.

Improved expertise in the perioperative management of the obese patient has allowed centers to transfer services previously available only in an ICU to less critical settings. No consensus exists about which type of patient should be considered for admission to the ICU after bariatric surgery (392 [EL 4], 470 [EL 4], 471 [EL 4]).

9.9.1. Monitoring for Surgical Complications

Anastomotic leak is a potentially fatal complication after bariatric surgery. It is reported to occur in up to 5% of RYGB procedures, but recent attempts to identify and correct leaks intraoperatively have been shown to reduce the postoperative incidence to 0% (472-477 [EL 2-3]). Symptoms can be subtle and difficult to distinguish from other postoperative complications such as PE. A high degree of suspicion is necessary. Tachycardia (pulse rate greater than 120 beats/min) in the setting of new or worsening abdominal symptoms should prompt immediate evaluation. Another clue may be an increasing ratio of blood urea nitrogen to creatinine in the absence of oli-

guria. Left shoulder pain may be a worrisome symptom after RYGB, BPD, or BPD/DS. If an anastomotic leak is unrecognized, oliguria, sepsis with multiorgan failure, and death can ensue (108 [EL 4], 191 [EL 2], 453 [EL 4], 470 [EL 4], 471 [EL 4]).

Evaluation is guided by the clinical presentation. If the patient is clinically stable, radiologic tests such as Gastrografin studies can be performed, although they are helpful only if a leak is identified. Indeed, the false-negative rate exceeds 4%. For identification of leaks from the excluded stomach, CT scanning may be preferable to UGI contrast swallow studies. In the setting of a negative study but a high index of suspicion or a clinically unstable patient, exploratory laparoscopy or laparotomy is indicated (19 [EL 4], 453 [EL 4], 470 [EL 4], 471 [EL 4], 478 [EL 3], 479 [EL 3]). Several authors have proposed performing a limited UGI contrast study to examine the anastomosis and identify subclinical leaks before discharge of the patient from the hospital, but this practice is not universally accepted and does not appear to be cost-effective (471 [EL 4], 480 [EL 4], 481 [EL 3]). Identification of a leak usually necessitates emergent surgical reexploration, either laparoscopically or with an open procedure. Occasionally, a patient can be managed expectantly or with percutaneous drainage; however, the signs and symptoms of sepsis must resolve promptly and completely, and there should be a low threshold for reexploration.

Wound complications after open bariatric surgery are common, and their incidence is significantly diminished by a laparoscopic approach (156 [EL 4], 453 [EL 4]). Procedures with large vertical incisions are associated with a high incidence of seromas (156 [EL 4], 392 [EL 4]). Although seromas often drain spontaneously, removal of excess fluid is recommended to decrease the risks for major wound infections (156 [EL 4], 392 [EL 4], 480 [EL 4]). For treatment of wound infections, aggressive management, with incision and drainage and orally administered antibiotics, is important. Partial opening of the incision in several locations is usually necessary for adequate drainage of a subcutaneous infection. Efforts should be made to avoid opening the entire incision because healing may require several months. In contrast, if segmental drainage is ineffective, then that approach must be abandoned. Healing occurs by secondary intention and can often take weeks to months (392 [EL 4], 480 [EL 4]). As in all abdominal surgical procedures, the patient should be given a broad-spectrum cephalosporin immediately before the incision and continued for up to 24 hours postoperatively (157 [EL 4], 482 [EL 4]). Wound dehiscence occurs most frequently in the setting of a wound or subcutaneous infection; however, the increased tension exerted on the wound by excess weight can itself lead to dehiscence. Some surgeons have modified their approach at reinforcing suture lines to avoid this complication (392 [EL 4]). Major wound infections are extremely rare with laparoscopic procedures (57 [EL 3], 62 [EL 2], 189 [EL 2], 419 [EL 3]).

Prophylactic antibiotics that cover skin organisms should be administered at the time of LAGB to prevent wound infection at the adjustment port site. Any hint of infection at this site necessitates immediate and aggressive treatment so that sepsis, reservoir removal, and peritoneal cavity adjustment tubing placement can be avoided. The band itself would not need to be removed. When sepsis has resolved, the tubing can be recovered laparoscopically, and a new adjustment reservoir can be attached.

9.9.2. Type 2 Diabetes Mellitus

Patients requiring insulin before bariatric surgery should have their blood glucose concentrations monitored regularly and insulin administered to control significant hyperglycemia. In the ICU, euglycemia can be maintained with a nurse-driven, dynamic intensive insulin therapy protocol targeting a blood glucose level of 80 to 110 mg/dL (483 [EL 1], 484 [EL 1]). In non-ICU patients, target glycemic control is accomplished with subcutaneously administered insulin: “basal” insulinization with intermediate-acting NPH insulin or long-acting insulin glargine or insulin detemir; “bolus” preprandial insulinization with rapid-acting insulin aspart, glulisine, or lispro; and “correction” insulin every 3 to 6 hours also with a rapid-acting insulin (485 [EL 2]). In the non-ICU setting, the evidence for target blood glucose values of <80 to 110 mg/dL (preprandially) and <180 mg/dL peak (postprandially) is provided in the 2007 AACE Medical Guidelines for Clinical Practice for the Management of Diabetes Mellitus (427 [EL 4]) and the 2004 American College of Endocrinology Position Statement on Inpatient Diabetes and Metabolic Control (486 [EL 4]).

The surgeon and floor nurses should be familiar with glycemic targets and subcutaneous insulin protocols as well as the use of dextrose-free intravenous fluids and low-sugar liquid supplements. Parameters for initiating intravenous insulin therapy and requesting an endocrine consultation should be explicitly discussed. Because of the risks of stress hyperglycemia, prediabetic patients (those patients with fasting blood glucose levels of 100 to 125 mg/dL or 2-hour post-oral glucose challenge blood glucose levels of 140 to 199 mg/dL) and even patients without any evidence of impaired glucose regulation should be treated with the same insulin protocols as those with established T2DM.

Patients should be instructed in regular monitoring of metered blood glucose concentrations to guide adjustments in glucose-lowering therapy. In patients with persistent hyperglycemia, continued surveillance and preventive care as recommended by AACE (427 [EL 4]) and the American Diabetes Association (430 [EL 4]) are advised. If euglycemia is achieved, it is unclear whether current recommendations for the preventive care of patients with T2DM should be continued.

It is important to note that a small number of patients with type 1 diabetes mellitus (T1DM) are obese and require insulin for survival. In those who present for

bariatric surgery, the diagnosis of T1DM versus T2DM may not be entirely clear at the time of surgery. Therefore, it is recommended that insulin be withdrawn cautiously and that blood glucose concentrations be reported to a health-care provider at frequent intervals after discharge of the patient from the hospital so that adjustments can be made in the event of inadequate blood glucose control (487 [EL 4]). Obese patients with T1DM will also have a decrease in insulin requirements after bariatric surgery. It needs to be emphasized that all patients with T1DM must have insulin onboard at all times to prevent diabetic ketoacidosis. Accordingly, intermediate- or long-acting insulin should be dosed even when patients with T1DM are not receiving any dextrose or nutrition.

9.9.3. Cardiology and Hypertension

Despite the increased prevalence of cardiac risk factors in patients undergoing bariatric surgery, the incidence of cardiac ischemic events is surprisingly low. This disparity is primarily attributed to the relatively young age of patients who undergo this intervention—overall mean age of 37.5 ± 0.63 years (488 [EL 3]) and for the 57% with at least one metabolic CAD risk factor (489 [EL 3]), mean age of 38.1 to 40.3 years. Several investigators have reported significant improvements of various cardiovascular risk factors after bariatric surgical procedures (104 [EL 3], 140 [EL 4], 248 [EL 3], 252 [EL 2], 254 [EL 3], 258 [EL 3], 490-493 [EL 3]). Patients with known CAD and low perioperative risks on the basis of the Goldman cardiac risk index who have undergone bariatric surgery do not experience an increase in mortality when compared with obese adults without a history of CAD. A trend toward increased cardiac events (3 versus 0) was noted but did not reach statistical significance (494 [EL 3]). Diastolic dysfunction may be present as a result of myocardial hypertrophy, decreased compliance, and increased systemic arterial pressure from obesity and can increase the risks for perioperative complications (266 [EL 2], 470 [EL 4], 495 [EL 4], 496 [EL 3]). In 7 published reports, the mortality for patients who had undergone bariatric surgery was significantly less than for patients who had not lost weight. Moreover, several reports have noted a significant decrease in mortality after bariatric surgery in comparison with that in matched non-surgical cohorts, which has been related to a significant decrease in deaths related to MI as well as a decrease in deaths from cancer and diabetes (39 [EL 3], 65 [EL 3], 151-153 [EL 3], 154 [EL 2], 155 [EL 3]).

Patients with known or presumed CAD may be managed best in an ICU setting for the first 24 to 48 hours after bariatric surgery (392 [EL 4], 470 [EL 4], 471 [EL 4]). Medications used in the management of CAD or hypertension can be administered parenterally while the patient remains without oral intake. This approach is especially important for β -adrenergic blocking agents because their abrupt discontinuation can be associated with increased risks for cardiac complications. If a patient is not taking a

β -adrenergic blocking agent, therapy with this medication can often be initiated in an attempt to provide additional cardioprotection perioperatively (437 [EL 4]).

Initiation of orally administered medications for the management of CAD and hypertension should be pursued as soon as the patient is able to tolerate liquids orally. Changes in drug preparations may be required, particularly in patients with gastric restrictive procedures, as a result of intolerance of tablets. The majority of patients who received antihypertensive medications before bariatric surgery will still require them at discharge from the hospital for adequate control of blood pressure values. Diuretic agents should be either discontinued or reduced to avoid dehydration and electrolyte abnormalities during the first month or 2 postoperatively; they can be initiated again if hypertension persists.

9.9.4. Pulmonary

Obesity increases the risk for respiratory complications after an abdominal operation: pneumonia, atelectasis, respiratory failure (intubation beyond 24 hours postoperatively or reintubation), and PE. These complications constitute the most serious nonsurgical perioperative events. They are more frequent than cardiac complications in patients undergoing abdominal surgical procedures (497 [EL 2]), and all efforts must be made to identify and minimize the risk (498 [EL 3], 499 [EL 4], 500 [EL 4]). Prolonged mechanical ventilation with an extended weaning period may be necessary for patients with OHS, a situation that increases the risks for aspiration and pneumonia. Hypoxemia and apneic episodes are frequently observed in the sedated patient with or without a preexisting diagnosis of OSA or OHS. Atelectasis remains a common cause of fever and tachycardia during the first 24 hours after bariatric surgery (453 [EL 4], 471 [EL 4]).

Pulmonary management after bariatric surgery includes aggressive pulmonary toilet and incentive spirometry for the prevention of atelectasis (470 [EL 4], 501 [EL 3]). Oxygen supplementation and early institution of nasal CPAP improve respiratory function in this patient population (498 [EL 3], 502 [EL 2]). A review of the literature suggests that nasal CPAP can be used safely after RYGB in patients with sleep apnea without increasing the risk of a postoperative anastomotic leak; in contrast, the use of bilevel positive airway pressure may increase the risk of anastomotic leaks (503 [EL 3]).

Respiratory distress or failure to wean from ventilator support should alert the physician to the possibility of an acute postoperative complication, such as a PE or an anastomotic leak. A high level of suspicion is critical because symptoms may be subtle, such as new-onset tachycardia or tachypnea (453 [EL 4], 470 [EL 4], 471 [EL 4], 504 [EL 4]).

The incidence of PE in patients who have undergone bariatric surgical procedures has been reported as 0.1% to 2% (453 [EL 4], 470 [EL 4], 471 [EL 4], 504 [EL 4]).

After bariatric surgery, PE is one of the most common causes of mortality (39 [EL 3], 505 [EL 2]). Obesity, lower extremity venous stasis, high pulmonary artery pressures, hypercoagulation, and immobilization contribute to the increased risk observed in this patient population (453 [EL 4], 470 [EL 4], 471 [EL 4], 504 [EL 4]). The use of thin-cut, thoracic spiral CT has improved the diagnosis of PE because of superior sensitivity and specificity in comparison with ventilation-perfusion scans (506 [EL 4]).

Treatment of PE after bariatric surgery should follow currently accepted guidelines. Thrombolytic agents should be avoided during the first 10 to 14 days postoperatively. Anticoagulation can be pursued within days after surgery, with rapid achievement of therapeutic levels within 24 hours after initiation of therapy (453 [EL 4], 470 [EL 4], 471 [EL 4], 504 [EL 4]).

Prophylaxis against DVT is an important component of the perioperative management after bariatric surgery; however, there is no consensus about a specific regimen. Most accepted regimens include a combination of sequential compression devices and subcutaneously administered unfractionated heparin or low-molecular-weight heparin before and after bariatric surgery (507 [EL 3], 508 [EL 2]). Clear evidence that preoperative heparin therapy is superior to postoperative administration is lacking. Early ambulation remains important in the prevention of DVT (453 [EL 4], 470 [EL 4], 471 [EL 4], 509 [EL 3]). Prophylactic placement of an inferior vena cava filter has been proposed, but not universally accepted, for the subgroup of patients with high mortality risk after PE or DVT, patients with known elevated pulmonary artery pressures >40 mm Hg, or those with hypercoagulable states (240 [EL 3], 453 [EL 4], 455 [EL 3], 504 [EL 4], 510 [EL 3]).

9.9.5. Fluids and Electrolytes

The management of perioperative fluid and electrolytes follows currently accepted practices. Such management should be modified on the basis of an individual patient's medical history. Current practices monitor urine output, attempt to maintain a urine output of 30 mL/h or 240 mL per 8-hour shift, and avoid volume overload. Renal failure can occur after bariatric surgery if patients have received inadequate volume replacement. Perioperative fluid requirements for patients after bariatric surgery are substantially greater than for their nonobese counterparts.

9.9.6. Anemia

Decreases in hemoglobin in the early postoperative period are not uncommon. In the absence of any nutritional deficiencies (iron, folate, vitamin B₁₂), these decreased hemoglobin values should resolve by 12 weeks postoperatively. Persistent abnormalities should prompt further evaluation to identify potential complications, such as nutritional deficiencies or unrecognized blood loss.

9.9.7. Rhabdomyolysis

Nonphysiologic surgical positioning during laparoscopic bariatric surgery has been associated with rhabdomyolysis. This condition is thought to be due to the presence of certain risk factors, such as prolonged muscle compression, muscle-compartment syndrome, and crush syndrome in superobese patients with a long duration of the operation, especially when they have peripheral vascular disease, diabetes, or hypertension. Mognol et al (511 [EL 3]) screened 66 consecutive patients undergoing LAGB or laparoscopic RYGB with CK levels on postoperative days 1 and 3. They found a 23% incidence of chemical rhabdomyolysis (CK >1,050 U/L)—in 3 of 50 patients (6%) with gastric banding and in 12 of 16 (75%) with gastric bypass ($P<.01$) (511 [EL 3]). Clinically significant rhabdomyolysis, however, rarely occurs; no patient in this series had acute renal failure. Thus, prophylactic measures in high-risk patients include the following: (1) use of a staged procedure with shorter operative times (for example, sleeve gastrectomy as a first stage in patients with superobesity or super-superobesity), (2) adequate padding at all pressure points, (3) postoperative screening with CK levels on days 1 and 3, and (4) aggressive fluid replacement (76 [EL 3], 512 [EL 3]). At the present time, there are insufficient data to recommend CK screening routinely or even prophylactic measures in high-risk patients, although such measures may be considered on a case-by-case basis by the bariatric surgery team. If rhabdomyolysis is suspected, however, then CK levels should be assessed.

9.9.8. Oral Nutrition

After LAGB, patients should sip fluids when fully awake and can be discharged from the hospital only if satisfactorily tolerating fluids orally. Occasionally, edema and tissue within a recently placed adjustable gastric band cause obstruction. This problem usually resolves spontaneously during a period of days, but continued intravenous administration of fluids is needed. Currently, however, this problem is rare because several sizes of bands are available and the surgeon can choose an appropriate size at the time of surgical intervention.

Historically, a nasogastric tube has been placed after open RYGB (but not after laparoscopic procedures) and has been removed on the first or second postoperative day. This practice, however, has been demonstrated to be unnecessary (513 [EL 3], 514 [EL 3]). Oral intake with ice chips and sips of water is generally started after removal of the nasogastric tube. Once tolerated, clear liquids are started, and intravenous administration of fluids is discontinued. Clear liquids are usually begun the morning after all bariatric surgical procedures. Although most centers have individual protocols for meal progression after bariatric surgery, particularly after gastric restrictive procedures, most centers follow the same general guidelines, which involve gradual progression of food consistencies over weeks and months (392 [EL 4], 471 [EL 4], 515 [EL

3]). Gradual progression of food consistencies allows the patient to adjust to a restrictive meal plan and minimizes vomiting, which can threaten the integrity of the anastomosis (392 [EL 4]).

9.10. Late Postoperative Management

Continuity of care after bariatric surgery is vital to ensure long-term success (392 [EL 4], 516 [EL 3]). This continuity serves to monitor weight loss, assess the status of preexisting medical conditions, monitor for surgical and nutritional complications, and provide guidance and support as patients pursue lifestyle changes. Many patients have maladaptive eating behaviors, nutritional deficiencies, or other nutritional inadequacies preoperatively, which may persist after a bariatric procedure. Some patients who underwent VBG were noted to develop maladaptive eating behaviors because sweets and ice cream would pass through their restriction without difficulty. For patients with the LAGB procedure, this challenge can be managed by adjusting the band along with continued nutritional counseling (187 [EL 2], 517 [EL 3]). In a review of prospectively collected data involving patients undergoing a LAGB procedure, EBW loss was comparable between those who ate sweets and those who did not (165 [EL 3]). In general, the bariatric surgery patient should adhere to recommendations for a healthful lifestyle, including increased consumption of fresh fruits and vegetables, limitation of foods high in saturated fats, reduction of stress, and participation in exercise 30 minutes a day or more to achieve optimal body weight. Increased physical activity was found to improve body composition in bariatric surgery patients, as measured by bioelectrical impedance analysis (518 [EL 3]).

Knowledge and experience are needed for appropriate LAGB adjustment. During the first postoperative year, regular consultations for advice and adjustments are critical in providing good weight loss (519 [EL 3]). Patients need to have follow-up visits every 2 to 4 weeks until a satisfactorily stable optimal zone adjustment level is achieved (520 [EL 4], 521 [EL 3]). Patients with LAGB should have follow-up examinations every year indefinitely. There is a slow diffusion from all bands, which will cause a gradual reduction in restriction over many months to years (522 [EL 3]). Adjustments for special circumstances, including major surgical procedures, intercurrent illness, pregnancy, and remote travel, can be beneficial (306 [EL 2], 520 [EL 4]).

The frequency of follow-up visits proposed varies among surgeons. Most surgeons agree on the need for frequent visits during the first year after bariatric surgery when rapid changes are occurring, usually within 2 weeks after surgery, at 6 months, and at 12 months postoperatively. After the first year, despite the absence of clinically evident complications, annual follow-up visits should always be encouraged, even after intestinal adaptation has occurred (140 [EL 4], 392 [EL 4]). The outcomes that should be evaluated routinely include initial weight loss,

maintenance of weight loss, nutritional status including micronutrient blood levels, comorbidities, and psychosocial status (393 [EL 4]).

Typically, the perioperative management of the bariatric surgery patient is multidisciplinary, and patients can be overwhelmed with the magnitude of postoperative follow-up visits and the number of physicians including covering physicians as well as physician-extenders and nutritionists. The bariatric surgeon, the obesity specialist, and the team's registered dietitian generally function as the primary caregivers postoperatively. Patients may need regular follow-up with various consultants for active problems, but these visits should be monitored and coordinated by the primary team to avoid excessive ordering of tests. Mental health professionals should be available to help patients adjust to the myriad of psychosocial changes they experience postoperatively. Some published data show an increased risk of suicide after RYGB, BPD, or BPD/DS (154 [EL 2]). Depression can diminish during the first year after LAGB-induced weight loss (523 [EL 3]). Regardless of the bariatric procedure, psychiatric counseling can benefit all bariatric surgery patients.

Late surgical complications include anastomotic stricture, staple-line dehiscence, pouch dilation, internal hernia in conjunction with intestinal obstruction (complete or partial), anastomotic leaks, and incisional hernias (10% to 20%) (453 [EL 4], 480 [EL 4]). An internal hernia after RYGB, BPD, or BPD/DS is a potentially fatal complication attributable to bowel infarction and peritonitis. The symptoms are those of a small bowel obstruction with cramping pain, usually periumbilical. An internal hernia can occur at 3 locations: at the jejunojejunostomy, through the mesocolon, or between the Roux limb mesentery, the mesocolon, and the retroperitoneum (Petersen hernia). Diagnosis may be obtained with a Gastrografin UGI study or abdominal CT; however, as with a leak, these studies are often misleading (453 [EL 4]). The best course of management is often an exploratory laparotomy or laparoscopy for recurrent cramping abdominal pain.

The restrictive component of gastric bypass surgery involves partitioning of the stomach to create a small reservoir. In-continuity RYGB without transection has been associated with staple-line failure (524 [EL 3], 525 [EL 3]) and a stomal ulceration rate of up to 16% (525 [EL 3], 526 [EL 3]). Staple-line disruption and gastrogastic fistulas can also occur after gastric transection and increase the risk of marginal ulceration (525 [EL 3], 527 [EL 3]). More recent stapling techniques, however, only rarely result in staple-line failure, although no clear guidance regarding the optimal stapling method is available.

9.10.1. Goals for Weight Loss and Nutritional Prescription

The methods for reporting weight outcomes have varied over the years. Currently, changes in BMI, weight loss as percent of EBW, and weight loss as percent of initial weight are the most common methods. There is no con-

sensus on the definition of minimal weight loss to justify the operative risk, nor is there consensus on the minimal duration of maintenance of weight loss. Some investigators have defined success after bariatric surgery as the loss of at least 50% of EBW (528 [EL 4], 529 [EL 4]). Most agree that clinically useful weight loss outcomes should be reported with a minimal follow-up of 3 to 5 years and with at least 80% retention (continued follow-up). Most surgical procedures performed today, with the exception of some restrictive operations, have been reported to lead to this degree of weight loss in a majority of patients (70 [EL 2], 89 [EL 3], 122 [EL 3], 156 [EL 4], 164 [EL 2], 192 [EL 2], 392 [EL 4], 398 [EL 3], 504 [EL 4], 527 [EL 3], 530-532 [EL 2], 533 [EL 4], 534-536 [EL 2]). Malabsorptive procedures, such as the long-limb or very, very long-limb RYGB and the BPD or BPD/DS, have yielded the greatest percentage of weight loss reported (72 [EL 3], 207 [EL 3], 533 [EL 4], 537-539 [EL 3]). Success, however, should probably be related to factors other than mere weight loss, such as improvement or resolution of comorbidities, decreased mortality, enhanced quality of life, and positive psychosocial changes.

Weight loss after bariatric surgery can be dramatic. The fastest rate of weight loss occurs during the first 3 months postoperatively, when dietary intake remains very restrictive (70 [EL 2], 156 [EL 4], 515 [EL 3], 540 [EL 4], 541 [EL 4]). After malabsorptive procedures, patients can lose 0.5 to 1 lb (0.23 to 0.45 kg) per day or 40 to 90 lb (18 to 40.5 kg) by 3 months postoperatively. This rapid weight loss decreases by 6 to 9 months after bariatric surgery, and the peak in weight loss is achieved at 12 to 18 months after the procedure (64 [EL 3], 156 [EL 4], 392 [EL 4]). After LAGB, a weight loss of 2.5 lb (1.13 kg) per week is advised. Hypometabolism is common during the first 6 months after bariatric surgery. Cold intolerance, hair loss, and fatigue are common complaints, which tend to diminish as weight loss stabilizes. Reassurance and support are often all that is necessary.

Inadequate weight loss after bariatric surgery may be observed after nonadjustable gastric restriction procedures (namely, VBG) attributable to loss of integrity of the gastric remnant and development of maladaptive eating behaviors (increased caloric intake or increased consumption of calorically dense foods) (48 [EL 4], 117 [EL 4], 341 [EL 3], 504 [EL 4], 517 [EL 3], 542 [EL 4]). Clinical assessment then involves (1) evaluation of current eating practices, (2) psychologic evaluation, and (3) imaging studies of the UGI tract (471 [EL 4], 543 [EL 3]).

Some recidivism is also observed 3 to 5 years after RYGB, although long-term weight maintenance is greater than that reported with purely gastric restrictive procedures (64 [EL 3], 392 [EL 4], 504 [EL 4], 517 [EL 3], 527 [EL 3]). Contributing factors to weight regain after RYGB have not been well studied but are influenced by the decrease in frequency of dumping symptoms, resolution of food intolerances, and return to preoperative eating and other lifestyle patterns that originally contributed to the

development of obesity (341 [EL 3], 471 [EL 4], 504 [EL 4], 541 [EL 4]). Reported weight maintenance after BPD or BPD/DS appears to be superior to that after gastric restrictive procedures and RYGB because weight loss is predominantly attributable to malabsorption and not caloric restriction; however, this observation has never been subjected to a randomized, prospective trial (207 [EL 3], 210 [EL 3]).

9.10.2. Routine Metabolic and Nutritional Management

9.10.2.1. General statements

The extent of metabolic and nutritional evaluation completed after bariatric surgery should be guided by the type of surgical procedure performed. Purely gastric restrictive procedures are not associated with alterations in intestinal continuity and do not alter normal digestive physiologic processes. As a result, selective nutritional deficiencies are uncommon. The anatomic changes imposed by malabsorptive surgical procedures increase the risk for various nutrient deficiencies, which can occur commonly within the first year postoperatively (210 [EL 3], 389 [EL 3], 471 [EL 4], 544-547 [EL 3], 548 [EL 1]). Routine laboratory surveillance for nutritional deficiencies is recommended after LAGB, RYGB, BPD, or BPD/DS procedures (Table 13), even in the absence of caloric or nutritional restriction, vomiting, or diarrhea.

For surgical procedures with a gastric restrictive component, regular visits with a registered dietitian provide guidance as the meal plan is progressed. The limited volume capacity of the gastric pouch (30 to 60 mL) results in substantial restrictions in the amount of food consumed and the rate at which food can be eaten (515 [EL 3], 541 [EL 4]). During the first few months after bariatric surgery, episodes of regurgitation, typically without nausea or true vomiting, are common if food is consumed in large volumes, eaten too quickly, or not chewed thoroughly. Gastric dumping occurs initially in 70% to 76% of patients who have had a RYGB (70 [EL 2], 122 [EL 3], 341 [EL 3], 548 [EL 1], 549 [EL 3]). Nevertheless, the frequency of clinically troublesome complaints is unknown. Some reports suggest that the dumping syndrome may not occur in all patients or may occur only transiently during the first postoperative year (341 [EL 3]). For some patients, dumping may be considered a desired side effect because it discourages ingestion of calorically dense liquids that could minimize the loss of weight. A previous opinion was that dumping symptoms were the result of the hyperosmolarity of intestinal contents, which led to an influx of fluid into the intestinal lumen with subsequent intestinal distention, fluid sequestration in the intestinal lumen, decreased intravascular volume, and hypotension. More recent data suggest that food bypassing the stomach and entering the small intestine leads to the release of gut peptides that are responsible for these “dumping” symptoms, inasmuch as these symptoms can often be blocked by subcutaneous administration of somatostatin (550 [EL 3]). Abdominal pain and cramping,

nausea, diarrhea, light-headedness, flushing, tachycardia, and syncope—symptoms indicative of dumping—are reported frequently and serve to discourage the intake of energy-dense foods and beverages (70 [EL 2], 193 [EL 2], 541 [EL 4]). These symptoms tend to become less prominent with time (541 [EL 4]). Symptoms can usually be controlled with certain nutritional changes, such as (1) eating small, frequent meals, (2) avoiding ingestion of liquids within 30 minutes of a solid-food meal, (3) avoiding simple sugars and increasing intake of fiber and complex carbohydrates, and (4) increasing protein intake (551 [EL 4]). If these measures are unsuccessful, then octreotide, 50 μ g subcutaneously 30 minutes before meals, may reduce symptoms in some patients (552 [EL 4]). Late dumping symptoms can be due to “reactive hypoglycemia” and can often be managed with nutritional manipulation or be treated prophylactically by having the patient drink half a glass of orange juice (or taking the equivalent small sugar supplement) about 1 hour after eating. A report by Service et al (553 [EL 3]) described 6 patients with severe, intractable postprandial symptoms associated with endogenous hyperinsulinemic hypoglycemia. This complication, believed to be attributable to the RYGB anatomy, in some patients has necessitated partial pancreatectomy for relief of the symptoms and hypoglycemia. Pathologic examination has shown pancreatic islet cell hyperplasia. This complication may manifest from 2 to 9 years after RYGB (554 [EL 3]). Patients who present with postprandial symptoms of hypoglycemia, particularly neuroglycopenic symptoms, after RYGB should undergo further evaluation for the possibility of insulin-mediated hypoglycemia.

Food intolerances are common, frequently involving meat products. Intake of alternative protein sources should be encouraged, although meals have been reported to remain deficient in protein for a year after bariatric surgery (515 [EL 3], 543 [EL 3], 555 [EL 3]). The use of protein supplements has been proposed but is not practiced universally (515 [EL 3], 556 [EL 3]). Continuous reinforcement of new nutritional habits will help minimize the frequency of bothersome gastrointestinal symptoms. Professional guidance remains important to optimize nutritional intake in patients who have had a malabsorptive procedure because of the risk for clinically important nutritional deficiencies (140 [EL 4]).

Chronic vomiting, generally described by the patient as “spitting up” or “the food getting stuck,” can occur. One-third to two-thirds of patients report postoperative vomiting (346 [EL 3], 363 [EL 3], 543 [EL 3]). Vomiting is thought to occur most commonly during the first few postoperative months (557 [EL 3]), during which time the patients are adapting to a small gastric pouch. This vomiting is not believed to be a purging behavior as seen with bulimia nervosa. Instead, patients may vomit in response to intolerable foods or in an effort to clear food that has become lodged in the upper digestive track. Frequent vomiting that persists longer than 6 months may suggest

(1) obstruction, necessitating evaluation with a gastrointestinal contrast study, before any endoscopic procedure in patients with LAGB; (2) reflux, inflammation, stomal erosion or ulceration, or stenosis, necessitating endoscopy; or (3) gastric dysmotility, necessitating a radionuclide gastric-emptying study. Regurgitation that occurs after a LAGB procedure can be managed with appropriate band adjustments and nutritional advice.

After RYGB, supplementation with a multivitamin-mineral preparation, iron, vitamin B₁₂, and calcium with vitamin D is common (471 [EL 4], 515 [EL 3], 558 [EL 3], 559 [EL 3]). Best practice guidelines published recently recommend a daily multivitamin and calcium supplementation with added vitamin D for all patients who have had a weight loss surgical procedure (560 [EL 4]). After BPD or BPD/DS, routine supplementation regimens recommended include a multivitamin-mineral preparation, iron, vitamin B₁₂, calcium, and fat-soluble vitamins (471 [EL 4], 515 [EL 3], 558 [EL 3]).

The multivitamin-mineral preparations used should have the recommended daily requirements for vitamins and minerals. Initially, 1 to 2 tablets of a chewable preparation are advised because they are better tolerated after malabsorptive procedures. Alternatively, however, nonchewable preparations or products with fortified amounts of folic acid and iron, such as prenatal vitamins, can be used. Regardless of the preparation, multivitamin supplements providing 800 to 1,000 µg/d of folate can effectively prevent the development of folate deficiency after RYGB (515 [EL 3], 545 [EL 3], 561 [EL 2]). More recent studies suggest that folic acid deficiency is uncommon (involving only 10% to 35%) after RYGB and BPD or BPD/DS despite the absence of folic acid supplementation (562 [EL 3]). This finding suggests that the intake of folic acid is often sufficient to prevent folic acid deficiency. Recent guidelines recommend regular use of iron supplements for patients at risk of iron or folic acid deficiency (560 [EL 4]).

With multiple nutrient deficiencies, specific diagnosis and treatment become difficult. One condition thought to be due to multiple nutritional factors is “acute post-gastric reduction surgery neuropathy” (563 [EL 3], 564 [EL 3]). This complication of bariatric surgery is characterized by vomiting, weakness, hyporeflexia, pain, numbness, incontinence, visual loss, hearing loss, attention loss, memory loss, nystagmus, and severe proximal symmetric weakness of the lower extremities (563 [EL 3], 564 [EL 3]). Because all symptoms may not be ameliorated with thiamine treatment alone, additional nutritional deficiencies may be involved in the underlying cause.

9.10.2.2. Protein depletion and supplementation

Protein-deficient meals are common after RYGB. This is generally noted at 3 to 6 months after bariatric surgery and is largely attributed to the development of intolerance of protein-rich foods (540 [EL 4]). Seventeen percent of patients experience persistent intolerance of

protein-rich foods and thus limit their intake of protein to less than 50% of that recommended (540 [EL 4]). Fortunately, most food intolerances diminish by 1 year postoperatively (540 [EL 4]). Even patients who experience complete resolution of food intolerances often do not meet the daily recommended intake of protein. Regular assessment of nutritional intake should be performed, and supplementation with protein modular sources should be pursued if protein intake remains below 60 g daily (540 [EL 4]). Nevertheless, hypoalbuminemia is rare after a standard RYGB.

Protein malnutrition remains the most severe macronutrient complication associated with malabsorptive surgical procedures. It is seen in 13% of superobese patients 2 years after a distal RYGB with a Roux limb ≥150 cm and in <5% with a Roux limb <150 cm (72 [EL 3], 562 [EL 3]), as well as in 3% to 18% of patients after BPD (208 [EL 2], 211 [EL 3], 453 [EL 4], 471 [EL 4], 547 [EL 3], 565 [EL 3]). Other studies have found only a 0% to 6% incidence of protein deficiency after RYGB up to 43 months postoperatively (190 [EL 2], 545 [EL 3], 566 [EL 3]). Prevention involves regular assessment of protein intake and encouraging the ingestion of protein-rich foods (>60 g/d) and use of modular protein supplements. Nutritional support with PN for at least 3 to 4 weeks may be required after BPD/DS but rarely after RYGB (515 [EL 3]). If a patient remains dependent on PN, then surgical revision and lengthening of the common channel to decrease malabsorption would be warranted (125 [EL 3], 453 [EL 4], 567 [EL 3]).

9.10.2.3. Skeletal and mineral homeostasis, including nephrolithiasis

At present, there are no conclusive data regarding the association of altered calcium and vitamin D homeostasis with LAGB surgery. In 2 reports, LAGB was not associated with significant reduction in bone mineral density (BMD) (568 [EL 3], 569 [EL 2]).

Calcium deficiency and metabolic bone disease can occur in patients who have undergone RYGB (515 [EL 3], 559 [EL 3], 570 [EL 3], 571 [EL 3], 572 [EL 2]). The onset is insidious and results from a decrease in the intake of calcium-rich foods, bypass of the duodenum and proximal jejunum where calcium is preferentially absorbed, and malabsorption of vitamin D (515 [EL 3], 559 [EL 3], 570 [EL 3], 573 [EL 4]). An increase in serum intact PTH level is indicative of negative calcium balance or a vitamin D deficiency (or both), although PTH is also required for bone mineralization. Elevations of bone-specific alkaline phosphatase and osteocalcin levels, which are indicative of increased osteoblastic activity and bone formation, are often the initial abnormalities (559 [EL 3], 570 [EL 3]). Thus, measurement of bone turnover markers has been proposed as a useful screening technique for metabolic bone disease after RYGB because serum calcium and phosphorus levels are often normal (515 [EL 3], 559 [EL 3], 570 [EL 3], 573 [EL 4], 574 [EL 3]). After gastric

restrictive procedures, urinary C-telopeptide levels, indicative of increased bone resorption, are elevated (574 [EL 3]). After LAGB or RYGB, increased bone resorption with prolonged immobilization, especially in association with critical illness, might be associated with hypercalciuria and, if renal calcium excretion is impaired, frank hypercalcemia (575 [EL 3]). Rapid and extreme weight loss is associated with bone loss (576 [EL 2], 577 [EL 3], 578 [EL 3]), even in the presence of normal vitamin D and PTH levels (574 [EL 3]). This observation supports the claim that nutritional or hormonal factors are not the only causes of bone loss. Other factors, such as decreased weight-bearing activity, also contribute to bone loss and can be estimated with N- or C-telopeptide levels (574 [EL 3]). One interesting model of bone remodeling involves leptin-dependent sympathetic innervation of bone formation by means of activation of peripheral clock genes (579 [EL 4]).

After a malabsorptive bariatric procedure, patients might have continued secondary hyperparathyroidism, low 25-OHD levels, increased 1,25-(OH)₂D levels, and hypocalciuria (570 [EL 3], 571 [EL 3], 572 [EL 2], 574 [EL 3], 580 [EL 3], 581 [EL 2]). Left uncorrected, secondary hyperparathyroidism will promote bone loss and increase the risk for osteopenia and osteoporosis (571 [EL 3]). The presence of hypocalcemia in the setting of a vitamin D deficiency exacerbates mineralization defects and accelerates the development of osteomalacia (582 [EL 4]).

In an observational study (583 [EL 3]), 29% of patients were found to have secondary hyperparathyroidism and 0.9% had hypocalcemia beyond the third postoperative month after RYGB. Parada et al (584 [EL 3]) reported that 53% of patients had secondary hyperparathyroidism after RYGB. Also after RYGB, Youssef et al (585 [EL 2]) found that patients had a greater degree of secondary hyperparathyroidism and vitamin D deficiency with longer Roux limb length. After BPD/DS, up to one-third of patients will have deficiencies in fat-soluble vitamins including vitamin D (586 [EL 3], 587 [EL 3]). Up to 50% will have frank hypocalcemia, which is associated with secondary hyperparathyroidism and vitamin D deficiency.

In an early study by Compston et al (546 [EL 3]), 30 of 41 patients (73%) studied 1 to 5 years after BPD demonstrated defective bone mineralization, decreased bone formation rate, increased bone resorption, or some combination of these findings. Of the 41 patients, 9 (22%) had hypocalcemia, but none had low 25-OHD levels (546 [EL 3]). Reidt et al (588 [EL 3]) found that women who had undergone RYGB had decreased estradiol- and vitamin D-dependent intestinal calcium absorption. This disorder was associated with increased N-telopeptide (marker of bone resorption), increased osteocalcin (marker of bone formation), or an “uncoupling” effect on bone remodeling (588 [EL 3]).

Compston et al (546 [EL 3]) found an increased incidence of metabolic bone disease with standard BPD and a

50-cm common channel but without reduced serum 25-OHD levels. Thus, bone loss at the hip after BPD may be predominantly due to protein malnutrition (547 [EL 3]). In a series of 230 patients who underwent RYGB, Johnson et al (589 [EL 2]) found that, at 1 year postoperatively, the radius BMD was increased by 1.85% and the lumbar spine and hip BMD was decreased by 4.53% and 9.27%, respectively. Of interest, no further bone loss was noted by 2 years postoperatively (589 [EL 2]). Calcium balance may be only one of many components for maintaining bone mass after bariatric surgery, inasmuch as aggressive calcium and vitamin D supplementation resulting in normal PTH levels will still be associated with abnormal bone turnover markers and decreased bone mass (572 [EL 2]). Overall, after a malabsorptive bariatric procedure, a calcium deficiency develops in 10% to 25% of patients by 2 years and in 25% to 48% by 4 years; moreover, a vitamin D deficiency develops in 17% to 52% of patients by 2 years and in 50% to 63% by 4 years (72 [EL 3], 208 [EL 2], 221 [EL 4], 466 [EL 3], 586 [EL 3], 587 [EL 3]). Increased awareness regarding the prevalence of metabolic bone disease after malabsorptive procedures has led to routine recommendation of calcium supplementation (193 [EL 2], 411 [EL 4], 453 [EL 4], 515 [EL 3]).

After bariatric surgery, recommended dosages of elemental calcium containing vitamin D range from 1,200 to 2,000 mg daily (453 [EL 4], 515 [EL 3], 545 [EL 3], 588 [EL 3], 590 [EL 4]). Calcium carbonate preparations are readily available in chewable forms and are better tolerated than tablets shortly after bariatric surgery. Patients, however, must be instructed to take calcium carbonate preparations with meals in order to enhance intestinal absorption. Calcium citrate preparations are preferred because this salt is absorbed in the absence of gastric acid production but require consumption of more tablets (581 [EL 2], 591 [EL 4], 592 [EL 4]). Vitamin D deficiency and bone mineralization defects result from decreased exposure to sunlight, maldigestion, impaired mixing of pancreatic and biliary secretions, and decreased vitamin D absorption in the proximal small bowel (471 [EL 4], 559 [EL 3], 570 [EL 3], 593 [EL 4], 594 [EL 3], 595 [EL 2]). Vitamin D supplementation can be provided with ergocalciferol, 50,000 IU one to three times per week, although in severe cases of vitamin D malabsorption, dosing as high as 50,000 IU one to three times daily may be necessary. In the setting of significant malabsorption unresponsive to the foregoing measures, parenteral vitamin D supplementation can be used. A common regimen consists of weekly intramuscular injections of ergocalciferol, 100,000 IU, until 25-OHD levels normalize. Intramuscular vitamin D preparations are difficult to locate, may require a pharmacist to compound the medication, and can be uncomfortable when injected. Calcitriol [1,25-(OH)₂D] therapy is generally unnecessary and increases the risk of hypercalcemia and hyperphosphatemia. Intravenous (0.25 to 0.5 µg/d) or oral (0.25 to 1.0 µg once or twice daily) calcitriol therapy has been used in situations characterized by

symptomatic hypocalcemia and severe vitamin D malabsorption (**596 [EL 3]**). In asymptomatic patients, however, when 25-OHD fails to reach optimal levels (25-OHD >30 ng/mL), functionally normalize 1,25-(OH)₂D levels, and suppress elevated PTH levels, the use of calcitriol is unproved.

Adequate calcium and vitamin D supplementation has been achieved when levels for serum calcium, bone-specific alkaline phosphatase or osteocalcin, and 25-OHD as well as 24-hour urinary calcium excretion rates are normal. Serum PTH levels may persist above the normal range even with functionally replete vitamin D levels (25-OHD >30 ng/mL). This scenario can raise the specter of primary hyperparathyroidism when inappropriately elevated PTH levels accompany elevated serum calcium levels.

After BPD or BPD/DS, supplementation with elemental calcium, 2,000 mg/d, and vitamin D as outlined in the foregoing material usually corrects deficiencies in calcium and vitamin D metabolism, corrects deterioration in BMD, and improves osteoid volume and thickness without osteomalacia (**547 [EL 3]**). Nutritional status remains important in the prevention of metabolic bone disease; a low serum albumin level is a strong predictor of bone loss and metabolic bone disease after BPD or BPD/DS (**597 [EL 2]**).

Routine postoperative monitoring of bone metabolism and mineral homeostasis in patients who have undergone a malabsorptive procedure is summarized in Table 15. There are several clinical challenges in the management of metabolic bone disease in these patients: (1) intolerance of calcium supplements, (2) induction of hypercalciuria and precipitation of nephrocalcinosis and nephrolithiasis, (3) avoidance of vitamin A oversupplementation, which can increase bone resorption, and (4) inability to absorb orally administered medications and nutritional supplements. Moreover, recalcitrant protein, vitamin K, and copper deficiencies can impair recovery of bone physiologic processes.

The presence of malabsorption raises the possibility that usual dosing of orally administered bisphosphonates (ibandronate 150 mg monthly, alendronate 70 mg weekly, and either risedronate 35 mg weekly or risedronate 75 mg daily for 2 consecutive days, once a month) cannot achieve sufficient blood levels for a therapeutic effect. In one study involving non-bariatric surgery patients, risedronate was absorbed in the small bowel regardless of the site of exposure (**598 [EL 3]**). It is not known whether orally administered bisphosphonates actually increase the risk of gastric ulceration in bariatric surgery patients, but risedronate has been associated with fewer endoscopically discovered gastric erosions than alendronate (**599 [EL 3]**). Therefore, the use of newer intravenously administered bisphosphonates has received considerable attention in postoperative bariatric patients. Intravenously administered pamidronate has successfully managed resorptive hypercalcemia in patients who have undergone bariatric

surgery (**575 [EL 3]**) but is not approved by the US Food and Drug Administration for osteoporosis prevention or treatment. Pamidronate, 90 mg, is given by continuous intravenous infusion during a 4-hour period up to once every 3 months in non-bariatric surgery patients with osteoporosis and may cause a low-grade fever as well as muscle and joint pain. Zoledronate, 5 mg, is given intravenously during a 1-hour period up to once a year in non-bariatric surgery patients with osteoporosis and may cause similar adverse events. Intravenously administered ibandronate, 3 mg every 3 months, has recently become approved by the US Food and Drug Administration for the treatment of osteoporosis and confers far less risk for renal insufficiency than pamidronate or zoledronate (**600 [EL 4]**). Care must be exercised to ensure that vitamin D deficiency after gastric bypass is corrected before administration of bisphosphonates to avoid severe hypocalcemia, hypophosphatemia, and osteomalacia (**601 [EL 3]**, **602 [EL 3]**). Even with vitamin D sufficiency, a bypassed small bowel may not be capable of absorbing adequate calcium to offset the effects of bisphosphonate binding to bone matrix. Overall, there are no published clinical trial data regarding use of bisphosphonates in bariatric surgery patients.

Just as in patients with short bowel syndrome, patients who have had malabsorptive procedures are at risk for oxalosis and renal oxalate stones. Impaired binding of oxalate in the small bowel allows greater oxalate absorption in the colon, contributing to excessive excretion of oxalates by the kidneys. Dehydration also has a role as a result of restrictions imposed on amount and timing of fluid intake after gastric restrictive procedures. Treatment of this problem consists of low-oxalate meals, appropriate oral calcium supplementation, and orally administered potassium citrate. Increasing the urinary calcium too high with orally administered calcium and vitamin D supplementation, intended to reduce secondary hyperparathyroidism and treat presumed osteomalacia, can exacerbate calcium oxalate stone formation. Clinical studies have demonstrated an association of *O formigenes* colonization of the small bowel, or administration as a probiotic therapy, with decreased urinary oxalate excretion and stone formation (**603 [EL 4]**, **604 [EL 3]**, **605 [EL 3]**).

Magnesium is readily available from plant and animal sources and is absorbed throughout the entire small bowel independent of vitamin D. Hypomagnesemia can be associated with neuromuscular, intestinal, and cardiovascular symptoms and abnormalities in secretion of PTH. Hypomagnesemia has been reported after bariatric procedures, such as jejunioileal bypass and BPD, and usually occurs in the setting of persistent diarrhea (**208 [EL 2]**). Empiric supplementation with a mineral-containing multivitamin providing the daily recommended intake of magnesium (>300 mg in women; >400 mg in men) should prevent magnesium deficiency in the absence of complicating factors. In the setting of symptomatic and severe

magnesium deficiency, supplementation should be dictated by the clinical situation. Parenteral supplementation in accordance with currently accepted protocols should be pursued in the patient with neurologic and cardiac symptoms. Magnesium supplementation should be accompanied by careful monitoring of other minerals and electrolytes. In the asymptomatic patient with low magnesium levels, oral supplementation can be prescribed as tolerated. Unfortunately, oral magnesium supplementation can cause or worsen diarrhea (606 [EL 3], 607 [EL 4]).

Hypophosphatemia might be observed in patients with malnutrition or fat malabsorption (or both). Milk products are an excellent source of phosphorus for those patients who can tolerate oral intake (608 [EL 4]). Phosphorus is also present in protein-rich foods such as meat and cereal grains. Phosphorus is absorbed throughout the small intestine under the control of vitamin D and specific phosphate transporters. Hypophosphatemia with or without phosphorus deficiency is common in seriously ill patients. In the presence of phosphorus deficiency, hypophosphatemia can result from chronic malnutrition, chronic alcoholism, hyperparathyroidism, vitamin D deficiency, metabolic bone disease, and fat malabsorption. In the absence of phosphorus deficiency, hypophosphatemia can result from the effect of acid-base status on plasma phosphorus levels or the administration of substances that influence the uptake of serum phosphorus by the cell (glucose, amino acids, and insulin: the “refeeding syndrome”) (609 [EL 4]). Thus, nutrition support must be initiated cautiously in severely malnourished bariatric surgery patients because of the risk of the refeeding syndrome. Hypophosphatemia can cause rhabdomyolysis, respiratory insufficiency, nervous system dysfunction, and proximal myopathy.

9.10.2.4. *Fat malabsorption: essential fatty acids and vitamins A, E, and K*

No published clinical data specifically address the potential for deficiency of essential fatty acids (EFA) in bariatric surgery patients. The recommended intake to prevent or reverse symptoms of linoleic acid (18:2n-6) deficiency is approximately 3% to 5% of energy intake. The recommended intake to prevent or reverse symptoms of linolenic acid (18:3n-3) deficiency is approximately 0.5% to 1% of energy intake (610 [EL 4]). Elevation of the triene:tetraene ratio (20:3n-9 to 20:4n-6) >0.2 indicates deficiency of n-3 and n-6 fatty acids (FA). Dietary sources of n-3 and n-6 FA are the polyunsaturated FA-rich vegetable oils. Linoleic acid content (as percent of all FA) is particularly high in safflower (76%), sunflower (68%), soybean (54%), and corn (54%) oils (611 [EL 4]). Because safflower, sunflower, and corn oils contain very little n-3 FA (<1%) and can therefore result in an n-3 FA deficiency, soybean, linseed, and canola oils, which contain relatively high amounts of both n-3 and n-6 FA, are better choices for long-term consumption (611 [EL 4]). Clinical symptoms of EFA deficiency in adults, applicable

to bariatric surgery patients, include dry and scaly skin, hair loss, decreased immunity and increased susceptibility to infections, anemia, mood changes, and unexplained cardiac, hepatic, gastrointestinal, and neurologic dysfunction (612 [EL 4]). Beyond consumption of the aforementioned whole foods rich in EFA, there are no data regarding optimal supplementation with EFA-containing nutraceuticals in bariatric surgery patients. Topical administration of safflower oil has been demonstrated to prevent EFA deficiency in patients receiving home total parenteral nutrition (613 [EL 3]) and therefore may be a reasonable alternative in symptomatic patients who have undergone extensive malabsorptive procedures, such as BPD/DS.

Steatorrhea induced by malabsorptive surgical procedures can frequently lead to deficiencies in fat-soluble vitamins, typically manifested by an eczematous rash (140 [EL 4], 392 [EL 4], 471 [EL 4]), but may also be associated with night blindness or full-blown loss of vision from profound vitamin A deficiency. Fat-soluble vitamins in their water-soluble form should be administered to all patients who have undergone a BPD or BPD/DS procedure. Fat-soluble vitamin levels, especially vitamin A, should be monitored annually after such operations (471 [EL 4], 545 [EL 3], 546 [EL 3]). No randomized, prospective studies have evaluated the efficacy of specific doses of fat-soluble vitamin supplementation in preventing deficiencies. Combined supplementation of vitamins A, D, E, and K can be achieved with ADEK tablets, each containing the following: vitamin A (palmitate), 4,000 IU; β -carotene, 3 mg; vitamin D (cholecalciferol), 400 IU; vitamin E (succinate), 150 IU; and vitamin K, 0.15 mg (as well as vitamin C, 60 mg; folic acid, 0.2 mg; thiamine, 1.2 mg; riboflavin, 1.3 mg; niacin, 10 mg; pantothenic acid, 10 mg; pyridoxine, 1.5 mg; biotin, 50 μ g; vitamin B₁₂, 12 μ g; and zinc oxide, 7.5 mg). As with all combination medications, serum levels need to be monitored carefully for both underreplacement and overreplacement of the various ingredients.

Vitamin A deficiency after bariatric surgery results from poor nutritional intake, maldigestion, malabsorption, and impaired hepatic release of vitamin A. In 2 series, there was a 61% to 69% incidence of vitamin A deficiency 2 to 4 years after BPD, with or without DS (208 [EL 2], 586 [EL 3]). In another series, however, the incidence was as low as 5% by 4 years (78 [EL 3]). Although data are scarce, mild vitamin A deficiency can also occur after distal RYGB procedures and is easily corrected with oral supplementation (392 [EL 4]). Nevertheless, prophylactic supplementation does not prevent the development of vitamin A deficiency in all patients; thus, continued biochemical monitoring is indicated (392 [EL 4]). Symptoms of vitamin A deficiency include ocular xerosis and night blindness. Oral supplementation of vitamin A, 5,000 to 10,000 IU/d, is recommended until the serum vitamin A level normalizes. Empiric supplementation with vitamin A, 25,000 IU/d, has been used after BPD/DS (84 [EL 3], 140 [EL 4], 547 [EL 3]). With symptoms, aggressive oral

supplementation, up to 65,000 IU/d of vitamin A, can normalize dark adaptation and the serum vitamin A level after 2 to 3 months. In the presence of severe malnutrition necessitating PN, supplementation with 25,000 IU/d can correct vitamin A deficiency within weeks (614-616 [EL 3]). When resulting from fat malabsorption, a vitamin A deficiency may be ameliorated by lengthening of the 50-cm common channel to 150 to 200 cm (586 [EL 3]).

In patients who have had a BPD or BPD/DS, vitamin K deficiency occurs in approximately 50% to 70% within 2 to 4 years postoperatively (208 [EL 2], 586 [EL 3]). Vitamin K supplementation is recommended when international normalized ratio values increase above 1.4 (140 [EL 4], 471 [EL 4]). Vitamin K deficiency can lead to easy bruising, bleeding, and metabolic bone disease (617 [EL 4]).

In approximately 95% of patients who have undergone BPD or BPD/DS who are already taking a multivitamin, vitamin E levels remain normal (208 [EL 2], 586 [EL 3]). Vitamin E deficiency can lead to anemia, ophthalmoplegia, and peripheral neuropathy. Administration of vitamin E (800 to 1,200 IU/d) should be initiated when deficiency is documented and continued until the serum levels reach the normal range. Overreplacement of vitamin E can exacerbate the coagulopathy associated with a concomitant vitamin K deficiency (618 [EL 4]).

9.10.2.5. Nutritional anemia: iron, vitamin B₁₂, folic acid, selenium, and copper

Iron deficiency and iron deficiency anemia are common after RYGB, BPD, or BPD/DS, especially in women with menorrhagia. For this reason, prophylactic iron supplementation is required (194 [EL 2], 545 [EL 3], 619 [EL 2], 620 [EL 3]). Decreased liberation and absorption of heme are caused from bypass of the acid environment in the lower portion of the stomach and the absorptive surfaces of the duodenum and upper jejunum (530 [EL 2], 621-624 [EL 4]). Moreover, after malabsorptive procedures, patients frequently eat meals low in meats, leading to decreased intake of heme. Iron deficiency may also be exacerbated in these patients as a result of a nutrient-nutrient inhibitory absorptive interaction between iron and calcium, another mineral that is routinely supplemented during the postoperative period. Most (625 [EL 3], 626 [EL 3]), but not all (627 [EL 3]), studies show that non-heme- and heme-iron absorption is inhibited up to 50% to 60% when consumed in the presence of calcium supplements or with dairy products. Calcium at doses of 300 to 600 mg has a direct dose-related inhibiting effect on iron absorption. This effect has been noted with calcium carbonate, calcium citrate, and calcium phosphate. The risk for iron deficiency increases with time, with some series reporting more than half of the subjects with low ferritin levels at 4 years after RYGB, BPD, or BPD/DS (545 [EL 3]). Iron deficiency after RYGB is influenced by multiple factors and can persist to 7 years postoperatively (555 [EL 3]). Iron deficiency has been reported to occur in up to

50% of patients after RYGB, most frequently in women with menorrhagia as previously stated (388 [EL 3], 559 [EL 3], 561 [EL 2], 628 [EL 3]). Thus, empiric iron supplementation is recommended after RYGB, BPD, or BPD/DS procedures (619 [EL 2], 620 [EL 3]).

In a randomized, controlled trial, iron supplementation (65 mg of elemental iron orally twice a day) prevented the development of iron deficiency, although it did not always prevent the development of iron deficiency anemia (619 [EL 2]). Supplementation with lower doses (80 mg/d) does not universally prevent iron deficiency after RYGB, BPD, or BPD/DS (545 [EL 3]). Nevertheless, low-dose iron supplementation (80 mg/d) was associated with a lower risk for low ferritin levels. Vitamin C increases iron absorption and should be empirically included with iron supplementation (590 [EL 4], 620 [EL 3]). Because oral iron supplementation is associated with poor absorption and adverse gastrointestinal effects, and intramuscular injections are painful, intermittent intravenous iron infusion may be necessary. Iron dextran (INFeD), ferric gluconate (Ferrlecit), or ferric sucrose (Venofer) may be administered intravenously. Supplementation should follow currently accepted guidelines to normalize the hemoglobin concentration. Continued surveillance of hemoglobin levels and iron studies is recommended (619 [EL 2]).

Vitamin B₁₂ deficiencies can occur after bariatric surgical procedures that bypass the lower part of the stomach. Impairment of vitamin B₁₂ absorption after RYGB results from decreased digestion of protein-bound cobalamins and impaired formation of intrinsic factor-vitamin B₁₂ complexes required for absorption (221 [EL 4], 515 [EL 3], 629 [EL 3], 630 [EL 3]). Whole-body storage (2,000 µg) is considerably greater than the daily needs (2 µg/d). Nonetheless, after the first postoperative year, 30% of patients with RYGB receiving only a multivitamin supplement will have a vitamin B₁₂ deficiency (631 [EL 3]). In other studies, the incidence of vitamin B₁₂ deficiency after RYGB at postoperative year 1 has been 33% to 40% (561 [EL 2], 628 [EL 3]) and by years 2 to 4 has been 8% to 37% (72 [EL 3], 544 [EL 3], 545 [EL 3], 632 [EL 3]). In patients with BPD, there was a 22% incidence of vitamin B₁₂ deficiency at 4 years (545 [EL 3]), and in patients with VBG (N = 26), there were no instances of vitamin B₁₂ deficiency at 1 year (633 [EL 3]). Anemias as a result of vitamin B₁₂ deficiency have been reported to occur in more than 30% of patients 1 to 9 years after RYGB (471 [EL 4]).

There is some controversy regarding the routine supplementation of vitamin B₁₂ after RYGB or BPD, and there are no evidence-based recommendations. Most bariatric surgery groups, however, advise the initiation of vitamin B₁₂ supplementation within 6 months postoperatively. Orally administered crystalline vitamin B₁₂ at a dose of at least 350 µg has been shown to maintain normal plasma vitamin B₁₂ levels (561 [EL 2], 634 [EL 3], 635 [EL 3], 636 [EL 2]). Optimal dosing of oral, sublingual,

or intranasal forms of vitamin B₁₂ supplementation has not been well studied. In a study of postoperative RYGB patients by Clements et al (637 [EL 3]), however, 1,000 µg of vitamin B₁₂ intramuscularly every 3 months or 500 µg of intranasally administered vitamin B₁₂ every week resulted in a lower incidence of vitamin B₁₂ deficiency (3.6% at 1 year and 2.3% at 2 years) in comparison with the frequency of 12% to 37% described by Brolin and Leung (558 [EL 3]). Functional markers of vitamin B₁₂ nutriture, which are more sensitive than plasma vitamin B₁₂ levels, may be followed and include methylmalonic acid and homocysteine. Both, however, might be low because of protein malnutrition, and the latter might be elevated because of vitamin B₆, folate, choline, or betaine deficiencies. Even with prophylactic supplementation, vitamin B₁₂ status should be monitored, inasmuch as deficiencies can develop and necessitate dosing modifications (545 [EL 3]). Routine monitoring of vitamin B₁₂ levels early during the postoperative course after RYGB, BPD, or BPD/DS (<3 to 6 months) may be justified if preoperative vitamin B₁₂ deficiency is suspected. In the absence of strong evidence, vitamin B₁₂ recommendations are based on subjective impressions of the prevalence and clinical significance of sequelae from vitamin B₁₂ deficiencies.

In comparison with vitamin B₁₂, deficiencies in folate are less common because folate absorption occurs throughout the entire small bowel; thus, deficiency is unlikely if the patient is taking a daily multivitamin as instructed. Biomarker monitoring is not necessary (392 [EL 4], 515 [EL 3], 623 [EL 4]). Hyperhomocysteinemia, suggestive of a functional folate deficiency, is an independent risk factor for cardiovascular disease, but intervention with folic acid remains controversial. The pregnant bariatric patient should also have routine additional folic acid supplementation because of the risk of fetal neural tube defects.

Other micronutrient deficiencies have been associated with anemias in non-bariatric surgery patients and include copper, vitamin A, vitamin B₁, vitamin E, and selenium (624 [EL 4], 638 [EL 4], 639 [EL 4]). Selenium is an antioxidant, and its status is closely associated with vitamin E status. In a series of patients who had undergone BPD or BPD/DS, selenium deficiency was found in 14.5% of patients without any clinical sequelae (208 [EL 2]). Proven selenium deficiency-associated anemias have not been reported in bariatric surgery patients. Copper deficiency can induce anemia (normocytic or macrocytic) and neutropenia (640 [EL 3], 641 [EL 3]). The detrimental effects of copper deficiency on tissue release, resulting in elevated ferritin levels, may be mediated by hepcidin, a ceruloplasmin homologue, and divalent metal transporter-1 (642 [EL 4]). Kumar et al (643 [EL 3]) described 2 patients who had undergone gastric surgery and had copper deficiency, leading to neurologic features similar to a vitamin B₁₂ deficiency. One of the 2 patients had undergone a gastric bypass procedure several years before clinical presentation.

9.10.2.6. Zinc

Plasma zinc levels represent only <0.1% of whole-body zinc and are a poor biomarker for zinc status (644 [EL 4]). With systemic inflammation, this insensitivity is exacerbated with increased hepatic zinc uptake (645 [EL 3]). Because zinc is lost in the feces, patients with chronic diarrhea are at risk for zinc deficiency (646 [EL 4]). In the absence of pancreatic exocrine secretions, however, zinc absorption remains normal (647 [EL 3]). Although it would appear rational to provide zinc empirically to patients with malabsorption, this intervention can induce a copper deficiency (648 [EL 3]). Thus, injudicious supplementation with zinc can lead to a copper deficiency-related anemia, which may be erroneously treated empirically with increasing amounts of iron, exacerbating an iron-overload condition and eventuating in organ damage. In contrast, 10% to 50% of patients who have undergone BPD/DS may experience zinc deficiency (208 [EL 2], 586 [EL 3]). Hair loss and rash are symptoms of zinc deficiency, but they can be nonspecific (649 [EL 4]).

9.10.2.7. Thiamine

Thiamine deficiency can occur as a result of bypass of the jejunum, where thiamine is primarily absorbed, or as a result of impaired nutritional intake from recurrent emesis (650 [EL 3], 651 [EL 4]). Two studies have shown decreased thiamine levels before bariatric surgery (388 [EL 3], 652 [EL 3]). Although thiamine deficiency has multiple manifestations, neurologic symptoms are predominant in this patient population (515 [EL 3], 638 [EL 4], 641 [EL 3], 650 [EL 3], 651 [EL 4], 653 [EL 4]). Acute neurologic deficits as a result of thiamine deficiency have been reported as soon as 1 to 3 months after bariatric surgery (654-663 [EL 3-4]). Early recognition is paramount to initiate appropriate supplementation and avoid potential complications resulting from the administration of dextrose-containing solutions (650 [EL 3]). Although not often performed, assessment of thiamine status is best done by determining erythrocyte transketolase activity. Parenteral supplementation with thiamine (100 mg/d) should be initiated in the patient with active neurologic symptoms (664 [EL 3], 665 [EL 3]). After a 7- to 14-day course, an oral preparation (10 mg/d) can be used until neurologic symptoms resolve (515 [EL 3], 666 [EL 3], 667 [EL 4]). Severe thiamine deficiency most commonly occurs in patients who develop severe, intractable vomiting after bariatric surgery, usually due to a mechanical problem such as stomal stenosis after RYGB or BPD/DS or excessive band tightness or slippage after LAGB. It is important that persistent vomiting be resolved aggressively to prevent this devastating complication.

9.10.3. Cardiology and Hypertension

Improvements in serum lipids are observed by 6 months after gastric restrictive procedures. Reported reductions in total cholesterol and triglyceride levels are >15% and >50%, respectively. No significant changes in

HDL cholesterol levels are observed early postoperative-ly; however, gradual and significant improvements occur after 12 months (20%) (252 [EL 2], 493 [EL 3]). The greatest reduction in lipid values is observed in patients with high preoperative values (252 [EL 2]). The underlying physiologic factors for the observed improvements in lipid values are multifactorial and include rapid weight loss, nutritional changes, and decreased insulin resistance (248 [EL 3], 252 [EL 2], 490 [EL 3], 493 [EL 3]). Improvements in lipid values have been reported despite suboptimal weight loss (<50% of excess weight) or weight regain (252 [EL 2]). The SOS Study has shown a significant decrease in the incidence of hypertriglyceridemia and low-HDL syndrome at 2 years in surgical patients compared with weight-matched control subjects (253 [EL 1]). Improvements in triglyceride and HDL cholesterol levels were still observed 10 years after bariatric surgery (64 [EL 3]). Statistically significant lowering of LDL cholesterol levels has been reported after BPD (256 [EL 3]). Continued monitoring of lipid values and the need for hypolipidemic medication on a periodic basis is advised, especially in patients with a history of T2DM or vascular disease.

Several investigators have reported long-term improvements of hypertension after bariatric surgery. Remission rates, however, are much lower than those reported with T2DM (64 [EL 3], 99 [EL 1], 263 [EL 3], 668-670 [EL 3]). The SOS Study initially reported a decrease in the incidence of hypertension in the surgical cohort in comparison with control subjects at 2 years. At 8 years, however, this protection was lost, with no significant difference in the incidence of hypertension between the 2 cohorts, despite a weight loss maintenance of 16% in the surgically treated patients (264 [EL 2]). In contrast, in another study in which the 6% of patients who had undergone a RYGB in the SOS Study and lost significantly more weight than the patients who had purely restrictive procedures (94%), a significant decrease in both systolic and diastolic blood pressures persisted at 8 years (64 [EL 3]). Continued surveillance of blood pressure and of adequacy of antihypertensive treatment is recommended.

9.10.4. Pulmonary

Therapy with nasal CPAP or bilevel positive airway pressure should be continued until repeated assessment with overnight polysomnography can confirm complete resolution. Unfortunately, many patients are unwilling or unable to comply with repeated overnight polysomnography. Many patients stop therapy on their own as a result of subjective improvement. At present, there are no clear guidelines regarding the timing for repeated sleep studies in bariatric surgery patients (243 [EL 2]). Overnight oximetry, although not studied in this situation, may provide some reassurance in that normal study results are unlikely in the presence of sleep apnea. Patients with mild to moderate sleep apnea (RDI <40) will usually have complete resolution, whereas those with more severe sleep

apnea will have residual apneic episodes but are usually asymptomatic without nasal CPAP (504 [EL 4]).

9.10.5. Gastrointestinal

9.10.5.1. Diarrhea and steatorrhea

Diarrhea and steatorrhea are common complications of malabsorptive procedures, reported in up to 60% of patients after jejunoileal bypass surgery (614 [EL 3], 671 [EL 3]). In one study, diarrhea with malodorous stools after BPD or BPD/DS was reported by 13% of patients (547 [EL 3]). Diarrhea is uncommon after RYGB and should prompt evaluation for the presence of significant macronutrient malabsorption and steatorrhea (672 [EL 3]). Diagnostic considerations include lactose intolerance, bacterial overgrowth, or a concurrent diagnosis such as celiac sprue (673 [EL 3]). Celiac sprue has also been recognized as a common cause for iron deficiency anemia (674 [EL 3]). Upper endoscopy in conjunction with small bowel biopsies showing the classic histologic findings of celiac sprue remains the standard diagnostic technique. Although classically duodenal biopsies are pursued in the diagnosis of celiac sprue, small bowel histologic features should also be representative if celiac sprue is present. There are no data to suggest that the prevalence of celiac sprue is any different among patients undergoing bariatric surgery than that expected in the general population. The use of serologic markers, however, has been gaining acceptance (675 [EL 3]). Once celiac sprue has been identified, management involves implementation of a gluten-free meal plan, which is associated with relief of gastrointestinal symptoms and improvement of abnormal histologic features in the majority of patients (676 [EL 3], 677 [EL 3]).

9.10.5.2. Stomal stenosis and ulceration

Potential causes of persistent and severe gastrointestinal symptoms include stomal stenosis and ulceration (392 [EL 4], 453 [EL 4], 471 [EL 4]). Stomal stenosis is common (12%) and results from the restrictive size of the gastric pouch and associated edema. This complication is more common after laparoscopic versus open RYGB (678 [EL 4]). Endoscopy is preferred in the evaluation of stomal obstruction because it can be used for diagnosis and treatment with transendoscopic balloon dilation. Repeated dilations may be required. After VBG, balloon dilation is often unsuccessful, and surgical intervention is required (453 [EL 4], 471 [EL 4], 525 [EL 3]).

Marginal ulcers between the stomach pouch and the small intestine are a frequent source of epigastric pain, blood loss, and iron deficiency, accounting for 27% of patients referred for endoscopy in one study (679 [EL 3]). The most likely cause for stomal ulceration is anastomotic ischemia, usually due to tension of the anastomosis when the gastrojejunostomy was created (680 [EL 4]). Stomal ulcers are also caused by retained acid-producing gastric tissues in large pouches. Other potential causes of stomal stenosis and ulceration should also be considered,

including *H pylori*, which can be treated with proton pump inhibitors, sucralfate, and antibiotics (453 [EL 4]). These ulcerations can be caused or exacerbated by the concomitant use of nonsteroidal antiinflammatory drugs, aspirin, and cyclooxygenase-2 inhibitors.

9.10.5.3. Gastric obstruction

Gastric obstruction associated with LAGB is due to a bolus of food lodging at the banded area, excessive inflation of the balloon, or gastric prolapse around the band. Vomiting releases the obstructed food and is temporary. Symptoms attributable to a tight band or band prolapse include obstruction and nongastrointestinal symptoms, such as sleep disturbance, night cough, asthma, and recurrent bronchitis or pneumonia. They can occur without any reflux or gastrointestinal symptoms (681 [EL 3], 682 [EL 3]). Removing saline from the band, by means of the adjustment reservoir, usually provides immediate resolution of the excessive band inflation. Nevertheless, a major prolapse of stomach through the band causing a very large proximal gastric pouch can lead to complete obstruction. Gastric obstruction is usually associated with pain and may necessitate emergency surgical reexploration because of the risk of gastric necrosis. The diagnosis is suggested by abdominal plain films showing an abnormal band position. Bands are placed such that they lie parallel to a line drawn from the patient's right hip to the left shoulder, with the tubing pointing toward the left shoulder. When this orientation is lost, prolapse should be expected. Because ischemia may ensue, the band should be immediately emptied and surgical exploration considered if the patient's abdominal pain does not resolve.

9.10.5.4. Intestinal obstruction

Small bowel obstruction can occur after RYGB, BPD, or BPD/DS (392 [EL 4], 480 [EL 4]). Evaluation, however, can be challenging as a result of the limitations in imaging imposed by the altered anatomy. UGI studies and CT scans may not confirm an obstruction when present. The symptom of cramping periumbilical pain should prompt strong consideration of reexploration by either the open or the laparoscopic technique because of the danger of bowel infarction, peritonitis, and death (392 [EL 4]). This complication may be attributable to adhesions or to an incarcerated internal hernia through one of three potential mesenteric defects (683-685 [EL 3]), especially during pregnancy (686 [EL 4], 687 [EL 3], 688 [EL 3]).

9.10.5.5. Gallbladder disease

Obesity is a risk factor for benign gallbladder disease, which is frequently identified in patients seeking bariatric surgery (689 [EL 3]). Gallstone formation is common after significant weight loss and is related to the rate of weight loss (690 [EL 4]). This relationship prevails for patients who have undergone RYGB, BPD, or BPD/DS surgery (392 [EL 4], 453 [EL 4], 471 [EL 4], 691 [EL 2]) but not LAGB (692 [EL 3]). Gallstone and sludge formation has been reported in 30% of patients 6 months after

RYGB, BPD, or BPD/DS procedures (693 [EL 3], 694 [EL 1]). As a result, some surgeons have advocated performing a prophylactic cholecystectomy at the time of these procedures (193 [EL 2], 453 [EL 4], 695 [EL 3]). In a randomized, placebo-controlled trial, medical therapy with ursodiol (300 mg twice daily) has been shown to be effective in decreasing the incidence of gallstone formation from 30% to 2% at 6 months (694 [EL 1]). Currently, it is an accepted alternative to prophylactic cholecystectomy in this patient population (392 [EL 4], 694 [EL 1], 696 [EL 3]). Ursodiol, however, is relatively expensive, often not well tolerated, and therefore associated with poor adherence (691 [EL 2]). Because few adhesions form after laparoscopic bariatric surgery, performance of laparoscopic cholecystectomy is not very difficult when symptoms of cholecystitis develop, and most surgeons are not recommending treatment with ursodiol at this time (696 [EL 3], 697 [EL 3]). Nonetheless, there is a risk of common bile duct stones developing, which may be very difficult to address after either RYGB or BPD/DS.

9.10.5.6. Bacterial overgrowth

Bacterial overgrowth can occur with malabsorptive procedures, although any structural change to gut continuity is a recognized risk factor (140 [EL 4], 471 [EL 4], 673 [EL 3]). It can contribute to additional complications such as inflammatory arthritis as a result of antibody-antigen deposition from translocation of endotoxin fragments into the bloodstream from the bypassed limb (453 [EL 4]). Symptoms include persistent diarrhea in conjunction with proctitis and abdominal distention. Diagnosis can be difficult but should involve upper endoscopy and performance of intestinal aspirate cultures. D-Xylose and hydrogen breath tests are available but have limited sensitivity when used alone (673 [EL 3], 698-701 [EL 3]). Empiric antibiotic therapy, particularly with metronidazole, is usually effective at controlling symptoms and supports the presence of bacterial overgrowth (140 [EL 4], 702 [EL 3], 703 [EL 3]). The role of probiotics in decreasing complications after gastrointestinal surgery, especially when bacterial overgrowth has occurred, has been reviewed by Correia and Nicoli (704 [EL 4]). At present, there is still inconclusive level 3 evidence for use of probiotics in the general surgery population and no data in bariatric surgery patients.

9.10.5.7. Incisional hernias

Incisional hernias are the most common complication after open bariatric surgical procedures (in 10% to 20% of patients), which is significantly reduced by use of the laparoscopic approach (57 [EL 3], 62 [EL 2], 191 [EL 2], 421 [EL 3], 480 [EL 4], 705 [EL 3]). Their cause is multifactorial, including factors such as increased intra-abdominal pressure and poor wound healing. In the asymptomatic patient, repair is often deferred until the patient has achieved maximal weight loss. In the symptomatic patient, prompt repair is recommended (453 [EL 4]).

9.10.5.8. Staple-line disruption

The prevalence of staple-line disruption, which is a problem only with open bariatric procedures in which the stomach is not transected, varies widely and is often asymptomatic (392 [EL 4], 453 [EL 4]). It is thought that modifications in technique have led to a decrease in the prevalence of this complication. Placement of three superimposed rows rather than one row of staples at the anastomosis when the stomach is not transected (453 [EL 4], 504 [EL 4], 706 [EL 3], 707 [EL 2]) reduces the risk of this complication. Transection of the stomach, however, decreases the risk of this complication and is the standard procedure when a laparoscopic or open RYGB is performed (57 [EL 3], 62 [EL 2], 191 [EL 2], 421 [EL 3], 708 [EL 3]). Gastrogastric fistulas can occur after stomach transection and have been reported in 1% to 6% of cases (706 [EL 3], 709 [EL 3], 710 [EL 3]).

9.11. Pregnancy

Pregnancy should be discouraged during periods of rapid weight loss (12 to 18 months postoperatively) (711 [EL 3], 712 [EL 4], 713 [EL 3]). Nevertheless, patients who may have had subfertility, with or without PCOS, before bariatric surgery are more likely to conceive postoperatively (711 [EL 3], 714 [EL 3], 715 [EL 3]). Postoperative patients desiring pregnancy should be counseled to adhere with their nutritional regimen, including use of micronutrient supplements. Folic acid and vitamin B₁₂ status should be monitored in these patients during pregnancy and also during the breastfeeding period. Hyperhomocysteinemia can result from deficiencies in folic acid, vitamin B₁₂, and other micronutrients, and in non-bariatric surgery patients, this condition is associated with placental vascular disease and recurrent early pregnancy loss and fetal neural tube defects (716-718 [EL 3]). Obstetricians should monitor post-bariatric surgery pregnant women for the potential development of internal hernias (688 [EL 3]) and small bowel ischemia (687 [EL 3]).

Complications of pregnancy after bariatric surgery include persistent vomiting, gastrointestinal bleeding (719 [EL 3]), anemia (720 [EL 3]), intrauterine growth restriction (720 [EL 3]), various micronutrient deficiencies including vitamin A (721 [EL 3]), vitamin B₁₂, folic acid, and iron (722 [EL 4]), and fetal neural tube defects (723 [EL 3], 724 [EL 3]). Bariatric surgery, however, may reduce the risks for gestational diabetes, hypertension, DVT, stress incontinence, preeclampsia, cephalopelvic disproportion, macrosomia, and cesarean delivery (281 [EL 3], 307-310 [EL 3], 725 [EL 3]). Routine weight management and periodic band adjustments during pregnancy have proved beneficial (305 [EL 3], 306 [EL 2], 309 [EL 3], 726 [EL 2]).

9.12. Body-Contouring Surgery

For some patients, the massive weight loss as a result of bariatric surgery is associated with physical discomfort and body image dissatisfaction related to loose, sagging

skin. These untoward experiences are believed to have a central role in the decision to seek body-contouring surgery (727 [EL 4]). According to the American Society of Plastic Surgeons (728 [EL 3]), more than 65,000 individuals underwent body-contouring surgery after massive weight loss in 2006. The most common surgical intervention was breast reduction-breast lift procedures, performed on 29,712 women (728 [EL 3]). Plastic surgeons also reported performing 19,046 extended abdominoplasty-lower body lifts, 9,274 upper arm lifts, and 7,920 thigh lifts (728 [EL 3]).

There is growing interest in these procedures within the plastic surgery community (729 [EL 4]). Typically, the procedures are recommended for patients whose weight has been stable for 3 to 6 months. It is unknown whether persons who have undergone bariatric surgery and who elect to undergo body-contouring surgery experience additional physical and psychosocial benefits. There is an increased risk for venous thromboembolism with body-contouring surgery after gastric bypass procedures, and tobacco use can increase this risk further (450 [EL 4], 730 [EL 3]). Typically, body contouring after bariatric surgery is not covered by third-party payers without prior authorization stating medical necessity, and it often remains a noncovered service.

9.13. Psychologic Issues

Literature reviews and numerous empirical studies have described significant improvements in psychosocial functioning after bariatric surgery (48 [EL 4], 117-119 [EL 4], 356 [EL 4]). Patients typically report decreases in symptoms of anxiety and depression and significant improvements in health-related quality of life (336 [EL 3], 379 [EL 1], 523 [EL 3], 731-735 [EL 3]). The presence of formal psychopathologic conditions appears to be reduced, although this has been investigated in only a limited number of studies (334 [EL 3]). Patients also typically report improvements in body image as well as marital and sexual functioning (357 [EL 3], 364 [EL 3], 378 [EL 3], 736-738 [EL 3]).

These generally positive reports are contradicted by other findings. In a significant minority of patients, a negative psychologic response to bariatric surgery has been reported (334 [EL 3], 739 [EL 3], 740 [EL 3]). For some patients, improvements in psychosocial status dissipate 2 to 3 years postoperatively (339 [EL 3], 340 [EL 4], 379 [EL 1], 741 [EL 4]). Other studies have documented suicides postoperatively (122 [EL 3], 336 [EL 3], 363 [EL 3]). The factors contributing to these less positive outcomes remain unclear and necessitate additional investigation.

Postoperative psychosocial status also may affect postoperative eating behavior. Several studies have suggested that patients struggle to adhere to the recommended postoperative eating plan (218 [EL 3], 543 [EL 3], 655 [EL 3], 742 [EL 3], 743 [EL 2]). Increased caloric consumption above patients' postoperative caloric demands

may contribute to suboptimal weight loss or even weight regain, which may begin as early as the second postoperative year (64 [EL 3], 339 [EL 3], 340 [EL 4], 345 [EL 3]). Some patients may experience a return of disordered eating behaviors, which may contribute to untoward events such as nausea, vomiting, and gastric dumping (70 [EL 2], 187 [EL 2], 346 [EL 3], 363 [EL 3], 543 [EL 3], 557 [EL 3]).

9.14. Criteria for Readmission to Hospital

9.14.1. Severe Protein Deficiency

Protein malnutrition causes a hospitalization rate of 1% per year after BPD or BPD/DS and leads to significant morbidity (140 [EL 4], 471 [EL 4]). Hospitalization with initiation of PN support is often necessary (744 [EL 4]). No currently accepted guidelines or clinical studies guiding nutritional therapy after weight loss surgery have been published. Most clinicians follow generally accepted guidelines for the initiation and administration of PN at their institutions. For avoidance of the refeeding syndrome, caution must be exercised with the initiation of solutions containing high amounts (more than 100 to 200 g per day) of dextrose in the setting of severe malnutrition. Symptoms of the refeeding syndrome include swelling with signs of volume overload associated with hypokalemia, hypophosphatemia, and hypomagnesemia. This constellation of clinical features results from the insulin-mediated influx of electrolytes into cells and renal salt and water retention (515 [EL 3]). Aggressive replacement to correct these abnormalities is advised, particularly with cautious initiation of PN. Calories provided can be gradually increased toward total caloric requirements after several days to a week. Surgical revision is advised, with lengthening of the common channel to ameliorate malabsorption (125 [EL 3], 515 [EL 3], 567 [EL 3]).

9.14.2. Parenteral Nutrition

Parenteral nutrition is recommended in the malnourished patient who is unable to maintain a normal weight or adequate nutrition with oral intake and in whom enteral tube feeding is not indicated or tolerated. No published studies have evaluated the optimal composition of a PN formula in this clinical situation. Consequently, formulas provided should generally follow accepted clinical guidelines tailored to meet the special needs of a bariatric surgery patient. Cautious monitoring is advised to avoid refeeding complications (see previous section). Evidence-based CPG for the use of PN have been compiled by the American Society for Parenteral and Enteral Nutrition (745 [EL 4], 746 [EL 4]).

9.14.3. Semielemental Oral Feedings

Semielemental oral feedings in the form of nutritional supplements have theoretical advantages attributable to a lower long-chain triglyceride:medium-chain triglyceride content and amino acid/peptide-based nitrogen source. Because they have not been formally evaluated in bariatric

surgery patients, their use is not evidence-based. In fact, there is a large repertoire of enteral nutrition preparations available but without clinical evidence suggesting that one is superior to another in bariatric surgery care. Thus, the choice about which preparation should be tried should be guided by patient preference, patient tolerance, and physician experience.

9.14.4. Inpatient Metabolic Work-up

The hospitalized patient with malnutrition after bariatric surgery should undergo evaluation for the presence of vitamin deficiencies, and appropriate supplementation should be initiated. In the absence of a malabsorptive procedure, other potential causes for malnutrition should be pursued. Potential diagnostic studies include a 72-hour fecal fat collection for fat malabsorption (note that an enteral intake of >100 g of fat daily is required to validate this test), a D-xylose test for carbohydrate malabsorption, a breath test for bacterial overgrowth, and various biochemical assays, such as for fat-soluble vitamins and malabsorption of other specific nutrients. The differential diagnosis includes bacterial overgrowth, celiac sprue, and pancreatic insufficiency to name a few. Bacterial overgrowth responds to rotating antibiotic agents and using probiotics and prebiotics. Celiac sprue responds to use of a gluten-free meal plan, and pancreatic insufficiency responds to supplementation with pancreatic enzymes.

Maladaptive eating behaviors have become increasingly recognized after bariatric surgery. Their presence can contribute to major nutritional deficiencies. If suspected, prompt evaluation by a trained mental health professional should be completed (747 [EL 3]).

9.15. Reoperation

9.15.1. Inadequate Weight Loss

In a prospective, randomized trial, 4% of patients undergoing RYGB had <20% loss of EBW and 21% of patients had <40% loss of EBW at 5 years (504 [EL 4]). In a large nonrandomized study, 8.8% of patients undergoing RYGB had <5% loss of initial weight and 26.5% of patients had <20% loss of initial weight at 10 years (64 [EL 3]). There is no consensus about what constitutes inadequate weight loss after bariatric surgery, but a range from <20% to 40% of EBW has been suggested in the literature.

The initial evaluation in patients with inadequate weight loss should include a thorough nutritional history and radiologic assessment of the pouch integrity. Communication between the upper and lower portions of the stomach may occur in as many as 12% of patients when the stomach is only stapled across to occlude the lumen but still remains in anatomic continuity. This rate can be reduced to 2% with a divided gastric bypass, but division of the stomach does not completely eliminate the problem (527 [EL 3], 706 [EL 3]). When this complication is present, a revisional surgical procedure is reason-

able to consider. Pouch size or stomal diameter may also be important if the pouch is intact; making it smaller or narrowing the anastomotic diameter, however, may not result in further weight loss (748 [EL 3]). Studies are currently under way to evaluate techniques of endoscopic suturing to narrow a dilated gastrojejunal anastomosis (749 [EL 3]). Patients who have undergone a gastroplasty can be considered for revision even if the gastroplasty is intact, inasmuch as further weight loss is common after conversion of gastroplasty to gastric bypass (517 [EL 3]). Some physicians recommend conversion to a malabsorptive distal RYGB for patients with RYGB who do not have adequate weight loss; however, a significant risk of protein-calorie malnutrition is associated with this procedure, and conversion should be considered only for patients with severe, life-threatening obesity (71 [EL 3]). Others have suggested converting a RYGB to a BPD/DS, but data are too few to analyze satisfactorily.

9.15.2. Stricture or Small Bowel Obstruction

Endoscopic dilation has become the preferred method for managing stomal strictures because it is a safe and commonly effective therapy (750 [EL 3], 751 [EL 2]). The reoperation rate from failed endoscopic dilation of stomal stenosis is low (5%). The rate of reoperation for stomal stenosis is less than 1% (752 [EL 3]).

Small bowel obstruction is a common late surgical complication after bariatric surgery. There are several potential causes of such obstruction, including adhesions, strictures, internal hernias, mesenteric volvulus, and intussusception (392 [EL 4], 480 [EL 4], 678 [EL 4], 753 [EL 4], 754 [EL 4], 755 [EL 3]). A rare cause of small bowel obstruction after gastric bypass is intussusception of the common channel (distal small bowel) proximally into the enteroenterostomy (755 [EL 3]). Other potential sites for intussusception are the biliopancreatic or gastric limbs into the enteroenterostomy, although such involvement would be uncommon. After BPD, the surgical rate for enterolysis of adhesions is reported as 1% (125 [EL 3], 756 [EL 3], 757 [EL 4]). Although operation rates are not commonly reported, surgical intervention is often required in the management of strictures at the site of the jejunojejunostomy and internal hernias into mesenteric defects. It is hoped that changes in surgical techniques during laparoscopic procedures (closing of mesenteric defects) will be associated with lower rates of these complications (678 [EL 4]). Overall, the rate of small bowel obstruction after open RYGB is no higher than that after any major gastric operation.

9.15.3. Ulceration

Anastomotic ulceration after bariatric surgery is commonly managed medically and rarely needs reoperation. Surgical modifications to the RYGB, including a smaller gastric pouch containing little or no acid, have been associated with lower prevalences of this complication (758 [EL 3]). Disruption of a stapled gastric pouch or a gastro-

gastric fistula will significantly increase the risk of marginal ulceration (525 [EL 3]).

9.15.4. Surgical Revision

In a series of their first 92 BPD-treated patients, Marceau et al (547 [EL 3]) reported that surgical revision was necessary in 14 patients to diminish diarrhea, improve low serum albumin levels, or both. In 11 of these patients, the common channel was increased from 50 cm to 100 cm and was successful in achieving the aforementioned results, without substantial weight gain (547 [EL 3]). Thus, lengthening of the common channel is generally recommended by most investigators to ameliorate severe malabsorption (125 [EL 3], 208 [EL 2], 515 [EL 3], 567 [EL 3]).

There are several indications for surgical revision. The most common indications are inadequate long-term weight loss in the presence of weight-mediated medical problems, metabolic complications of malabsorptive surgery, or significant side effects, technical complications, or both of the initial procedure. In these patients, revisions are often effective without excessive risks (758-762 [EL 3-4]). Inadequate weight loss is most frequently noted after solely gastric restrictive procedures and often attributed to staple-line dehiscence (758 [EL 3], 763 [EL 3], 764 [EL 3]). Generally, conversion to a RYGB is advised because it has been shown to lead to acceptable weight loss (758 [EL 3], 759 [EL 3], 763 [EL 3], 765 [EL 3], 766 [EL 3]). Roller and Provost (767 [EL 3]) reported their experience involving patients with failed gastric restrictive procedures who underwent revision to a RYGB procedure. They found a 16.7% complication rate and a 54.3% loss of EBW in patients who had undergone multiple revisional procedures, in comparison with a 9.3% complication rate and a 60.6% loss of EBW in patients who had undergone only one revisional procedure (767 [EL 3]).

The jejunoileal bypass is no longer recommended for the treatment of obesity because of the high incidence of serious complications (615 [EL 3]). Nevertheless, it is estimated that more than 100,000 patients underwent this operation in the United States in the past and are at risk for metabolic complications if the bypass is still intact (762 [EL 3]). Reversal of jejunoileal bypass can be performed safely. It can lead to improvement of most metabolic complications, with the exception of the immune complex arthropathy (758 [EL 3], 761 [EL 3]). VBG reversal is generally associated with considerable weight gain. Therefore, conversion to a RYGB is advised (517 [EL 3], 758 [EL 3], 768-770 [EL 3]). Severe protein malnutrition associated with BPD or BPD/DS is an indication for surgical revision involving lengthening of the common and alimentary channels to improve absorption (125 [EL 3], 453 [EL 4], 567 [EL 3]).

Other complications of initial bariatric procedures necessitating surgical revision include stomal stenosis unresponsive to nonsurgical therapy, alkaline-mediated

gastroesophageal reflux, and erosion of hardware specifically with banding procedures. Gastric restrictive procedures can be converted to RYGB with acceptable outcomes in amelioration of these symptoms and weight control (758 [EL 3], 771 [EL 3]). Nonetheless, perioperative complications including PE and anastomotic leaks have been reported (758 [EL 3], 765 [EL 3]). Anastomotic ulcerations after RYGB have become less common as a result of a decrease in the size of the gastric pouch containing little or no acid (758 [EL 3]). In general, there are higher risks for complications after revision of a bariatric procedure, in comparison with the primary procedure; therefore, these revisions must be performed only by experienced bariatric surgeons.

9.15.5. Surgical Reversal

Restoration of normal gut continuity should be performed in the presence of complications not amenable or responsive to surgical revision or conversion with appropriate medical management. Examples of complications necessitating surgical reversal include severe malnutrition, organ failure, or psychiatric emergencies (753 [EL 4]).

9.15.6. Increased Malabsorption Procedure

Laparoscopic reoperation and conversion to a RYGB can successfully induce weight loss and can be performed safely in patients with inadequate restrictive bariatric procedures (72 [EL 3], 772-774 [EL 3]). In patients who have failed to achieve long-term weight loss after a LAGB procedure, conversion to BPD/DS has been successful. This conversion is also safe because the proximal duodenal anastomosis is away from the gastric band, as opposed to performance of a RYGB conversion (775 [EL 3]). In a series of 57 patients who lost an average of 87% weight after conversion from a restrictive procedure to BPD, however, 24% required PN, 22% developed hypoalbuminemia, and 16% had a late bowel obstruction (776 [EL 3]).

A failed RYGB can also be converted to a distal bypass. Among 1,450 patients undergoing RYGB by Sapala et al (777 [EL 3]), 805 had primary operations and 645 were converted from restrictive procedures. In 38 patients who failed to lose weight with the RYGB, conversion to a modified BPD was performed without dismantling the original gastric exclusion, resulting in a sustained weight loss (777 [EL 3]).

10. PHYSICIAN RESOURCES

Interested physicians may refer to several key textbooks, journals, Web sites, and guidelines for information regarding various aspects in the care of bariatric surgical patients (Table 21). In general, the textbooks provide basic concepts, whereas certain journals are replete with pertinent and specific reports. Many of the journal articles contain sound experimental design and valid conclusions, although careful scrutiny is advised before extrapolation

of their results to a specific clinical practice. Web sites are, for the most part, biased toward the experience of the clinical group sponsoring the educational material. Nevertheless, the experiences of these groups, typically regional surgical teams, are worthwhile and can be adapted to any clinical practice. Material on many of the Web sites has been written by the dietitians working with the bariatric surgeons, and their experience is invaluable. Physicians, in general, have not had formal nutrition training. Therefore, nutritional strategies should be reviewed and studied by interested physicians. There are also several symposia on bariatric surgery organized each year in major medical institutions throughout the United States. Lastly, clinical guidelines are generally evidence-based and sponsored by a clinical society or governmental agency, such as the National Institutes of Health. These guidelines are valuable tools for developing a standard of care and monitoring innovations over time.

DISCLOSURE

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Table 21
Educational Resources on Bariatric Surgery

Textbooks

- Buchwald H, Cowan GSM Jr, Pories WJ, eds.** *Surgical Management of Obesity*. Philadelphia, PA: Saunders, 2007.
- DeMaria EJ, Latifi R, Sugerman HJ.** *Laparoscopic Bariatric Surgery: Techniques and Outcomes*. Austin, TX: Landes Bioscience, 2002.
- Farraye F, Forse A, eds.** *Bariatric Surgery: A Primer for Your Medical Practice*. Thorofare, NJ: SLACK Incorporated, 2006.
- Inabnet WB, DeMaria EJ, Ikramuddin S, eds.** *Laparoscopic Bariatric Surgery*. Philadelphia, PA: Lippincott Williams & Wilkins, 2004.
- Mitchell JE, de Zwann M, eds.** *Bariatric Surgery: A Guide for Mental Health Professionals*. New York, NY: Routledge, Taylor & Francis Group, 2005.
- Sugerman HJ, Nguyen N, eds.** *Management of Morbid Obesity*. Philadelphia, PA: Taylor & Francis Group, 2005.

Society Web sites

- | | |
|---|---|
| American Association of Clinical Endocrinologists | http://www.aace.com |
| American Dietetic Association | http://www.eatright.org |
| American Obesity Association | http://www.obesity1.tempdomainname.com/ |
| American Society for Metabolic & Bariatric Surgery | http://www.asbs.org/ |
| Association for Morbid Obesity Support | http://www.obesityhelp.com/ |
| International Federation for the Surgery of Obesity | http://www.obesity-online.com/ifso/ |
| Obesity Action Coalition | http://www.obesityaction.org |
| The Obesity Society | http://www.obesity.org |

Clinical practice guidelines

- Guidelines for the Clinical Application of Laparoscopic Bariatric Surgery
http://www.guideline.gov/summary/summary.aspx?doc_id=4383&nbr=3301&string=bariatric+AND+surgery
- Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report
<http://www.ncbi.nlm.nih.gov/books/bookres.fcgi/obesity/obesity.pdf>
- VA/DoD Clinical Practice Guideline for Management of Overweight and Obesity
http://www.oqp.med.va.gov/cpg/OBE/OBE_CPG/GOL.htm
- SAGES/ASBS Guideline for Laparoscopic and Conventional Surgical Treatment of Morbid Obesity
http://www.asbs.org/html/lab_guidelines.html
- Rationale for the Surgical Treatment of Morbid Obesity
http://www.asbs.org/Newsite07/patients/resources/asbs_rationale.htm
- Guidelines for Granting Privileges in Bariatric Surgery
<http://www.asbs.org/html/about/grantingprivileges.html>
- Suggestions for the Pre-Surgical Psychological Assessment of Bariatric Surgery Candidates
<http://www.asbs.org/html/pdf/PsychPreSurgicalAssessment.pdf>
- A.S.P.E.N. Clinical Guidelines, Standards, and Safe Practices for Parenteral Nutrition
<http://www.nutritioncare.org/lcontent.aspx?id=540>
- Commonwealth of Massachusetts Betsy Lehman Center for Patient Safety and Medical Error Reduction Expert Panel on Weight Loss Surgery, Executive Report, December 12, 2007, Prepublication Copy
http://www.mass.gov/Eeohhs2/docs/dph/patient_safety/weight_loss_executive_report_dec07/pdf

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REFERENCES

Note: All reference sources are followed by an evidence level [EL] rating of 1, 2, 3, or 4, as outlined in Table 5. The strongest evidence levels (EL 1 and EL 2) appear in red for easier recognition.

1. **AAACE/ACE Obesity Task Force.** AAACE/ACE position statement on the prevention, diagnosis, and treatment of obesity. *Endocr Pract.* 1997;3:162-208. [EL 4]
2. **AAACE/ACE Obesity Task Force.** AAACE/ACE position statement on the prevention, diagnosis, and treatment of obesity (1998 revision). *Endocr Pract.* 1998;4:297-350. [EL 4]
3. **National Institutes of Health-National Heart, Lung, and Blood Institute and North American Association for the Study of Obesity.** The practical guide: identification, evaluation, and treatment of overweight and obesity in adults. http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_b.pdf. Published October 2000. Accessed for verification April 8, 2008. [EL 4]
4. **Greenway FL.** Surgery for obesity. *Endocrinol Metab Clin North Am.* 1996;25:1005-1027. [EL 4]
5. **Kremen AJ, Linner JH, Nelson CH.** An experimental evaluation of the nutritional importance of proximal and distal small intestine. *Ann Surg.* 1954;140:439-448. [EL 3]
6. **Balsiger BM, Murr MM, Poggio JL, Sarr MG.** Bariatric surgery: surgery for weight control in patients with morbid obesity. *Med Clin North Am.* 2000;84:477-489. [EL 4]
7. **Griffen WO Jr, Young VL, Stevenson CC.** A prospective comparison of gastric and jejunoileal bypass procedures for morbid obesity. *Ann Surg.* 1977;186:500-509. [EL 3]
8. **Santry HP, Gillen DL, Lauderdale DS.** Trends in bariatric surgical procedures. *JAMA.* 2005;294:1909-1917. [EL 4]
9. **Alt SJ.** Bariatric surgery programs growing quickly nationwide. *Health Care Strateg Manage.* 2001;19:7-23. [EL 4]
10. **DeMaria EJ, Jamal MK.** Surgical options for obesity. *Gastroenterol Clin North Am.* 2005;34:127-142. [EL 4]

11. **Angrisani L, Lorenzo M, Borrelli V.** Laparoscopic adjustable gastric banding versus Roux-en-Y gastric bypass: 5-year results of a prospective randomized trial [with discussion]. *Surg Obes Relat Dis.* 2007;3:127-133. [EL 2]
12. **Santry HP, Lauderdale DS, Cagney KA, Rathouz PJ, Alverdy JC, Chin MH.** Predictors of patient selection in bariatric surgery. *Ann Surg.* 2007;245:59-67. [EL 3]
13. **Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM.** Prevalence of overweight and obesity in the United States, 1999-2004. *JAMA.* 2006;295:1549-1555. [EL 3]
14. **Greenway SE, Greenway FL III, Klein S.** Effects of obesity surgery on non-insulin-dependent diabetes mellitus. *Arch Surg.* 2002;137:1109-1117. [EL 3]
15. **Tichansky DS, DeMaria EJ, Fernandez AZ, et al.** Postoperative complications are not increased in super-obese patients who undergo laparoscopic Roux-en-Y gastric bypass. *Surg Endosc.* 2005;19:939-941. [EL 3]
16. **World Health Organization.** *Obesity: Preventing and Managing the Global Epidemic; Report of a WHO Consultation.* Technical Report Series No. 894. Geneva, Switzerland: World Health Organization, 2000. [EL 4]
17. **Sturm R.** Increases in morbid obesity in the USA: 2000-2005. *Public Health.* 2007;121:492-496. [EL 4]
18. **Sturm R.** Increases in clinically severe obesity in the United States, 1986-2000. *Arch Intern Med.* 2003;163:2146-2148. [EL 4]
19. **Knol JA.** Management of the problem patient after bariatric surgery. *Gastroenterol Clin North Am.* 1994;23:345-369. [EL 4]
20. **Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB.** Years of life lost due to obesity. *JAMA.* 2003;289:187-193. [EL 4]
21. **McTigue K, Larson JC, Valoski A.** Mortality and cardiac and vascular outcomes in extremely obese women. *JAMA.* 2006;296:79-86. [EL 3]
22. **Gastrointestinal surgery for severe obesity: National Institutes of Health Consensus Development Conference Statement.** *Am J Clin Nutr.* 1992;55(2 suppl):615S-619S. [EL 4]
23. **Consensus Development Conference Panel.** NIH conference: gastrointestinal surgery for severe obesity. *Ann Intern Med.* 1991;115:956-961. [EL 4]
24. **Ontario Health Technology Advisory Committee.** Bariatric surgery: report summary. http://www.health.gov.on.ca/english/providers/program/ohtac/tech/reviews/s_um_baria_010105.html. Published January 2005. Accessed for verification April 8, 2008. [EL 4]
25. **National Institutes of Health.** Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. *Obes Res.* 1998;6(suppl 2):51S-209S. [EL 4]
26. **Newer techniques in bariatric surgery for morbid obesity.** *TEC Bull (Online).* 2003;20:4-6. [EL 4]
27. **Blue Cross/Blue Shield.** Special report: the relationship between weight loss and changes in morbidity following bariatric surgery for morbid obesity. http://www.diet.ie/component/option,com_docman/task,doc_view/gid,374/. Published 2003. Accessed for verification April 8, 2008. [EL 4]
28. **Agency for Healthcare Research and Quality.** Pharmacological and surgical treatment of obesity. <http://www.ahrq.gov/clinic/epcsums/obesphsum.pdf>. Published July 2004. Accessed for verification April 8, 2008. [EL 4]
29. **U. S. Preventive Services Task Force, Agency for Healthcare Research and Quality.** Screening and interventions to prevent obesity in adults. <http://www.ahrq.gov/clinic/uspstf/uspsobes.htm>. Published December 2003. Accessed for verification April 8, 2008. [EL 4]
30. **Brechner RJ, Farris C, Harrison S, Tillman K, Salive M, Phurrough S.** Summary of evidence—bariatric surgery. <http://www.cms.hhs.gov/FACA/downloads/id26c.pdf>. Published November 4, 2004. Accessed for verification April 8, 2008. [EL 4]
31. **Avenell A, Broom J, Brown TJ, et al.** Systematic review of the long-term effects and economic consequences of treatments for obesity and implications for health improvement. *Health Technol Assess.* 2004;8:1-182. <http://www.nchta.org/fullmono/mon821.pdf>. Accessed for verification April 25, 2008. [EL 4]
32. **Clegg AJ, Colquitt J, Sidhu MK, Royle P, Loveman E, Walker A.** The clinical effectiveness and cost-effectiveness of surgery for people with morbid obesity: a systematic review and economic evaluation. *Health Technol Assess.* 2002;6:1-153. <http://www.nchta.org/fullmono/mon612.pdf>. Accessed for verification April 25, 2008. [EL 4]
33. **U.S. Department of Health and Human Services.** The Surgeon General's call to action to prevent and decrease overweight and obesity, 2001. <http://www.surgeongeneral.gov/topics/obesity/calltoaction/CalltoAction.pdf>. Accessed for verification April 8, 2008. [EL 4]
34. **Lehman Center Weight Loss Surgery Expert Panel.** Commonwealth of Massachusetts Betsy Lehman Center for Patient Safety and Medical Error Reduction Expert Panel on Weight Loss Surgery: executive report. *Obes Res.* 2005;13:2005-2026. [EL 4]
35. **ECRI Institute.** *Bariatric Surgery for Obesity: Technology Assessment Report.* Plymouth Meeting, PA: Emergency Care Research Institute, 2005. [EL 4]
36. **Brechner RJ, Farris C, Harrison S, Tillman K, Salive M, Phurrough S.** A graded, evidence-based summary of evidence for bariatric surgery. *Surg Obes Relat Dis.* 2005;1:430-441. [EL 4]
37. **Livingston EH, Huerta S, Arthur D, Lee S, De Shields S, Heber D.** Male gender is a predictor of morbidity and age a predictor of mortality for patients undergoing gastric bypass surgery. *Ann Surg.* 2002;236:576-582. [EL 3]
38. **Livingston EH.** Procedure incidence and in-hospital complication rates of bariatric surgery in the United States. *Am J Surg.* 2004;188:105-110. [EL 3]
39. **Flum DR, Dellinger EP.** Impact of gastric bypass operation on survival: a population-based analysis. *J Am Coll Surg.* 2004;199:543-551. [EL 3]
40. **Flum DR, Salem L, Elrod JA, Dellinger EP, Cheadle A, Chan L.** Early mortality among Medicare beneficiaries undergoing bariatric surgical procedures. *JAMA.* 2005;294:1903-1908. [EL 3]
41. **Murr MM, Siadati MR, Sarr MG.** Results of bariatric surgery for morbid obesity in patients older than 50 years. *Obes Surg.* 1995;5:399-402. [EL 2]
42. **Gonzalez R, Lin E, Mattar SG, Venkatesh KR, Smith CD.** Gastric bypass for morbid obesity in patients 50 years or older: is laparoscopic technique safer? *Am Surg.* 2003;69:547-553. [EL 3]
43. **Macgregor AM, Rand CS.** Gastric surgery in morbid obesity: outcome in patients aged 55 and older. *Arch Surg.* 1993;128:1153-1157. [EL 3]
44. **Sugerman HJ, DeMaria EJ, Kellum JM, Sugerman EL, Meador JG, Wolfe LG.** Effects of bariatric surgery in older patients. *Ann Surg.* 2004;240:243-247. [EL 3]
45. **Fatima J, Houghton SG, Iqbal CW, et al.** Bariatric surgery at the extremes of age. *J Gastrointest Surg.* 2006;10:1392-1396. [EL 3]

46. **Centers for Medicare & Medicaid Services.** Medicare expands national coverage for bariatric surgery procedures. <https://www.cms.hhs.gov/apps/media/press/release.asp?Counter=1786>. Press release February 21, 2006. Accessed for verification April 8, 2008. [EL 4]
47. **Department of Veterans Affairs and Department of Defense.** VA/DoD clinical practice guideline for management of overweight and obesity. Version 1.0. http://www.oqp.med.va.gov/cpg/OBE/OBE_CPG/GOL.htm. Published November 2006. Accessed for verification April 8, 2008. [EL 4—evidence-based CPG]
48. **Sarwer DB, Wadden TA, Fabricatore AN.** Psychosocial and behavioral aspects of bariatric surgery. *Obes Res.* 2005;13:639-648. [EL 4]
49. **Alvarado R, Alami RS, Hsu G, et al.** The impact of preoperative weight loss in patients undergoing laparoscopic Roux-en-Y gastric bypass. *Obes Surg.* 2005;15:1282-1286. [EL 3]
50. **Still CD, Benotti P, Wood GC, et al.** Outcomes of preoperative weight loss in high-risk patients undergoing gastric bypass surgery. *Arch Surg.* 2007;142:994-998. [EL 2]
51. **Alami RS, Morton JM, Schuster R, et al.** Is there a benefit to preoperative weight loss in gastric bypass patients? A prospective randomized trial. *Surg Obes Relat Dis.* 2007;3:141-145. [EL 2]
52. **Jamal MK, DeMaria EJ, Johnson JM, et al.** Insurance-mandated preoperative dietary counseling does not improve outcome and increases dropout rates in patients considering gastric bypass surgery for morbid obesity. *Surg Obes Relat Dis.* 2006;2:122-127. [EL 3]
53. **Gibbons LM, Sarwer DB, Crerand CE, et al.** Previous weight loss experiences of bariatric surgery candidates: how much have patients dieted prior to surgery? *Obesity.* 2006;14(suppl 2):70S-76S. [EL 3]
54. **Colles SL, Dixon JB, Marks P, Strauss BJ, O'Brien PE.** Preoperative weight loss with a very-low-energy diet: quantitation of changes in liver and abdominal fat by serial imaging. *Am J Clin Nutr.* 2006;84:304-311. [EL 2]
55. **Schauer PR, Ikramuddin S.** Laparoscopic surgery for morbid obesity. *Surg Clin North Am.* 2001;81:1145-1179. [EL 4]
56. **Dávila-Cervantes A, Borunda D, Domínguez-Cherit G, et al.** Open versus laparoscopic vertical banded gastroplasty: a randomized controlled double blind trial. *Obes Surg.* 2002;12:812-818. [EL 2]
57. **Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J.** Outcomes after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Ann Surg.* 2000;232:515-529. [EL 3]
58. **Fried M, Peskova M.** Gastric banding: advantages and complications; a 5-year and 10-year follow-up. *Obes Surg.* 1995;5:372-374. [EL 3]
59. **Fried M, Peskova M.** Gastric banding in the treatment of morbid obesity. *Hepatogastroenterology.* 1997;44:582-587. [EL 3]
60. **Gorecki P, Wise L, Brolin RE, Champion JK.** Complications of combined gastric restrictive and malabsorptive procedures: part 1. *Curr Surg.* 2003;60:138-144. [EL 4]
61. **Higa KD, Ho T, Boone KB.** Internal hernias after laparoscopic Roux-en-Y gastric bypass: incidence, treatment and prevention. *Obes Surg.* 2003;13:350-354. [EL 3]
62. **Higa KD, Boone KB, Ho T.** Complications of the laparoscopic Roux-en-Y gastric bypass: 1,040 patients—what have we learned? *Obes Surg.* 2000;10:509-513. [EL 2]
63. **O'Brien PE, Dixon JB, Laurie C, et al.** Treatment of mild to moderate obesity with laparoscopic adjustable gastric banding or an intensive medical program: a randomized trial. *Ann Intern Med.* 2006;144:625-633. [EL 2]
64. **Sjöström L, Lindroos AK, Peltonen M, et al (Swedish Obese Subjects Study Scientific Group).** Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med.* 2004;351:2683-2693. [EL 3]
65. **Sjöström L, Narbro K, Sjöström CD, et al (Swedish Obese Subjects Study).** Effects of bariatric surgery on mortality in Swedish obese subjects. *N Engl J Med.* 2007;357:741-752. [EL 3]
66. **Thomusch O, Keck T, Dobschütz EV, Wagner C, Rückauer KD, Hopt UT.** Risk factors for the intermediate outcome of morbid obesity after laparoscopically placed adjustable gastric banding. *Am J Surg.* 2005;189:214-218. [EL 2]
67. **Silecchia G, Greco F, Bacci V, et al.** Results after laparoscopic adjustable gastric banding in patients over 55 years of age. *Obes Surg.* 2005;15:351-356. [EL 3]
68. **O'Brien PE, Dixon JB, Laurie C, Anderson M.** A prospective randomized trial of placement of the laparoscopic adjustable gastric band: comparison of the perigastric and pars flaccida pathways. *Obes Surg.* 2005;15:820-826. [EL 2]
69. **Talieh J, Kirgan D, Fisher BL.** Gastric bypass for morbid obesity: a standard surgical technique by consensus. *Obes Surg.* 1997;7:198-202. [EL 3]
70. **Sugerman HJ, Starkey JV, Birkenhauer R.** A randomized prospective trial of gastric bypass versus vertical banded gastroplasty for morbid obesity and their effects on sweets versus non-sweets eaters. *Ann Surg.* 1987;205:613-624. [EL 2]
71. **Sugerman HJ, Kellum JM, DeMaria EJ.** Conversion of proximal to distal gastric bypass for failed gastric bypass for superobesity [with discussion]. *J Gastrointest Surg.* 1997;1:517-526. [EL 3]
72. **Brolin RE, LaMarca LB, Kenler HA, Cody RP.** Malabsorptive gastric bypass in patients with superobesity. *J Gastrointest Surg.* 2002;6:195-203. [EL 3]
73. **Capella JF, Capella RF.** An assessment of vertical banded gastroplasty-Roux-en-Y gastric bypass for the treatment of morbid obesity. *Am J Surg.* 2002;183:117-123. [EL 3]
74. **Fobi MA, Lee H, Felahy B, Che K, Ako P, Fobi N.** Choosing an operation for weight control, and the transected banded gastric bypass. *Obes Surg.* 2005;15:114-121. [EL 4]
75. **White S, Brooks E, Jurikova L, Stubbs RS.** Long-term outcomes after gastric bypass. *Obes Surg.* 2005;15:155-163. [EL 3]
76. **Mognol P, Chosidow D, Marmuse JP.** Laparoscopic conversion of laparoscopic gastric banding to Roux-en-Y gastric bypass: a review of 70 patients. *Obes Surg.* 2004;14:1349-1353. [EL 3]
77. **Gonzalez R, Gallagher SF, Haines K, Murr MM.** Operative technique for converting a failed vertical banded gastroplasty to Roux-en-Y gastric bypass. *J Am Coll Surg.* 2005;201:366-374. [EL 3]
78. **DeMaria EJ, Sugerman HJ, Kellum JM, Meader JG, Wolfe LG.** Results of 281 consecutive total laparoscopic Roux-en-Y gastric bypasses to treat morbid obesity [with discussion]. *Ann Surg.* 2002;235:640-647. [EL 3]
79. **Scopinaro N, Gianetta E, Civalleri D, Bonalui U, Bachì V.** Bilio-pancreatic bypass for obesity: II. Initial experience in man. *Br J Surg.* 1979;66:618-620. [EL 3]

80. **Noya G, Cossu ML, Coppola M, et al.** Biliopancreatic diversion preserving the stomach and pylorus in the treatment of hypercholesterolemia and diabetes type II: results in the first 10 cases. *Obes Surg.* 1998;8:67-72. [EL 3]
81. **Tataranni PA, Mingrone G, Raguso CA, et al.** Twenty-four-hour energy and nutrient balance in weight stable postobese patients after biliopancreatic diversion. *Nutrition.* 1996;12:239-244. [EL 3]
82. **Hess DS, Hess DW.** Biliopancreatic diversion with duodenal switch. *Obes Surg.* 1998;8:267-282. [EL 3]
83. **DeMeester TR, Fuchs KH, Ball CS, Albertucci M, Smyrk TC, Marcus JN.** Experimental and clinical results with proximal end-to-end duodenojejunosomy for pathologic duodenogastric reflux. *Ann Surg.* 1987;206:414-426. [EL 3]
84. **Marceau P, Hould FS, Simard S, et al.** Biliopancreatic diversion with duodenal switch. *World J Surg.* 1998;22:947-954. [EL 3]
85. **Gabriel SG, Karaindros CA, Papaioannou MA, et al.** Biliopancreatic diversion with duodenal switch combined with laparoscopic adjustable gastric banding. *Obes Surg.* 2005;15:517-522. [EL 3]
86. **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. [EL 3]
87. **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. [EL 3]
88. **Almogy G, Crookes PF, Anthonie GJ.** Longitudinal gastrectomy as a treatment for the high-risk super-obese patient. *Obes Surg.* 2004;14:492-497. [EL 3]
89. **Mognol P, Chosidow D, Marmuse JP.** Laparoscopic gastric bypass versus laparoscopic adjustable gastric banding in the super-obese: a comparative study of 290 patients. *Obes Surg.* 2005;15:76-81. [EL 3]
90. **Mognol P, Chosidow D, Marmuse JP.** Laparoscopic sleeve gastrectomy (LSG): review of a new bariatric procedure and initial results. *Surg Technol Int.* 2006;15:47-52. [EL 3]
91. **Hamoui N, Anthonie GJ, Kaufman HS, Crookes PF.** Sleeve gastrectomy in the high-risk patient. *Obes Surg.* 2006;16:1445-1449. [EL 3]
92. **Cottam D, Qureshi FG, Matter SG, et al.** Laparoscopic sleeve gastrectomy as an initial weight-loss procedure for high-risk patients with morbid obesity. *Surg Endosc.* 2006;20:859-863. [EL 3]
93. **Nguyen NT, Longoria M, Gelfand DV, Sabio A, Wilson SE.** Staged laparoscopic Roux-en-Y: a novel two-stage bariatric operation as an alternative in the super-obese with massively enlarged liver. *Obes Surg.* 2005;15:1077-1081. [EL 4]
94. **Himpens J, Dapri G, Cadiere GB.** A prospective randomized study between laparoscopic gastric banding and laparoscopic isolated sleeve gastrectomy: results after 1 and 3 years. *Obes Surg.* 2006;16:1450-1456. [EL 2]
95. **Milone L, Strong V, Gagner M.** Laparoscopic sleeve gastrectomy is superior to endoscopic intragastric balloon as a first stage procedure for super-obese patients (BMI \geq 50). *Obes Surg.* 2005;15:612-617. [EL 3]
96. **Zingmond DS, McGory ML, Ko CY.** Hospitalization before and after gastric bypass surgery. *JAMA.* 2005;294:1918-1924. [EL 3]
97. **Pratt GM, McLees B, Pories WJ.** The ASBS Bariatric Surgery Centers of Excellence program: a blueprint for quality improvement. *Surg Obes Relat Dis.* 2006;2:497-503. [EL 4]
98. **Zhao Y, Encinosa W.** Bariatric surgery utilization and outcomes in 1998 and 2004. Statistical Brief #23. Agency for Healthcare Research and Quality, Rockville, MD. <http://www.hcup-us.ahrq.gov/reports/statbriefs/sb23.pdf>. Published January 2007. Accessed for verification April 8, 2008. [EL 3]
99. **Buchwald H, Avidor Y, Braunwald E, et al.** Bariatric surgery: a systematic review and meta-analysis [published correction appears in *JAMA.* 2005;293:1728]. *JAMA.* 2004;292:1724-1737. [EL 1]
100. **Nguyen NT, Silver M, Robinson M, et al.** Result of a national audit of bariatric surgery performed at academic centers: a 2004 University HealthSystem Consortium Benchmarking Project. *Arch Surg.* 2006;141:445-449. [EL 3]
101. **DeMaria EJ, Portenier D, Wolfe L.** Obesity surgery mortality risk score: proposal for a clinically useful score to predict mortality risk in patients undergoing gastric bypass. *Surg Obes Relat Dis.* 2007;3:134-140. [EL 4]
102. **Belle S (LABS Consortium).** The NIDDK bariatric surgery clinical research consortium (LABS). *Surg Obes Relat Dis.* 2005;1:145-147. [EL 4]
103. **Pories WJ, MacDonald KG Jr, Morgan EJ, et al.** Surgical treatment of obesity and its effect on diabetes: 10-y follow-up. *Am J Clin Nutr.* 1992;55(2 suppl):582S-585S. [EL 3]
104. **Foley EF, Benotti PN, Borlase BC, Hollingshead J, Blackburn GL.** Impact of gastric restrictive surgery on hypertension in the morbidly obese. *Am J Surg.* 1992;163:294-297. [EL 3]
105. **Gleysteen JJ.** Results of surgery: long-term effects on hyperlipidemia. *Am J Clin Nutr.* 1992;55(2 suppl):591S-593S. [EL 3]
106. **Charuzi I, Lavie P, Peiser J, Peled R.** Bariatric surgery in morbidly obese sleep-apnea patients: short- and long-term follow-up. *Am J Clin Nutr.* 1992;55(2 suppl):594S-596S. [EL 3]
107. **Sugerman HJ, Fairman RP, Sood RK, Engle K, Wolfe L, Kellum JM.** Long-term effects of gastric surgery for treating respiratory insufficiency of obesity. *Am J Clin Nutr.* 1992;55(2 suppl):597S-601S. [EL 3]
108. **Sugerman HJ, DeMaria EJ.** Gastric surgery for morbid obesity. In: Zinner MJ, Schwartz SI, Ellis H, eds. *Maingot's Abdominal Operations.* 10th ed. Stamford, CT: Appleton & Lange, 1997: 1057-1077. [EL 4]
109. **Sugerman H, Windsor A, Bessos M, Wolfe L.** Intra-abdominal pressure, sagittal abdominal diameter, and obesity comorbidity. *J Intern Med.* 1997;241:71-79. [EL 2]
110. **Sugerman HJ, DeMaria EJ, Felton WL III, Nakatsuka M, Sismanis A.** Increased intra-abdominal pressure and cardiac filling pressures in obesity-associated pseudotumor cerebri. *Neurology.* 1997;49:507-511. [EL 2]
111. **Sugerman H, Windsor A, Bessos M, Kellum J, Reines H, DeMaria E.** Effects of surgically induced weight loss on urinary bladder pressure, sagittal abdominal diameter and obesity co-morbidity. *Int J Obes Relat Metab Disord.* 1998;22:230-235. [EL 2]
112. **Sugerman HJ, Felton WL III, Sismanis A, Kellum JM, DeMaria EJ, Sugerman EL.** Gastric surgery for pseudotumor cerebri associated with severe obesity [with discussion]. *Ann Surg.* 1999;229:634-642. [EL 3]
113. **Sugerman HJ.** Effects of increased intra-abdominal pressure in severe obesity. *Surg Clin North Am.* 2001;81:1063-1075, vi. [EL 4]
114. **Lambert DM, Marceau S, Forse RA.** Intra-abdominal pressure in the morbidly obese. *Obes Surg.* 2005;15:1225-1232. [EL 2]

115. **Cummings DE, Overduin J, Foster-Schubert KE.** Gastric bypass for obesity: mechanisms of weight loss and diabetes resolution. *J Clin Endocrinol Metab.* 2004;89:2608-2615. [EL 4]
116. **Cummings DE, Overduin J, Foster-Schubert KE, Carlson MJ.** Role of the bypassed proximal intestine in the anti-diabetic effects of bariatric surgery. *Surg Obes Relat Dis.* 2007;3:109-115. [EL 4]
117. **Bocchieri LE, Meana M, Fisher BL.** A review of psychosocial outcomes of surgery for morbid obesity. *J Psychosom Res.* 2002;52:155-165. [EL 4]
118. **Herpertz S, Kielmann R, Wolf AM, Hebebrand J, Senf W.** Do psychosocial variables predict weight loss or mental health after obesity surgery? A systematic review. *Obes Res.* 2004;12:1554-1569. [EL 4]
119. **Herpertz S, Kielmann R, Wolf AM, Langkafel M, Senf W, Hebebrand J.** Does obesity surgery improve psychosocial functioning? A systematic review. *Int J Obes Relat Metab Disord.* 2003;27:1300-1314. [EL 4]
120. **Maggard MA, Shugarman LR, Suttorp M, et al.** Meta-analysis: surgical treatment of obesity. *Ann Intern Med.* 2005;142:547-559. [EL 1]
121. **Sjöström CD, Peltonen M, Sjöström L.** Blood pressure and pulse pressure during long-term weight loss in the obese: the Swedish Obese Subjects (SOS) Intervention Study. *Obes Res.* 2001;9:188-195. [EL 2]
122. **Pories WJ, Swanson MS, MacDonald KG, et al.** Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. *Ann Surg.* 1995;222:339-350. [EL 3]
123. **Hickey MS, Pories WJ, MacDonald KG Jr, et al.** A new paradigm for type 2 diabetes mellitus: could it be a disease of the foregut [with discussion]? *Ann Surg.* 1998;227:637-644. [EL 3]
124. **Cowan GS Jr, Buffington CK.** Significant changes in blood pressure, glucose, and lipids with gastric bypass surgery. *World J Surg.* 1998;22:987-992. [EL 3]
125. **Scopinaro N, Adami GF, Marinari GM, et al.** Biliopancreatic diversion. *World J Surg.* 1998;22:936-946. [EL 3]
126. **Yashkov YI, Timoshin AD, Opperl TA.** Vertical banded gastroplasty: first experience in Russia. *Obes Surg.* 1997;7:317-320. [EL 3]
127. **Bourdages H, Goldenberg F, Nguyen P, Buchwald H.** Improvement in obesity-associated medical conditions following vertical banded gastroplasty and gastrointestinal bypass. *Obes Surg.* 1994;4:227-231. [EL 3]
128. **DeMaria EJ.** Laparoscopic adjustable silicone gastric banding. *Surg Clin North Am.* 2001;81:1129-1144, vii. [EL 4]
129. **Torquati A, Lutfi R, Abumrad N, Richards WO.** Is Roux-en-Y gastric bypass surgery the most effective treatment for type 2 diabetes mellitus in morbidly obese patients [with discussion]? *J Gastrointest Surg.* 2005;9:1112-1118. [EL 3]
130. **Schauer PR, Burguera B, Ikramuddin S, et al.** Effect of laparoscopic Roux-en-Y gastric bypass on type 2 diabetes mellitus. *Ann Surg.* 2003;238:467-484. [EL 3]
131. **Sanderson I, Deitel M.** Insulin response in patients receiving concentrated infusions of glucose and casein hydrolysate for complete parenteral nutrition. *Ann Surg.* 1974;179:387-394. [EL 3]
132. **Deitel M, Sidhu P, Stone E.** Effect of vertical banded gastroplasty on diabetes in the morbidly obese [abstract]. *Obes Surg.* 1991;1:113-114. [EL 3]
133. **Jensen K, Mason E, Scott D.** Changes in the postoperative hypoglycemic and anti-hypertensive medication requirements in morbidly obese patients after VBG [abstract]. *Obes Surg.* 1991;1:114. [EL 3]
134. **Smith SC, Edwards CB, Goodman GN.** Changes in diabetic management after Roux-en-Y gastric bypass. *Obes Surg.* 1996;6:345-348. [EL 3]
135. **Mingrone G, DeGaetano A, Greco AV, et al.** Reversibility of insulin resistance in obese diabetic patients: role of plasma lipids. *Diabetologia.* 1997;40:599-605. [EL 3]
136. **Herbst CA, Hughes TA, Gwynne JT, Buckwalter JA.** Gastric bariatric operation in insulin-treated adults. *Surgery.* 1984;95:209-214. [EL 3]
137. **Pories WJ, MacDonald KG Jr, Flickinger EG, et al.** Is type II diabetes mellitus (NIDDM) a surgical disease? *Ann Surg.* 1992;215:633-642. [EL 3]
138. **Sirinek KR, O'Dorisio TM, Hill D, Mcfee AS.** Hyperinsulinism, glucose-dependent insulinotropic polypeptide, and the enteroinsular axis in morbidly obese patients before and after gastric bypass. *Surgery.* 1986;100:781-787. [EL 3]
139. **Sugerman HJ, Wolfe LG, Sica DA, Clore JN.** Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. *Ann Surg.* 2003;237:751-756. [EL 3]
140. **Marceau P, Hould FS, Lebel S, Marceau S, Biron S.** Malabsorption obesity surgery. *Surg Clin North Am.* 2001;81:1113-1127. [EL 4]
141. **Dixon JB, O'Brien PE, Playfair J, et al.** Adjustable gastric banding and conventional therapy for type 2 diabetes: a randomized controlled trial. *JAMA.* 2008;299:316-323. [EL 2]
142. **Dixon JB, O'Brien PE.** Health outcomes of severely obese type 2 diabetic subjects 1 year after laparoscopic adjustable gastric banding. *Diabetes Care.* 2002;25:358-363. [EL 2]
143. **Champault A, Duwat O, Polliand C, Rizk N, Champault GG.** Quality of life after laparoscopic gastric banding: prospective study (152 cases) with a follow-up of 2 years. *Surg Laparosc Endosc Percutan Tech.* 2006;16:131-136. [EL 2]
144. **Pontiroli AE, Pizzocri P, Librenti MC, et al.** Laparoscopic adjustable gastric banding for the treatment of morbid (grade 3) obesity and its metabolic complications: a three-year study. *J Clin Endocrinol Metab.* 2002;87:3555-3561. [EL 2]
145. **Pontiroli AE, Folli F, Paganelli M, et al.** Laparoscopic gastric banding prevents type 2 diabetes and arterial hypertension and induces their remission in morbid obesity: a 4-year case-controlled study. *Diabetes Care.* 2005;28:2703-2709. [EL 2]
146. **Long SD, O'Brien K, MacDonald KG Jr, et al.** Weight loss in severely obese subjects prevents the progression of impaired glucose tolerance to type II diabetes: a longitudinal interventional study. *Diabetes Care.* 1994;17:372-375. [EL 2]
147. **Muscelli E, Mingrone G, Camastra S, et al.** Differential effect of weight loss on insulin resistance in surgically treated obese patients. *Am J Med.* 2005;118:51-57. [EL 3]
148. **Mattar SG, Velcu LM, Rabinovitz M, et al.** Surgically-induced weight loss significantly improves nonalcoholic fatty liver disease and the metabolic syndrome [with discussion]. *Ann Surg.* 2005;242:610-620. [EL 3]
149. **Tataranni PA, Mingrone G, Greco AV, et al.** Glucose-induced thermogenesis in postobese women who have undergone biliopancreatic diversion. *Am J Clin Nutr.* 1994;60:320-326. [EL 3]

150. Scopinaro N, Marinari GM, Camerini GB, Papadia FS, Adami GF. Specific effects of biliopancreatic diversion on the major components of metabolic syndrome: a long-term follow-up. *Diabetes Care.* 2005;28:2406-2411. [EL 3]
151. MacDonald KG Jr, Long SD, Swanson MS, et al. The gastric bypass operation reduces the progression and mortality of non-insulin-dependent diabetes mellitus. *J Gastrointest Surg.* 1997;1:213-220. [EL 3]
152. Sowemimo OA, Yood SM, Courtney J, et al. Natural history of morbid obesity without surgical intervention. *Surg Obes Relat Dis.* 2007;3:73-77. [EL 3]
153. Christou NV, Sampalis JS, Liberman M, et al. Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients [with discussion]. *Ann Surg.* 2004;240:416-424. [EL 3]
154. Adams TD, Gress RE, Smith SC, et al. Long-term mortality following gastric bypass surgery. *N Engl J Med.* 2007;357:753-761. [EL 2]
155. Busetto L, Miribelli D, Petroni ML, et al. Comparative long-term mortality after laparoscopic adjustable gastric banding versus nonsurgical controls. *Surg Obes Relat Dis.* 2007;3:496-502. [EL 3]
156. Doherty C. Vertical banded gastroplasty. *Surg Clin North Am.* 2001;81:1097-1112. [EL 4]
157. Polk HC Jr, Simpson CJ, Simmons BP, Alexander JW. Guidelines for prevention of surgical wound infection. *Arch Surg.* 1983;118:1213-1217. [EL 4]
158. Van Dielen FM, Soeters PB, de Brauw LM, Greve JW. Laparoscopic adjustable gastric banding versus open vertical banded gastroplasty: a prospective randomized trial. *Obes Surg.* 2005;15:1292-1298. [EL 2]
159. Nocca D, Aggarwal R, Blanc P, et al. Laparoscopic vertical banded gastroplasty: a multicenter prospective study of 200 procedures. *Surg Endosc.* 2007;21:870-874. [EL 2]
160. van Mastrigt GA, van Dielen FM, Severens JL, Voss GB, Greve JW. One-year cost-effectiveness of surgical treatment of morbid obesity: vertical banded gastroplasty versus Lap-Band. *Obes Surg.* 2006;16:75-84. [EL 2]
161. O'Brien PE, McPhail T, Chaston TB, Dixon JB. Systematic review of medium-term weight loss after bariatric operations. *Obes Surg.* 2006;16:1032-1040. [EL 1]
162. Chapman AE, Kiroff G, Game P, et al. Laparoscopic adjustable gastric banding in the treatment of obesity—a systematic literature review. *Surgery.* 2004;135:326-351. [EL 4]
163. Puzifferri N, Austrheim-Smith IT, Wolfe BM, Wilson SE, Nguyen NT. Three-year follow-up of a prospective randomized trial comparing laparoscopic versus open gastric bypass. *Ann Surg.* 2006;243:181-188. [EL 2]
164. Jan JC, Hong D, Pereira N, Patterson EJ. Laparoscopic adjustable gastric banding versus laparoscopic gastric bypass for morbid obesity: a single-institution comparison study of early results [with discussion]. *J Gastrointest Surg.* 2005;9:30-41. [EL 2]
165. Kim TH, Daud A, Ude AO, et al. Early U.S. outcomes of laparoscopic gastric bypass versus laparoscopic adjustable silicone gastric banding for morbid obesity. *Surg Endosc.* 2006;20:202-209. [EL 3]
166. Ren CJ, Horgan S, Ponce J. US experience with the LAP-BAND system. *Am J Surg.* 2002;184:46S-50S. [EL 4]
167. Belachew M, Belva PH, Desai C. Long-term results of laparoscopic adjustable gastric banding for the treatment of morbid obesity. *Obes Surg.* 2002;12:564-568. [EL 3]
168. Melvin WS. Roux-en-Y gastric bypass is the operation of choice for bariatric surgery [with discussion]. *J Gastrointest Surg.* 2004;8:398-400, 404-405. [EL 4]
169. Biertho L, Steffen R, Branson R, et al. Management of failed adjustable gastric banding. *Surgery.* 2005;137:33-41. [EL 3]
170. Ceelen W, Walder J, Cardon A, et al. Surgical treatment of severe obesity with a low-pressure adjustable gastric band: experimental data and clinical results in 625 patients. *Ann Surg.* 2003;237:10-16. [EL 3]
171. Dargent J. Surgical treatment of morbid obesity by adjustable gastric band: the case for a conservative strategy in the case of failure—a 9-year series. *Obes Surg.* 2004;14:986-990. [EL 3]
172. Fox SR, Fox KM, Srikanth MS, Rumbaut R. The Lap-Band system in a North American population. *Obes Surg.* 2003;13:275-280. [EL 3]
173. Frigg A, Peterli R, Peters T, Ackermann C, Tondelli P. Reduction in co-morbidities 4 years after laparoscopic adjustable gastric banding. *Obes Surg.* 2004;14:216-223. [EL 3]
174. Greenslade J, Kow L, Toouli J. Surgical management of obesity using a soft adjustable gastric band. *ANZ J Surg.* 2004;74:195-199. [EL 2]
175. Holloway JA, Forney GA, Gould DE. The Lap-Band is an effective tool for weight loss even in the United States. *Am J Surg.* 2004;188:659-662. [EL 2]
176. Mittermair RP, Weiss H, Aigner F, Weissenboeck E, Lanthaler M, Nehoda H. Is it necessary to deflate the adjustable gastric band for subsequent operations? *Am J Surg.* 2003;185:50-53. [EL 2]
177. Ponce J, Haynes B, Paynter S, et al. Effect of Lap-Band-induced weight loss on type 2 diabetes mellitus and hypertension. *Obes Surg.* 2004;14:1335-1342. [EL 3]
178. Steffen R, Biertho L, Ricklin T, Picc G, Horber FF. Laparoscopic Swedish adjustable gastric banding: a five-year prospective study. *Obes Surg.* 2003;13:404-411. [EL 2]
179. Suter M, Giusti V, Worreth M, Héraief E, Calmes JM. Laparoscopic gastric banding: a prospective, randomized study comparing the Lap-Band and the SAGB; early results. *Ann Surg.* 2005;241:55-62. [EL 2]
180. Vertruyen M. Experience with Lap-Band System up to 7 years. *Obes Surg.* 2002;12:569-572. [EL 3]
181. Victorzon M, Tolonen P. Intermediate results following laparoscopic adjustable gastric banding for morbid obesity [with discussion]. *Dig Surg.* 2002;19:354-358. [EL 2]
182. Weiner R, Blanco-Engert R, Weiner S, Matkowitz R, Schaefer L, Pomhoff I. Outcome after laparoscopic adjustable gastric banding—8 years experience. *Obes Surg.* 2003;13:427-434. [EL 2]
183. Zinzindohoue F, Chevallier JM, Douard R, et al. Laparoscopic gastric banding: a minimally invasive surgical treatment for morbid obesity; prospective study of 500 consecutive patients. *Ann Surg.* 2003;237:1-9. [EL 3]
184. Naef M, Naef U, Mouton WG, Wagner HE. Outcome and complications after laparoscopic Swedish adjustable gastric banding: 5-year results of a prospective clinical trial. *Obes Surg.* 2007;17:195-201. [EL 2]
185. Lee WJ, Wang W, Wei PL, Huang MT. Weight loss and improvement of obesity-related illness following laparoscopic adjustable gastric banding procedure for morbidly obese patients in Taiwan. *J Formos Med Assoc.* 2006;105:887-894. [EL 2]
186. Parikh MS, Fielding GA, Ren CJ. U.S. experience with 749 laparoscopic adjustable gastric bands: intermediate outcomes. *Surg Endosc.* 2005;19:1631-1635. [EL 2]
187. Sugerma HJ, Londrey GL, Kellum JM, et al. Weight loss with vertical banded gastroplasty and Roux-Y gastric bypass for morbid obesity with selective versus random assignment. *Am J Surg.* 1989;157:93-102. [EL 2]

188. **Smith SC, Edwards CB, Goodman GN.** Symptomatic and clinical improvement in morbidly obese patients with gastroesophageal reflux disease following Roux-en-Y gastric bypass. *Obes Surg.* 1997;7:479-484. [EL 3]
189. **Nguyen NT, Goldman C, Rosenquist CJ, et al.** Laparoscopic versus open gastric bypass: a randomized study of outcomes, quality of life, and costs [with discussion]. *Am Surg.* 2001;234:279-291. [EL 2]
190. **Brolin RE, Kenler HA, Gorman JH, Cody RP.** Long-limb gastric bypass in the superobese: a prospective randomized study. *Ann Surg.* 1992;215:387-395. [EL 2]
191. **Wittgrove AC, Clark GW.** Laparoscopic gastric bypass, Roux-en-Y—500 patients: technique and results, with 3-60 month follow-up. *Obes Surg.* 2000;10:233-239. [EL 2]
192. **Olbers T, Fagevik-Olsén M, Maleckas A, Lönroth H.** Randomized clinical trial of laparoscopic Roux-en-Y gastric bypass versus laparoscopic vertical banded gastroplasty for obesity. *Br J Surg.* 2005;92:557-562. [EL 2]
193. **Balsiger BM, Kennedy FP, Abu-Lebdeh HS, et al.** Prospective evaluation of Roux-en-Y gastric bypass as primary operation for medically complicated obesity. *Mayo Clin Proc.* 2000;75:673-680. [EL 2]
194. **Skroubis G, Anesidis S, Kehagias I, Mead N, Vagenas K, Kalfarentzos F.** Roux-en-Y gastric bypass versus a variant of biliopancreatic diversion in a non-superobese population: prospective comparison of the efficacy and the incidence of metabolic deficiencies. *Obes Surg.* 2006;16:488-495. [EL 2]
195. **Gleysteen JJ.** Four-year weight loss Roux-Y-gastric bypass: anastomotic reinforcement not additive. *Gastroenterol Clin North Am.* 1987;16:525-527. [EL 3]
196. **Higa KD, Ho T, Boone KB.** Laparoscopic Roux-en-Y gastric bypass: technique and 3-year follow-up. *J Laparoendosc Adv Surg Tech A.* 2001;11:377-382. [EL 2]
197. **Jones KB Jr.** Experience with the Roux-en-Y gastric bypass, and commentary on current trends. *Obes Surg.* 2000;10:183-185. [EL 3]
198. **Linner JH.** Comparative effectiveness of gastric bypass and gastroplasty: a clinical study. *Arch Surg.* 1982;117:695-700. [EL 3]
199. **Oh C, Kim HJ, Oh S.** Weight loss following transected gastric bypass with proximal Roux-en-Y. *Obes Surg.* 1997;7:142-147. [EL 3]
200. **Rabkin RA.** Distal gastric bypass/duodenal switch procedure, Roux-en-Y gastric bypass and biliopancreatic diversion in a community practice. *Obes Surg.* 1998;8:53-59. [EL 3]
201. **Alami RS, Morton JM, Sanchez BR, Curet MJ, Wren SM, Safadi BY.** Laparoscopic Roux-en-Y gastric bypass at a Veterans Affairs and high-volume academic facilities: a comparison of institutional outcomes. *Am J Surg.* 2005;190:821-825. [EL 3]
202. **Inabnet WB, Quinn T, Gagner M, Urban M, Pomp A.** Laparoscopic Roux-en-Y gastric bypass in patients with BMI <50: a prospective randomized trial comparing short and long limb lengths. *Obes Surg.* 2005;15:51-57. [EL 2]
203. **Lee WJ, Yu PJ, Wang W, Chen TC, Wei PL, Huang MT.** Laparoscopic Roux-en-Y versus mini-gastric bypass for the treatment of morbid obesity: a prospective randomized controlled clinical trial. *Ann Surg.* 2005;242:20-28. [EL 2]
204. **Nelson LG, Lopez PP, Haines K, et al.** Outcomes of bariatric surgery in patients ≥ 65 years. *Surg Obes Relat Dis.* 2006;2:384-388. [EL 3]
205. **Obeid F, Falvo A, Dabideen H, Stocks J, Moore M, Wright M.** Open Roux-en-Y gastric bypass in 925 patients without mortality. *Am J Surg.* 2005;189:352-356. [EL 3]
206. **Choban PS, Jackson B, Poplawski S, Bistolarides P.** Bariatric surgery for morbid obesity: why, when, how, where, and then what? *Cleve Clin J Med.* 2002;69:897-903. [EL 4]
207. **Scopinaro N, Gianetta E, Adami GF, et al.** Biliopancreatic diversion for obesity at eighteen years. *Surgery.* 1996;119:261-268. [EL 3]
208. **Dolan K, Hatzifotis M, Newbury L, Fielding G.** A comparison of laparoscopic adjustable gastric banding and biliopancreatic diversion in superobesity. *Obes Surg.* 2004;14:165-169. [EL 2]
209. **Baltasar A, Bou R, Bengochea M, et al.** Duodenal switch: an effective therapy for morbid obesity—intermediate results. *Obes Surg.* 2001;11:54-58. [EL 3]
210. **Lagacé M, Marceau P, Marceau S, et al.** Biliopancreatic diversion with a new type of gastrectomy: some previous conclusions revisited. *Obes Surg.* 1995;5:411-418. [EL 3]
211. **Nanni G, Balduzzi GF, Capoluongo R, et al.** Biliopancreatic diversion: clinical experience. *Obes Surg.* 1997;7:26-29. [EL 3]
212. **Hess DS, Hess DW, Oakley RS.** The biliopancreatic diversion with the duodenal switch: results beyond 10 years. *Obes Surg.* 2005;15:408-416. [EL 4]
213. **Dolan K, Fielding G.** Biliopancreatic diversion following failure of laparoscopic adjustable gastric banding. *Surg Endosc.* 2004;18:60-63. [EL 4]
214. **Dolan K, Hatzifotis M, Newbury L, Lowe N, Fielding G.** A clinical and nutritional comparison of biliopancreatic diversion with and without duodenal switch. *Ann Surg.* 2004;240:51-56. [EL 3]
215. **Bajardi G, Ricevuto G, Mastrandrea G, et al.** Surgical treatment of morbid obesity with biliopancreatic diversion and gastric banding: report on an 8-year experience involving 235 cases. *Ann Chir.* 2000;125:155-162. [EL 3]
216. **Anthone GJ, Lord RV, DeMeester TR, Crookes PF.** The duodenal switch operation for the treatment of morbid obesity. *Ann Surg.* 2003;238:618-627. [EL 3]
217. **Hudson SM, Dixon JB, O'Brien PE.** Sweet eating is not a predictor of outcome after Lap-Band placement: can we finally bury the myth? *Obes Surg.* 2002;12:789-794. [EL 3]
218. **Lindroos AK, Lissner L, Sjöström L.** Weight change in relation to intake of sugar and sweet foods before and after weight reducing gastric surgery. *Int J Obes Relat Metab Disord.* 1996;20:634-643. [EL 3]
219. **American Association of Clinical Endocrinologists Ad Hoc Task Force for Standardized Production of Clinical Practice Guidelines.** American Association of Clinical Endocrinologists protocol for standardized production of clinical practice guidelines. *Endocr Pract.* 2004;10:353-361. [EL 4]
220. **Johnson N.** New approaches to the development of treatment guidelines. *Formulary.* 1998;33:665-678. [EL 4]
221. **Bloomberg RD, Fleischman A, Nalle JE, Herron DM, Kini S.** Nutritional deficiencies following bariatric surgery: what have we learned? *Obes Surg.* 2005;15:145-154. [EL 4]
222. **Fujioka K.** Follow-up of nutritional and metabolic problems after bariatric surgery. *Diabetes Care.* 2005;28:481-484. [EL 4]
223. **Mason ME, Jalagani H, Vinik AI.** Metabolic complications of bariatric surgery: diagnosis and management issues. *Gastroenterol Clin North Am.* 2005;34:25-33. [EL 4]
224. **Alvarez-Leite JI.** Nutrient deficiencies secondary to bariatric surgery. *Curr Opin Clin Nutr Metab Care.* 2004;7:569-575. [EL 4]

225. **Cohen RV, Schiavon CA, Pinheiro JS, Correa JL, Rubino F.** Duodenal-jejunal bypass for the treatment of type 2 diabetes in patients with body mass index of 22-34 kg/m²: a report of 2 cases. *Surg Obes Relat Dis.* 2007;3:195-197. [EL 3]
226. **Rubino F, Gagner M, Gentileschi P, et al.** The early effect of the Roux-en-Y gastric bypass on hormones is involved in body weight regulation and glucose metabolism. *Ann Surg.* 2004;240:236-242. [EL 3]
227. **Wickremesekera K, Miller G, Naotunne TD, Knowles G, Stubbs RS.** Loss of insulin resistance after Roux-en-Y gastric bypass surgery: a time course study. *Obes Surg.* 2005;15:474-481. [EL 3]
228. **MacDonald PE, El-Kholy M, Riedel MJ, Salapatek AM, Light PE, Wheeler MB.** The multiple actions of GLP-1 on the process of glucose-stimulated insulin secretion. *Diabetes.* 2002;51(suppl 3):S434-S442. [EL 4]
229. **de Paula AL, Macedo AL, Prudente AS, Queiroz L, Schraibman V, Pinus J.** Laparoscopic sleeve gastrectomy with ileal interposition ("neuroendocrine brake")—pilot study of a new operation. *Surg Obes Relat Dis.* 2006;2:464-467. [EL 3]
230. **Infanger D, Baldinger R, Branson R, Barbier T, Steffen R, Horber FF.** Effect of significant intermediate-term weight loss on serum leptin levels and body composition in severely obese subjects. *Obes Surg.* 2003;13:879-888. [EL 2]
231. **Holdstock C, Engström BE, Ohrvall M, Lind L, Sundbom M, Karlsson FA.** Ghrelin and adipose tissue regulatory peptides: effect of gastric bypass surgery in obese humans. *J Clin Endocrinol Metab.* 2003;88:3177-3183. [EL 3]
232. **Guldstrand M, Ahrén B, Adamson U.** Improved beta-cell function after standardized weight reduction in severely obese subjects. *Am J Physiol Endocrinol Metab.* 2003;284:E557-E565. [EL 3]
233. **Faraj M, Havel PJ, Phélis S, Blank D, Sniderman AD, Cianflone K.** Plasma acylation-stimulating protein, adiponectin, leptin, and ghrelin before and after weight loss induced by gastric bypass surgery in morbidly obese subjects. *J Clin Endocrinol Metab.* 2003;88:1594-1602. [EL 3]
234. **Vendrell J, Broch M, Vilarrasa N, et al.** Resistin, adiponectin, ghrelin, leptin, and proinflammatory cytokines: relationships in obesity. *Obes Res.* 2004;12:962-971. [EL 2]
235. **Rubino F, Gagner M.** Potential of surgery for curing type 2 diabetes mellitus. *Ann Surg.* 2002;236:554-559. [EL 4]
236. **Rubino F, Marescaux J.** Effect of duodenal-jejunal exclusion in a non-obese animal model of type 2 diabetes: a new perspective for an old disease. *Ann Surg.* 2004;239:1-11. [EL 4]
237. **Rubino F, Zizzari P, Tomasetto C, et al.** The role of the small bowel in the regulation of circulating ghrelin levels and food intake in the obese Zucker rat. *Endocrinology.* 2005;146:1745-1751. [EL 4]
238. **Dixon JB, Schachter LM, O'Brien PE.** Polysomnography before and after weight loss in obese patients with severe sleep apnea. *Int J Obes.* 2005;29:1048-1054. [EL 3]
239. **Peiser J, Ovnat A, Uwyyed K, Lavie P, Charuzi I.** Cardiac arrhythmias during sleep in morbidly obese sleep-apneic patients before and after gastric bypass surgery. *Clin Cardiol.* 1985;8:519-521. [EL 3]
240. **Sugerman HJ, Fairman RP, Baron PL, Kwentus JA.** Gastric surgery for respiratory insufficiency of obesity. *Chest.* 1986;90:81-86. [EL 3]
241. **Sugerman HJ, Baron PL, Fairman RP, Evans CR, Vetrovec GW.** Hemodynamic dysfunction in obesity hypoventilation syndrome and the effects of treatment with surgically induced weight loss [with discussion]. *Ann Surg.* 1988;207:604-613. [EL 3]
242. **Charuzi I, Fraser D, Peiser J, Ovnat A, Lavie P.** Sleep apnea syndrome in the morbidly obese undergoing bariatric surgery. *Gastroenterol Clin North Am.* 1987;16:517-519. [EL 4]
243. **Rasheid S, Banasiak M, Gallagher SF, et al.** Gastric bypass is an effective treatment for obstructive sleep apnea in patients with clinically significant obesity. *Obes Surg.* 2003;13:58-61. [EL 2]
244. **Kalra M, Inge T.** Effect of bariatric surgery on obstructive sleep apnoea in adolescents. *Paediatr Respir Rev.* 2006;7:260-267. [EL 4]
245. **Fritscher LG, Mottin CC, Canani S, Chatkin JM.** Obesity and obstructive sleep apnea-hypopnea syndrome: the impact of bariatric surgery. *Obes Surg.* 2007;17:95-99. [EL 4]
246. **Dixon JB, Chapman L, O'Brien P.** Marked improvement in asthma after Lap-Band surgery for morbid obesity. *Obes Surg.* 1999;9:385-389. [EL 3]
247. **Dixon JB, O'Brien PE.** Lipid profile in the severely obese: changes with weight loss after Lap-Band surgery. *Obes Res.* 2002;10:903-910. [EL 3]
248. **Gleysteen JJ, Barboriak JJ.** Improvement in heart disease risk factors after gastric bypass. *Arch Surg.* 1983;118:681-684. [EL 3]
249. **Buffington CK, Cowan GS Jr, Smith H.** Significant changes in the lipid-lipoprotein status of premenopausal morbidly obese females following gastric bypass surgery. *Obes Surg.* 1994;4:328-335. [EL 3]
250. **Wolf AM, Beisiegel U, Kortner B, Kuhlmann HW.** Does gastric restriction surgery reduce the risks of metabolic diseases? *Obes Surg.* 1998;8:9-13. [EL 3]
251. **Busetto L, Pisent C, Rinaldi D, et al.** Variation in lipid levels in morbidly obese patients operated with the Lap-Band adjustable gastric banding system: effects of different levels of weight loss. *Obes Surg.* 2000;10:569-577. [EL 2]
252. **Brolin RE, Bradley LJ, Wilson AC, Cody RP.** Lipid risk profile and weight stability after gastric restrictive operations for morbid obesity. *J Gastroenterol Surg.* 2000;4:464-469. [EL 2]
253. **Sjöström CD, Lissner L, Wedel H, Sjöström L.** Reduction in incidence of diabetes, hypertension and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS Intervention Study. *Obes Res.* 1999;7:477-484. [EL 1]
254. **Lubrano C, Cornoldi A, Pili M, et al.** Reduction of risk factors for cardiovascular diseases in morbid-obese patients following biliary-intestinal bypass: 3 years' follow-up. *Int J Obes Relat Metab Disord.* 2004;28:1600-1606. [EL 3]
255. **Palomar R, Fernández-Fresnedo G, Dominguez-Diez A, et al.** Effects of weight loss after biliopancreatic diversion on metabolism and cardiovascular profile. *Obes Surg.* 2005;15:794-798. [EL 3]
256. **Corradini SG, Eramo A, Lubrano C, et al.** Comparison of changes in lipid profile after bilio-intestinal bypass and gastric banding in patients with morbid obesity. *Obes Surg.* 2005;15:367-377. [EL 3]
257. **Zlabeck JA, Grimm MS, Larson CJ, Mathiason MA, Lambert PJ, Kothari SN.** The effect of laparoscopic gastric bypass surgery on dyslipidemia in severely obese patients. *Surg Obes Relat Dis.* 2005;1:537-542. [EL 2]

258. Vogel JA, Franklin BA, Zalesin KC, et al. Reduction in predicted coronary heart disease risk after substantial weight reduction after bariatric surgery. *Am J Cardiol.* 2007;99:222-226. [EL 3]
259. Williams DB, Hagedorn JC, Lawson EH, et al. Gastric bypass reduces biochemical cardiac risk factors. *Surg Obes Relat Dis.* 2007;3:8-13. [EL 2]
260. Dixon JB, O'Brien P. A disparity between conventional lipid and insulin resistance markers at body mass index levels greater than 34 kg/m². *Int J Obes Relat Metab Disord.* 2001;25:793-797. [EL 2]
261. Carson JL, Ruddy ME, Duff AE, Holmes NJ, Cody RP, Broolin RE. The effect of gastric bypass surgery on hypertension in morbidly obese patients [published correction appears in *Arch Intern Med.* 1994;154:1770]. *Arch Intern Med.* 1994;154:193-200. [EL 3]
262. Ben-Dov I, Grossman E, Stein A, Shachor D, Gaides M. Marked weight reduction lowers resting and exercise blood pressure in morbidly obese subjects. *Am J Hypertens.* 2000;13:251-255. [EL 3]
263. Fernstrom JD, Courcoulas AP, Houck PR, Fernstrom MH. Long-term changes in blood pressure in extremely obese patients who have undergone bariatric surgery. *Arch Surg.* 2006;141:276-283. [EL 3]
264. Sjöström CD, Peltonen M, Wedel H, Sjöström L. Differentiated long-term effects of intentional weight loss on diabetes and hypertension. *Hypertension.* 2000;36:20-25. [EL 2]
265. Messerli FH, Sundgaard-Riise K, Reisin ED, et al. Dimorphic cardiac adaptation to obesity and arterial hypertension. *Ann Intern Med.* 1983;99:757-761. [EL 3]
266. Alpert MA, Lambert CR, Terry BE, et al. Influence of left ventricular mass on left ventricular diastolic filling in normotensive morbid obesity. *Am Heart J.* 1995;130:1068-1073. [EL 2]
267. Karason K, Wallentin I, Larsson B, Sjöström L. Effects of obesity and weight loss on left ventricular mass and relative wall thickness: survey and intervention study. *BMJ.* 1997;315:912-916. [EL 3]
268. Karason K, Wallentin I, Larsson B, Sjöström L. Effects of obesity and weight loss on cardiac function and valvular performance. *Obes Res.* 1998;6:422-429. [EL 2]
269. Kanoupakis E, Michaloudis D, Fraidakis O, Parthenakis F, Vardas P, Melissas J. Left ventricular function and cardiopulmonary performance following surgical treatment of morbid obesity. *Obes Surg.* 2001;11:552-558. [EL 3]
270. Cunha LdeC, da Cunha CL, de Souza AM, Chiminacio Neto N, Pereira RS, Suplicy HL. Evolutive echocardiographic study of the structural and functional heart alterations in obese individuals after bariatric surgery [article in Portuguese]. *Arq Bras Cardiol.* 2006;87:615-622. [EL 3]
271. Leichman JG, Aguilar D, King TM, et al. Improvements in systemic metabolism, anthropometrics, and left ventricular geometry 3 months after bariatric surgery. *Surg Obes Relat Dis.* 2006;2:592-599. [EL 3]
272. Iyengar S, Leier CV. Rescue bariatric surgery for obesity-induced cardiomyopathy. *Am J Med.* 2006;119:e5-e6. [EL 3]
273. Manson JE, Colditz GA, Stampfer MJ, et al. A prospective study of obesity and risk of coronary heart disease in women. *N Engl J Med.* 1990;322:882-889. [EL 2]
274. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. *N Engl J Med.* 1995;333:677-685. [EL 2]
275. Sampalis JS, Sampalis F, Christou N. Impact of bariatric surgery on cardiovascular and musculoskeletal morbidity. *Surg Obes Relat Dis.* 2006;2:587-591. [EL 2]
276. Frezza EE, Ikramuddin S, Gourash W, et al. Symptomatic improvement in gastroesophageal reflux disease (GERD) following laparoscopic Roux-en-Y gastric bypass. *Surg Endosc.* 2002;16:1027-1031. [EL 3]
277. Nelson LG, Gonzalez R, Haines K, Gallagher SF, Murr MM. Amelioration of gastroesophageal reflux symptoms following Roux-en-Y gastric bypass for clinically significant obesity [with discussion]. *Am Surg.* 2005;71:950-954. [EL 3]
278. Lenglinger J, Eisler M, Riegler M. Obesity and GERD: implications and consequences for bariatric surgery? *Am J Gastroenterol.* 2005;100:2600-2601. [EL 4]
279. Cobey F, Oelschlager B. Complete regression of Barrett's esophagus after Roux-en-Y gastric bypass. *Obes Surg.* 2005;15:710-712. [EL 3]
280. Deitel M, Khanna RK, Hagen J, Ilves R. Vertical banded gastroplasty as an antireflux procedure. *Am J Surg.* 1988;155:512-516. [EL 3]
281. Gholam PM, Kotler DP, Flancbaum LJ. Liver pathology in morbidly obese patients undergoing Roux-en-Y gastric bypass surgery. *Obes Surg.* 2002;12:49-51. [EL 3]
282. Shaffer EA. Bariatric surgery: a promising solution for nonalcoholic steatohepatitis in the very obese. *J Clin Gastroenterol.* 2006;40(3 suppl 1):S44-S50. [EL 4]
283. Adams LA, Angulo P. Treatment of non-alcoholic fatty liver disease. *Postgrad Med J.* 2006;82:315-322. [EL 4]
284. Dixon JB. Surgical treatment for obesity and its impact on non-alcoholic steatohepatitis. *Clin Liver Dis.* 2007;11:141-154, ix-x. [EL 4]
285. Dixon JB, Bhathal PS, Hughes NR, O'Brien PE. Nonalcoholic fatty liver disease: improvement in liver histological analysis with weight loss. *Hepatology.* 2004;39:1647-1654. [EL 2]
286. Ranlov I, Hardt F. Regression of liver steatosis following gastroplasty or gastric bypass for morbid obesity. *Digestion.* 1990;47:208-214. [EL 3]
287. Silverman EM, Sapala JA, Appelman HD. Regression of hepatic steatosis in morbidly obese persons after gastric bypass. *Am J Clin Pathol.* 1995;104:23-31. [EL 2]
288. Luyckx FH, Desaive C, Thiry A, et al. Liver abnormalities in severely obese subjects: effect of drastic weight loss after gastroplasty. *Int J Obes Relat Metab Disord.* 1998;22:222-226. [EL 3]
289. Duchini A, Brunson ME. Roux-en-Y gastric bypass for recurrent nonalcoholic steatohepatitis in liver transplant recipients with morbid obesity. *Transplantation.* 2001;72:156-159. [EL 3]
290. Clark JM, Alkhuraishi AR, Solga SF, Alli P, Diehl AM, Magnuson TH. Roux-en-Y gastric bypass improves liver histology in patients with non-alcoholic fatty liver disease. *Obes Res.* 2005;13:1180-1186. [EL 3]
291. Barker KB, Palekar NA, Bowers SP, Goldberg JE, Pulcini JP, Harrison SA. Non-alcoholic steatohepatitis: effect of Roux-en-Y gastric bypass surgery. *Am J Gastroenterol.* 2006;101:368-373. [EL 3]
292. Jaskiewicz K, Raczynska S, Rzepko R, Sledziński Z. Nonalcoholic fatty liver disease treated by gastroplasty. *Dig Dis Sci.* 2006;51:21-26. [EL 3]
293. Mathurin P, Gonzalez F, Kerdraon O, et al. The evolution of severe steatosis after bariatric surgery is related to insulin resistance. *Gastroenterology.* 2006;130:1617-1624. [EL 2]
294. Ehrmann DA. Polycystic ovary syndrome. *N Engl J Med.* 2005;352:1223-1236. [EL 4]

295. Nestler JE, Jakubowicz DJ. Lean women with polycystic ovary syndrome respond to insulin reduction with decreases in ovarian P450c17 alpha activity and serum androgens. *J Clin Endocrinol Metab.* 1997;82:4075-4079. [EL 2]
296. Legro RS, Barnhart HX, Schlaff WD, et al (Cooperative Multicenter Reproductive Medicine Network). Clomiphene, metformin, or both for infertility in the polycystic ovary syndrome. *N Engl J Med.* 2007;356:551-566. [EL 1]
297. Dixon JB, O'Brien PE. Neck circumference a good predictor of raised insulin and free androgen index in obese premenopausal women: changes with weight loss. *Clin Endocrinol (Oxf).* 2002;57:769-778. [EL 2]
298. Deitel M, To TB, Stone E, Sutherland DJ, Wilk EJ. Sex hormonal changes accompanying loss of massive excess weight. *Gastroenterol Clin North Am.* 1987;16:511-515. [EL 4]
299. Escobar-Morreale HF, Botella-Carretero JI, Alvarez-Blasco F, Sancho J, San Millán JL. The polycystic ovary syndrome associated with morbid obesity may resolve after weight loss induced by bariatric surgery. *J Clin Endocrinol Metab.* 2005;90:6364-6369. [EL 3]
300. Eid GM, Cottam DR, Velcu LM, et al. Effective treatment of polycystic ovarian syndrome with Roux-en-Y gastric bypass. *Surg Obes Relat Dis.* 2005;1:77-80. [EL 3]
301. Teitelman M, Grotgut CA, Williams NN, Lewis JD. The impact of bariatric surgery on menstrual patterns. *Obes Surg.* 2006;16:1457-1463. [EL 3]
302. Kopp HP, Kryzanowska K, Scherthner GH, Kriwanek S, Scherthner G. Relationship of androgens to insulin resistance and chronic inflammation in morbidly obese premenopausal women: studies before and after vertical banded gastroplasty. *Obes Surg.* 2006;16:1214-1220. [EL 2]
303. Alagna S, Cossu ML, Gallo P, et al. Biliopancreatic diversion: long-term effects on gonadal function in severely obese men. *Surg Obes Relat Dis.* 2006;2:82-86. [EL 3]
304. Strain GW, Zumoff B. The effect of bariatric surgery on the abnormalities of the pituitary-gonadal axis in obese men. *Surg Obes Relat Dis.* 2006;2:75-77. [EL 4]
305. Dixon JB, Dixon ME, O'Brien PE. Pregnancy after Lap-Band surgery: management of the band to achieve healthy weight outcomes. *Obes Surg.* 2001;11:59-65. [EL 3]
306. Dixon JB, Dixon ME, O'Brien PE. Birth outcomes in obese women after laparoscopic adjustable gastric banding. *Obstet Gynecol.* 2005;106(5, pt 1):965-972. [EL 2]
307. Friedman D, Cunco S, Valenzano M, et al. Pregnancies in an 18-year follow-up after biliopancreatic diversion. *Obes Surg.* 1995;5:308-313. [EL 3]
308. Wittgrove AC, Jester L, Wittgrove P, Clark GW. Pregnancy following gastric bypass for morbid obesity. *Obes Surg.* 1998;8:461-464. [EL 3]
309. Skull AJ, Slater GH, Duncombe JE, Fielding GA. Laparoscopic adjustable banding in pregnancy: safety, patient tolerance and effect on obesity-related pregnancy outcomes. *Obes Surg.* 2004;14:230-235. [EL 3]
310. Sheiner E, Levy A, Silverberg D, et al. Pregnancy after bariatric surgery is not associated with adverse perinatal outcome. *Am J Obstet Gynecol.* 2004;190:1335-1340. [EL 3]
311. Dao T, Kuhn J, Ehmer D, Fisher T, McCarty T. Pregnancy outcomes after gastric-bypass surgery. *Am J Surg.* 2006;192:762-766. [EL 3]
312. Kral JG, Biron S, Simard S, et al. Large maternal weight loss from obesity surgery prevents transmission of obesity to children who were followed from 2 to 18 years. *Pediatrics.* 2006;118:1644-1649. [EL 3]
313. Sugerman HJ, Sugerman EL, Wolfe L, Kellum JM Jr, Schweitzer MA, DeMaria EJ. Risks and benefits of gastric bypass in morbidly obese patients with severe venous stasis disease. *Ann Surg.* 2001;234:41-46. [EL 3]
314. Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ. Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion [with discussion]. *J Trauma.* 1996;40:936-943. [EL 4]
315. Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ. A proposed relationship between increased intra-abdominal, intrathoracic, and intracranial pressure. *Crit Care Med.* 1997;25:496-503. [EL 4]
316. Rosenberg ML, Corbett JJ, Smith C, et al. Cerebrospinal fluid diversion procedures in pseudotumor cerebri. *Neurology.* 1993;43:1071-1072. [EL 3]
317. Nadkarni T, Rekate HL, Wallace D. Resolution of pseudotumor cerebri after bariatric surgery for related obesity: case report. *J Neurosurg.* 2004;101:878-880. [EL 4]
318. Chandra V, Dutta S, Albanese CT, Shepard E, Farrales-Nguyen S, Morton J. Clinical resolution of severely symptomatic pseudotumor cerebri after gastric bypass in an adolescent. *Surg Obes Relat Dis.* 2007;3:198-200. [EL 3]
319. Bump RC, Sugerman HJ, Fantl JA, McClish DK. Obesity and lower urinary tract function in women: effect of surgically induced weight loss. *Am J Obstet Gynecol.* 1992;167:392-397. [EL 3]
320. Richter HE, Burgio KL, Clements RH, Goode PS, Redden DT, Varner RE. Urinary and anal incontinence in morbidly obese women considering weight loss surgery. *Obstet Gynecol.* 2005;106:1272-1277. [EL 3]
321. Deitel M, Stone E, Kassam HA, Wilk EJ, Sutherland DJ. Gynecologic-obstetric changes after loss of massive excess weight following bariatric surgery. *J Am Coll Nutr.* 1988;7:147-153. [EL 3]
322. Nevitt MC. Obesity outcomes in disease management: clinical outcomes for osteoarthritis. *Obes Res.* 2002;10(suppl 1):33S-37S. [EL 4]
323. Winiarsky R, Barth P, Lotke P. Total knee arthroplasty in morbidly obese patients. *J Bone Joint Surg Am.* 1998;80:1770-1774. [EL 3]
324. McKee MD, Waddell JP. Intramedullary nailing of femoral fractures in morbidly obese patients. *J Trauma.* 1994;36:208-210. [EL 3]
325. Böstman OM. Body mass index and height in patients requiring surgery for lumbar intervertebral disc herniation. *Spine.* 1993;18:851-854. [EL 3]
326. Parvizi J, Trousdale RT, Sarr MG. Total joint arthroplasty in patients surgically treated for morbid obesity. *J Arthroplasty.* 2000;15:1003-1008. [EL 3]
327. Peltonen M, Lindroos AK, Torgerson JS. Musculoskeletal pain in the obese: a comparison with a general population and long-term changes after conventional and surgical obesity treatment. *Pain.* 2003;104:549-557. [EL 3]
328. Abu-Abeid S, Wishnitzer N, Szold A, Liebergall M, Manor O. The influence of surgically-induced weight loss on the knee joint [published correction appears in *Obes Surg.* 2006;16:530]. *Obes Surg.* 2005;15:1437-1442. [EL 2]
329. Hooper MM, Stellato TA, Hallowell PT, Seitz BA, Moskowitz RW. Musculoskeletal findings in obese subjects before and after weight loss following bariatric surgery. *Int J Obes (Lond).* 2007;31:114-120. [EL 3]

330. **Melissas J, Volakakis E, Hadjipavlou A.** Low-back pain in morbidly obese patients and the effect of weight loss following surgery. *Obes Surg.* 2003;13:389-393. [EL 3]
331. **Serés L, Lopez-Ayerbe J, Coll R, et al.** Increased exercise capacity after surgically induced weight loss in morbid obesity. *Obes Res.* 2006;14:273-279. [EL 2]
332. **Maniscalco M, Zedda A, Giardiello C, et al.** Effect of bariatric surgery on the six-minute walk test in severe uncomplicated obesity. *Obes Surg.* 2006;16:836-841. [EL 3]
333. **Teasdale N, Hue O, Marcotte J, et al.** Reducing weight increases postural stability in obese and morbid obese men. *Int J Obes (Lond).* 2007;31:153-160. [EL 3]
334. **Larsen F.** Psychosocial function before and after gastric banding surgery for morbid obesity: a prospective psychiatric study. *Acta Psychiatr Scand Suppl.* 1990;359:1-57. [EL 3]
335. **Black DW, Goldstein RB, Mason EE.** Prevalence of mental disorder in 88 morbidly obese bariatric clinic patients. *Am J Psychiatry.* 1992;149:227-234. [EL 3]
336. **Powers PS, Rosemurgy A, Boyd F, Perez A.** Outcome of gastric restriction procedures: weight, psychiatric diagnoses, and satisfaction. *Obes Surg.* 1997;7:471-477. [EL 3]
337. **Sarwer DB, Cohn NI, Gibbons LM, et al.** Psychiatric diagnoses and psychiatric treatment among bariatric surgery candidates. *Obes Surg.* 2004;14:1148-1156. [EL 3]
338. **Adami GF, Gandolfo P, Bauer B, Scopinaro N.** Binge eating in massively obese patients undergoing bariatric surgery. *Int J Eat Disord.* 1995;17:45-50. [EL 3]
339. **Hsu LK, Sullivan SP, Benotti PN.** Eating disturbances and outcome of gastric bypass surgery: a pilot study. *Int J Eat Disord.* 1997;21:385-390. [EL 3]
340. **Hsu LK, Benotti PN, Dwyer J, et al.** Nonsurgical factors that influence the outcome of bariatric surgery: a review. *Psychosom Med.* 1998;60:338-346. [EL 4]
341. **Hsu LK, Mulliken B, McDonagh B, et al.** Binge eating disorder in extreme obesity. *Int J Obes Relat Metab Disord.* 2002;26:1398-1403. [EL 3]
342. **Huang CS, Farraye FA.** Endoscopy in the bariatric surgical patient. *Gastroenterol Clin North Am.* 2005;34:151-166. [EL 4]
343. **Kalarchian MA, Wilson GT, Brolin RE, Bradley L.** Binge eating in bariatric surgery patients. *Int J Eat Disord.* 1998;23:89-92. [EL 3]
344. **Kalarchian MA, Wilson GT, Brolin RE, Bradley L.** Assessment of eating disorders in bariatric surgery candidates: self-report questionnaire versus interview. *Int J Eat Disord.* 2000;28:465-469. [EL 3]
345. **Kalarchian MA, Marcus MD, Wilson GT, Labouvie EW, Brolin RE, LaMarca LB.** Binge eating among gastric bypass patients at long-term follow-up. *Obes Surg.* 2002;12:270-275. [EL 3]
346. **Powers PS, Perez A, Boyd F, Rosemurgy A.** Eating pathology before and after bariatric surgery: a prospective study. *Int J Eat Disord.* 1999;25:293-300. [EL 3]
347. **Allison KC, Wadden TA, Sarwer DB, et al.** Night eating syndrome and binge eating disorder among persons seeking bariatric surgery: prevalence and related features. *Obesity (Silver Spring).* 2006;14(suppl 2):77S-82S. [EL 3]
348. **de Zwaan M, Mitchell JE, Howell LM, et al.** Characteristics of morbidly obese patients before gastric bypass surgery. *Compr Psychiatry.* 2003;44:428-434. [EL 3]
349. **Fabricatore AN, Wadden TA, Sarwer DB, Faith MS.** Health-related quality of life and symptoms of depression in extremely obese persons seeking bariatric surgery. *Obes Surg.* 2005;15:304-309. [EL 3]
350. **Wadden TA, Sarwer DB, Womble LG, Foster GD, McGuckin BG, Schimmel A.** Psychosocial aspects of obesity and obesity surgery. *Surg Clin North Am.* 2001;81:1001-1024. [EL 4]
351. **Fontaine KR, Barofsky I.** Obesity and health-related quality of life. *Obes Rev.* 2001;2:173-182. [EL 4]
352. **Sarwer DB, Thompson JK, Cash TF.** Body image and obesity in adulthood. *Psychiatr Clin North Am.* 2005;28:69-87, viii. [EL 4]
353. **Puhl R, Brownell KD.** Bias, discrimination, and obesity. *Obes Res.* 2001;9:788-805. [EL 4]
354. **Klesges RC, Klem ML, Hanson CL, et al.** The effects of applicant's health status and qualifications on simulating hiring decisions. *Int J Obes.* 1990;14:527-535. [EL 3]
355. **Pingitore R, Dugoni BL, Tindale RS, Spring B.** Bias against overweight job applicants in a simulated employment interview. *J Appl Psychol.* 1994;79:909-917. [EL 3]
356. **van Hout GC, Boekestein P, Fortuin FA, Pelle AJ, van Heck GL.** Psychosocial functioning following bariatric surgery. *Obes Surg.* 2006;16:787-794. [EL 4]
357. **Rand CS, Macgregor A, Hankins G.** Gastric bypass surgery for obesity: weight loss, psychosocial outcome, and morbidity one and three years later. *South Med J.* 1986;79:1511-1514. [EL 3]
358. **Dixon JB, Dixon ME, O'Brien PE.** Body image: appearance orientation and evaluation in the severely obese; changes with weight loss. *Obes Surg.* 2002;12:65-71. [EL 3]
359. **Dziurowicz-Kozłowska A, Lisik W, Wierzbicki Z, Kosieradzki M.** Health-related quality of life after surgical treatment of obesity. *J Physiol Pharmacol.* 2005;56(suppl 6):127-134. [EL 3]
360. **Mamplakou E, Komesidou V, Bissias Ch, Papakonstantinou A, Melissas J.** Psychological condition and quality of life in patients with morbid obesity before and after surgical weight loss. *Surg Obes.* 2005;15:1177-1184. [EL 3]
361. **Velcu LM, Adolphine R, Mourelo R, Cottam DR, Angus LD.** Weight loss, quality of life and employment status after Roux-en-Y gastric bypass: a 5-year analysis. *Surg Obes Relat Dis.* 2006;1:413-416. [EL 3]
362. **Sanchez-Santos R, Del Barrio MJ, Gonzalez C, et al.** Long-term health-related quality of life following gastric bypass: influence of depression. *Obes Surg.* 2006;16:580-585. [EL 3]
363. **Mitchell JE, Lancaster KL, Burgard MA, et al.** Long-term follow-up of patients' status after gastric bypass. *Obes Surg.* 2001;11:464-468. [EL 3]
364. **Rand CS, Kuldau JM, Robbins L.** Surgery for obesity and marriage quality. *JAMA.* 1982;247:1419-1422. [EL 3]
365. **Peeters A, O'Brien PE, Laurie C, et al.** Substantial intentional weight loss and mortality in the severely obese. *Ann Surg.* 2007;246:1028-1033. [EL 3]
366. **Kushner RF, Roth JL.** Assessment of the obese patient. *Endocrinol Metab Clin North Am.* 2003;32:915-933. [EL 4]
367. **Schwartz TL, Nihalani N, Jindal S, Virk S, Jones N.** Psychiatric medication-induced obesity: a review. *Obes Rev.* 2004;5:115-121. [EL 4]
368. **Schwartz TL, Nihalani N, Virk S, Jindal S, Chilton M.** Psychiatric medication-induced obesity: treatment options. *Obes Rev.* 2004;5:233-238. [EL 4]
369. **Wadden TA, Foster GD.** Weight and lifestyle inventory (WALDI). *Obesity (Silver Spring).* 2006;14(suppl 2):99S-118S. [EL 4]

370. **Sarwer DB, Fabricatore AN, Wadden TA.** The behavioral evaluation of bariatric surgery candidates. *Obes Manag.* 2006;2:103-109. [EL 4]
371. **Wadden TA, Sarwer DB.** Behavioral assessment of candidates for bariatric surgery: a patient-oriented approach. *Obesity (Silver Spring).* 2006;14(suppl 2):53S-62S. [EL 4]
372. **Foster GD, Wadden TA, Phelan S, Sarwer DB, Sanderson RS.** Obese patients' perceptions of treatment outcomes and the factors that influence them. *Arch Intern Med.* 2001;161:2133-2139. [EL 3]
373. **Sogg S, Mori DL.** The Boston interview for gastric bypass: determining the psychological suitability of surgical candidates. *Obes Surg.* 2004;14:370-380. [EL 4]
374. **Fabricatore AN, Crerand CE, Wadden TA, Sarwer DB, Krasucki JL.** How do mental health professionals evaluate candidates for bariatric surgery? Survey results. *Obes Surg.* 2006;16:567-573. [EL 3]
375. **Bauchowitz AU, Gonder-Frederick LA, Olbrisch ME, et al.** Psychosocial evaluation of bariatric surgery candidates: a survey of present practices. *Psychosom Med.* 2005;67:825-832. [EL 3]
376. **Larsson U, Karlsson J, Sullivan M.** Impact of overweight and obesity on health-related quality of life—a Swedish population study. *Int J Obes Relat Metab Disord.* 2002;26:417-424. [EL 3]
377. **Sullivan M, Karlsson J, Sjöström L, et al.** Swedish Obese Subjects (SOS)—an intervention study of obesity: baseline evaluation of health and psychosocial functioning in the first 1743 subjects examined. *Int J Obes Relat Metab Disord.* 1993;17:503-512. [EL 3]
378. **Camps MA, Zervos E, Goode S, Rosemurgy AS.** Impact of bariatric surgery on body image perception and sexuality in morbidly obese patients and their partners. *Obes Surg.* 1996;6:356-360. [EL 3]
379. **Karlsson J, Sjöström L, Sullivan M.** Swedish Obese Subjects (SOS)—an intervention study of obesity: two-year follow-up of health-related quality of life (HRQL) and eating behavior after gastric surgery for severe obesity. *Int J Obes Relat Metab Disord.* 1998;22:113-126. [EL 1]
380. **Pawlow LA, O'Neil PM, White MA, Byrne TK.** Findings and outcomes of psychological evaluations of gastric bypass applicants [with discussion]. *Surg Obes Relat Dis.* 2005;1:523-529. [EL 3]
381. **Surrat PM, Findley LJ.** Clinical manifestations and diagnosis of obesity hypoventilation syndrome. UpToDate, 2003. <http://www.uptodate.com/online>. Accessed for verification April 22, 2008. [EL 4]
382. **Raff H, Findling JW.** A physiologic approach to diagnosis of the Cushing syndrome. *Ann Intern Med.* 2003;138:980-991. [EL 4]
383. **Dunaif A, Lobo RA.** Toward optimal health: the experts discuss polycystic ovary syndrome. *J Womens Health Gend Based Med.* 2002;11:579-584. [EL 4]
384. **American Gastroenterological Association.** American Gastroenterological Association medical position statement: nonalcoholic fatty liver disease. *Gastroenterology.* 2002;123:1702-1704. [EL 4]
385. **Clark JM, Diehl AM.** Nonalcoholic fatty liver disease an underrecognized cause of cryptogenic cirrhosis. *JAMA.* 2003;289:3000-3004. [EL 3]
386. **Dindo D, Muller MK, Weber M, Clavien PA.** Obesity in general elective surgery. *Lancet.* 2003;361:2032-2035. [EL 3]
387. **American Society of Anesthesiologists Task Force on Preanesthesia Evaluation.** Practice advisory for preanesthesia evaluation: a report by the American Society of Anesthesiologists Task Force on Preanesthesia Evaluation. *Anesthesiology.* 2002;96:485-496. [EL 4]
388. **Flancbaum L, Belsley S, Drake V, Colarusso T, Tayler E.** Preoperative nutritional status of patients undergoing Roux-en-Y gastric bypass for morbid obesity. *J Gastrointest Surg.* 2006;10:1033-1037. [EL 3]
389. **Madan AK, Orth WS, Tichansky DS, Ternovits CA.** Vitamin and trace mineral levels after laparoscopic gastric bypass. *Obes Surg.* 2006;16:603-606. [EL 3]
390. **Zeni TM, Frantzides CT, Mahr C, et al.** Value of preoperative upper endoscopy in patients undergoing laparoscopic gastric bypass. *Obes Surg.* 2006;16:142-146. [EL 3]
391. **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. [EL 3]
392. **Brolin RE.** Gastric bypass. *Surg Clin North Am.* 2001;81:1077-1095. [EL 4]
393. **Sauerland S, Angrisani L, Belachew M, et al (European Association for Endoscopic Surgery).** Obesity surgery: evidence-based guidelines of the European Association for Endoscopic Surgery (EAES). *Surg Endosc.* 2005;19:200-221. [EL 4]
394. **O'Brien PE, Brown WA, Smith A, McMurrick PJ, Stephens M.** Prospective study of a laparoscopically placed, adjustable gastric band in the treatment of morbid obesity. *Br J Surg.* 1999;86:113-118. [EL 2]
395. **Buchwald H.** A bariatric surgery algorithm. *Obes Surg.* 2002;12:733-746. [EL 4]
396. **Fielding GA.** Laparoscopic adjustable gastric banding for massive superobesity (>60 body mass index kg/m²). *Surg Endosc.* 2003;17:1541-1545. [EL 3]
397. **Parikh MS, Shen R, Weiner M, Siegel N, Ren CJ.** Laparoscopic bariatric surgery in super-obese patients (BMI >50) is safe and effective: a review of 332 patients. *Obes Surg.* 2005;15:858-863. [EL 3]
398. **Hall JC, Watts JM, O'Brien PE, et al.** Gastric surgery for morbid obesity: the Adelaide Study. *Ann Surg.* 1990;211:419-427. [EL 3]
399. **Howard L, Malone M, Michalek A, Carter J, Alger S, Van Woert J.** Gastric bypass and vertical banded gastroplasty: a prospective randomized comparison and 5-year follow-up. *Obes Surg.* 1995;5:55-60. [EL 2]
400. **MacLean LD, Rhode BM, Forse RA, Nohr R.** Surgery for obesity—an update of a randomized trial. *Obes Surg.* 1995;5:145-150. [EL 2]
401. **Weber M, Muller MK, Bucher T, et al.** Laparoscopic gastric bypass is superior to laparoscopic gastric banding for treatment of morbid obesity. *Ann Surg.* 2004;240:975-982. [EL 2]
402. **Jan JC, Hong D, Bardaro SJ, July LV, Patterson EJ.** Comparative study between laparoscopic adjustable gastric banding and laparoscopic gastric bypass: single-institution, 5-year experience in bariatric surgery [published correction appears in *Surg Obes Relat Dis.* 2007;3:203]. *Surg Obes Relat Dis.* 2007;3:42-50. [EL 2]
403. **Baltasar A, del Rio J, Escrivá C, Arlandis F, Martínez R, Serra C.** Preliminary results of the duodenal switch. *Obes Surg.* 1997;7:500-504. [EL 3]
404. **Michielson D, Van Hee R, Hendrickx L.** Complications of biliopancreatic diversion surgery as proposed by Scopinaro in the treatment of morbid obesity. *Obes Surg.* 1996;6:416-420. [EL 3]
405. **Antal SC.** Prevention and reversal of liver damage following biliopancreatic diversion for obesity. *Obes Surg.* 1994;4:285-290. [EL 3]

406. **Torres JC.** Prevention and reversal of liver damage following bypass diversion for obesity. *Obes Surg.* 1995;5:81. [EL 3]
407. **Kral JG, Thung SN, Biron S, et al.** Effects of surgical treatment of the metabolic syndrome on liver fibrosis and cirrhosis. *Surgery.* 2004;135:48-58. [EL 2]
408. **Martinez Castro R, Baltasar A, Vidal V, Sánchez Cuenca J, Liedó JL.** Gastric emptying in patients with morbid obesity treated with a duodenal switch [article in Spanish]. *Rev Esp Enferm Dig.* 1997;89:413-414. [EL 3]
409. **Marceau P, Biron S, Bourque RA, Potvin M, Hould FS, Simard S.** Biliopancreatic diversion with a new type of gastrectomy. *Obes Surg.* 1993;3:29-35. [EL 4]
410. **Rabkin RA, Rabkin JM, Metcalf B, Lazo M, Rossi M, Lehmanbecker LB.** Laparoscopic technique for performing duodenal switch with gastric reduction. *Obes Surg.* 2003;13:263-268. [EL 3]
411. **Sugerman HJ.** Bariatric surgery for severe obesity. *J Assoc Acad Minor Phys.* 2001;12:129-136. [EL 4]
412. **Azagra JS, Goergen M, Ansay J, et al.** Laparoscopic gastric reduction surgery: preliminary results of a randomized, prospective trial of laparoscopic vs open vertical banded gastroplasty. *Surg Endosc.* 1999;13:555-558. [EL 2]
413. **de Wit LT, Mathus-Vliegen L, Hey C, Rademaker B, Gouma DJ, Obertop H.** Open versus laparoscopic adjustable silicone gastric banding: a prospective randomized trial for treatment of morbid obesity [with discussion]. *Ann Surg.* 1999;230:800-807. [EL 2]
414. **Luján JA, Frutos MD, Hernández Q, et al.** Laparoscopic versus open gastric bypass in the treatment of morbid obesity: a randomized prospective study. *Ann Surg.* 2004;239:433-437. [EL 2]
415. **Sundbom M, Gustavsson S.** Randomized clinical trial of hand-assisted laparoscopic versus open Roux-en-Y gastric bypass for the treatment of morbid obesity. *Br J Surg.* 2004;91:418-423. [EL 2]
416. **Nguyen NT, Ho HS, Palmer LS, Wolfe BM.** A comparison study of laparoscopic versus open gastric bypass for morbid obesity. *J Am Coll Surg.* 2000;191:149-157. [EL 3]
417. **Nguyen NT, Ho HS, Fleming NW, et al.** Cardiac function during laparoscopic vs open gastric bypass. *Surg Endosc.* 2002;16:78-83. [EL 2]
418. **Marema RT.** Laparoscopic Roux-en-Y gastric bypass: a step-by-step approach. *J Am Coll Surg.* 2005;200:979-982. [EL 4]
419. **Wittgrove AC, Clark GW.** Laparoscopic gastric bypass, Roux-en-Y: experience of 27 cases, with 3-18 months follow-up. *Obes Surg.* 1996;6:54-57. [EL 3]
420. **Wittgrove AC, Clark GW, Schubert KR.** Laparoscopic gastric bypass, Roux-en-Y: technique and results in 75 patients with 3-30 months follow-up. *Obes Surg.* 1996;6:500-504. [EL 3]
421. **Marema RT, Perez M, Buffington CK.** Comparison of the benefits and complications between laparoscopic and open Roux-en-Y gastric bypass surgeries. *Surg Endosc.* 2005;19:525-530. [EL 3]
422. **Kim WW, Gagner M, Kini S, Inabnet WB, Quinn T, Herron D.** Laparoscopic vs. open biliopancreatic diversion with duodenal switch: a comparative study. *J Gastrointest Surg.* 2003;7:552-557. [EL 3]
423. **Benotti PN, Burchard KW, Kelly JJ, Thayer BA.** Obesity. *Arch Surg.* 2004;139:406-414. [EL 4]
424. **Schauer P, Ikramuddin S, Hamad G, Gourash W.** The learning curve for laparoscopic Roux-en-Y gastric bypass is 100 cases. *Surg Endosc.* 2003;17:212-215. [EL 3]
425. **Shikora SA, Kim JJ, Tarnoff ME, Raskin E, Shore R.** Laparoscopic Roux-en-Y gastric bypass: results and learning curve of a high-volume academic program. *Arch Surg.* 2005;140:362-367. [EL 3]
426. **Ballantyne GH, Ewing D, Capella RF, et al.** The learning curve measured by operating times for laparoscopic and open gastric bypass: roles of surgeon's experience, institutional experience, body mass index and fellowship training. *Obes Surg.* 2005;15:172-182. [EL 3]
427. **AAACE Diabetes Mellitus Clinical Practice Guidelines Task Force.** American Association of Clinical Endocrinologists medical guidelines for clinical practice for the management of diabetes mellitus. *Endocr Pract.* 2007;13(suppl 1):1-68. [EL 4]
428. **Vora AC, Saleem TM, Polomano RC, et al.** Improved perioperative glycemic control by continuous insulin infusion under supervision of an endocrinologist does not increase costs in patients with diabetes. *Endocr Pract.* 2004;10:112-118. [EL 3]
429. **Pomposelli JJ, Baxter JK III, Babineau TJ, et al.** Early postoperative glucose control predicts nosocomial infection rate in diabetic patients. *JPEN J Parenter Enteral Nutr.* 1998;22:77-81. [EL 3]
430. **American Diabetes Association.** Standards of medical care in diabetes—2006 [published correction appears in *Diabetes Care.* 2006;29:1192]. *Diabetes Care.* 2006;29(suppl 1):S4-S42. [EL 4]
431. **Dronge AS, Perkal MF, Kancir S, Concato J, Aslan M, Rosenthal RA.** Long-term glycemic control and postoperative infectious complications. *Arch Surg.* 2006;141:375-380. [EL 3]
432. **Raftopoulos Y, Gagné DJ, Pappasavvas P, et al.** Improvement of hypothyroidism after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Obes Surg.* 2004;14:509-513. [EL 3]
433. **Szomstein S, Avital S, Brasesco O, Mehran A, Cabral JM, Rosenthal R.** Laparoscopic gastric bypass in patients on thyroid replacement for subnormal thyroid function—prevalence and short-term outcome. *Obes Surg.* 2004;14:95-97. [EL 3]
434. **Moulin de Moraes CM, Mancini MC, de Melo ME, et al.** Prevalence of subclinical hypothyroidism in a morbidly obese population and improvement after weight loss induced by Roux-en-Y gastric bypass. *Obes Surg.* 2005;15:1287-1291. [EL 3]
435. **Helfand M, Redfern CC.** Clinical guideline, part 2: screening for thyroid disease; an update. American College of Physicians [published correction appears in *Ann Intern Med.* 1999;130:246]. *Ann Intern Med.* 1998;129:144-158. [EL 4]
436. **National Cholesterol Education Program.** Third report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): ATP III at-a-glance; quick desk reference. <http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.htm>. Accessed for verification April 21, 2008. [EL 4]
437. **Eagle KA, Berger PB, Calkins H, et al (American College of Cardiology/American Heart Association Task Force on Practice Guidelines [Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery]).** ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery) [published correction appears in *Circulation.* 2006;113:e486]. *Circulation.* 2002;105:1257-1267. [EL 4]

438. **Auerbach A, Goldman L.** Assessing and reducing the cardiac risk of noncardiac surgery. *Circulation.* 2006;113:1361-1376. [EL 4]
439. **Lee TH, Marcantonio ER, Mangione CM, et al.** Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation.* 1999;100:1043-1049. [EL 2]
440. **U.S. Department of Health & Human Services.** Dietary Guidelines for Americans, 2005, Chapter 4 Physical Activity. <http://www.health.gov/dietaryguidelines/dga2005/document/html/chapter4.htm>. Accessed for verification April 22, 2008. [EL 4]
441. **Kertai MD, Boersma E, Bax JJ, et al.** A meta-analysis comparing the prognostic accuracy of six diagnostic tests for predicting perioperative cardiac risk in patients undergoing major vascular surgery. *Heart.* 2003;89:1327-1334. [EL 1]
442. **McGory ML, Maggard MA, Ko CY.** A meta-analysis of perioperative beta blockade: what is the actual risk reduction? *Surgery.* 2005;138:171-179. [EL 1]
443. **U.S. Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute.** The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. NIH Publication No. 04-5230, August, 2004. <http://www.nhlbi.nih.gov/guidelines/hypertension/jnc7full.pdf>. Accessed for verification April 25, 2008. [EL 4]
444. **Qaseem A, Snow V, Fitterman N, et al (Clinical Efficacy Assessment Subcommittee of the American College of Physicians).** Risk assessment for and strategies to reduce perioperative pulmonary complications for patients undergoing noncardiothoracic surgery: a guideline from the American College of Physicians. *Ann Intern Med.* 2006;144:575-580. [EL 4]
445. **Nguyen NT, Hinojosa M, Fayad C, Varela E, Wilson SE.** Use and outcomes of laparoscopic versus open gastric bypass at academic medical centers. *J Am Coll Surg.* 2007;205:248-255. [EL 3]
446. **Doyle RL.** Assessing and modifying the risk of postoperative pulmonary complications. *Chest.* 1999;115(5 suppl):77S-81S. [EL 4]
447. **Smetana GW, Lawrence VA, Cornell JE (American College of Physicians).** Preoperative pulmonary risk stratification for noncardiothoracic surgery: systematic review for the American College of Physicians. *Ann Intern Med.* 2006;144:581-595. [EL 1]
448. **Lautz DB, Jackson TD, Clancy KA, et al.** Bariatric operations in Veterans Affairs and selected university medical centers: results of the Patient Safety in Surgery Study. *J Am Coll Surg.* 2007;204:1261-1272. [EL 3]
449. **Livingston EH, Arterburn D, Schiffner TL, Henderson WG, DePalma RG.** National Surgical Quality Improvement Program analysis of bariatric operations: modifiable risk factors contribute to bariatric surgical adverse outcomes. *J Am Coll Surg.* 2006;203:625-633. [EL 3]
450. **Geerts WH, Pineo GF, Heit JA, et al.** Prevention of venous thromboembolism: the seventh ACCP conference on antithrombotic and thrombolytic therapy. *Chest.* 2004;126(3 suppl):338S-400S. [EL 4]
451. **Martin LF, Gouda BP.** What we know and don't know about deep venous thrombosis and pulmonary embolism! *Obes Surg.* 2005;15:565-566. [EL 4]
452. **Ogunnaike BO, Jones SB, Jones DB, Provost D, Whitten CW.** Anesthetic considerations for bariatric surgery. *Anesth Analg.* 2002;95:1793-1805. [EL 4]
453. **Byrne TK.** Complications of surgery for obesity. *Surg Clin North Am.* 2001;81:1181-1193, vii-viii. [EL 4]
454. **Atluri P, Raper SE.** Factor V Leiden and postoperative deep vein thrombosis in patients undergoing open Roux-en-Y gastric bypass surgery. *Obes Surg.* 2005;15:561-564. [EL 3]
455. **Keeling WB, Haines K, Stone PA, Armstrong PA, Murr MM, Shames ML.** Current indications for preoperative inferior vena cava filter insertion in patients undergoing surgery for morbid obesity. *Obes Surg.* 2005;15:1009-1012. [EL 3]
456. **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. [EL 3]
457. **Yang CS, Lee WJ, Wang HH, Huang SP, Lin JT, Wu MS.** The influence of *Helicobacter pylori* infection on the development of gastric ulcer in symptomatic patients after bariatric surgery. *Obes Surg.* 2006;16:735-739. [EL 2]
458. **Wang HH, Lee WJ, Liew PL, et al.** The influence of *Helicobacter pylori* infection and corpus gastritis on the postoperative outcomes of laparoscopic vertical banded gastroplasty. *Obes Surg.* 2006;16:297-307. [EL 2]
459. **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. [EL 3]
460. **Green RM, Flamm S.** AGA technical review on the evaluation of liver chemistry tests. *Gastroenterology.* 2002;123:1367-1384. [EL 4]
461. **Dallal RM, Mattar SG, Lord JL, et al.** Results of laparoscopic gastric bypass in patients with cirrhosis. *Obes Surg.* 2004;14:47-53. [EL 3]
462. **Tichansky DS, Madan AK.** Laparoscopic Roux-en-Y gastric bypass is safe and feasible after orthotopic liver transplantation. *Obes Surg.* 2005;15:1481-1486. [EL 3]
463. **McGoey BV, Deitel M, Saplys RJ, Kliman ME.** Effect of weight loss on musculoskeletal pain in the morbidly obese. *J Bone Joint Surg.* 1990;72:322-323. [EL 3]
464. **Amaral JF, Thompson WR, Caldwell MD, Martin HF, Randall HT.** Prospective metabolic evaluation of 150 consecutive patients who underwent gastric exclusion. *Am J Surg.* 1984;147:468-476. [EL 3]
465. **Starkloff GB, Donovan JF, Ramach KR, Wolfe BM.** Metabolic intestinal surgery: its complications and management. *Arch Surg.* 1975;110:652-657. [EL 3]
466. **Hamoui N, Kim K, Anthon G, Crookes PF.** The significance of elevated levels of parathyroid hormone in patients with morbid obesity before and after bariatric surgery. *Arch Surg.* 2003;138:891-897. [EL 3]
467. **Hamoui N, Anthon G, Crookes PF.** Calcium metabolism in the morbidly obese. *Obes Surg.* 2004;14:9-12. [EL 3]
468. **Compston JE, Vedi S, Ledger JE, Webb A, Gazet JC, Pilkington TR.** Vitamin D status and bone histomorphometry in gross obesity. *Am J Clin Nutr.* 1981;34:2359-2363. [EL 3]
469. **Miller PD.** Guidelines for the diagnosis of osteoporosis: T-scores vs fracture. *Rev Endocr Metab Disord.* 2006;7:75-89. [EL 4]
470. **Levi D, Goodman ER, Patel M, Savransky Y.** Critical care of the obese and bariatric surgical patient. *Crit Care Clin.* 2003;19:11-32. [EL 4]
471. **Stocker DJ.** Management of the bariatric surgery patient. *Endocrinol Metab Clin North Am.* 2003;32:437-457. [EL 4]

472. **Sekhar N, Torquati A, Lutfi R, Richards WO.** Endoscopic evaluation of the gastrojejunostomy in laparoscopic gastric bypass: a series of 340 patients without postoperative leak. *Surg Endosc.* 2006;20:199-201. [EL 2]
473. **Fernandez AZ Jr, DeMaria EJ, Tichansky DS, et al.** Experience with over 3,000 open and laparoscopic bariatric procedures: multivariate analysis of factors related to leak and related mortality. *Surg Endosc.* 2004;18:193-197. [EL 3]
474. **Marshall JS, Srivastava A, Gupta SK, Rossi TR, DeBord JR.** Roux-en-Y gastric bypass leak complications. *Arch Surg.* 2003;138:520-523. [EL 3]
475. **Hamilton EC, Sims TL, Hamilton TT, Mullican MA, Jones DB, Provost DA.** Clinical predictors of leak after laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Surg Endosc.* 2003;17:679-684. [EL 3]
476. **Edwards MA, Jones DB, Ellsmere J, Grinbaum R, Schneider BE.** Anastomotic leak following antecolic versus retrocolic laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Obes Surg.* 2007;17:292-297. [EL 3]
477. **Carucci LR, Turner MA, Conklin RC, DeMaria EJ, Kellum JM, Sugerman HJ.** Roux-en-Y gastric bypass surgery for morbid obesity: evaluation of postoperative extraluminal leaks with upper gastrointestinal series. *Radiology.* 2006;238:119-127. [EL 3]
478. **Ganci-Cerrud G, Herrera MF.** Role of radiologic contrast studies in the early postoperative period after bariatric surgery. *Obes Surg.* 1999;9:532-534. [EL 3]
479. **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. [EL 3]
480. **Brolin RE.** Complications of surgery for severe obesity. *Prob Gen Surg.* 2000;17:55-61. [EL 4]
481. **Toppino M, Cesarani F, Comba A, et al.** The role of early radiological studies after gastric bariatric surgery. *Obes Surg.* 2001;11:447-454. [EL 3]
482. **Polk HC Jr, Christmas AB.** Prophylactic antibiotics in surgery and surgical wound infections. *Am Surg.* 2000;66:105-111. [EL 4]
483. **Van den Berghe G, Wouters P, Weekers F, et al.** Intensive insulin therapy in critically ill patients. *N Engl J Med.* 2001;345:1359-1367. [EL 1]
484. **Van den Berghe G, Wilmer A, Hermans G, et al.** Intensive insulin therapy in the medical ICU. *N Engl J Med.* 2006;354:449-461. [EL 1]
485. **Datta S, Qaadir A, Villanueva G, Baldwin D.** Once-daily insulin glargine versus 6-hour sliding scale regular insulin for control of hyperglycemia after a bariatric surgical procedure: a randomized clinical trial. *Endocr Pract.* 2007;13:225-231. [EL 2]
486. **Garber AJ, Moghissi ES, Bransome ED Jr, et al (American College of Endocrinology Task Force on Inpatient Diabetes and Metabolic Control).** American College of Endocrinology position statement on inpatient diabetes and metabolic control. *Endocr Pract.* 2004;10(suppl 2):4-9. [EL 4]
487. **McDonnell ME, Apovian CM, Hess DT Jr.** Medical management of the patient after gastric-bypass surgery. In: Farraye FA, Forse RA, eds. *Bariatric Surgery: A Primer for Your Medical Practice.* Thorofare, NJ: SLACK Incorporated, 2006: chapter 9. [EL 4]
488. **Residori L, García-Lorda P, Flancbaum L, Pi-Sunyer FX, Laferrère B.** Prevalence of co-morbidities in obese patients before bariatric surgery: effect of race. *Obes Surg.* 2003;13:333-340. [EL 3]
489. **Pope GD, Birkmeyer JD, Finlayson SR.** National trends in utilization and in-hospital outcomes of bariatric surgery. *J Gastrointest Surg.* 2002;6:855-860. [EL 3]
490. **Nanji AA, Freeman JB.** Rate of weight loss after vertical banded gastroplasty in morbid obesity: relationship to serum lipids and uric acid. *Int Surg.* 1985;70:323-325. [EL 3]
491. **Kelly TM, Jones SB.** Changes in serum lipids after gastric bypass surgery: lack of a relationship to weight loss. *Int J Obes.* 1986;10:443-452. [EL 3]
492. **Gleysteen JJ, Barboriak JJ, Sasse EA.** Sustained coronary-risk-factor reduction after gastric bypass for morbid obesity. *Am J Clin Nutr.* 1990;51:774-778. [EL 3]
493. **Brolin RE, Kenler HA, Wilson AC, Kuo PT, Cody RP.** Serum lipids after gastric bypass surgery for morbid obesity. *Int J Obes.* 1990;14:939-950. [EL 3]
494. **Lopez-Jimenez F, Bhatia S, Collazo-Clavell ML, Sarr MG, Somers VK.** Safety and efficacy of bariatric surgery in patients with coronary artery disease. *Mayo Clin Proc.* 2005;80:1157-1162. [EL 3]
495. **Alpert MA, Hashimi MW.** Obesity and the heart. *Am J Med Sci.* 1993;306:117-123. [EL 4]
496. **Backman L, Freyschuss U, Hallberg D, Melcher A.** Cardiovascular function in extreme obesity. *Acta Med Scand.* 1973;193:437-446. [EL 3]
497. **Lawrence VA, Hilsenbeck SG, Mulrow CD, Dhanda R, Sapp J, Page CP.** Incidence and hospital stay for cardiac and pulmonary complications after abdominal surgery. *J Gen Intern Med.* 1995;10:671-678. [EL 2]
498. **Blouw EL, Rudolph AD, Narr BJ, Sarr MG.** The frequency of respiratory failure in patients with morbid obesity undergoing gastric bypass. *AANA J.* 2003;71:45-50. [EL 3]
499. **Oberg B, Poulsen TD.** Obesity: an anaesthetic challenge. *Acta Anaesthesiol Scand.* 1996;40:191-200. [EL 4]
500. **Marik P, Varon J.** The obese patient in the ICU. *Chest.* 1998;113:492-498. [EL 4]
501. **Torrington KG, Sorenson DE, Sherwood LM.** Postoperative chest percussion with postural drainage in obese patients following gastric stapling. *Chest.* 1984;86:891-895. [EL 3]
502. **Huerta S, DeShields S, Shpiner R, et al.** Safety and efficacy of postoperative continuous positive airway pressure to prevent pulmonary complications after Roux-en-Y gastric bypass. *J Gastrointest Surg.* 2002;6:354-358. [EL 2]
503. **Schumann R, Jones SB, Ortiz VE, et al.** Best practice recommendations for anesthetic perioperative care and pain management in weight loss surgery. *Obes Res.* 2005;13:254-266. [EL 3]
504. **Sugerman HJ, Kellum JM, Engle KM, et al.** Gastric bypass for treating severe obesity. *Am J Clin Nutr.* 1992;55(2 suppl):560S-566S. [EL 4]
505. **Fobi MA, Lee H, Igwe D Jr, Stanczyk M, Tambi JN.** Prospective comparative evaluation of stapled versus transected Silastic ring gastric bypass: 6-year follow-up. *Obes Surg.* 2001;11:18-24. [EL 2]
506. **Capan LM, Miller SM.** Monitoring for suspected pulmonary embolism. *Anesthesiol Clin North Am.* 2001;19:673-703. [EL 4]
507. **Scholten DJ, Hoedema RM, Scholten SE.** A comparison of two different prophylactic dose regimens of low molecular weight heparin in bariatric surgery. *Obes Surg.* 2002;12:19-24. [EL 3]
508. **Kalfarentzos F, Stavropoulou F, Yarmenitis S, et al.** Prophylaxis of venous thromboembolism using two different doses of low-molecular-weight heparin (nadroparin) in bariatric surgery: a prospective randomized trial. *Obes Surg.* 2001;11:670-676. [EL 2]

509. **Miller MT, Rovito PF.** An approach to venous thromboembolism prophylaxis in laparoscopic Roux-en-Y gastric bypass surgery. *Obes Surg.* 2004;14:731-737. [EL 3]
510. **Ferrell A, Byrne TK, Robison JG.** Placement of inferior vena cava filters in bariatric surgical patients—possible indications and technical considerations. *Obes Surg.* 2004;14:738-743. [EL 3]
511. **Mognol P, Vignes S, Chosidow D, Marmuse JP.** Rhabdomyolysis after laparoscopic bariatric surgery. *Obes Surg.* 2004;14:91-94. [EL 3]
512. **de Freitas Carvalho DA, Valezi AC, de Brito EM, de Souza JC, Masson AC, Matsuo T.** Rhabdomyolysis after bariatric surgery. *Obes Surg.* 2006;16:740-744. [EL 3]
513. **Yeats M, Wedergren S, Fox N, Thompson JS.** The use and modification of clinical pathways to achieve specific outcomes in bariatric surgery. *Am Surg.* 2005;71:152-154. [EL 3]
514. **Huerta S, Arteaga JR, Sawicki MP, Liu CD, Livingston EH.** Assessment of routine elimination of postoperative nasogastric decompression after Roux-en-Y gastric bypass. *Surgery.* 2002;132:844-848. [EL 3]
515. **Kushner R.** Managing the obese patient after bariatric surgery: a case report of severe malnutrition and review of the literature. *JPEN J Parenter Enteral Nutr.* 2000;24:126-132. [EL 3]
516. **Wolf AM, Kortner B, Kuhlmann HW.** Results of bariatric surgery. *Int J Obes Relat Metab Disord.* 2001;25(suppl 1):S113-S114. [EL 3]
517. **Sugerman HJ, Kellum JM Jr, DeMaria EJ, Reines HD.** Conversion of failed or complicated vertical banded gastroplasty to gastric bypass in morbid obesity. *Am J Surg.* 1996;171:263-269. [EL 3]
518. **Metcalfe B, Rabkin RA, Rabkin JM, Metcalfe LJ, Lehman-Becker LB.** Weight loss composition: the effects of exercise following obesity surgery as measured by bioelectrical impedance analysis. *Obes Surg.* 2005;15:183-186. [EL 3]
519. **Shen R, Dugay G, Rajaram K, Cabrera I, Siegel N, Ren CJ.** Impact of patient follow-up on weight loss after bariatric surgery. *Obes Surg.* 2004;14:514-519. [EL 3]
520. **Favretti F, O'Brien PE, Dixon JB.** Patient management after LAP-BAND placement. *Am J Surg.* 2002;184:38S-41S. [EL 4]
521. **Dixon AF, Dixon JB, O'Brien PE.** Laparoscopic adjustable gastric banding induces prolonged satiety: a randomized blind crossover study. *J Clin Endocrinol Metab.* 2005;90:813-819. [EL 3]
522. **Dixon JB, O'Brien PE.** Permeability of the silicone membrane in laparoscopic adjustable gastric bands has important clinical implications. *Obes Surg.* 2005;15:624-629. [EL 3]
523. **Dixon JB, Dixon ME, O'Brien PE.** Depression in association with severe obesity: changes with weight loss. *Arch Intern Med.* 2003;163:2058-2065. [EL 3]
524. **Pories WJ, MacDonald KG, Swanson MS, Morgan EJ, Long S, Brown B.** Staple-line failure: an avoidable complication of the gastric bypass. *Obes Surg.* 1993;3:95. [EL 3]
525. **MacLean LD, Rhode BM, Nohr C, Katz S, McLean AP.** Stomal ulcer after gastric bypass. *J Am Coll Surg.* 1997;185:1-7. [EL 3]
526. **Lublin M, McCoy M, Waldrep DJ.** Perforating marginal ulcers after laparoscopic gastric bypass. *Surg Endosc.* 2006;20:51-54. [EL 3]
527. **MacLean LD, Rhode BM, Nohr CW.** Late outcome of isolated gastric bypass. *Ann Surg.* 2000;231:524-528. [EL 3]
528. **Deitel M.** Overview of operations for morbid obesity. *World J Surg.* 1998;22:913-918. [EL 4]
529. **Deitel M, Shahi B.** Morbid obesity: selection of patients for surgery. *J Am Coll Nutr.* 1992;11:457-462. [EL 4]
530. **Lechner GW, Callender AK.** Subtotal gastric exclusion and gastric partitioning: a randomized prospective comparison of one hundred patients. *Surgery.* 1981;90:637-644. [EL 2]
531. **Näslund I.** The size of the gastric outlet and the outcome of surgery for obesity. *Acta Chir Scand.* 1986;152:205-210. [EL 2]
532. **Pories WJ, Flickinger EG, Meelheim D, Van Rij AM, Thomas FT.** The effectiveness of gastric bypass over gastric partition in morbid obesity: consequence of distal gastric and duodenal exclusion. *Ann Surg.* 1982;196:389-399. [EL 2]
533. **Marceau P, Biron S, Hould FS, Lebel S, Marceau S.** Malabsorptive procedure in surgical treatment of morbid obesity. *Prob Gen Surg.* 2000;17:29-39. [EL 4]
534. **Lee WJ, Huang MT, Yu PJ, Wang W, Chen TC.** Laparoscopic vertical banded gastroplasty and laparoscopic gastric bypass: a comparison. *Obes Surg.* 2004;14:626-634. [EL 2]
535. **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. [EL 2]
536. **Watkins BM, Montgomery KF, Ahroni JH.** Laparoscopic adjustable gastric banding: early experience in 400 consecutive patients in the USA. *Obes Surg.* 2005;15:82-87. [EL 2]
537. **Murr MM, Balsiger BM, Kennedy FP, Mai JL, Sarr MG.** Malabsorptive procedures for severe obesity: comparison of pancreaticobiliary bypass and very very long limb Roux-en-Y gastric bypass. *J Gastrointest Surg.* 1999;3:607-612. [EL 3]
538. **Guedea ME, Arribas del Amo D, Solanas JA, et al.** Results of biliopancreatic diversion after five years. *Obes Surg.* 2004;14:766-772. [EL 3]
539. **Nelson WK, Fatima J, Houghton SG, et al.** The malabsorptive very, very long limb Roux-en-Y gastric bypass for super obesity: results in 257 patients [with discussion]. *Surgery.* 2006;140:517-523. [EL 3]
540. **Bock MA.** Roux-en-Y gastric bypass: the dietitian's and patient's perspectives. *Nutr Clin Pract.* 2003;18:141-144. [EL 4]
541. **Collene A, Hertzler S.** Metabolic outcomes of gastric bypass. *Nutr Clin Pract.* 2003;18:136-140. [EL 4]
542. **Shamblin JR, Shamblin WR.** Bariatric surgery should be more widely accepted. *South Med J.* 1987;80:861-865. [EL 4]
543. **Brolin RL, Robertson LB, Kenler HA, Cody RP.** Weight loss and dietary intake after vertical banded gastroplasty and Roux-en-Y gastric bypass. *Ann Surg.* 1994;220:782-790. [EL 3]
544. **Brolin RE, Gorman RC, Milgrim LM, Kenler HA.** Multivitamin prophylaxis in prevention of post-gastric bypass vitamin and mineral deficiencies. *Int J Obes.* 1991;15:661-667. [EL 3]
545. **Skroubis G, Sakellaropoulos G, Pougouras K, Mead N, Nikiforidis G, Kalfarentzos F.** Comparison of nutritional deficiencies after Roux-en-Y gastric bypass and after biliopancreatic diversion with Roux-en-Y gastric bypass. *Obes Surg.* 2002;12:551-558. [EL 3]
546. **Compston JE, Vedi S, Gianetta E, Watson G, Civalleri D, Scopinaro N.** Bone histomorphometry and vitamin D status after biliopancreatic bypass for obesity. *Gastroenterology.* 1984;87:350-356. [EL 3]

547. **Marceau P, Biron S, Lagacé M, et al.** Biliopancreatic diversion, with distal gastrectomy, 250 cm and 50 cm limbs: long-term results. *Obes Surg.* 1995;5:302-307. [EL 3]
548. **Monteforte MJ, Turkelson CM.** Bariatric surgery for morbid obesity. *Obes Surg.* 2000;10:391-401. [EL 1]
549. **Mallory GN, Macgregor AM, Rand CS.** The influence of dumping on weight loss after gastric restrictive surgery for morbid obesity. *Obes Surg.* 1996;6:474-478. [EL 3]
550. **Didden P, Penning C, Masclee AA.** Octreotide therapy in dumping syndrome: analysis of long-term results. *Aliment Pharmacol Ther.* 2006;24:1367-1375. [EL 3]
551. **Ukleja A.** Dumping syndrome: pathophysiology and treatment. *Nutr Clin Pract.* 2005;20:517-525. [EL 4]
552. **Carvajal SH, Mulvihill SJ.** Postgastrectomy syndromes: dumping and diarrhea. *Gastroenterol Clin North Am.* 1994;23:261-279. [EL 4]
553. **Service GJ, Thompson GB, Service FJ, Andrews JC, Collazo-Clavell ML, Lloyd RV.** Hyperinsulinemic hypoglycemia with nesidioblastosis after gastric-bypass surgery. *N Engl J Med.* 2005;353:249-254. [EL 3]
554. **Patti ME, McMahon G, Mun EC, et al.** Severe hypoglycaemia post-gastric bypass requiring partial pancreatectomy: evidence for inappropriate insulin secretion and pancreatic islet hyperplasia. *Diabetologia.* 2005;48:2236-2240. [EL 3]
555. **Avinoah E, Ovnat A, Charuzi I.** Nutritional status seven years after Roux-en-Y gastric bypass surgery. *Surgery.* 1992;111:137-142. [EL 3]
556. **Moize V, Geliebter A, Gluck ME, et al.** Obese patients have inadequate protein intake related to protein intolerance up to 1 year following Roux-en-Y gastric bypass. *Obes Surg.* 2003;13:23-28. [EL 3]
557. **Stunkard A, Foster G, Glassman J, Rosato E.** Retrospective exaggeration of symptoms: vomiting after gastric surgery for obesity. *Psychosom Med.* 1985;47:150-155. [EL 3]
558. **Brolin RE, Leung M.** Survey of vitamin and mineral supplementation after gastric bypass and biliopancreatic diversion for morbid obesity. *Obes Surg.* 1999;9:150-154. [EL 3]
559. **Crowley LV, Seay J, Mullin G.** Late effects of gastric bypass for obesity. *Am J Gastroenterol.* 1984;79:850-860. [EL 3]
560. **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. [EL 4]
561. **Amaral JF, Thompson WR, Caldwell MD, Martin HF, Randall HT.** Prospective hematologic evaluation of gastric exclusion surgery for morbid obesity. *Ann Surg.* 1985;201:186-193. [EL 2]
562. **Faintuch J, Matsuda M, Cruz ME, et al.** Severe protein-calorie malnutrition after bariatric procedures. *Obes Surg.* 2004;14:175-181. [EL 3]
563. **Chang CG, Adams-Huet B, Provost DA.** Acute post-gastric reduction surgery (APGARS) neuropathy. *Obes Surg.* 2004;14:182-189. [EL 3]
564. **Akhtar M, Collins MP, Kissel JT.** Acute postgastric reduction surgery (APGARS) neuropathy: a polynutritional, multisystem disorder [abstract]. *Neurology.* 2002;58(suppl 3):A68-A69. [EL 3]
565. **Marinari GM, Murelli F, Camerini G, et al.** A 15-year evaluation of biliopancreatic diversion according to the Bariatric Analysis Reporting Outcome System (BAROS). *Obes Surg.* 2004;14:325-328. [EL 3]
566. **Kalfarentzos F, Dimakopoulos A, Kehagias I, Loukidi A, Mead N.** Vertical banded gastroplasty versus standard or distal Roux-en-Y gastric bypass based on specific selection criteria in the morbidly obese: preliminary results. *Obes Surg.* 1999;9:433-442. [EL 3]
567. **Vanuytsel JL, Nobels FR, Van Gaal LF, De Leeuw IH.** A case of malnutrition after biliopancreatic diversion for morbid obesity. *Int J Obes Relat Metab Disord.* 1993;17:425-426. [EL 3]
568. **Strauss BJ, Marks SJ, Growcott JP, et al.** Body composition changes following laparoscopic gastric banding for morbid obesity. *Acta Diabetol.* 2003;40(suppl 1):S266-S269. [EL 3]
569. **Dixon JB, Strauss BJ, Laurie C, O'Brien PE.** Changes in body composition with weight loss: obese subjects randomized to surgical and medical programs. *Obesity (Silver Spring).* 2007;15:1187-1198. [EL 2]
570. **Ott MT, Fanti P, Malluche HH, et al.** Biochemical evidence of metabolic bone disease in women following Roux-Y gastric bypass for morbid obesity. *Obes Surg.* 1992;2:341-348. [EL 3]
571. **Shaker JL, Norton AJ, Woods MF, Fallon MD, Findling JW.** Secondary hyperparathyroidism and osteopenia in women following gastric exclusion surgery for obesity. *Osteoporos Int.* 1991;1:177-181. [EL 3]
572. **Coates PS, Fernstrom JD, Fernstrom MH, Schauer PR, Greenspan LS.** Gastric bypass surgery for morbid obesity leads to an increase in bone turnover and a decrease in bone mass. *J Clin Endocrinol Metab.* 2004;89:1061-1065. [EL 2]
573. **Charles P.** Calcium absorption and calcium bioavailability. *J Intern Med.* 1992;231:161-168. [EL 4]
574. **Pugnale N, Giusti V, Suter M, et al.** Bone metabolism and risk of secondary hyperparathyroidism 12 months after gastric banding in obese pre-menopausal women. *Int J Obes Relat Metab Disord.* 2003;27:110-116. [EL 3]
575. **Alborzi F, Leibowitz AB.** Immobilization hypercalcemia in critical illness following bariatric surgery. *Obes Surg.* 2002;12:871-873. [EL 3]
576. **Liel Y, Edwards J, Shary J, Spicer KM, Gordon L, Bell NH.** The effects of race and body habitus on bone mineral density of the radius, hip, and spine in premenopausal women. *J Clin Endocrinol Metab.* 1988;66:1247-1250. [EL 2]
577. **Gossain VV, Rao DS, Carella MJ, Divine G, Rovner DR.** Bone mineral density (BMD) in obesity: effect of weight loss. *J Med.* 1999;30:367-376. [EL 3]
578. **Bell NH, Epstein S, Greene A, Shary J, Oexmann MJ, Shaw S.** Evidence for alteration of the vitamin D-endocrine system in obese subjects. *J Clin Invest.* 1985;76:370-373. [EL 3]
579. **Fu L, Patel MS, Bradley A, Wagner EF, Karsenty G.** The molecular clock mediates leptin-regulated bone formation. *Cell.* 2005;122:803-815. [EL 4]
580. **De Prisco C, Levine SN.** Metabolic bone disease after gastric bypass surgery for obesity. *Am J Med Sci.* 2005;329:57-61. [EL 3]
581. **Goode LR, Brolin RE, Chowdhury HA, Shapses SA.** Bone and gastric bypass surgery: effects of dietary calcium and vitamin D. *Obes Res.* 2004;12:40-47. [EL 2]
582. **Fitzpatrick LA.** Secondary causes of osteoporosis. *Mayo Clin Proc.* 2002;77:453-468. [EL 4]
583. **Diniz MdeF, Diniz MT, Sanches SR, et al.** Elevated serum parathormone after Roux-en-Y gastric bypass. *Obes Surg.* 2004;14:1222-1226. [EL 3]
584. **Parada P, Maruri I, Morales MJ, Otero I, Delgado C, Casal JE.** Nutritional complications after bariatric surgery [abstract 33]. *Obes Surg.* 2003;13:525. [EL 3]
585. **Youssef Y, Richards WO, Sekhar N, et al.** Risk of secondary hyperparathyroidism after laparoscopic gastric bypass surgery in obese women. *Surg Endosc.* 2007;21:1393-1396. [EL 2]

586. Slater GH, Ren CJ, Siegel N, et al. Serum fat-soluble vitamin deficiency and abnormal calcium metabolism after malabsorptive bariatric surgery. *J Gastrointest Surg.* 2004;8:48-55. [EL 3]
587. Newbury L, Dolan K, Hatzifotis M, Low N, Fielding G. Calcium and vitamin D depletion and elevated parathyroid hormone following biliopancreatic diversion. *Obes Surg.* 2003;13:893-895. [EL 3]
588. Reidt CS, Brolin RE, Sherrell RM, Field MP, Shapses SA. True fractional calcium absorption is decreased after Roux-en-Y gastric bypass surgery. *Obesity (Silver Spring).* 2006;14:1940-1948. [EL 3]
589. Johnson JM, Maher JW, Samuel I, Heitshusen D, Doherty C, Downs RW. Effects of gastric bypass procedures on bone mineral density, calcium, parathyroid hormone, and vitamin D [with discussion]. *J Gastrointest Surg.* 2005;9:1106-1111. [EL 2]
590. Shikora SA, Kim JJ, Tarnoff ME. Nutrition and gastrointestinal complications of bariatric surgery. *Nutr Clin Pract.* 2007;22:29-40. [EL 4]
591. Miller AD, Smith KM. Medication and nutrient administration considerations after bariatric surgery. *Am J Health Syst Pharm.* 2006;63:1852-1857. [EL 4]
592. Levenson DI, Bockman RS. A review of calcium preparations [published correction appears in *Nutr Rev.* 1994;52:364]. *Nutr Rev.* 1994;52:221-232. [EL 4]
593. Dawson-Hughes B. Calcium and vitamin D. In: Favus MJ, ed. *Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism.* 6th ed. Washington, DC: American Society for Bone and Mineral Research, 2006: 257-259. [EL 4]
594. Ybarra J, Sánchez-Hernández J, Gich I, et al. Unchanged hypovitaminosis D and secondary hyperparathyroidism in morbid obesity after bariatric surgery. *Obes Surg.* 2005;15:330-335. [EL 3]
595. Sánchez-Hernández J, Ybarra J, Gich I, et al. Effects of bariatric surgery on vitamin D status and secondary hyperparathyroidism: a prospective study. *Obes Surg.* 2005;15:1389-1395. [EL 2]
596. Rojas-Marcos PM, Rubio MA, Kreskshi WI, Cabrerizo L, Sánchez-Pernaute A. Severe hypocalcemia following total thyroidectomy after biliopancreatic diversion. *Obes Surg.* 2005;15:431-434. [EL 3]
597. Marceau P, Biron S, Lebel S, et al. Does bone change after biliopancreatic diversion? *J Gastrointest Surg.* 2002; 6:690-698. [EL 2]
598. Mitchell DY, Eusebio RA, Dunlap LE, et al. Risedronate gastrointestinal absorption is independent of site and rate of administration. *Pharm Res.* 1998;15:228-232. [EL 3]
599. Lanza FL, Hunt RH, Thomson AB, Provenza JM, Blank MA. Endoscopic comparison of esophageal and gastroduodenal effects of risedronate and alendronate in postmenopausal women. *Gastroenterology.* 2000;119: 631-638. [EL 3]
600. Maricic M. New and emerging treatments for osteoporosis. *Curr Opin Rheumatol.* 2007;19:364-369. [EL 4]
601. Rosen CJ, Brown S. Severe hypocalcemia after intravenous bisphosphonate therapy in occult vitamin D deficiency. *N Engl J Med.* 2003;348:1503-1504. [EL 3]
602. Collazo-Clavell ML, Jimenez A, Hodgson SF, Sarr MG. Osteomalacia after Roux-en-Y gastric bypass. *Endocr Pract.* 2004;10:195-198. [EL 3]
603. Duncan SH, Richardson AJ, Kaul P, Holmes RP, Allison MJ, Stewart CS. Oxalobacter formigenes and its potential role in human health. *Appl Environ Microbiol.* 2002;68:3841-3847. [EL 4]
604. Campieri C, Campieri M, Bertuzzi V, et al. Reduction of oxaluria after an oral course of lactic acid bacteria at high concentration. *Kidney Int.* 2001;60:1097-1105. [EL 3]
605. Lieske JC, Goldfarb DS, De Simone C, Regnier C. Use of a probiotic to decrease enteric hyperoxaluria. *Kidney Int.* 2005;68:1244-1249. [EL 3]
606. Spencer H, Lesniak M, Gatzka CA, Osis D, Lender M. Magnesium absorption and metabolism in patients with chronic renal failure and in patients with normal renal function. *Gastroenterology.* 1980;79:26-34. [EL 3]
607. Chassany O, Michaux A, Bergmann JF. Drug-induced diarrhoea. *Drug Saf.* 2000;22:53-72. [EL 4]
608. New York State Osteoporosis Prevention and Education Program. Phosphorus. http://www.nysopep.org/Prevention_Phosphorus.shtm. Accessed for verification April 25, 2008. [EL 4]
609. Apovian CM, McMahon MM, Bistrrian BR. Guidelines for refeeding the marasmic patient. *Crit Care Med.* 1990;18:1030-1033. [EL 4]
610. Food and Agriculture Organization/World Health Organization. Fat in human nutrition: report of an expert consultation. Rome, Italy: Publications Division, Food and Agriculture Organization, 1993. [EL 4]
611. Innis SM. Essential dietary lipids. In: Ziegler EE, Filer LJ Jr, eds. *Present Knowledge in Nutrition.* 7th ed. Washington, DC: ILSI Press, 1996: 58-66. [EL 4]
612. Smit EN, Muskiet FA, Boersma ER. The possible role of essential fatty acids in the pathophysiology of malnutrition: a review. *Prostaglandins Leukot Essent Fatty Acids.* 2004;71:241-250. [EL 4]
613. Miller DG, Williams SK, Palombo JD, Griffin RE, Bistrrian BR, Blackburn GL. Cutaneous application of safflower oil in preventing essential fatty acid deficiency in patients on home parenteral nutrition. *Am J Clin Nutr.* 1987;46:419-423. [EL 3]
614. Bradley JE, Brown RO, Luther RW. Multiple nutritional deficiencies and metabolic complications 20 years after jejunioleal bypass surgery. *JPEN J Parenter Enteral Nutr.* 1987;11:494-498. [EL 3]
615. Halverson JD, Scheff RJ, Gentry K, Alpers DH. Long-term follow-up of jejunioleal bypass patients. *Am J Clin Nutr.* 1980;33(2 suppl):472-475. [EL 3]
616. Rogers EL, Douglass W, Russell RM, Bushman L, Hubbard TB, Iber FL. Deficiency of fat soluble vitamins after jejunioleal bypass surgery for morbid obesity. *Am J Clin Nutr.* 1980;33:1208-1214. [EL 3]
617. Kaneki M, Hosoi T, Ouchi Y, Orimo H. Pleiotropic actions of vitamin K: protector of bone health and beyond? *Nutrition.* 2006;22:845-852. [EL 4]
618. Bendich A, Machlin LJ. Safety of oral intake of vitamin E. *Am J Clin Nutr.* 1988;48:612-619. [EL 4]
619. Brolin RE, Gorman JH, Gorman RC, et al. Prophylactic iron supplementation after Roux-en-Y gastric bypass: a prospective, double-blind, randomized study. *Arch Surg.* 1998;133:740-744. [EL 2]
620. Rhode BM, Shustik C, Christou NV, MacLean LD. Iron absorption and therapy after gastric bypass. *Obes Surg.* 1999;9:17-21. [EL 3]
621. Beard JL, Dawson H, Piñero DJ. Iron metabolism: a comprehensive review. *Nutr Rev.* 1996;54:295-317. [EL 4]
622. Lash A, Saleem A. Iron metabolism and its regulation: a review. *Ann Clin Lab Sci.* 1995;25:20-30. [EL 4]
623. Herbert V. Absorption of vitamin B12 and folic acid. *Gastroenterology.* 1968;54:110-115. [EL 4]

624. **Fondu P, Hariga-Muller C, Mozes N, Neve J, Van Steirteghem A, Mandelbaum IM.** Protein-energy malnutrition and anemia in Kivu. *Am J Clin Nutr.* 1978;31:46-56. [EL 4]
625. **Hallberg L, Brune M, Erlandsson M, Sandberg AS, Rossander-Hultén L.** Calcium: effect of different amounts on nonheme- and heme-iron absorption in humans. *Am J Clin Nutr.* 1991;53:112-119. [EL 3]
626. **Cook JD, Dassenko SA, Whittaker P.** Calcium supplementation: effect on iron absorption. *Am J Clin Nutr.* 1991;53:106-111. [EL 3]
627. **Reddy MB, Cook JD.** Effect of calcium intake on nonheme-iron absorption from a complete diet. *Am J Clin Nutr.* 1997;65:1820-1825. [EL 3]
628. **Halverson JD.** Micronutrient deficiencies after gastric bypass for morbid obesity. *Am Surg.* 1986;52:594-598. [EL 3]
629. **Smith CD, Herkes SB, Behrns KE, Fairbanks VF, Kelly KA, Sarr MG.** Gastric acid secretion and vitamin B12 absorption after vertical Roux-en-Y gastric bypass for morbid obesity. *Ann Surg.* 1993;218:91-96. [EL 3]
630. **Behrns KE, Smith CD, Sarr MG.** Prospective evaluation of gastric acid secretion and cobalamin absorption following gastric bypass for clinically severe obesity. *Dig Dis Sci.* 1994;39:315-320. [EL 3]
631. **Provenzale D, Reinhold RB, Golner B, et al.** Evidence for diminished B12 absorption after gastric bypass: oral supplementation does not prevent low plasma B12 levels in bypass patients. *J Am Coll Nutr.* 1992;11:29-35. [EL 3]
632. **Marcuard SP, Sinar DR, Swanson MS, Silverman JF, Levine JS.** Absence of luminal intrinsic factor after gastric bypass surgery for morbid obesity. *Dig Dis Sci.* 1989;34:1238-1242. [EL 3]
633. **Cooper PL, Brearley LK, Jamieson AC, Ball MJ.** Nutritional consequences of modified vertical gastroplasty in obese subjects. *Int J Obes Relat Metab Disord.* 1999;23:382-388. [EL 3]
634. **Rhode BM, Tamin H, Gilfix BM, Sampalis JS, Nohr C, MacLean LD.** Treatment of vitamin B12 deficiency after gastric surgery for severe obesity. *Obes Surg.* 1995;5:154-158. [EL 3]
635. **Rhode BM, Arseneau P, Cooper BA, Katz M, Gilfix BM, MacLean LD.** Vitamin B-12 deficiency after gastric surgery for obesity. *Am J Clin Nutr.* 1996;63:103-109. [EL 3]
636. **Kuzminski AM, Del Giacco EJ, Allen RH, Stabler SP, Lindenbaum J.** Effective treatment of cobalamin deficiency with oral cobalamin. *Blood.* 1998;92:1191-1198. [EL 2]
637. **Clements RH, Katasani VG, Palepu R, et al.** Incidence of vitamin deficiency after laparoscopic Roux-en-Y gastric bypass in a university hospital setting. *Am Surg.* 2006;72:1196-1202. [EL 3]
638. **Mejia LA.** Role of vitamin A in iron deficiency anemia. In: Fomon SJ, Zlotkin S, eds. *Nutritional Anemias.* New York, NY: Raven Press, 1992: 93-104. [EL 4]
639. **Shaw JC.** Copper deficiency in term and preterm infants. In: Fomon SJ, Zlotkin S, eds. *Nutritional Anemias.* New York, NY: Raven Press, 1992: 105-120. [EL 4]
640. **Todd LM, Godber IM, Gunn IR.** Iatrogenic copper deficiency causing anaemia and neutropenia. *Ann Clin Biochem.* 2004;41(pt 5):414-416. [EL 3]
641. **Nagano T, Toyoda T, Tanabe H, et al.** Clinical features of hematological disorders caused by copper deficiency during long-term enteral nutrition. *Intern Med.* 2005;44:554-559. [EL 3]
642. **Sharp P.** The molecular basis of copper and iron interactions. *Proc Nutr Soc.* 2004;63:563-569. [EL 4]
643. **Kumar N, Ahlskog JE, Gross JB Jr.** Acquired hypocupremia after gastric surgery. *Clin Gastroenterol Hepatol.* 2004;2:1074-1079. [EL 3]
644. **King JC, Shames DM, Woodhouse LR.** Zinc homeostasis in humans. *J Nutr.* 2000;130(5S suppl):1360S-1366S. [EL 4]
645. **McMillan DC, Sattar N, Talwar D, O'Reilly DS, McArdle CS.** Changes in micronutrient concentrations following anti-inflammatory treatment in patients with gastrointestinal cancer. *Nutrition.* 2000;16:425-428. [EL 3]
646. **Hambidge KM.** Zinc and diarrhea. *Acta Paediatr Suppl.* 1992;381:82-86. [EL 4]
647. **Vanderhoof JA, Scopinaro N, Tuma DJ, Gianetta E, Civalleri D, Antonson DL.** Hair and plasma zinc levels following exclusion of biliopancreatic secretions from functioning gastrointestinal tract in humans. *Dig Dis Sci.* 1983;28:300-305. [EL 3]
648. **Willis MS, Monaghan SA, Miller ML, et al.** Zinc-induced copper deficiency: a report of three cases initially recognized on bone marrow examination. *Am J Clin Pathol.* 2005;123:125-131. [EL 3]
649. **Jeejeebhoy KN.** Human zinc deficiency. *Nutr Clin Pract.* 2007;22:65-67. [EL 4]
650. **Angstadt JD, Bodziner RA.** Peripheral polyneuropathy from thiamine deficiency following laparoscopic Roux-en-Y gastric bypass. *Obes Surg.* 2005;15:890-892. [EL 3]
651. **Shuster MH, Vázquez JA.** Nutritional concerns related to Roux-en-Y gastric bypass: what every clinician needs to know. *Crit Care Nurs Q.* 2005;28:227-260. [EL 4]
652. **Carrodeguas L, Kaidar-Person O, Szomstein S, Antozzi P, Rosenthal R.** Preoperative thiamine deficiency in obese population undergoing laparoscopic bariatric surgery. *Surg Obes Relat Dis.* 2005;1:517-522. [EL 3]
653. **Gollobin C, Marcus WY.** Bariatric beriberi. *Obes Surg.* 2002;12:309-311. [EL 4]
654. **Mason EE.** Starvation injury after gastric reduction for obesity. *World J Surg.* 1998;22:1002-1007. [EL 4]
655. **MacLean LD, Rhode BM, Shizgal HM.** Nutrition following gastric operations for morbid obesity. *Ann Surg.* 1983;198:347-355. [EL 3]
656. **Feit H, Glasberg M, Ireton C, Rosenberg RN, Thal E.** Peripheral neuropathy and starvation after gastric partitioning for morbid obesity. *Ann Intern Med.* 1982;96:453-455. [EL 3]
657. **Fawcett S, Young GB, Holliday RL.** Wernicke's encephalopathy after gastric partitioning for morbid obesity. *Can J Surg.* 1984;27:169-170. [EL 3]
658. **Villar HV, Ranne RD.** Neurologic deficit following gastric partitioning: possible role of thiamine. *JPEN J Parenter Enteral Nutr.* 1984;8:575-578. [EL 3]
659. **Somer H, Bergström L, Mustajoki P, Rovamo L.** Morbid obesity, gastric plication and a severe neurological deficit. *Acta Med Scand.* 1985;217:575-576. [EL 3]
660. **Paulson GW, Martin EW, Mojzisek C, Carey LC.** Neurologic complications of gastric partitioning. *Arch Neurol.* 1985;42:675-677. [EL 3]
661. **Oczkowski WJ, Kertesz A.** Wernicke's encephalopathy after gastroplasty for morbid obesity. *Neurology.* 1985;35:99-101. [EL 3]
662. **Abarbanel JM, Berginer VM, Osimani A, Solomon H, Charuzi I.** Neurologic complications after gastric restriction surgery for morbid obesity. *Neurology.* 1987;37:196-200. [EL 3]
663. **Singh S, Kumar A.** Wernicke encephalopathy after obesity surgery: a systematic review. *Neurology.* 2007;68:807-811. [EL 4]

664. **Chaves LC, Faintuch J, Kahwage S, Alencar DdeA.** A cluster of polyneuropathy and Wernicke-Korsakoff syndrome in a bariatric unit. *Obes Surg.* 2002;12:328-334. [EL 3]
665. **Primavera A, Brusa G, Novello P, et al.** Wernicke-Korsakoff encephalopathy following biliopancreatic diversion. *Obes Surg.* 1993;3:175-177. [EL 3]
666. **Rindi G, Bordi C, Rappel S, La Rosa S, Stolte M, Solcia E.** Gastric carcinoids and neuroendocrine carcinomas: pathogenesis, pathology, and behavior. *World J Surg.* 1996;20:168-172. [EL 3]
667. **Heye N, Terstege K, Sirtl C, McMonagle U, Schreiber K, Meyer-Gessner M.** Wernicke's encephalopathy—causes to consider. *Intensive Care Med.* 1994;20:282-286. [EL 4]
668. **Melissas J, Christodoulakis M, Schoretsanitis G, et al.** Obesity-associated disorders before and after weight reduction by vertical banded gastroplasty in morbidly vs super obese individuals. *Obes Surg.* 2001;11:475-481. [EL 3]
669. **Adami GF, Papadia F, Carlini F, Murelli F, Scopinaro N.** Effect of biliopancreatic diversion on hypertension in severely obese patients. *Hypertens Res.* 2005;28:119-123. [EL 3]
670. **Valera-Mora ME, Simeoni B, Gagliardi L, et al.** Predictors of weight loss and reversal of comorbidities in malabsorptive bariatric surgery. *Am J Clin Nutr.* 2005;81:1292-1297. [EL 3]
671. **Hocking MP, Duerson MC, O'Leary JP, Woodward ER.** Jejunoileal bypass for morbid obesity: late follow-up in 100 cases. *N Engl J Med.* 1983;308:995-999. [EL 3]
672. **Armbrecht U, Lundell L, Lindstedt G, Stockbruegger RW.** Causes of malabsorption after total gastrectomy with Roux-en-Y reconstruction. *Acta Chir Scand.* 1988;154:37-41. [EL 3]
673. **Teo M, Chung S, Chitti L, et al.** Small bowel bacterial overgrowth is a common cause of chronic diarrhea. *J Gastroenterol Hepatol.* 2004;19:904-909. [EL 3]
674. **Ransford RA, Hayes M, Palmer M, Hall MJ.** A controlled, prospective screening study of celiac disease presenting as iron deficiency anemia. *J Clin Gastroenterol.* 2002;35:228-233. [EL 3]
675. **Scoglio R, Di Pasquale G, Pagano G, Lucanto MC, Magazzù G, Sferlazzas C.** Is intestinal biopsy always needed for diagnosis of celiac disease? *Am J Gastroenterol.* 2003;98:1325-1331. [EL 3]
676. **Murray JA, Watson T, Clearman B, Mitros F.** Effect of a gluten-free diet on gastrointestinal symptoms in celiac disease. *Am J Clin Nutr.* 2004;79:669-673. [EL 3]
677. **Wahab PJ, Meijer JW, Mulder CJ.** Histologic follow-up of people with celiac disease on a gluten-free diet: slow and incomplete recovery. *Am J Clin Pathol.* 2002;118:459-463. [EL 3]
678. **Podnos YD, Jimenez JC, Wilson SE, Stevens CM, Nguyen NT.** Complications after laparoscopic gastric bypass: a review of 3464 cases. *Arch Surg.* 2003;138:957-961. [EL 4]
679. **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. [EL 3]
680. **Livingston EH.** Complications of bariatric surgery. *Surg Clin North Am.* 2005;85:853-868, vii. [EL 4]
681. **Nemni J.** Severe chronic cough after Lap-Band gastric surgery. *Can Respir J.* 2007;14:171-172. [EL 3]
682. **Gentil B, Etienne-Mastroianni B, Cordier JF.** Chronic cough after laparoscopic adjustable gastric banding [article in French]. *Rev Mal Respir.* 2003;20(3, pt 1):451-454. [EL 3]
683. **Jones KB.** Biliopancreatic limb obstruction in gastric bypass at or proximal to the jejunojunostomy: a potentially deadly, catastrophic event. *Obes Surg.* 1996;6:485-493. [EL 3]
684. **Champion JK, Williams M.** Small bowel obstruction and internal hernias after laparoscopic Roux-en-Y gastric bypass. *Obes Surg.* 2003;13:596-600. [EL 3]
685. **Nelson LG, Gonzalez R, Haines K, Gallagher SF, Murr MM.** Spectrum and treatment of small bowel obstruction after Roux-en-Y gastric bypass. *Surg Obes Relat Dis.* 2006;2:377-383. [EL 3]
686. **Ahmed AR, O'Malley W.** Internal hernia with Roux loop obstruction during pregnancy after gastric bypass surgery. *Obes Surg.* 2006;16:1246-1248. [EL 4]
687. **Charles A, Domingo S, Goldfadden A, Fader J, Lampmann R, Mazzeo R.** Small bowel ischemia after Roux-en-Y gastric bypass complicated by pregnancy: a case report. *Am Surg.* 2005;71:231-234. [EL 3]
688. **Kakarla N, Dailey C, Marino T, Shikora SA, Chelmow D.** Pregnancy after gastric bypass surgery and internal hernia formation. *Obstet Gynecol.* 2005;105(5, pt 2):1195-1198. [EL 3]
689. **Dittrick GW, Thompson JS, Campos D, Bremers D, Sudan D.** Gallbladder pathology in morbid obesity. *Obes Surg.* 2005;15:238-242. [EL 3]
690. **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. [EL 4]
691. **Wudel LJ Jr, Wright JK, Debelak JP, Allos TM, Shyr Y, Chapman WC.** Prevention of gallstone formation in morbidly obese patients undergoing rapid weight loss: results of a randomized controlled pilot study. *J Surg Res.* 2002;102:50-56. [EL 2]
692. **O'Brien PE, Dixon JB.** A rational approach to cholelithiasis in bariatric surgery: its application to the laparoscopically placed adjustable gastric band. *Arch Surg.* 2003;138:908-912. [EL 3]
693. **Shiffman ML, Sugerman HJ, Kellum JH, Brewer WH, Moore EW.** Gallstones in patients with morbid obesity: relationship to body weight, weight loss and gallbladder bile cholesterol solubility. *Int J Obes Relat Metab Disord.* 1993;17:153-158. [EL 3]
694. **Sugerman HJ, Brewer WH, Shiffman ML, et al.** A multicenter, placebo-controlled, randomized, double-blind, prospective trial of prophylactic ursodiol for the prevention of gallstone formation following gastric bypass-induced rapid weight loss [with discussion]. *Am J Surg.* 1995;169:91-97. [EL 1]
695. **Fobi M, Lee H, Igwe D, et al.** Prophylactic cholecystectomy with gastric bypass operation: incidence of gallbladder disease. *Obes Surg.* 2002;12:350-353. [EL 3]
696. **Villegas L, Schneider B, Provost D, et al.** Is routine cholecystectomy required during laparoscopic gastric bypass? *Obes Surg.* 2004;14:206-211. [EL 3]
697. **Ellner SJ, Myers TT, Piorkowski JR, Mavanur AA, Barba CA.** Routine cholecystectomy is not mandatory during morbid obesity surgery. *Surg Obes Relat Dis.* 2007;3:456-460. [EL 3]
698. **Riordan SM, McIver CJ, Duncombe VM, Thomas MC, Bolin TD.** Evaluation of the rice breath hydrogen test for small intestinal bacterial overgrowth. *Am J Gastroenterol.* 2000;95:2858-2864. [EL 3]
699. **Kerlin P, Wong L.** Breath hydrogen testing in bacterial overgrowth of the small intestine. *Gastroenterology.* 1988;95:982-988. [EL 3]

700. **Rumessen JJ, Gudmand-Høyer E, Bachmann E, Justesen T.** Diagnosis of bacterial overgrowth of the small intestine: comparison of the 14C-D-xylose breath test and jejunal cultures in 60 patients. *Scand J Gastroenterol.* 1985;20:1267-1275. [EL 3]
701. **Schneider A, Novis B, Chen V, Leichtman G.** Value of the 14C-D-xylose breath test in patients with intestinal bacterial overgrowth. *Digestion.* 1985;32:86-91. [EL 3]
702. **Attar A, Flourié B, Rambaud JC, Franchisseur C, Ruszniewski P, Bouhnik Y.** Antibiotic efficacy in small intestinal bacterial overgrowth-related chronic diarrhea: a crossover, randomized trial. *Gastroenterology.* 1999;117:794-797. [EL 3]
703. **Di Stefano M, Miceli E, Missanelli A, Mazzocchi S, Corazza GR.** Absorbable vs. non-absorbable antibiotics in the treatment of small intestine bacterial overgrowth in patients with blind-loop syndrome. *Aliment Pharmacol Ther.* 2005;21:985-992. [EL 3]
704. **Correia MI, Nicoli JR.** The role of probiotics in gastrointestinal surgery. *Curr Opin Clin Nutr Metab Care.* 2006;9:618-621. [EL 4]
705. **Sugerman HJ, Kellum JM Jr, Reines HD, DeMaria EJ, Newsome HH, Lowry JW.** Greater risk of incisional hernia with morbidly obese than steroid-dependent patients and low recurrence with prefascial polypropylene mesh. *Am J Surg.* 1996;171:80-84. [EL 3]
706. **Cucchi SG, Pories WJ, MacDonald KG, Morgan EJ.** Gastrogastric fistulas: a complication of divided gastric bypass surgery. *Ann Surg.* 1995;221:387-391. [EL 3]
707. **MacLean LD, Rhode BM, Sampalis J, Forse RA.** Results of the surgical treatment of obesity. *Am J Surg.* 1993;165:155-160. [EL 2]
708. **Flanbaum L, Belsley S.** Factors affecting morbidity and mortality of Roux-en-Y gastric bypass for clinically severe obesity: an analysis of 1,000 consecutive open cases by a single surgeon. *J Gastrointest Surg.* 2007;11:500-507. [EL 3]
709. **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. [EL 3]
710. **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. [EL 3]
711. **Martin LF, Finigan KM, Nolan TE.** Pregnancy after adjustable gastric banding. *Obstet Gynecol.* 2000;95(6, pt 1):927-930. [EL 3]
712. **American College of Obstetricians and Gynecologists.** ACOG Committee opinion number 315, September 2005: obesity in pregnancy. *Obstet Gynecol.* 2005;106:671-675. [EL 4]
713. **Gerrits EG, Ceulemans R, van Hee R, Hendrickx L, Totté E.** Contraceptive treatment after biliopancreatic diversion needs consensus. *Obes Surg.* 2003;13:378-382. [EL 3]
714. **Weiss HG, Nehoda H, Labeck B, Hourmont K, Marth C, Aigner F.** Pregnancies after adjustable gastric banding. *Obes Surg.* 2001;11:303-306. [EL 3]
715. **Bilenka B, Ben-Schlomo I, Cozacov C, Gold CH, Zohar S.** Fertility, miscarriage and pregnancy after vertical banded gastroplasty operation for morbid obesity. *Acta Obstet Gynecol Scand.* 1995;74:42-44. [EL 3]
716. **Nelen WL, Blom HJ, Steegers EA, den Heijer M, Eskes TK.** Hyperhomocysteinemia and recurrent early pregnancy loss: a meta-analysis. *Fertil Steril.* 2000;74:1196-1199. [EL 3]
717. **Khong TY, Hague WM.** The placenta in maternal hyperhomocysteinemia. *Br J Obstet Gynaecol.* 1999;106:273-278. [EL 3]
718. **Mathews TJ, Honein MA, Erickson JD.** Spina bifida and anencephaly prevalence—United States, 1991-2001. *MMWR Recomm Rep.* 2002;51(RR-13):9-11. [EL 3]
719. **Ramirez MM, Turrentine MA.** Gastrointestinal hemorrhage during pregnancy in a patient with a history of vertical-banded gastroplasty. *Am J Obstet Gynecol.* 1995;173:1630-1631. [EL 3]
720. **Gurewitsch ED, Smith-Levitin M, Mack J.** Pregnancy following gastric bypass surgery for morbid obesity. *Obstet Gynecol.* 1996;88(4, pt 2):658-661. [EL 3]
721. **Huerta S, Rogers LM, Li Z, Heber D, Liu C, Livingston EH.** Vitamin A deficiency in a newborn resulting from maternal hypovitaminosis A after biliopancreatic diversion for the treatment of morbid obesity. *Am J Clin Nutr.* 2002;76:426-429. [EL 3]
722. **Woodard CB.** Pregnancy following bariatric surgery. *J Perinat Neonatal Nurs.* 2004;18:329-340. [EL 4]
723. **Haddow JE, Hill LE, Kloza EM, Thanhauser D.** Neural tube defects after gastric bypass. *Lancet.* 1986;1:1330. [EL 3]
724. **Martin L, Chavez GF, Adams MJ Jr, et al.** Gastric bypass surgery as maternal risk factor for neural tube defects. *Lancet.* 1988;1:640-641. [EL 3]
725. **Marceau P, Kaufman D, Biron S, et al.** Outcome of pregnancies after biliopancreatic diversion. *Obes Surg.* 2004;14:318-324. [EL 3]
726. **Bar-Zohar D, Azem F, Klausner J, Abu-Abeid S.** Pregnancy after laparoscopic adjustable gastric banding: perinatal outcome is favorable also for women with relatively high gestational weight gain. *Surg Endosc.* 2006;20:1580-1583. [EL 2]
727. **Wu JK.** Body contouring after bariatric surgery is critical for optimal cosmetic results. *MedGenMed.* 2006;8:77. [EL 4]
728. **American Society of Plastic Surgeons.** Body contouring after massive weight loss. <http://www.plasticsurgery.org/loader.cfm?url=/commonspot/security/getfile.cfm&p ageID=16766>. Accessed for verification April 23, 2008. [EL 3]
729. **Rohrich RJ.** Body contouring after massive weight loss supplement. *Plast Reconstr Surg.* 2006;117(suppl 1):1S-86S. [EL 4]
730. **Shermak MA, Chang DC, Heller J.** Factors impacting thromboembolism after bariatric body contouring surgery. *Plast Reconstr Surg.* 2007;119:1590-1596. [EL 3]
731. **Dymek MP, le Grange D, Neven K, Alverdy J.** Quality of life and psychosocial adjustment in patients after Roux-en-Y gastric bypass: a brief report. *Obes Surg.* 2001;11:32-39. [EL 3]
732. **van Gemert WG, Adang EM, Greve JW, Soeters PB.** Quality of life assessment of morbidly obese patients: effect of weight-reducing surgery. *Am J Clin Nutr.* 1998;67:197-201. [EL 3]
733. **Choban PS, Onyejekwe J, Burge JC, Flanbaum L.** A health status assessment of the impact of weight loss following Roux-en-Y gastric bypass for clinically severe obesity. *J Am Coll Surg.* 1999;188:491-497. [EL 3]
734. **Schok M, Geenen R, van Antwerpen T, de Wit P, Brand N, van Ramshorst B.** Quality of life after laparoscopic adjustable gastric banding for severe obesity: post-operative and retrospective preoperative evaluations. *Obes Surg.* 2000;10:502-508. [EL 3]

735. **Wyss C, Laurent-Jacard A, Burckhardt P, Jayet A, Gazzola L.** Long-term results on quality of life of surgical treatment of obesity with vertical banded gastroplasty. *Obes Surg.* 1995;5:387-392. [EL 3]
736. **Solow C, Silberfarb PM, Swift K.** Psychosocial effects of intestinal bypass surgery for severe obesity. *N Engl J Med.* 1974;290:300-304. [EL 3]
737. **Adami GF, Meneghelli A, Bressani A, Scopinaro N.** Body image in obese patients before and after stable weight reduction following bariatric surgery. *J Psychosom Res.* 1999;46:275-281. [EL 3]
738. **Kinzl JF, Trefalt E, Fiala M, Hotter A, Biebl W, Aigner F.** Partnership, sexuality, and sexual disorders in morbidly obese women: consequences of weight loss after gastric banding. *Obes Surg.* 2001;11:455-458. [EL 3]
739. **Valley V, Grace DM.** Psychosocial risk factors in gastric surgery for obesity: identifying guidelines for screening. *Int J Obes.* 1987;11:105-113. [EL 3]
740. **Kuldau JM, Barnard G, Kreutziger S, Rand CS.** Psychosocial effects of jejunoileal bypass for obesity: six-month follow-up. *Psychosomatics.* 1979;20:462, 467-472. [EL 3]
741. **Pories WJ, MacDonald KG.** The surgical treatment of morbid obesity. In: Daly M, ed. *Current Opinion in General Surgery.* Philadelphia, PA: Current Science Ltd., 1993: 195-205. [EL 4]
742. **Andersen T, Larsen U.** Dietary outcome in obese patients treated with a gastroplasty program. *Am J Clin Nutr.* 1989;50:1328-1340. [EL 3]
743. **Näslund I, Järnmark I, Andersson H.** Dietary intake before and after gastric bypass and gastroplasty for morbid obesity in women. *Int J Obes.* 1988;12:503-513. [EL 2]
744. **Brolin RE.** Bariatric surgery and long-term control of morbid obesity. *JAMA.* 2002;288:2793-2796. [EL 4]
745. **American Society for Parenteral and Enteral Nutrition.** A.S.P.E.N. Guidelines and Standards Library. <http://www.nutritioncare.org/Library.aspx>. Accessed for verification April 23, 2008. [EL 4]
746. **Merritt R, ed.** *The A.S.P.E.N. Nutrition Support Practice Manual.* 2nd ed. Silver Spring, MD: American Society for Parenteral and Enteral Nutrition, 2005. [EL 4]
747. **Segal A, Kinoshita Kusunoki D, Larino MA.** Post-surgical refusal to eat: anorexia nervosa, bulimia nervosa or a new eating disorder? A case series. *Obes Surg.* 2004;14:353-360. [EL 3]
748. **Schwartz RW, Strodel WE, Simpson WS, Griffen WO Jr.** Gastric bypass revision: lessons learned from 920 cases. *Surgery.* 1988;104:806-812. [EL 3]
749. **Schweitzer M.** Endoscopic intraluminal suture plication of the gastric pouch and stoma in postoperative Roux-en-Y gastric bypass patients. *J Laparoendosc Adv Surg Tech A.* 2004;14:223-226. [EL 3]
750. **Sanyal AJ, Sugerman HJ, Kellum JM, Engle KM, Wolfe L.** Stomal complications of gastric bypass: incidence and outcome of therapy. *Am J Gastroenterol.* 1992;87:1165-1169. [EL 3]
751. **Dresel A, Kuhn JA, McCarty TM.** Laparoscopic Roux-en-Y gastric bypass in morbidly obese and super morbidly obese patients. *Am J Surg.* 2004;187:230-232. [EL 2]
752. **Go MR, Muscarella P II, Needleman BJ, Cook CH, Melvin WS.** Endoscopic management of stomal stenosis after Roux-en-Y gastric bypass. *Surg Endosc.* 2004;18:56-59. [EL 3]
753. **Van Hee RH.** Biliopancreatic diversion in the surgical treatment of morbid obesity. *World J Surg.* 2004;28:435-444. [EL 4]
754. **Becker C.** Stapling standards: group issues bariatric best-practice guidelines. *Mod Healthc.* 2004;34:12. [EL 4]
755. **Edwards MA, Grinbaum R, Ellsmere J, Jones DB, Schneider BE.** Intussusception after Roux-en-Y gastric bypass for morbid obesity: case report and literature review of rare complication. *Surg Obes Relat Dis.* 2006;2:483-489. [EL 3]
756. **Totté E, Hendrickx L, van Hee R, et al.** Biliopancreatic diversion for treatment of morbid obesity: experience in 180 consecutive cases. *Obes Surg.* 1999;9:161-165. [EL 3]
757. **Gianetta E, Friedman D, Adami GF, et al.** Etiological factors of protein malnutrition after biliopancreatic diversion. *Gastroenterol Clin North Am.* 1987;16:503-504. [EL 4]
758. **Behrns KE, Smith CD, Kelly KA, Sarr MG.** Reoperative bariatric surgery: lessons learned to improve patient selection and results. *Ann Surg.* 1993;218:646-653. [EL 3]
759. **Yale CE.** Conversion surgery for morbid obesity: complications and long-term weight control. *Surgery.* 1989;106:474-480. [EL 3]
760. **Pessa M, Robertson J, Woodward ER.** Surgical management of the failed jejunoileal bypass. *Am J Surg.* 1986;151:364-367. [EL 4]
761. **Kirkpatrick JR.** Jejunioleal bypass: a legacy of late complications. *Arch Surg.* 1987;122:610-614. [EL 3]
762. **Yale CE.** Gastric bypass combined with reversal of intestinal bypass for morbid obesity. *World J Surg.* 1980;4:723-727. [EL 3]
763. **Sugerman HJ, Wolper JL.** Failed gastroplasty for morbid obesity: revised gastroplasty versus Roux-Y gastric bypass. *Am J Surg.* 1984;148:331-336. [EL 3]
764. **Brolin RE, Ravitch MM.** Experimental evaluation of techniques of gastric partitioning for morbid obesity. *Surg Gynecol Obstet.* 1981;153:877-882. [EL 3]
765. **Buckwalter JA, Herbst CA Jr.** Leaks occurring after gastric bariatric operations. *Surgery.* 1988;103:156-160. [EL 3]
766. **Torres JC, Oca CF, Honer HM.** Gastroplasty conversion to Roux-en-Y gastric bypass at the lesser curvature due to weight loss failure. *Am Surg.* 1985;51:559-562. [EL 3]
767. **Roller JE, Provost DA.** Revision of failed gastric restrictive operations to Roux-en-Y gastric bypass: impact of multiple prior bariatric operations on outcome. *Obes Surg.* 2006;16:865-869. [EL 3]
768. **Hanni CL, Pool LR, Dean RE, Cronquist JC.** Treatment of jejunoileal bypass failure by reanastomosis and gastroplasty in a single-stage procedure: review of 47 cases. *Am Surg.* 1984;50:354-357. [EL 3]
769. **Robertson JW, Woodward ER.** Gastric partition after reversal of jejunoileal bypass for morbid obesity: three-year follow-up. *South Med J.* 1985;78:1314-1316. [EL 3]
770. **Nesset EM, Kendrick ML, Houghton SG, et al.** A two-decade spectrum of revisional bariatric surgery at a tertiary referral center. *Surg Obes Relat Dis.* 2007;3:25-30. [EL 3]
771. **Kim CH, Sarr MG.** Severe reflux esophagitis after vertical banded gastroplasty for treatment of morbid obesity. *Mayo Clin Proc.* 1992;67:33-35. [EL 3]
772. **de Csepel J, Nahouraii R, Gagner M.** Laparoscopic gastric bypass as a reoperative bariatric surgery for failed open restrictive procedures. *Surg Endosc.* 2001;15:393-397. [EL 3]
773. **Gagner M, Gentileschi P, de Csepel J, et al.** Laparoscopic reoperative bariatric surgery: experience from 27 consecutive patients. *Obes Surg.* 2002;12:254-260. [EL 3]
774. **Kothari SN, DeMaria EJ, Sugerman HJ, Kellum JM, Meador J, Wolfe L.** Lap-Band failures: conversion to gastric bypass and their preliminary outcomes. *Surgery.* 2002;131:625-629. [EL 3]
775. **de Csepel J, Quinn T, Pomp A, Gagner M.** Conversion to a laparoscopic biliopancreatic diversion with a duodenal switch for failed laparoscopic adjustable silicone gastric banding. *J Laparoendosc Adv Surg Tech A.* 2002;12:237-240. [EL 3]
776. **Fox SR.** The use of the biliopancreatic diversion as a treatment for failed gastric partitioning in the morbidly obese. *Obes Surg.* 1991;1:89-93. [EL 3]
777. **Sapala JA, Sapala MA, Resto Soto AD, Quarermus JF.** A technique for converting the Roux-en Y gastric bypass to a modified biliopancreatic diversion. *Obes Surg.* 1991;1:311-313. [EL 3]