

Endocrine and Nutritional Management of the Post-Bariatric Surgery Patient: An Endocrine Society Clinical Practice Guideline

David Heber, Frank L. Greenway, Lee M. Kaplan, Edward Livingston, Javier Salvador, and Christopher Still

David Geffen School of Medicine at University of California (D.H.), Los Angeles, California 90095; Pennington Biomedical Research Center (F.L.G.), Louisiana State University System, Baton Rouge, Louisiana; Massachusetts General Hospital (L.M.K.), Boston, Massachusetts; University of Texas Southwestern Medical Center (E.L.), Dallas, Texas; University Clinic of Navarra (J.S.), Pamplona, Spain; and Geisinger Medical Center (C.S.), Danville, Pennsylvania

Objective: We sought to provide guidelines for the nutritional and endocrine management of adults after bariatric surgery, including those with diabetes mellitus. The focus is on the immediate postoperative period and long-term management to prevent complications, weight regain, and progression of obesity-associated comorbidities. The treatment of specific disorders is only summarized.

Participants: The Task Force was composed of a chair, five additional experts, a methodologist, and a medical writer. It received no corporate funding or remuneration.

Conclusions: Bariatric surgery is not a guarantee of successful weight loss and maintenance. Increasingly, patients regain weight, especially those undergoing restrictive surgeries such as laparoscopic banding rather than malabsorptive surgeries such as Roux-en-Y bypass. Active nutritional patient education and clinical management to prevent and detect nutritional deficiencies are recommended for all patients undergoing bariatric surgery. Management of potential nutritional deficiencies is particularly important for patients undergoing malabsorptive procedures, and strategies should be employed to compensate for food intolerance in patients who have had a malabsorptive procedure to reduce the risk for clinically important nutritional deficiencies. To enhance the transition to life after bariatric surgery and to prevent weight regain and nutritional complications, all patients should receive care from a multidisciplinary team including an experienced primary care physician, endocrinologist, or gastroenterologist and consider enrolling postoperatively in a comprehensive program for nutrition and lifestyle management. Future research should address the effectiveness of intensive postoperative nutritional and endocrine care in reducing morbidity and mortality from obesity-associated chronic diseases. (*J Clin Endocrinol Metab* 95: 4823–4843, 2010)

Summary of Recommendations

1.0 Prevention and treatment of weight regain (WR)

1.1 We recommend that a technically proficient surgical team, preferably accredited by a national certifying

organization, and an integrated medical support team able to provide dietary instruction and behavior modification be available postoperatively and during long-term follow-up (1|⊕⊕⊕⊕).

1.2 We recommend that treatment of WR postoperatively should include a multidisciplinary approach to med-

ISSN Print 0021-972X ISSN Online 1945-7197

Printed in U.S.A.

Copyright © 2010 by The Endocrine Society

doi: 10.1210/jc.2009-2128 Received October 5, 2009. Accepted July 2, 2010.

Abbreviations: AGB, Adjustable gastric banding; BPD, biliopancreatic diversion; 1,25-D, 1,25-dihydroxyvitamin D; 25-D, 25-hydroxyvitamin D; DS, duodenal switch; GBS, gastric bypass surgery; GLP-1, glucagon-like peptide-1; GS, gastric sleeve; HbA1c, glycated hemoglobin; LAGB, laparoscopic AGB; LDL, low-density lipoprotein; NAFLD, nonalcoholic fatty liver disease; RYGB, Roux-en-Y gastric bypass; T2DM, type 2 diabetes mellitus; WR, weight regain.

ical weight loss, including diet instruction, increased activity, behavior modification, and pharmacological therapy (1|⊕⊕⊕⊕).

1.3 We suggest, in cases of severe or unremitting postoperative weight gain, the determination of whether the surgical manipulation of the gastrointestinal tract remains anatomically intact [e.g. absence of gastrogastic fistula after Roux-en-Y gastric bypass (RYGB), integrity of band after a restrictive procedure]. If not intact, a multidisciplinary team should consider all options, including patient education, behavior modification, additional weight loss therapies, or referral for revisionary surgery as clinically indicated (2|⊕○○○).

2.0 Postoperative nutritional management

2.1 We recommend that nutritional management should include: an average of 60–120 g of protein daily in all patients to maintain lean body mass during weight loss and for the long term. This is especially important in those treated with malabsorptive procedures to prevent protein malnutrition and its effects (1|⊕⊕⊕⊕).

2.2 We recommend that long-term vitamin and mineral supplementation be considered in all patients undergoing bariatric surgery, with those who have had malabsorptive procedures requiring potentially more extensive replacement therapy to prevent nutritional deficiencies (1|⊕⊕⊕⊕).

2.3 We recommend periodic clinical and biochemical monitoring (see Table 2) for micro- and macronutritional deficiencies after bariatric surgery (1|⊕⊕⊕⊕).

3.0 Management of diabetes mellitus and lipids

3.1 We recommend that postoperative glycemic control should consist of achieving glycated hemoglobin (HbA1c) of 7% or less, with fasting blood glucose no greater than 110 mg/dl and postprandial glucose no greater than 180 mg/dl (1|⊕⊕⊕⊕).

3.2 We suggest that physicians and floor nurses be familiar with glycemic targets and insulin protocols, as well as the use of dextrose-free iv fluids and low-sugar liquid supplements (2|⊕○○○).

3.3 We recommend that obese patients with type 1 diabetes receive scheduled insulin therapy during their hospital stay, as required (1|⊕⊕⊕⊕).

3.4 We recommend that lipid abnormalities should be treated according to the National Cholesterol Education Program (NCEP) guidelines [Adult Treatment Panel III (ATP III)] and that existing lipid-lowering therapy for low-density lipoprotein (LDL)-cholesterol and triglyceride values should be continued after surgery if levels remain above desired goals (1|⊕⊕⊕⊕).

4.0 Bone health and gout

4.1 We recommend that patients who have undergone malabsorptive [*i.e.* RYGB, gastric sleeve (GS), biliopancreatic diversion (BPD)] obesity surgical procedures should have vitamin D, calcium, phosphorus, PTH, and alkaline phosphatase levels followed every 6 months and have a dual-energy x-ray absorptiometry for bone density performed yearly until stable (1|⊕⊕⊕⊕).

4.2 We recommend vitamin D and calcium supplementation postoperatively for malabsorptive obesity surgical procedures and that the doses be adjusted by a qualified medical professional based on serum markers and measures of bone density (1|⊕⊕⊕⊕).

4.3 We suggest that patients with frequent attacks of gout should have prophylactic therapy to lessen the chance of acute gout postoperatively as they lose weight (2|⊕○○○).

5.0 Gastroenterological and eating behavior considerations

5.1 We recommend that bariatric surgery patients should sip fluids in the immediate postoperative period when fully awake after surgery and that they can only be discharged if satisfactorily tolerating oral fluids (1|⊕⊕⊕⊕).

5.2 Particularly after procedures with a gastric restrictive component, we recommend that gradual progression of food consistency over weeks to months be used to allow patients to adjust to a restrictive meal plan and to minimize vomiting, which can damage surgical anastomoses or lead to gastroesophageal reflux after restrictive procedures (1|⊕⊕⊕⊕).

5.3 We suggest continuous reinforcement of new nutritional habits that discourage the intake of simple carbohydrate-dense foods and beverages, to minimize the frequency of bothersome gastrointestinal symptoms due to dumping, including abdominal pain and cramping, nausea, diarrhea, lightheadedness, flushing, tachycardia, and syncope (2|⊕○○○).

5.4 We suggest that patients, who present with postprandial symptoms of hypoglycemia, particularly neuroglycopenic symptoms, should undergo further evaluation for the possibility of insulin-mediated hypoglycemia (2|⊕○○○).

Method of Development of Evidence-Based Recommendations

The Clinical Guidelines Subcommittee of The Endocrine Society deemed endocrine and nutritional management of the post-bariatric surgery patient a priority area in need of

practice guidelines and appointed a Task Force to formulate evidence-based recommendations. The Task Force followed the approach recommended by the Grading of Recommendations, Assessment, Development, and Evaluation group, an international group with expertise in development and implementation of evidence-based guidelines.

The Task Force used the best available research evidence that members identified to inform the recommendations and consistent language and graphical descriptions of both the strength of a recommendation and the quality of evidence. To indicate the strength of the recommendation, strong recommendations use the phrase “we recommend” and the number 1, and weak recommendations use the phrase “we suggest” and the number 2. Cross-filled circles represent the quality of the evidence, such that ⊕○○○ denotes very low quality evidence; ⊕⊕○○, low quality; ⊕⊕⊕○, moderate quality; and ⊕⊕⊕⊕, high quality. The Task Force has confidence that patients who receive care according to the strong recommendations will derive, on average, more good than harm. Weak recommendations require more careful consideration of the patient’s circumstances, values, and preferences to determine the best course of action. A detailed description of this grading scheme has been published elsewhere.

Linked to each *recommendation* is a description of the *evidence*, the *values* that panelists considered in making the recommendation (when making these explicit was necessary), and *remarks*, a section in which panelists offer technical suggestions for testing conditions, dosing, and monitoring. These technical comments reflect the best available evidence applied to a typical patient. Often, this evidence comes from the unsystematic observations of the panelists and should, therefore, be considered suggestions.

Introduction

The incidence of severe obesity has increased more rapidly than the incidence of nonsevere obesity. Between 1999 and 2004, obesity increased by 24% in the United States, whereas the incidence of severe obesity is rising even more rapidly (1). Bariatric surgery has gained wide acceptance as a treatment for severe obesity, especially when complicated by type 2 diabetes mellitus (T2DM). An estimated 200,000 operations will be performed in 2009 alone at a cost of about \$5 billion. After surgery, patients are cared for by their primary care physicians, endocrinologists, or gastroenterologists. Frequently, these patients present with associated comorbidities, including T2DM, polycystic ovarian disease, metabolic bone disease, lipid abnormalities, fatty liver, degenerative joint disease, hypertension, gastroesophageal reflux disease, and obstructive sleep apnea.

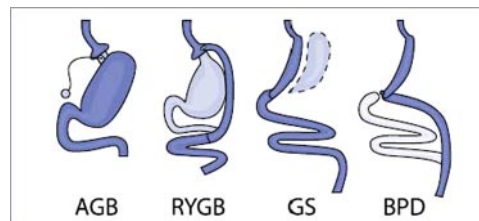


FIG. 1. Diagram of surgical options. [Adapted with permission from W. J. Pories: *J Clin Endocrinol Metab* 93:S89–S96, 2008 (248). © The Endocrine Society.]

Bariatric surgery is not a guarantee of success, and patients require postoperative care. To reduce the likelihood of weight regain (WR) and to ensure that comorbid conditions are adequately managed, all patients should receive careful medical follow-up postoperatively. To guide patients through the transition to life after bariatric surgery, a multidisciplinary team that includes an experienced primary care physician, endocrinologist, or gastroenterologist should provide care, and patients should consider enrolling postoperatively in a comprehensive program for nutrition and lifestyle management. Such support can ease the transition to life after bariatric surgery and may help prevent WR.

Common operations include various banding procedures, which restrict the amount of food entering the stomach, the RYGB, the duodenal switch (DS)/GS, or the BPD (Fig. 1). The modifications of gastrointestinal function after these surgeries are least with banding, greater with RYGB, and greatest with BPD or DS/GS. As the physiological alterations of gastrointestinal function increase, there is an impression that less medical, dietary, and behavioral intervention is needed to induce weight loss. Pure restrictive operations such as adjustable gastric banding are more commonly associated with WR and weight loss failure than techniques with a malabsorptive component such as RYGB. However, the use of routine algorithms in postoperative care is essential to reduce the risk of WR and postoperative complications.

Postoperative management of the bariatric surgery patient begins by having the proper team in place before the operation is performed. To enhance the likelihood of long-term success, the bariatric surgeon should be part of a comprehensive team that provides pre- and postoperative care. Patient support groups have the additional advantage of maintaining contact between the patients and their primary care physicians, endocrinologists, or gastroenterologists who provide care for medical needs. Support groups may also aid in the prevention of WR by keeping patients focused on lifestyle issues over the long term, but this has not been demonstrated in clinical trials. In addition, the facility where the surgeon practices must have experience with bariatric patients and a familiarity with routine postoperative care.

Physicians referring patients to bariatric surgery should request specific experience and performance data from the bariatric surgeon or program regarding the procedure being considered. Various resources are available to locate a suitable bariatric surgeon on the Internet or by contacting the Surgical Review Corporation, American Society for Metabolic and Bariatric Surgery, American College of Surgeons, or the Obesity Society. These resources should be a starting point for finding surgeons to work in a collaborative fashion with endocrinologists, gastroenterologists, and primary care physicians interested in the postoperative care of patients after bariatric surgery.

1.0 Prevention and Treatment of WR

Recommendations

1.1 We recommend that a technically proficient surgical team, preferably accredited by a national certifying organization, and an integrated medical support team able to provide dietary instruction and behavior modification be available postoperatively and during long-term follow-up (1|⊕⊕⊕⊕).

1.2 We recommend that treatment of WR postoperatively should include a multidisciplinary approach to medical weight loss, including diet instruction, increased activity, behavior modification, and pharmacological therapy (1|⊕⊕⊕⊕).

1.3 We suggest in cases of severe or unremitting postoperative weight gain the determination of whether the surgical manipulation of the gastrointestinal tract remains anatomically intact (e.g. absence of gastrogastic fistula after RYGB and integrity of band after a restrictive procedure). If not intact, a multidisciplinary team should consider all options, including patient education, behavior modification, additional weight loss therapies, or referral for revisionary surgery as clinically indicated (2|⊕○○○).

1.1–1.3 Evidence

WR is not uncommon in patients undergoing bariatric surgery, and it can be expected that 20–25% of the lost weight will be regained over a period of 10 yr. The impact of this WR on comorbid conditions is dependent on individual risk factors. The vast majority of long-term studies after either pure restrictive or mixed techniques show WR (2–5), which in some cases may lead to a percentage of excess weight loss lower than 50% (5–7). Although WR prevalence has been reported in 7–50% of cases (7, 8), this classification as WR is based on an arbitrary amount of WR (50% of the lost weight). On the other hand, loss of patients to follow-up at late stages may underestimate the true prevalence of WR. Significant WR is accompanied by reversal or reduction of

TABLE 1. Causes and prevention of WR

Causes
Noncompliance with dietary and lifestyle recommendations
Physiological factors (variations in response to surgery)
Surgical failure
Prevention
Optimizing patient selection criteria
Realistic preoperative expectations
Consideration of benefits of bypass vs. restrictive procedures
Adherence to scheduled visits

surgically improved obesity comorbidities, including common medical conditions and psychosocial functioning, which may lead to a decrease in quality of life (3, 9).

Causes of WR

WR is most commonly related to noncompliance with dietary and lifestyle instructions, although differences in physiological responses and occasionally surgical failure can be the cause (Table 1). Food records show that calorie intake is reduced after bariatric surgery, but increases at 1–2 yr after surgery coincide with WR (3). In general, patients report greater physical activity over the long term compared with the preoperative period (3, 10). Some studies have suggested an influence of genetic factors (11, 12). Although it has been suggested that gastrointestinal hormones such as ghrelin, glucagon-like peptide-1 (GLP-1), and peptide YY 3-36 may be involved in postoperative weight homeostasis (13–15) due to observed decreases in ghrelin concentrations and increases in GLP-1 and peptide YY after RYGB and BPD, other studies do not confirm a clear relationship between these changes, appetite/satiety scores, and weight reduction (16). A reduction in leptin and insulin serum concentrations may also play a role (17). Weight loss is always accompanied by a reduction in resting energy expenditure, but this decrease is proportional to the loss of lean body mass, and therefore, there is no evidence of adaptive decreases in resting metabolic rate due to surgery that could explain WR (17, 18). Mechanical problems such as band slippage or pouch and stomal dilation, especially in restrictive operations such as vertical banded gastroplasty, gastric banding, sleeve gastrectomy, and RYGB could potentially impair gastric neural signals driving satiety sensations to the central nervous system, favoring increased food intake and WR. No conclusive evidence that WR is due to surgical factors has been found (19, 20). Adaptive intestinal mechanisms leading to changes in the absorptive capacity of the small bowel can also influence WR (21, 22). In general, pure restrictive operations are more commonly associated with WR and weight loss failure than other techniques with a malabsorptive component (4, 23, 24). A recent meta-analysis of 14 studies (25) found that excess body weight loss at 1 yr was 76% after RYGB compared with 50% after laparoscopic adjustable banding and

that long-term reoperation rates were lower after RYGB (16 vs. 24%). Psychological factors and eating disorders can also promote WR, especially when developed in the postsurgical period (26). Although individuals differ in their response to surgery, postsurgery adherence to scheduled visits and compliance, more than personality disorders, was found to predict outcome of bariatric restrictive surgery in severely obese patients (27).

Prevention and treatment of WR

Prevention of WR is essential to maintain the benefits of bariatric surgery on a long-term basis. Key factors are preoperative realistic expectations, adherence to scheduled visits (27), compliance with nutritional recommendations, maintenance of regular physical activity of at least 150 min/wk (28), and periodic assessment to prevent or treat eating or other psychiatric disorders (27, 29). In general, bariatric surgery has a favorable impact on psychological condition (30, 31), although some improvements may disappear over time. From the nutritional point of view, a low glycemic load, moderately high protein content diet, combined with a physical activity program has been shown to effectively treat WR in the short term (32). Promoting adherence to diet and lifestyle recommendations by collecting food records and monitoring body weight carefully is also useful. Participation in support groups could also be helpful in the prevention and treatment of WR (33, 34).

Because patients with a mechanically intact malabsorptive operation who have experienced WR are not likely to achieve sustained weight loss after pouch revision, revisional surgery is inadvisable for them (35). When WR is severe and unremitting, consideration should be given to revisional bariatric surgery, and this should be discussed

with a surgeon experienced in revisional surgery. In some cases, RYGB or DS (36, 37) can be indicated after failure of a previous restrictive operation. Nevertheless, application of conventional strategies and the risk of serious postoperative complications must be carefully evaluated before making this decision (36).

Assumed values and preferences

Our recommendation places a high value on potential benefits derived from maintenance of weight reduction to control obesity-associated comorbidities and to improve psychological function, general health, and quality of life.

2.0 Postoperative Nutritional Management

Recommendations

2.1 We recommend that nutritional management should include an average of 60–120 g of protein daily in all patients to maintain lean body mass during weight loss and for the long term. This is especially important in those treated with malabsorptive procedures to prevent protein malnutrition and its effects (1|⊕⊕⊕⊕).

2.2 We recommend that long-term vitamin and mineral supplementation be considered in all patients undergoing bariatric surgery, with those who have had malabsorptive procedures requiring potentially more extensive replacement therapy to prevent nutritional deficiencies (1|⊕⊕⊕⊕).

2.3 We recommend periodic clinical and biochemical monitoring (Table 2) for micro- and macronutritional deficiencies after bariatric surgery (1|⊕⊕⊕⊕).

TABLE 2. Schedule for clinical and biochemical monitoring

	Preoperative	1 month	3 months	6 months	12 months	18 months	24 months	Annually
Complete blood count	X	X	X	X	X	X	X	X
LFTs	X	X	X	X	X	X	X	X
Glucose	X	X	X	X	X	X	X	X
Creatinine	X	X	X	X	X	X	X	X
Electrolytes	X	X	X	X	X	X	X	X
Iron/ferritin	X			X ^a	X ^a	X ^a	X ^a	X ^a
Vitamin B12	X			X ^a	X ^a	X ^a	X ^a	X ^a
Folate	X			X ^a	X ^a	X ^a	X ^a	X ^a
Calcium	X			X ^a	X ^a	X ^a	X ^a	X ^a
Intact PTH	X			X ^a	X ^a	X ^a	X ^a	X ^a
25-D	X			X ^a	X ^a	X ^a	X ^a	X ^a
Albumin/prealbumin	X			X ^a	X ^a	X ^a	X ^a	X ^a
Vitamin A	X						Optional	Optional
Zinc	X			Optional	Optional		Optional	Optional
Bone mineral density and body composition	X				X ^a		X ^a	X ^a
Vitamin B1			Optional	Optional	Optional	Optional	Optional	Optional

Data indicate the suggested schedule for laboratory monitoring after bariatric surgery. LFT, Liver function tests.

^a Examinations should only be performed after RYGB, BPD, or BPD/DS. All of them are considered as suggested for patients submitted to restrictive surgery where frank deficiencies are less common.

2.1–2.3 Evidence

Protein intake

Protein malnutrition, defined by hypoalbuminemia (albumin < 3.5 mg/dl), remains the most severe macronutrient complication associated with malabsorptive surgical procedures. Some studies have reported it in 13% of superobese patients 2 yr after a distal RYGB with Rouxlimb at least 150 cm, less than 5% of patients with a Rouxlimb less than 150 cm (38, 39), and 3–18% of patients after BPD (40–45). Other studies have found only a 0–6% incidence of protein deficiency after RYGB up to 43 months postoperatively (46–48). Protein malnutrition causes an annual hospitalization rate of 1% per year after malabsorptive procedures and leads to significant morbidity (42, 49).

When it occurs, protein malnutrition is generally observed at 3–6 months after surgery and is largely attributed to the development of food intolerance to protein-rich foods (50). Protein-deficient meals are common after RYGB. Purely restrictive procedures [adjustable gastric banding (AGB) and sleeve gastrectomy], for example, can induce digestive symptoms, food intolerance, or maladaptive eating behaviors due to pre- or postsurgical eating disorders (51). Prevention of protein malnutrition requires regular assessment of protein intake and counseling regarding ingestion of protein from protein-rich foods and modular protein supplements. Protein needs for adults relate to body weight. Dietary protein need is often presented as a percentage of energy intake. The dietary reference intakes represent the acceptable protein range as 10–35% of total energy. However, protein needs are constant across all energy intakes. So at low energy intake, protein needs to be a higher percentage of total calories, and at high energy intake, protein can be reduced as a percentage of total calories. In general, dietary protein should be established first in any diet in proportion to body weight, and then carbohydrates and fats should be added as determined by energy needs. Protein is an important part of good nutrition at every meal. Vitamins and minerals can fulfill nutrient needs on a once-per-day basis, but for protein, the body has no ability to store a daily supply. To maintain healthy muscles and bones for adults, at least 30 g of protein should be consumed at more than one meal. Breakfast is an important meal for dietary protein because the body is in a catabolic state after an overnight fast. A meal with at least 30 g protein is required to initiate repletion of body proteins. Protein at breakfast is also critical for regulation of appetite and daily food intake. The recommended dietary allowance represents the minimum daily intake for active healthy adults. For most adults, replacing some dietary carbohydrates with protein

will help to maintain body composition and mobility, improve blood lipids and lipoproteins, and help to control food intake (52–55).

Modular protein supplements can be sorted into four categories: 1) protein concentrates derived from a complete protein such as milk, soy, or eggs; 2) protein concentrates derived from collagen, either alone or in combination with a complete protein; 3) doses of one or more dispensable (nonessential) amino acids; and 4) hybrids of the complete or collagen-based proteins and amino acid dose. Modular protein supplements are generally provided either as a substrate for protein synthesis or as a source of one or more amino acids that may be conditionally indispensable (conditionally essential) (50).

Hospitalization with initiation of parenteral nutrition support may be required (38) in cases of severe protein deficiency, but there are no currently accepted guidelines or clinical studies guiding nutritional therapy after weight loss surgery. Nutritional support with parenteral nutrition for at least 3–4 wk may rarely be required after RYGB when enteral nutrition is not successful (56). Caution must be exercised with the initiation of solutions containing high amounts (>100–200 g/d) of dextrose in the setting of severe malnutrition to avoid refeeding syndrome. Symptoms of 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 (57). If a patient requires prolonged parenteral nutrition, then surgical revision and lengthening of the common channel to decrease malabsorption is warranted (41), although this will increase the likelihood of WR.

Vitamin and mineral supplementation

The anatomic changes imposed by malabsorptive surgery increase the risk for various vitamin and mineral deficiencies, which can occur commonly within the first year after surgery (42, 43, 48, 58–62). After RYGB, screening and supplementation of deficiencies with a multivitamin-mineral, iron, vitamin B12, or calcium with vitamin D is routinely conducted, and prophylactic supplementation should be considered in patients at increased risk (*e.g.* existing osteoporosis and heavy menstruation) (42, 57, 63, 64). Best practice guidelines published recently recommend a daily multivitamin and calcium supplementation with added vitamin D for all weight-loss surgery patients (65).

Vitamin D in doses required to optimize vitamin D status should be carefully considered. Suboptimal vitamin D levels are now recognized to be a common condition in the general population and should be screened for before sur-

gery by measuring 25-hydroxyvitamin D (25-D) levels. Recommended doses of elemental calcium after bariatric surgery range from 1200–2000 mg daily, and these usually contain vitamin D as well (41, 48, 57, 66). Calcium and vitamin D can also be given as separate supplements. Calcium carbonate preparations are easily available in chewable forms and are better tolerated shortly after surgery. However, patients must be instructed to take calcium carbonate preparations with meals to enhance intestinal absorption. Calcium citrate preparations are preferred because this salt is better absorbed in the absence of gastric acid production (67–69).

The multivitamin-mineral preparations should have the recommended daily requirements for vitamins and minerals. Initially, one to two tablets of a chewable preparation are advised because they are better tolerated after malabsorptive procedures. However, nonchewable preparations or products with fortified amounts of folic acid and iron, such as prenatal vitamins, can be used.

Vitamin B12 deficiencies can occur after bariatric surgery procedures that bypass the lower stomach. Impairment of vitamin B12 absorption after RYGB results from decreased digestion of the protein-bound cobalamins and impaired formation of intrinsic factor-vitamin B12 complexes required for absorption (57, 70–72). According to one study, 30% of RYGB patients receiving only a multivitamin supplement will have a B12 deficiency after 1 yr (73). In other studies, the incidence of vitamin B12 deficiency after RYGB is 33–40% at postoperative yr 1 (74) and 8–37% by yr 2–4 (48, 60, 75, 76). In a study of vertical banded gastroplasty patients ($n = 26$), there were no instances of vitamin B12 deficiency at 1 yr (77). Anemias as a result of vitamin B12 deficiency have been reported to occur in more than 30% of patients 1–9 yr after RYGB (42, 78).

The initiation of vitamin B12 supplementation within 6 months postoperatively is recommended by most surgical groups in the absence of controlled studies. Oral crystalline vitamin B12 at a dose of at least 350 $\mu\text{g}/\text{d}$ has been shown to maintain normal plasma vitamin B12 levels (78–80). Optimal dosing of oral, sublingual, or intranasal forms of B12 supplementation has not been well studied. However, in a study of postoperative RYGB patients by Clements *et al.* (81), 1000 μg vitamin B12 im every 3 months or intranasal B12, 1000 μg every week, resulted in a lower incidence of vitamin B12 deficiency (3.6% at 1 yr and 2.3% at 2 yr) compared with the frequency of 12–37% described by Brolin and Leung (62). In many institutions, intranasal administration of vitamin B12 has been supplanted by sublingual administration of vitamin B12. One study demonstrated that oral and sublingual administration of 500 μg vitamin B12 were equally efficacious in correcting vitamin B12 deficiency (82).

Regardless of the preparation, multivitamin supplements providing 400 $\mu\text{g}/\text{d}$ folate can effectively prevent the development of folate deficiency after RYGB (48, 56, 83). This suggests that the intake of folic acid from the diet and routine multivitamins is generally sufficient to prevent folic acid deficiency.

Iron deficiency is common after Roux-en Y bypass, especially for women with menorrhagia due to excessive menstrual blood loss. For this reason, prophylactic iron supplementation is required to reduce the risk of iron deficiency anemia (84–86). Decreased liberation and absorption of heme from foods are caused from bypass of the acid environment in the lower stomach and the absorptive surfaces of the duodenum and upper jejunum (87–89). Moreover, meals after malabsorptive procedures are frequently low in meats, which results in decreased heme intake. Iron deficiency may also be exacerbated as a result of a nutrient-nutrient inhibitory absorptive interaction between iron and calcium, another mineral that should be given routinely during the postoperative period. Most studies (90, 91), but not all studies (92), show that nonheme- and heme-iron absorption is inhibited up to 50–60% when consumed in the presence of calcium supplements or with dairy products. Calcium at doses of 300–600 mg has a direct dose-related inhibiting effect on iron absorption. This has been seen with calcium carbonate, calcium citrate, and calcium phosphate. The risk for iron deficiency increases over time, with some series reporting that more than half of subjects had low ferritin levels 4 yr after the RYGB, BPD, or BPD/DS (48). Iron deficiency after RYGB is influenced by multiple factors and can persist to 7 yr postoperatively (93). Iron deficiency has been reported to occur in up to 50% of patients after RYGB, most frequently in women with menorrhagia (63, 64). Thus, empiric iron supplementation is recommended (84, 85). In a randomized, controlled trial, iron supplementation (65 mg elemental iron by mouth twice daily) prevented the development of iron deficiency, although it did not always prevent the development of anemia (85), suggesting that in some subjects after RYGB, anemia may be related to factors other than iron deficiency. Supplementation with lower doses (80 mg/d) does not universally prevent iron deficiency (48). Vitamin C increases iron absorption and should be included empirically with iron supplementation (65, 84). Because oral iron supplementation is associated with poor absorption and adverse gastrointestinal effects, and injections are painful, intermittent iv iron infusion may be required during treatment. Iron dextran, ferric gluconate, or ferric sucrose may be administered iv. Supplementation should follow currently accepted guidelines to normalize hemoglobin (85), and continued surveillance of hemoglobin and iron studies is recommended.

Steatorrhea induced by malabsorptive surgical procedures can lead to deficiencies in fat-soluble vitamins, which typically present as an eczematous rash (38, 42, 49). Vitamin A deficiency after bariatric surgery results from poor nutritional intake, maldigestion, malabsorption, and impaired hepatic release of vitamin A. In two series, the incidence of vitamin A deficiency was 61–69% 2–4 yr after BPD, with or without DS (40, 94). In a third series, the incidence was as low as 5% by 4 yr (62). Although data are scarce, mild vitamin A deficiency can also occur after distal RYGB procedures and is easily corrected with oral supplementation (62). Oral supplementation of vitamin A, 5,000–10,000 IU/d, is recommended until the vitamin A level normalizes. Vitamin K deficiency can also be common with BPD and BPD/DS. In a research setting, vitamin K levels have been measured, and levels were low in 50–60% of patients who underwent BPD or BPD/DS (68, 95). In that study, no clinical symptoms such as easy bruising, increased bleeding, clotting alterations, or metabolic bone disease because of the role of vitamin K in osteocalcin formation were observed. In the clinical setting, vitamin K should be supplemented orally or im when INR values rise above 1.4 as the measurement of vitamin K levels and effects on vitamin K-induced proteins are research procedures.

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 (96, 97). Acute neurological deficits as a result of thiamine deficiency have been reported as soon as 1–3 months after surgery (98–107). Early recognition is paramount to initiate appropriate supplementation and to avoid potential complications resulting from the administration of dextrose-containing solutions (108). Although not often evaluated, thiamine status is best assessed by determining erythrocyte transketolase activity. Parenteral supplementation with thiamine (100 mg/d) should be initiated in the patient with active neurological symptoms (109, 110). After a 7- to 14-d course, an oral preparation (10 mg/d) can be used until neurological symptoms resolve (56, 111, 112). 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 excessive band tightness or slippage after laparoscopic AGB (LAGB). It is important that persistent vomiting be resolved aggressively to prevent this devastating complication.

Biochemical and clinical monitoring

The extent of metabolic and nutritional evaluation completed after bariatric surgery should be guided by the surgical procedure performed. Purely gastric restrictive procedures are not associated with alterations in intestinal continuity

and do not alter normal digestive physiology. As a result, selective nutritional deficiencies are uncommon.

Regular monitoring and screening of laboratory values and nutritional intake before and after bariatric surgery are key to ensuring adequacy of nutrition. Therefore, they are recommended after bariatric surgeries, even if patients tolerate their diet well with no vomiting or diarrhea, to detect subclinical nutritional deficiencies and prevent development of frank deficiencies (113–116). Malabsorptive procedures can be associated with micronutrient and macronutrient deficiencies and require lifelong supplementation and monitoring of laboratory data by a team familiar with possible deficiencies (113, 114). Fat-soluble vitamin levels, especially vitamin A, should be monitored annually after malabsorptive procedures (48, 60). Restrictive procedures, often overlooked, such as LAGB, also require certain attention to supplementation and laboratory data secondary to decreased intake or poor tolerance of certain foods or food groups. Baseline data should be obtained before bariatric surgery to permit correction of deficiencies and to provide comparison values.

Selection and timing of preoperative laboratory tests is based on each patient's specific clinical indications because obesity alone is not a risk factor for postoperative complications (117). Evaluation by the anesthesiologist can reveal important preoperative risk factors including metabolic syndrome, respiratory diseases including asthma, and peripheral vascular or thrombotic predisposition. The use of a designated anesthesia team familiar with bariatric operations can help maximize perioperative management and minimize complications. There is insufficient evidence to recommend ordering routine preoperative tests (118), but in view of the high risk for development of micronutrient deficiencies after malabsorptive procedures, preoperative evaluation of iron status (Fe, total iron binding capacity, ferritin, and/or serum transferrin receptor), vitamin B12, 25-D, and PTH is recommended (Table 3). Preoperative micronutrient deficiencies have been described in bariatric surgery patients, *e.g.* 14–43.9% iron deficiency, 5–29% B12 deficiency, and 40–68.1% vitamin D deficiency (119, 120). Treatment for clinically significant deficiencies, *e.g.* iron deficiency anemia, should be initiated preoperatively.

3.0 Management of Diabetes Mellitus and Lipids

Recommendations

3.1 We recommend that postoperative glycemic control should consist of achieving glycated HbA1c of 7% or less with fasting blood glucose no greater than 110 mg/dl and postprandial glucose no greater than 180 mg/dl (1⊕⊕⊕○).

TABLE 3. Diagnosis and treatment of nutritional deficiencies

Deficiency	Symptoms and signs	Confirmation	Treatment first phase	Treatment second phase
Protein malnutrition	Weakness, decreased muscle mass, brittle hair, generalized edema	Serum albumin and prealbumin levels, serum creatinine	Protein supplements	Enteral or parenteral nutrition; reversal of surgical procedure
Calcium/vitamin D	Hypocalcemia, tetany, tingling, cramping, metabolic bone disease	Total and ionized calcium levels, intact PTH, 25-D, urinary N-telopeptide, bone densitometry	Calcium citrate, 1,200–2,000 mg, oral vitamin D, 50,000 IU/d	Calcitriol oral vitamin D 1,000 IU/d
Vitamin B12	Pernicious anemia, tingling in fingers and toes, depression, dementia	Blood cell count, vitamin B12 levels	Oral crystalline B12, 350 μ g/d	1,000–2,000 μ g/2–3 months im
Folic acid	Macrocytic anemia, palpitations, fatigue, eural tube defects	Cell blood count, folic acid levels, homocysteine	Oral folate, 400 μ g/d (included in multivitamin)	Oral folate, 1,000 μ g/d
Iron	Decreased work ability, palpitations, fatigue, koilonychia, pica, brittle hair, anemia	Blood cell count, serum iron, iron binding capacity, ferritin	Ferrous sulfate 300 mg 2–3 times/d, taken with vitamin C	Parenteral iron administration
Vitamin A	Xerophthalmia, loss of nocturnal vision, decreased immunity	Vitamin A levels	Oral vitamin A, 5,000–10,000 IU/d	Oral vitamin A, 50,000 IU/d

Details are shown for the diagnosis and treatment for specific nutritional deficiencies.

3.2 We suggest that physicians and floor nurses be familiar with glycemic targets and insulin protocols as well as the use of dextrose-free iv fluids and low-sugar liquid supplements (2|⊕○○○).

3.3 We recommend that obese patients with type 1 diabetes receive scheduled insulin therapy during their hospital stay, as required (1|⊕⊕⊕○).

3.4 We recommend that lipid abnormalities should be treated according to the NCEP guidelines (ATP III) and that existing lipid-lowering therapy for LDL-cholesterol and triglyceride values should be continued after surgery if levels remain above desired goals (1|⊕⊕⊕○).

3.1–3.4 Evidence

Type 2 diabetes mellitus

T2DM is commonly associated with severe obesity but can improve to the point that little or no medication is necessary in patients after RYGB (121–123). Fasting plasma glucose concentrations have been reported to return to normal before hospital dismissal and before significant weight loss (124–137). After RYGB or BPD/DS/GS, insulin-treated patients experience a significant decrease in insulin requirements; the majority of patients can discontinue insulin therapy by 6 wk after surgery (136, 138), and some may even be able to discontinue insulin before hospital discharge. The long-term effects of these bypass operations appear to include both weight loss-dependent and -independent effects (139).

By contrast, gastric restrictive operations such as banding appear to improve T2DM as a result of the weight loss itself. Therefore, the effects will likely be reversed if there is WR (140).

The longer T2DM has been present, the less likely it is to respond to surgically induced weight loss (124, 126), most likely due to destruction of pancreatic β -cells. Whether weight loss and/or bypass surgery itself will also slow the cellular and molecular events leading to β -cell destruction in the long term has not been established.

Improvements in hyperglycemia are observed almost immediately after RYGB, in part due to increased release of GLP-1 (141–143) and possibly other incretins. Rubino and Gagner (144) observed that RYGB and BPD achieved durable primary beneficial effects on glycemic control in 80–100% of patients with T2DM, independent of effects on body weight. These conclusions were supported by rat studies in which gastrojejunal bypass controlled T2DM independent of weight loss (145). In a subsequent study of 10 obese patients undergoing RYGB, a potential mechanism was elucidated (136). Bypass of the proximal small bowel was associated with a statistically significant increase 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 (136). These and/or other intestinal factors may also restore meal-induced suppression of ghrelin release

from the stomach, resulting in decreased food intake (146). One explanation for the immediate effects of RYGB and intestinal bypass on glucose metabolism is that secretion of incretins, including glucose-dependent insulinotropic polypeptide and GLP-1, recovers rapidly after surgery. Bypass of the duodenum without gastric bypass or ileal interposition has been found to improve diabetes in both animal models and patients (136, 143, 147). Although these changes in glucose homeostasis may play a physiological role, more research is needed to determine their contribution to glucose control under real-world conditions of rapid weight loss after RYGB.

Postoperative glycemic control

Achievement of postoperative glycemic control (HbA1c \leq 7%; blood glucose \leq 110 mg/dl fasting and \leq 180 mg/dl postprandial) represents a realistic goal (148, 149). Preoperative glycemic control represented by an HbA1c less than 7% has been associated with decreased perioperative infectious complications (150). Patients with poor control on oral medications or who require high doses of insulin preoperatively may require insulin for several days after surgery to maintain blood glucose concentrations in a desirable range.

Patients requiring insulin before surgery should have their blood glucose concentrations monitored regularly and insulin administered as needed to control hyperglycemia. In the intensive care unit, glycemic control can be maintained with a nurse-driven, dynamic intensive insulin therapy protocol targeting a blood glucose level of 140–180 mg/dl (151). In non-intensive care unit patients, target glycemic control is accomplished with sc insulin: basal insulinization insulin treatment with intermediate-acting NPH insulin, long-acting insulin glargine, or insulin detemir; bolus preprandial insulinization with rapid-acting insulin aspart, glulisine, or lispro; and correction insulin every 3–6 h, also with a rapid-acting insulin (152).

Physicians and floor nurses should be familiar with glycemic targets and insulin protocols as well as with the use of dextrose-free iv fluids and low-sugar liquid supplements. Parameters for starting iv insulin should follow established clinical protocols. Patients should be instructed on regular monitoring of metered blood glucose concentrations to guide adjustments in glucose-lowering therapy. In the patient with persistent hyperglycemia, continued surveillance and preventive care as recommended by the American Diabetes Association are advised. Sulfonylurea drugs should generally be avoided in the immediate postoperative period when insulin sensitivity may improve and increase the risk of hypoglycemia. These agents should be reintroduced later only if clinically indicated. The long-term management of patients who achieve

remission of their T2DM after surgery is not established, but routine follow-up should be continued.

Postoperative pregnancies

Women with a history of oligomenorrhea and androgenicity due to polycystic ovarian syndrome may become fertile during the postoperative period and should be counseled that unexpected pregnancies can occur unless contraceptive methods are employed. The management of pregnancy requires meeting the nutritional needs of a pregnant mother with attention to micronutrients and protein. We generally recommend that patients take precautions to avoid pregnancy for 12 to 18 months after surgery. Rates of many adverse maternal and neonatal outcomes may be lower in women who become pregnant after having had bariatric surgery compared with rates in pregnant women who are obese; however, further data are needed from rigorously designed studies (153).

Fatty liver disease and nonalcoholic fatty liver disease (NAFLD)

Many obese patients will have abnormal liver function tests with asymptomatic increases in serum alanine aminotransferase and aspartate aminotransferase. These changes are most commonly associated with fatty liver disease or NAFLD. At the time of surgery, 84% of severely obese subjects have steatosis on liver biopsy specimens (154), 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 cases (155–164). The clinical challenge is to determine which patients require additional evaluation, because fatty liver disease is a diagnosis of exclusion. Gallstones, chronic hepatitis B or C, alcohol use, and potential side effects of medications (such as acetaminophen, nonsteroidal inflammatory agents, and clopidogrel) are among the less common causes of liver disease. Patients with marked increases in liver function tests (generally considered at two to three times the upper limit of normal) should be considered for additional testing by hepatobiliary ultrasonography or computed tomography, and a hepatitis screen if this was not done before surgery (165). Patients with mild-to-moderate cirrhosis may benefit from bariatric surgery with acceptable complication risks (166). If cirrhosis is suspected, preoperative endoscopy should be undertaken to rule out esophageal or gastric varices and/or need for transplantation (158), and liver transplant patients may undergo successful bariatric surgery (167). NAFLD is being increasingly recognized as an important cause of liver-related morbidity and mortality (168) and may be the most common cause of cryptogenic cirrhosis in the obese pa-

tient (169). Abnormal transaminases should be followed at appropriate intervals until they fall into the normal range or stabilize.

Lipid disorders

Triglyceride and LDL-cholesterol decrease and high-density lipoprotein-cholesterol increases after LAGB, RYGB, BPD, or BPD/DS surgery (170–183). However, conventional lipid measurements of total and LDL-cholesterol may not be reflective of dyslipidemic risks and/or insulin resistance in obese people, as suggested by a cross-sectional study of 572 obese patients (184). The improvement in dyslipidemia appears to be related not only to the percentage of excess weight loss (170) but also to the decrease in insulin resistance (170). Given the improvement in cardiovascular mortality after bariatric surgery, these changes have likely led to a decreased risk of cardiovascular disease. Recent studies show decreased cardiovascular and myocardial infarction mortality in bariatric surgery patients (185). Previously unrecognized lipid abnormalities may be identified and can strengthen the case for medical necessity for these procedures. Lipid abnormalities should be treated according to the NCEP guidelines (186). Lipid-lowering therapy for LDL-cholesterol and triglyceride values that remain above desired goals after surgery should be continued. BPD and BPD/DS procedures have been associated with lower triglyceride and LDL values (128). Due to the dramatic reductions in lipid levels, the doses of lipid-lowering drugs should be periodically reevaluated.

4.0 Bone Health and Gout

Recommendations

4.1 We recommend that patients who have undergone malabsorptive (*i.e.* RYGB, GS, and BPD) obesity surgical procedures should have vitamin D, calcium, phosphorus, PTH, and alkaline phosphatase levels followed every 6 months and have a dual-energy x-ray absorptiometry for bone density performed yearly until stable (1|⊕⊕⊕⊕).

4.2 We recommend vitamin D and calcium supplementation postoperatively for malabsorptive obesity surgical procedures and that the doses be adjusted by a qualified medical professional based on serum markers and measures of bone density (1|⊕⊕⊕⊕).

4.3 We suggest that patients with frequent attacks of gout should have prophylactic therapy to lessen the chance of acute gout postoperatively as they lose weight (2|⊕○○○).

4.1–4.3 Evidence

Obesity is associated with greater bone density, but weight loss by diet or other means decreases bone density.

Bone loss is accompanied by an increase in bone turnover, but only malabsorptive procedures cause a disproportionate loss of bone compared with weight loss through dietary calorie restriction.

The Roux-en-Y procedure is the leading bariatric operation performed in the United States. In this surgery, the primary sites for calcium absorption are bypassed. Patients become calcium- and vitamin D-deficient, and the body then up-regulates PTH, causing increased production of vitamin D and increased calcium resorption from bone. Gastric banding uses a restrictive band and has not been shown to produce the same bone loss as the Roux-en-Y procedure, nor has there been evidence of secondary hyperparathyroidism (187–199).

Overall, after a malabsorptive bariatric procedure, 10–25% of patients develop a calcium deficiency by 2 yr and 25–48% by 4 yr; 17–52% of patients develop a vitamin D deficiency by 2 yr and 50–63% by 4 yr (40, 69, 74, 94, 200, 201). Increased awareness regarding the prevalence of metabolic bone disease after malabsorptive procedures has led to the recommendation that calcium supplementation be routinely provided (41, 56, 202, 203).

Vitamin D deficiency and bone mineralization defects result from decreased sunlight exposure, maldigestion, impaired mixing of pancreatic and biliary secretions, and decreased vitamin D absorption in the proximal small bowel (42, 63, 204–207). 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 a day may be necessary.

Indicators of bone loss in malabsorptive procedures can be detected in serum measures at 6 months. Supplementation with vitamin D and calcium can improve parameters of bone health, but large amounts may be needed in some individuals.

At present, there are no conclusive data regarding the association of altered calcium and vitamin D homeostasis with LAGB surgery. In two reports, LAGB was not associated with significant reduction in bone mineral density (208, 209). Calcium deficiency and metabolic bone disease can occur in RYGB patients (55, 62, 206, 210, 211). The onset of metabolic bone disease 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 (56, 63, 207, 212).

A rise in serum intact PTH indicates negative calcium balance and/or a vitamin D deficiency. Elevations of bone-specific alkaline phosphatase and osteocalcin levels, which indicate increased osteoblastic activity and bone formation, are often the initial abnormalities found (63,

207). The appropriate use of bone turnover markers has been proposed as a useful screening tool for metabolic bone disease after RYGB because serum calcium and phosphate levels are often normal, but this has not been established (56, 207, 212, 213).

After gastric restrictive procedures, urinary C-telopeptide levels, indicative of increased bone resorption, are elevated (213). In the event of prolonged immobilization after LAGB or RYGB, increased bone resorption, especially in association with critical illness, might be associated with hypercalciuria and, if renal calcium excretion is impaired, frank hypercalcemia (214). Rapid and extreme weight loss is associated with bone loss (215–217), even in the presence of normal vitamin D and PTH levels (213).

Decreased weight-bearing after surgery may also contribute to bone loss and can be estimated with N- or C-telopeptide levels (213). After a malabsorptive bariatric procedure, patients might have continued secondary hyperparathyroidism, low 25-D levels, increased 1,25-dihydroxyvitamin D (1,25-D) levels, and hypocalciuria (67, 207, 210, 211, 213, 218). Left uncorrected, secondary hyperparathyroidism will promote bone loss and increases the risk for osteopenia and osteoporosis (210). The presence of hypocalcemia in the setting of vitamin D deficiency exacerbates mineralization defects and accelerates the development of osteomalacia (219). In an observational study by Diniz Mde *et al.* (220), 29% of patients developed secondary hyperparathyroidism and 0.9% hypocalcemia beyond RYGB postoperative month 3. Parada *et al.* (221) reported that 53% of patients had secondary hyperparathyroidism after RYGB. Youssef *et al.* (222) found a greater degree of secondary hyperparathyroidism and vitamin D deficiency with longer Roux limb length after RYGB.

Riedt *et al.* (223) found that women who have had a RYGB experienced decreased estradiol- and vitamin D-dependent intestinal calcium absorption. This was associated with increased N-telopeptide (marker of bone resorption), increased osteocalcin (marker of bone formation), or an uncoupling effect on bone remodeling (223). Compston *et al.* (60) found an increased incidence of metabolic bone disease with standard BPD and a 50-cm common channel, but without reduced serum 25-D levels. After bariatric surgery, the most common cause of secondary hyperparathyroidism with normal vitamin D levels is calcium deficiency. A common regimen consists of weekly parenteral ergocalciferol, 100,000 IU, until 25-D levels normalize. Primary treatment is with ergocalciferol, but in individuals with persistently elevated PTH levels or bone loss, calcitriol (1,25-D) therapy has been used in this setting. However, appropriate use has not been estab-

lished. Intravenous (0.25–0.5 $\mu\text{g}/\text{d}$) or oral (0.25–1.0 μg daily or twice daily) calcitriol therapy has been used in situations characterized by symptomatic hypocalcemia and severe vitamin D malabsorption. Many obese patients have suboptimal levels of vitamin D, and it is important to normalize vitamin D levels preoperatively when the procedure contemplated is likely to result in vitamin D malabsorption. However, in asymptomatic patients in whom 25-D levels fail to reach optimal levels (25-D > 30 ng/ml), functionally normalize 1,25-D levels, and suppress elevated PTH levels, the use of calcitriol is unproven. Adequate calcium and vitamin D supplementation has been achieved when levels for serum calcium, bone-specific alkaline phosphatase or osteocalcin, 25-D, and 24-h urinary calcium excretion rates are normal. PTH levels may persist above the normal range, even with functionally replete vitamin D levels (25-D > 30 ng/ml). Monitoring of vitamin D and PTH levels should be accompanied by monitoring of calcium. If elevated calcium levels are found, then PTH levels should be measured to detect primary hyperparathyroidism. In most bariatric surgery patients, there will be secondary hyperparathyroidism secondary to negative calcium balance as indicated rather than primary hyperparathyroidism.

Obese patients with a body mass index greater than 40 kg/m² are at greater risk for osteoarthritis, progression of arthritis, and gout, which can improve with weight loss (224). After bariatric surgery, hip and knee pain may improve, and exercise capacity may increase (225–228). Moreover, serum uric acid levels decrease (81). Gout may be precipitated during weight loss after intestinal bypass (211), just as surgery itself is a risk factor for acute gout attacks. Therefore, patients with frequent attacks of gout should have prophylactic therapy started well in advance of surgery to lessen the chance of acute gout immediately after surgery.

5.0 Gastroenterological and Eating Behavior Considerations

Recommendations

5.1 We recommend that bariatric surgery patients should sip fluids in the immediate postoperative period when fully awake after surgery and that they can only be discharged if satisfactorily tolerating oral fluids (1⊕⊕⊕⊕).

5.2 Particularly after procedures with a gastric restrictive component, we recommend that gradual progression of food consistency over weeks to months be used to allow patients to adjust to a restrictive meal plan and to minimize vomiting, which can damage surgical anastomoses or lead to gastroesophageal reflux after restrictive procedures (1⊕⊕⊕⊕).

5.3 We suggest continuous reinforcement of new nutritional habits that discourage the intake of simple carbohydrate-dense foods and beverages to minimize the frequency of bothersome gastrointestinal symptoms due to dumping, including abdominal pain and cramping, nausea, diarrhea, lightheadedness, flushing, tachycardia, and syncope (2|⊕○○○).

5.4 We suggest that patients who present with postprandial symptoms of hypoglycemia, particularly neuroglycopenic symptoms, should undergo further evaluation for the possibility of insulin-mediated hypoglycemia (2|⊕○○○).

5.1–5.4 Evidence

Vomiting and surgical complications

Chronic vomiting, generally described by the patient as spitting up or the food gets stuck, can occur. One third to two thirds of patients report postoperative vomiting (229–231). Vomiting is thought to occur most commonly during the first few postoperative months (232), when 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 tract. Frequent vomiting may suggest: 1) obstruction, necessitating evaluation with a gastrointestinal contrast study, before any endoscopic procedure in LAGB patients; 2) reflux, inflammation, stoma erosion/ulceration, or stenosis, necessitating endoscopy; or 3) gastric dysmotility, necessitating a radionuclide gastric-emptying study. Regurgitation that occurs after a LAGB can be managed with appropriate band adjustments and nutritional advice.

Continuous reinforcement of new nutritional habits will help minimize the frequency of bothersome gastrointestinal symptoms. Guidance remains important to optimize nutritional intake in patients who have had a malabsorptive procedure because of the risk for clinically important nutritional deficiencies (233). For surgeries with a gastric restrictive component, regular visits with the clinical team provide guidance as the meal plan is progressed. The limited volume capacity of the gastric pouch (30–60 ml) results in marked restrictions in the amount and rate at which food can be eaten. During the first few months after surgery, episodes of regurgitation, typically without nausea or true vomiting, are common if food is consumed in large volumes or too quickly or if it is not chewed thoroughly.

RYGB has been associated with staple line failure (234, 235) and a stomal ulceration rate of up to 16% (234, 236). Staple line disruption and gastrogastic fistulas can also occur after gastric transection and increase the risk of mar-

ginal ulceration (234, 237). More recent stapling techniques only rarely result in staple line failure, although there is no clear guidance regarding the optimal stapling method.

Late surgical complications include anastomotic stricture, staple line dehiscence, pouch dilation, internal hernia with intestinal obstruction (complete or partial), anastomotic leaks, and incisional hernias (41, 238). An internal hernia after RYGB, BPD, or BPD/DS is a potentially fatal complication secondary to bowel infarction and peritonitis. The symptoms are those of a small bowel obstruction with cramping pain, usually periumbilical. There are three locations for an internal hernia: at the jejunojejunostomy, through the mesocolon, or between the Roux limb mesentery, mesocolon, and the retroperitoneum (Petersen hernia). Diagnosis may be obtained with a gastrografin upper gastrointestinal or abdominal computed tomography; however, as with a leak, these studies are often misleading (41). In many instances, the best course of management is an exploratory laparotomy or laparoscopy for recurrent cramping abdominal pain.

Dumping syndrome

Abdominal pain and cramping, nausea, diarrhea, lightheadedness, flushing, tachycardia, and syncope, indicative of dumping, are reported frequently and serve to discourage the intake of energy-dense foods and beverages (203, 239, 240). Gastric dumping occurs initially in 70–76% of patients who have had a RYGB (61, 124, 241). However, 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 (242). For some patients, dumping may be considered to be a desired side effect because it discourages ingestion of calorically dense liquids that could mitigate weight loss. It used to be thought that dumping symptoms were the result of the hyperosmolarity of intestinal contents, which resulted in 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 dumping symptoms because they can often be blocked with sc octreotide, a somatostatin analog (243).

Dumping symptoms tend to become less prominent with time (240) and can usually be controlled with certain nutritional changes, such as: 1) eating small, frequent meals; 2) avoiding ingestion of liquids within 30 min of a solid-food meal; 3) avoiding simple sugars and increasing intake of fiber and complex carbohydrates; and 4) increas-

ing protein intake (244). If these measures are unsuccessful, then octreotide, 50 μg sc 30 min before meals may reduce symptoms in some patients (244).

Late dumping can be due to reactive hypoglycemia and can often be managed with nutritional manipulation or be treated prophylactically by having the patient eat a small snack.

Postprandial hypoglycemia

Post-RYGB patients who present with postprandial symptoms of hypoglycemia, particularly neuroglycopenic symptoms, should undergo further evaluation for the possibility of insulin-mediated hypoglycemia. In a study conducted in one institution, only nine adult patients without a history of gastric bypass had surgically confirmed nesidioblastosis during the same period in which six patients were evaluated and treated for the condition after gastric bypass surgery (GBS) (245). The study described six patients with severe, intractable postprandial symptoms associated with endogenous hyperinsulinemic hypoglycemia. This complication, believed to be secondary to the RYGB anatomy in some patients, has necessitated partial pancreatectomy for relief of the symptoms and hypoglycemia. In these patients, histological examination demonstrated pancreatic islet cell hyperplasia. This complication may present from 2 to 9 yr after RYGB. In a recent study of 14 patients with hyperinsulinemic hypoglycemia, the glucose and insulin responses to mixed meals were measured (246). A subsequent study of six RYGB patients with postoperative hypoglycemia compared with lean and obese controls without hypoglycemia failed to find an increase in β -cell mass (247) and concluded that post-GBS hypoglycemia is not due to an increase in β -cell mass or formation. Rather, they concluded that postprandial hypoglycemia after GBS is due to a combination of gastric dumping and inappropriately increased insulin secretion, either as a failure to adaptively decrease insulin secretion after GBS or as an acquired phenomenon (247).

Suggested Directions for Future Research

Due to the nature of the physician-patient relationship in individualizing bariatric surgical approaches, research that compares different types of surgery in a randomized, prospective, controlled design study is challenging. However, it would be possible to design a postoperative study that assigns subjects randomly to standard intervention or intensive intervention to examine effects on WR, morbidity, and mortality. By stratifying the study to examine laparoscopic adjustable banding and RYGB, it would be possible to tailor the interventions to the needs of the two

types of operations as restrictive and malabsorptive. It is also possible that this type of research could be applied successfully to more aggressive malabsorptive procedures as long as the appropriate safety standards were incorporated.

Finally, treatment of diabetes and metabolic disease through surgical intervention requires greater study. The scientific rationale for the approach is sound, but questions remain pertaining to long-term outcome and the possible occurrence of nesidioblastosis after gastric bypass. These issues and the impact on overall mortality in diabetes deserve much more attention in future clinical research.

Acknowledgments

The members of the Task Force thank The Endocrine Society's Clinical Guidelines Subcommittee and Clinical Affairs Core Committee and Council for their careful, critical review of earlier versions of this manuscript and their helpful comments and suggestions. We also thank the leadership of the American Gastroenterological Association Institute, and the European Society of Endocrinology for their review and comments. In addition, we thank the many members of The Endocrine Society who reviewed the draft version of this manuscript when it was posted on the Society's website and who sent a great number of additional comments and suggestions, most of which were incorporated into the final version of the manuscript. Finally, we thank the staff at the Society office for their helpful support during the development of this guideline.

Address all correspondence to: The Endocrine Society, 8401 Connecticut Avenue, Suite 900, Chevy Chase, MD 20815. E-mail: govt-prof@endo-society.org, Telephone: 301-941-0200. Address all commercial reprint requests for orders 101 and more to: Walchli Tauber Group Inc. E-mail: Karen.burkhardt@wt-group.com. Address all reprint requests for orders for 100 or fewer to Society Services, Telephone: 301-941-0210, E-mail: societyservices@endo-society.org, or Fax: 301-941-0257.

Cosponsoring associations include the American Gastroenterological Association Institute, and European Society of Endocrinology.

Financial Disclosure of Task Force

David Heber, M.D., Ph.D. (chair) — Financial or Business/Organizational Interests: Herbalife International; Significant Financial Interest or Leadership Position: Medical Nutrition Council, American Society for Nutrition (Chair, 2006–2007); Frank L. Greenway, III, M.D. — Financial or Business/Organizational Interests: ABIC International Consultants, BAROnova, Basic Research Inc., Catalyst Pharmaceutical Partners, General Nutrition Corp., Health and Nutrition Technology, Lipothera, Nastech

Pharmaceuticals, Orexigen Therapeutics, GlaxoSmithKline, Leptos Biomedical, Novo Nordisk, Schering-Plough Research Institute; Significant Financial Interest or Leadership Position: Jenny Craig (Chairman); Lee M. Kaplan, M.D., Ph.D.—Financial or Business/Organizational Interests: Merck Research Labs, GI Dynamics, Johnson & Johnson, Davol, Stryker Development, Gelesis, Blue Cross & Blue Shield; Significant Financial Interest or Leadership Position: none declared; Edward Livingston, M.D., F.A.C.S.—Financial or Business/Organizational Interests: none declared; Significant Financial Interest or Leadership Position: none declared; Javier Salvador, M.D., Ph.D.—Financial or Business/Organizational Interests: none declared; Significant Financial Interest or Leadership Position: none declared; Christopher Still, D.O., M.S.—Financial or Business/Organizational Interests: Ethion-Endosurgery; Significant Financial Interest or Leadership Position: Obesity Action Coalition, Surgical Review Corp., ABPNS American Board of Physician Nutrition Specialists.

References

- Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM 2006 Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA* 295:1549–1555
- Sjöström L, Narbro K, Sjöström CD, Karason K, Larsson B, Wedel H, Lystig T, Sullivan M, Bouchard C, Carlsson B, Bengtsson C, Dahlgren S, Gummesson A, Jacobson P, Karlsson J, Lindroos AK, Lönnroth H, Näslund I, Olbers T, Stenlöf K, Torgerson J, Agren G, Carlsson LM; Swedish Obese Subjects Study 2007 Effects of bariatric surgery on mortality in Swedish obese subjects. *N Engl J Med* 357:741–752
- Sjöström L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, Dahlgren S, Larsson B, Narbro K, Sjöström CD, Sullivan M, Wedel H; Swedish Obese Subjects Study Scientific Group 2004 Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med* 351:2683–2693
- Maggard MA, Shugarman LR, Suttorp M, Maglione M, Sugarman HJ, Sugarman HJ, Livingston EH, Nguyen NT, Li Z, Mojica WA, Hilton L, Rhodes S, Morton SC, Shekelle PG 2005 Meta-analysis: surgical treatment of obesity. *Ann Intern Med* 142:547–559
- Pajcecki D, Dalcanelle L, Souza de Oliveira CP, Zilberstein B, Halpern A, Garrido Jr AB, Cecconello I 2007 Follow-up of Roux-en-Y gastric bypass patients at 5 or more years postoperatively. *Obes Surg* 17:601–607
- Larrad-Jiménez A, Díaz-Guerra CS, de Cuadros Borrajo P, Lesmes IB, Esteban BM 2007 Short-, mid- and long-term results of Larrad biliopancreatic diversion. *Obes Surg* 17:202–210
- Capella JF, Capella RF 1996 The weight reduction operation of choice: vertical banded gastroplasty or gastric bypass? *Am J Surg* 171:74–79
- Magro DO, Geloneze B, Delfini R, Pareja BC, Callejas F, Pareja JC 2008 Long-term weight regain after gastric bypass: a 5-year prospective study. *Obes Surg* 18:648–651
- Karlsson J, Taft C, Rydén A, Sjöström L, Sullivan M 2007 Ten-year trends in health-related quality of life after surgical and conventional treatment for severe obesity: the SOS intervention study. *Int J Obes (Lond)* 31:1248–1261
- Silver HJ, Torquati A, Jensen GL, Richards WO 2006 Weight, dietary and physical activity behaviors two years after gastric bypass. *Obes Surg* 16:859–864
- Parikh M, Lo H, Chang C, Collings D, Fielding G, Ren C 2006 Comparison of outcomes after laparoscopic adjustable gastric banding in African-Americans and whites. *Surg Obes Relat Dis* 2:607–610; discussion 610–612
- Xu Y, Ramos EJ, Middleton F, Romanova I, Quinn R, Chen C, Das U, Inui A, Meguid MM 2004 Gene expression profiles post Roux-en-Y gastric bypass. *Surgery* 136:246–252
- Cummings DE, Weigle DS, Frayo RS, Breen PA, Ma MK, Dellinger EP, Purnell JQ 2002 Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. *N Engl J Med* 346:1623–1630
- Frühbeck G, Rotellar F, Hernández-Lizoain JL, Gil MJ, Gómez-Ambrosi J, Salvador J, Cienfuegos JA 2004 Fasting plasma ghrelin concentrations 6 months after gastric bypass are not determined by weight loss or changes in insulinemia. *Obes Surg* 14:1208–1215
- le Roux CW, Welbourn R, Werling M, Osborne A, Kokkinos A, Laurenus A, Lönnroth H, Fändriks L, Ghatei MA, Bloom SR, Olbers T 2007 Gut hormones as mediators of appetite and weight loss after Roux-en-Y gastric bypass. *Ann Surg* 246:780–785
- Christou NV, Look D, McLean AP 2005 Pre- and post-prandial plasma ghrelin levels do not correlate with satiety or failure to achieve a successful outcome after Roux-en-Y gastric bypass. *Obes Surg* 15:1017–1023
- Coupaye M, Bouillot JL, Coussieu C, Guy-Grand B, Basdevant A, Oppert JM 2005 One-year changes in energy expenditure and serum leptin following adjustable gastric banding in obese women. *Obes Surg* 15:827–833
- Carey DG, Pliego GJ, Raymond RL 2006 Body composition and metabolic changes following bariatric surgery: effects on fat mass, lean mass and basal metabolic rate: six months to one-year follow-up. *Obes Surg* 16:1602–1608
- Madan AK, Tichansky DS, Phillips JC 2007 Does pouch size matter? *Obes Surg* 17:317–320
- Nishie A, Brown B, Barloon T, Kuehn D, Samuel I 2007 Comparison of size of proximal gastric pouch and short-term weight loss following routine upper gastrointestinal contrast study after laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 17:1183–1188
- O'Brien PE, McPhail T, Chaston TB, Dixon JB 2006 Systematic review of medium-term weight loss after bariatric operations. *Obes Surg* 16:1032–1040
- Borg CM, le Roux CW, Ghatei MA, Bloom SR, Patel AG 2007 Biliopancreatic diversion in rats is associated with intestinal hypertrophy and with increased GLP-1, GLP-2 and PYY levels. *Obes Surg* 17:1193–1198
- te Riele WW, Vogten JM, Boerma D, Wiezer MJ, van Ramshorst B 2008 Comparison of weight loss and morbidity after gastric bypass and gastric banding: a single center European experience. *Obes Surg* 18:11–16
- Prachand VN, Davee RT, Alverdy JC 2006 Duodenal switch provides superior weight loss in the super-obese (BMI \geq 50 kg/m²) compared with gastric bypass. *Ann Surg* 244:611–619
- Tice JA, Karliner L, Walsh J, Petersen AJ, Feldman MD 2008 Gastric banding or bypass? A systematic review comparing the two most popular bariatric procedures. *Am J Med* 121:885–893
- Burgmer R, Grigutsch K, Zipfel S, Wolf AM, de Zwaan M, Husemann B, Albus C, Senf W, Herpertz S 2005 The influence of eating behavior and eating pathology on weight loss after gastric restriction operations. *Obes Surg* 15:684–691
- Pontiroli AE, Fossati A, Vedani P, Fiorilli M, Folli F, Paganelli M, Marchi M, Maffei C 2007 Post-surgery adherence to scheduled visits and compliance, more than personality disorders, predict outcome of bariatric restrictive surgery in morbidly obese patients. *Obes Surg* 17:1492–1497
- Evans RK, Bond DS, Wolfe LG, Meador JG, Herrick JE, Kellum JM, Maher JW 2007 Participation in 150 min/wk of moderate or higher intensity physical activity yields greater weight loss after gastric bypass surgery. *Surg Obes Relat Dis* 3:526–530

29. van Hout GC, Verschure SK, van Heck GL 2005 Psychosocial predictors of success following bariatric surgery. *Obes Surg* 15:552–560
30. Mamplekou E, Komesidou V, Bissias Ch, Papakonstantinou A, Melissas J 2005 Psychological condition and quality of life in patients with morbid obesity before and after surgical weight loss. *Obes Surg* 15:1177–1184
31. van Hout GC, Fortuin FA, Pelle AJ, van Heck GL 2008 Psychosocial functioning, personality, and body image following vertical banded gastroplasty. *Obes Surg* 18:115–120
32. Faria SL, de Oliveira Kelly E, Lins RD, Faria OP 2010 Nutritional management of weight regain after bariatric surgery. *Obes Surg* 20:135–139
33. Orth WS, Madan AK, Taddeucci RJ, Coday M, Tichansky DS 2008 Support group meeting attendance is associated with better weight loss. *Obes Surg* 18:391–394
34. Elakkary E, Elhorr A, Aziz F, Gazayerli MM, Silva YJ 2006 Do support groups play a role in weight loss after laparoscopic adjustable gastric banding? *Obes Surg* 16:331–334
35. Schwartz RW, Strodel WE, Simpson WS, Griffen Jr WO 1988 Gastric bypass revision: lessons learned from 920 cases. *Surgery* 104:806–812
36. Iannelli A, Amato D, Addeo P, Buratti MS, Damhan M, Ben Amor I, Sejour E, Facchiano E, Gugenheim J 2008 Laparoscopic conversion of vertical banded gastroplasty (Mason MacLean) into Roux-en-Y gastric bypass. *Obes Surg* 18:43–46
37. Keshishian A, Zahriya K, Hartoonian T, Ayagian C 2004 Duodenal switch is a safe operation for patients who have failed other bariatric operations. *Obes Surg* 14:1187–1192
38. Brolin RE 2002 Bariatric surgery and long-term control of morbid obesity. *JAMA* 288:2793–2796
39. Faintuch J, Matsuda M, Cruz MELE, Silva MM, Teivelis MP, Garrido Jr AB, Gama-Rodrigues JJ 2004 Severe protein-calorie malnutrition after bariatric procedures. *Obes Surg* 14:175–181
40. Dolan K, Hatzifotis M, Newbury L, Fielding G 2004 A comparison of laparoscopic adjustable gastric banding and biliopancreatic diversion in superobesity. *Obes Surg* 14:165–169
41. Byrne TK 2001 Complications of surgery for obesity. *Surg Clin North Am* 81:1181–1193, vii–viii
42. Stocker DJ 2003 Management of the bariatric surgery patient. *Endocrinol Metab Clin North Am* 32:437–457
43. Marceau S, Biron S, Lagacé M, Hould FS, Potvin M, Bourque RA, Marceau P 1995 Biliopancreatic diversion, with distal gastrectomy, 250 cm and 50 cm limbs: long-term results. *Obes Surg* 5:302–307
44. Marinari GM, Murelli F, Camerini G, Papadia F, Carlini F, Stablini C, Adami GF, Scopinaro N 2004 A 15-year evaluation of biliopancreatic diversion according to the Bariatric Analysis Reporting Outcome System (BAROS). *Obes Surg* 14:325–328
45. Nanni G, Balduzzi GF, Capoluongo R, Scotti A, Rosso G, Botta C, Demichelis P, Daffara M, Coppo E 1997 Biliopancreatic diversion: clinical experience. *Obes Surg* 7:26–29
46. Kalfarentzos F, Dimakopoulos A, Kehagias I, Loukidi A, Mead N 1999 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* 9:433–442
47. Brolin RE, Kenler HA, Gorman JH, Cody RP 1992 Long-limb gastric bypass in the super obese. A prospective randomized study. *Ann Surg* 215:387–395
48. Skroubis G, Sakellaropoulos G, Pougouras K, Mead N, Nikiforidis G, Kalfarentzos F 2002 Comparison of nutritional deficiencies after Roux-en-Y gastric bypass and after biliopancreatic diversion with Roux-en-Y gastric bypass. *Obes Surg* 12:551–558
49. Marceau P, Hould FS, Lebel S, Marceau S, Biron S 2001 Malabsorption obesity surgery. *Surg Clin North Am* 81:1113–1127
50. Bock MA 2003 Roux-en-Y gastric bypass: the dietitian's and patient's perspectives. *Nutr Clin Pract* 18:141–144
51. Ziegler O, Sirveaux MA, Brunaud L, Reibel N, Quilliot D 2009 Medical follow up after bariatric surgery: nutritional and drug issues. General recommendations for the prevention and treatment of nutritional deficiencies. *Diabetes Metab* 35:544–557
52. Layman DK 2003 The role of leucine in weight loss diets and glucose homeostasis. *J Nutr* 133:261S–267S
53. Volpi E, Sheffield-Moore M, Rasmussen BB, Wolfe RR 2001 Basal muscle amino acid kinetics and protein synthesis in healthy young and older men. *JAMA* 286:1206–1212
54. Garlick PJ 2005 The role of leucine in the regulation of protein metabolism. *J Nutr* 135:1553S–1556S
55. Layman DK, Walker DA 2006 Protein importance of leucine in treatment of obesity and the metabolic syndrome. *J Nutr* 136:319S–323S
56. Kushner R 2000 Managing the obese patient after bariatric surgery: a case report of severe malnutrition and review of the literature. *JPN J Parenter Enteral Nutr* 24:126–132
57. Carrodeguas L, Kaidar-Person O, Szomstein S, Antozzi P, Rosenthal R 2005 Preoperative thiamine deficiency in obese population undergoing laparoscopic bariatric surgery. *Surg Obes Relat Dis* 1:517–522
58. Lagacé M, Marceau P, Marceau S, Hould FS, Potvin M, Bourque RA, Biron S 1995 Biliopancreatic diversion with a new type of gastrectomy: some previous conclusions revisited. *Obes Surg* 5:411–418
59. Brolin RE, Gorman RC, Milgrim LM, Kenler HA 1991 Multivitamin prophylaxis in prevention of postgastric bypass vitamin and mineral deficiencies. *Int J Obes* 15:661–667
60. Compston JE, Vedi S, Gianetta E, Watson G, Civalleri D, Scopinaro N 1984 Bone histomorphometry and vitamin D status after biliopancreatic bypass for obesity. *Gastroenterology* 87:350–356
61. Monteforte MJ, Turkelson CM 2000 Bariatric surgery for morbid obesity. *Obes Surg* 10:391–401
62. Brolin RE, Leung M 1999 Survey of vitamin and mineral supplementation after gastric bypass and biliopancreatic diversion for morbid obesity. *Obes Surg* 9:150–154
63. Crowley LV, Seay J, Mullin G 1984 Late effects of gastric bypass for obesity. *Am J Gastroenterol* 79:850–860
64. Saltzman E, Anderson W, Apovian CM, Boulton H, Chamberlain A, Cullum-Dugan D, Cummings S, Hatchigian E, Hodges B, Keroack CR, Pettus M, Thomason P, Veglia L, Young LS 2005 Criteria for patient selection and multidisciplinary evaluation and treatment of the weight loss surgery patient. *Obes Res* 13:234–243
65. Shikora SA, Kim JJ, Tarnoff ME 2007 Nutrition and gastrointestinal complications of bariatric surgery. *Nutr Clin Pract* 22:29–40
66. Levenson DI, Bockman RS 1994 A review of calcium preparations. *Nutr Rev* 52:221–232
67. Miller PD 2006 Guidelines for the diagnosis of osteoporosis: T-scores vs fracture. *Rev Endocr Metab Disord* 7:75–89
68. Goode LR, Brolin RE, Chowdhury HA, Shapses SA 2004 Bone and gastric bypass surgery: effects of dietary calcium and vitamin D. *Obes Res* 12:40–47
69. Bloomberg RD, Fleishman A, Nalle JE, Herron DM, Kini S 2005 Nutritional deficiencies following bariatric surgery: what have we learned? *Obes Surg* 15:145–154
70. Smith CD, Herkes SB, Behrns KE, Fairbanks VF, Kelly KA, Sarr MG 1993 Gastric acid secretion and vitamin B12 absorption after vertical Roux-en-Y gastric bypass for morbid obesity. *Ann Surg* 218:91–96
71. Behrns KE, Smith CD, Sarr MG 1994 Prospective evaluation of gastric acid secretion and cobalamin absorption following gastric bypass for clinically severe obesity. *Dig Dis Sci* 39:315–320
72. Provenzale D, Reinhold RB, Golner B, Irwin V, Dallal GE, Papathanasopoulos N, Sahyoun N, Samloff IM, Russell RM 1992 Evidence for diminished B12 absorption after gastric bypass: oral supplementation does not prevent low plasma B12 levels. *J Am Coll Nutr* 11:29–35
73. Halverson JD 1986 Micronutrient deficiencies after gastric bypass for morbid obesity. *Am Surg* 52:594–598

74. Brolin RE, LaMarca LB, Kenler HA, Cody RP 2002 Malabsorptive gastric bypass in patients with superobesity. *J Gastrointest Surg* 6:195–203; discussion 204–205
75. Marcuard SP, Sinar DR, Swanson MS, Silverman JF, Levine JS 1989 Absence of luminal intrinsic factor after gastric bypass surgery for morbid obesity. *Dig Dis Sci* 34:1238–1242
76. Cooper PL, Brearley LK, Jamieson AC, Ball MJ 1999 Nutritional consequences of modified vertical gastroplasty in obese subjects. *Int J Obes Relat Metab Disord* 23:382–388
77. Strauss RS, Bradley LJ, Brolin RE 2001 Gastric bypass surgery in adolescents with morbid obesity. *J Pediatr* 138:499–504
78. Rhode BM, Tamin H, Gilfix BM, Sampalis JS, Nohr C, MacLean LD 1995 Treatment of vitamin B12 deficiency after gastric surgery for severe obesity. *Obes Surg* 5:154–158
79. Rhode BM, Arseneau P, Cooper BA, Katz M, Gilfix BM, MacLean LD 1996 Vitamin B-12 deficiency after gastric surgery for obesity. *Am J Clin Nutr* 63:103–109
80. Kuzminski AM, Del Giacco EJ, Allen RH, Stabler SP, Lindenbaum J 1998 Effective treatment of cobalamin deficiency with oral cobalamin. *Blood* 92:1191–1198
81. Clements RH, Katasani VG, Palepu R, Leath RR, Leath TD, Roy BP, Vickers SM 2006 Incidence of vitamin deficiency after laparoscopic roux-en-Y gastric bypass in a university hospital setting. *Am Surg* 72:1196–1202; discussion 1203–1204
82. Sharabi A, Cohen E, Sulkes J, Garty M 2003 Replacement therapy for vitamin B12 deficiency: comparison between the sublingual and oral route. *Br J Clin Pharmacol* 56:635–638
83. Park AM, Storm DW, Fulmer BR, Still CD, Wood GC, Hartle 2nd JE 2009 A prospective study of risk factors for nephrolithiasis after Roux-en-Y gastric bypass surgery. *J Urol* 182:2334–2339
84. Rhode BM, Shustik C, Christou NV, MacLean LD 1999 Iron absorption and therapy after gastric bypass. *Obes Surg* 9:17–21
85. Brolin RE, Gorman JH, Gorman RC, Petschenik AJ, Bradley LB, Kenler HA, Cody RP 1998 Prophylactic iron supplementation after Roux-en-Y gastric bypass: a prospective, double-blind, randomized study. *Arch Surg* 133:740–744
86. Skroubis G, Anesidis S, Kehagias I, Mead N, Vagenas K, Kalfarentzos F 2006 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* 16:488–495
87. Lechner GW, Callender AK 1981 Subtotal gastric exclusion and gastric partitioning: a randomized prospective comparison of one hundred patients. *Surgery* 90:637–644
88. Herbert V 1968 Absorption of vitamin B12 and folic acid. *Gastroenterology* 54:110–115
89. Fondu P, Hariga-Muller C, Mozes N, Neve J, Van Steirteghem A, Mandelbaum IM 1978 Protein-energy malnutrition and anemia in Kivu. *Am J Clin Nutr* 31:46–56
90. Hallberg L, Brune M, Erlandsson M, Sandberg AS, Rossander-Hultén L 1991 Calcium: effect of different amounts on nonheme- and heme-iron absorption in humans. *Am J Clin Nutr* 53:112–119
91. Cook JD, Dassenko SA, Whittaker P 1991 Calcium supplementation: effect on iron absorption. *Am J Clin Nutr* 53:106–111
92. Reddy MB, Cook JD 1997 Effect of calcium intake on nonheme-iron absorption from a complete diet. *Am J Clin Nutr* 65:1820–1825
93. Avinoah E, Ovnat A, Charuzi I 1992 Nutritional status seven years after Roux-en-Y gastric bypass surgery. *Surgery* 111:137–142
94. Slater GH, Ren CJ, Siegel N, Williams T, Barr D, Wolfe B, Dolan K, Fielding GA 2004 Serum fat soluble vitamin deficiency and abnormal calcium metabolism after malabsorptive bariatric surgery. *J Gastrointest Surg* 8:48–55
95. Davies DJ, Baxter JM, Baxter JN 2007 Nutritional deficiencies after bariatric surgery. *Obes Surg* 17:1150–1158
96. Mejia LA 1992 Role of vitamin A in iron deficiency anemia. In: Fomon SJ, Zlotkin S, eds. *Nutritional anemias*. New York: Raven Press; 93–104
97. Shuster MH, Vázquez JA 2005 Nutritional concerns related to Roux-en-Y gastric bypass: what every clinician needs to know. *Crit Care Nurs Q* 28:227–260; quiz 261–262
98. Mason EE 1998 Starvation injury after gastric reduction for obesity. *World J Surg* 22:1002–1007
99. MacLean LD, Rhode BM, Shizgal HM 1983 Nutrition following gastric operations for morbid obesity. *Ann Surg* 198:347–355
100. Feit H, Glasberg M, Ireton C, Rosenberg RN, Thal E 1982 Peripheral neuropathy and starvation after gastric partitioning for morbid obesity. *Ann Intern Med* 96:453–455
101. Fawcett S, Young GB, Holliday RL 1984 Wernicke's encephalopathy after gastric partitioning for morbid obesity. *Can J Surg* 27:169–170
102. Villar HV, Ranne RD 1984 Neurologic deficit following gastric partitioning: possible role of thiamine. *JPEN J Parenter Enteral Nutr* 8:575–578
103. Somer H, Bergström L, Mustajoki P, Rovamo L 1985 Morbid obesity, gastric plication and a severe neurological deficit. *Acta Med Scand* 217:575–576
104. Paulson GW, Martin EW, Mojzisek C, Carey LC 1985 Neurologic complications of gastric partitioning. *Arch Neurol* 42:675–677
105. Oczkowski WJ, Kertesz A 1985 Wernicke's encephalopathy after gastroplasty for morbid obesity. *Neurology* 35:99–101
106. Abarbanel JM, Berginer VM, Osimani A, Solomon H, Charuzi I 1987 Neurologic complications after gastric restriction surgery for morbid obesity. *Neurology* 37:196–200
107. Singh S, Kumar A 2007 Wernicke encephalopathy after obesity surgery: a systematic review. *Neurology* 68:807–811
108. Angstadt JD, Bodziner RA 2005 Peripheral polyneuropathy from thiamine deficiency following laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 15:890–892
109. Chaves LC, Faintuch J, Kahwage S, Alencar Fde A 2002 A cluster of polyneuropathy and Wernicke-Korsakoff syndrome in a bariatric unit. *Obes Surg* 12:328–334
110. Primavera A, Brusa G, Novello P, Schenone A, Gianetta E, Marinari G, Cuneo S, Scopinaro N 1993 Wernicke-Korsakoff encephalopathy following biliopancreatic diversion. *Obes Surg* 3:175–177
111. Rindi G, Bordi C, Rappel S, La Rosa S, Stolte M, Solcia E 1996 Gastric carcinoids and neuroendocrine carcinomas: pathogenesis, pathology, and behavior. *World J Surg* 20:168–172
112. Heye N, Terstege K, Sirtl C, McMonagle U, Schreiber K, Meyer-Gessner M 1994 Wernicke's encephalopathy: causes to consider. *Intensive Care Med* 20:282–286
113. Ernst B, Thurnheer M, Schmid SM, Schultes B 2009 Evidence for the necessity to systematically assess micronutrient status prior to bariatric surgery. *Obes Surg* 19:66–73
114. Shah M, Simha V, Garg A 2006 Review: Long-term impact of bariatric surgery on body weight, comorbidities, and nutritional status. *J Clin Endocrinol Metab* 91:4223–4231
115. Xanthakos SA, Inge TH 2006 Nutritional consequences of bariatric surgery. *Curr Opin Clin Nutr Metab Care* 9:489–496
116. Alvarez-Leite JI 2004 Nutrient deficiencies secondary to bariatric surgery. *Curr Opin Clin Nutr Metab Care* 7:569–575
117. Dindo D, Muller MK, Weber M, Clavien PA 2003 Obesity in general elective surgery. *Lancet* 361:2032–2035
118. American Society of Anesthesiologists Task Force on Preanesthesia Evaluation 2002 A report by the American Society of Anesthesiologists task force on preanesthesia evaluation. *Practice advisory for preanesthesia evaluation*. *Anesthesiology* 96:485–496
119. Flancbaum L, Belsley S, Drake V, Colarusso T, Tayler E 2006 Preoperative nutritional status of patients undergoing Roux-en-Y gastric bypass for morbid obesity. *J Gastrointest Surg* 10:1033–1037
120. Madan AK, Orth WS, Tichansky DS, Ternovits CA 2006 Vitamin and trace mineral levels after laparoscopic gastric bypass. *Obes Surg* 16:603–606
121. Silecchia G, Greco F, Bacci V, Boru C, Pecchia A, Casella G, Rizzello

- M, Basso N 2005 Results after laparoscopic gastric banding in patients over 55 years of age. *Obes Surg* 15:351–356
122. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrback K, Schoelles K 2004 Bariatric surgery: a systematic review and meta-analysis. *JAMA* 292:1724–1737
 123. Torquati A, Lutfi R, Abumrad N, Richards WO 2005 Is Roux-en-Y gastric bypass surgery the most effective treatment for type 2 diabetes mellitus in morbidly obese patients? *J Gastrointest Surg* 9:1112–1118
 124. Pories WJ, Swanson MS, MacDonald KG, Long SB, Morris PG, Brown BM, Barakat HA, deRamon RA, Israel G, Dolezal JM 1995 Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. *Ann Surg* 222:339–350; discussion 350–352
 125. Hickey MS, Pories WJ, MacDonald Jr KG, Cory KA, Dohm GL, Swanson MS, Israel RG, Barakat HA, Considine RV, Caro JF, Houmard JA 1998 A new paradigm for type 2 diabetes mellitus: could it be a disease of the foregut? *Ann Surg* 227:637–643; discussion 643–644
 126. Schauer PR, Burguera B, Ikramuddin S, Cottam D, Gourash W, Hamad G, Eid GM, Mattar S, Ramanathan R, Barinas-Mitchel E, Rao RH, Kuller L, Kelley D 2003 Effect of laparoscopic Roux-en-Y gastric bypass on type 2 diabetes mellitus. *Ann Surg* 238:467–484; discussion 84–85
 127. Sanderson I, Deitel M 1974 Insulin response in patients receiving concentrated infusions of glucose and casein hydrolysate for complete parenteral nutrition. *Ann Surg* 179:387–394
 128. Deitel M, Sidhu P, Stone E 1991 Effect of vertical banded gastroplasty on diabetes in the morbidly obese. *Obes Surg* 1:113–114 (Abstract)
 129. Jensen K, Mason E, Scott D 1991 Changes in the postoperative hypoglycemic and antihypertensive medication requirements in morbidly obese patients after VBG. *Obes Surg* 1:113–114 (Abstract)
 130. Smith SC, Edwards CB, Goodman GN 1996 Changes in diabetic management after Roux-en-Y gastric bypass. *Obes Surg* 6:345–348
 131. Mingrone G, DeGaetano A, Greco AV, Capristo E, Benedetti G, Castagneto M, Gasbarrini G 1997 Reversibility of insulin resistance in obese diabetic patients: role of plasma lipids. *Diabetologia* 40:599–605
 132. Pories WJ, MacDonald Jr KG, Flickinger EG, Dohm GL, Sinha MK, Barakat HA, May HJ, Khazanie P, Swanson MS, Morgan E 1992 Is type II diabetes mellitus (NIDDM) a surgical disease? *Ann Surg* 215:633–642; discussion 643
 133. Pories WJ, MacDonald KG, Morgan EJ, Sinha MK, Dohm GL, Swanson MS, Barakat HA, Khazanie PG, Leggett-Frazier N, Long SD 1992 Surgical treatment of obesity and its effect on diabetes: 10-y follow-up. *Am J Clin Nutr* 55(Suppl 2):582S–585S
 134. Long SD, O'Brien K, MacDonald Jr KG, Leggett-Frazier N, Swanson MS, Pories WJ, Caro JF 1994 Weight loss in severely obese subjects prevents the progression of impaired glucose tolerance to type II diabetes. A longitudinal interventional study. *Diabetes Care* 17:372–375
 135. Muscelli E, Mingrone G, Camastra S, Manco M, Pereira JA, Pareja JC, Ferrannini E 2005 Differential effect of weight loss on insulin resistance in surgically treated obese patients. *Am J Med* 118:51–57
 136. Rubino F, Gagner M, Gentileschi P, Kini S, Fukuyama S, Feng J, Diamond E 2004 The early effect of the Roux-en-Y gastric bypass on hormones is involved in body weight regulation and glucose metabolism. *Ann Surg* 240:236–242
 137. Wickremesekera K, Miller G, Naotunne TD, Knowles G, Stubbs RS 2005 Loss of insulin resistance after Roux-en-Y gastric bypass surgery: a time course study. *Obes Surg* 15:474–481
 138. Herbst CA, Hughes TA, Gwynne JT, Buckwalter JA 1984 Gastric bariatric operation in insulin treated adults. *Surgery* 95:209–214
 139. Laferrère B, Teixeira J, McGinty J, Tran H, Egger JR, Colarusso A, Kovack B, Bawa B, Koshy N, Lee H, Yapp K, Olivan B 2008 Effect of weight loss by gastric bypass surgery versus hypocaloric diet on glucose and incretin levels in patients with type 2 diabetes. *J Clin Endocrinol Metab* 93:2479–2485
 140. Dixon JB, O'Brien PE, Playfair J, Chapman L, Schachter LM, Skinner S, Proietto J, Bailey M, Anderson M 2008 Adjustable gastric banding and conventional therapy for type 2 diabetes: a randomized controlled trial. *JAMA* 299:316–323
 141. Cummings DE, Overduin J, Foster-Schubert KE 2004 Gastric bypass for obesity: mechanisms of weight loss and diabetes resolution. *J Clin Endocrinol Metab* 89:2608–2615
 142. Cummings DE, Overduin J, Foster-Schubert KE, Carlson MJ 2007 Role of the bypassed proximal intestine in the anti-diabetic effects of bariatric surgery. *Surg Obes Relat Dis* 3:109–115
 143. Cohen RV, Schiavon CA, Pinheiro JS, Correa JL, Rubino F 2007 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* 3:195–197
 144. Rubino F, Gagner M 2002 Potential of surgery for curing type 2 diabetes mellitus. *Ann Surg* 236:554–559
 145. Rubino F, Marescaux J 2004 Effect of duodenal-jejunal exclusion in a non-obese animal model of type 2 diabetes. *Ann Surg* 239:1–11
 146. Rubino F, Zizzari P, Tomasetto C, Bluet-Pajot MT, Forgione A, Vix M, Grouelle D, Marescaux J 2005 The role of the small bowel in the regulation of circulating ghrelin levels and food intake in the obese Zucker rat. *Endocrinology* 146:1745–1751
 147. de Paula AL, Macedo AL, Prudente AS, Queiroz L, Schraibman V, Pinus J 2006 Laparoscopic sleeve gastrectomy with ileal interposition (“neuroendocrine brake”): pilot study of a new operation. *Surg Obes Relat Dis* 2:464–467
 148. Vora AC, Saleem TM, Polomano RC, Eddinger VL, Hollenbeak CS, Girdharry DT, Joshi R, Martin D, Gabbay RA 2004 Improved perioperative glycemic control by continuous insulin infusion under supervision of an endocrinologist does not increase costs in patients with diabetes. *Endocr Pract* 10:112–118
 149. Pomposelli JJ, Baxter 3rd JK, Babineau TJ, Pomfret EA, Driscoll DF, Forse RA, Bistrrian BR 1998 Early postoperative glucose control predicts nosocomial infection rate in diabetic patients. *JPEN J Parenter Enteral Nutr* 22:77–81
 150. Dronge AS, Perkal MF, Kancir S, Concato J, Aslan M, Rosenthal RA 2006 Long-term glycemic control and postoperative infectious complications. *Arch Surg* 141:375–380
 151. NICE-SUGAR Study Investigators; Finfer S, Chittock DR, Su SY, Blair D, Foster D, Dhingra V, Bellomo R, Cook D, Dodek P, Henderson WR, Hébert PC, Heritier S, Heyland DK, McArthur C, McDonald E, Mitchell I, Myburgh JA, Norton R, Potter J, Robinson BG, Ronco JJ 2009 Intensive versus conventional glucose control in critically ill patients. *N Engl J Med* 360:1283–1297
 152. Datta S, Qaadir A, Villanueva G, Baldwin D 2007 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* 13:225–231
 153. Maggard MA, Yermilov I, Li Z, Maglione M, Newberry S, Suttorp M, Hilton L, Santry HP, Morton JM, Livingston EH, Shekelle PG 2008 Pregnancy and fertility following bariatric surgery: a systematic review. *JAMA* 300:2286–2296
 154. Gholam PM, Kotler DP, Flancbaum LJ 2002 Liver pathology in morbidly obese patients undergoing Roux-en-Y gastric bypass surgery. *Obes Surg* 12:49–51
 155. Mattar SG, Velcu LM, Rabinovitz M, Demetris AJ, Krasinskas AM, Barinas-Mitchell E, Eid GM, Ramanathan R, Taylor DS, Schauer PR 2005 Surgically-induced weight loss significantly improves nonalcoholic fatty liver disease and the metabolic syndrome. *Ann Surg* 242:610–620
 156. Adams LA, Angulo P 2006 Treatment of non-alcoholic fatty liver disease. *Postgrad Med J* 82:315–322
 157. Ranløv I, Hardt F 1990 Regression of liver steatosis following

- gastroplasty or gastric bypass for morbid obesity. *Digestion* 47:208–214
158. Silverman EM, Sapala JA, Appelman HD 1995 Regression of hepatic steatosis in morbidly obese persons after gastric bypass. *Am J Clin Pathol* 104:23–31
 159. Luyckx FH, Desai C, Thiry A, Dewé W, Scheen AJ, Gielen JE, Lefebvre PJ 1998 Liver abnormalities in severely obese subjects: effect of drastic weight loss after gastroplasty. *Int J Obes Relat Metab Disord* 22:222–226
 160. Duchini A, Brunson ME 2001 Roux-en-Y gastric bypass for recurrent nonalcoholic steatohepatitis in liver transplant recipients with morbid obesity. *Transplantation* 72:156–159
 161. Clark JM, Alkhuraishi AR, Solga SF, Alli P, Diehl AM, Magnuson TH 2005 Roux-en-Y gastric bypass improves liver histology in patients with non-alcoholic fatty liver disease. *Obes Res* 13:1180–1186
 162. Barker KB, Palekar NA, Bowers SP, Goldberg JE, Pulcini JP, Harrison SA 2006 Non-alcoholic steatohepatitis: effect of Roux-en-Y gastric bypass surgery. *Am J Gastroenterol* 101:368–373
 163. Jaskiewicz K, Raczynska S, Rzepko R, Sledziński Z 2006 Nonalcoholic fatty liver disease treated by gastroplasty. *Dig Dis Sci* 51:21–26
 164. Mathurin P, Gonzalez F, Kerdraon O, Leteurtre E, Arnalsteen L, Hollebecque A, Louvet A, Dharancy S, Cocq P, Jany T, Boitard J, Deltenre P, Romon M, Pattou F 2006 The evolution of severe steatosis after bariatric surgery is related to insulin resistance. *Gastroenterology* 130:1617–1624
 165. Green RM, Flamm S 2002 AGA technical review on the evaluation of liver chemistry tests. *Gastroenterology* 123:1367–1384
 166. Dallal RM, Mattar SG, Lord JL, Watson AR, Cottam DR, Eid GM, Hamad G, Rabinovitz M, Schauer PR 2004 Results of laparoscopic gastric bypass in patients with cirrhosis. *Obes Surg* 14:47–53
 167. Tichansky DS, Madan AK 2005 Laparoscopic Roux-en-Y gastric bypass is safe and feasible after orthotopic liver transplantation. *Obes Surg* 15:1481–1486
 168. American Gastroenterological Association 2002 American Gastroenterological Association medical position statement: nonalcoholic fatty liver disease. *Gastroenterology* 123:1702–1704
 169. Clark JM, Diehl AM 2003 Nonalcoholic fatty liver disease an under recognized cause of cryptogenic cirrhosis. *JAMA* 289:3000–3004
 170. Dixon JB, O'Brien PE 2002 Lipid profile in the severely obese: changes with weight loss after lap-band surgery. *Obes Res* 10:903–910
 171. Gleysteen JJ, Barboriak JJ 1983 Improvement in heart disease risk factors after gastric bypass. *Arch Surg* 118:681–684
 172. Gleysteen JJ 1992 Results of surgery: long term effects on hyperlipidemia. *Am J Clin Nutr* 55(Suppl 2):591S–593S
 173. Buffington CK, Cowan Jr GS, Smith H 1994 Significant changes in the lipid-lipoprotein status of premenopausal morbidly obese females following gastric bypass surgery. *Obes Surg* 4:328–335
 174. Wolf AM, Beisiegel U, Kortner B, Kuhlmann HW 1998 Does gastric restriction surgery reduce the risks of metabolic diseases? *Obes Surg* 8:9–13
 175. Busetto L, Pisent C, Rinaldi D, Longhin PL, Segato G, De Marchi F, Foletto M, Favretti F, Lise M, Enzi G 2000 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* 10:569–577
 176. Brolin RE, Bradley LJ, Wilson AC, Cody RP 2000 Lipid risk profile and weight stability after gastric restrictive operations for morbid obesity. *J Gastrointest Surg* 4:464–469
 177. Sjöström CD, Lissner L, Wedel H, Sjöström L 1999 Reduction in incidence of diabetes, hypertension and lipid disturbances after intentional weight loss induced by bariatric surgery: the SOS Intervention Study. *Obes Res* 7:477–484
 178. Lubrano C, Cornoldi A, Pili M, Falcone S, Brandetti F, Fabbrini E, Ginanni-Cornoldi S, Eramo A, Marini M, Migliaccio S, Giancotti V, Badiali M, Falsetto N, Prossomariti G, Spera G 2004 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* 28:1600–1606
 179. Palomar R, Fernández-Fresnedo G, Domínguez-Diez A, López-Deogracias M, Olmedo F, Martín de Francisco AL, Sanz de Castro S, Casado Martín F, Gómez-Fleitas M, Arias M, Fernández-Escalante C 2005 Effects of weight loss after biliopancreatic diversion on metabolism and cardiovascular profile. *Obes Surg* 15:794–798
 180. Corradini SG, Eramo A, Lubrano C, Spera G, Cornoldi A, Grossi A, Liguori F, Siciliano M, Pisanelli MC, Salen G, Batta AK, Attili AF, Badiali M 2005 Comparison of changes in lipid profile after bilio-intestinal bypass and gastric banding in patients with morbid obesity. *Obes Surg* 15:367–377
 181. Zlabek JA, Grimm MS, Larson CJ, Mathiason MA, Lambert PJ, Kothari SN 2005 The effect of laparoscopic gastric bypass surgery on dyslipidemia in severely obese patients. *Surg Obes Relat Dis* 1:537–542
 182. Vogel JA, Franklin BA, Zalesin KC, Trivax JE, Krause KR, Changelis DL, McCullough PA 2007 Reduction in predicted coronary heart disease after substantial weight reduction after bariatric surgery. *Am J Cardiol* 99:222–226
 183. Williams DB, Hagedorn JC, Lawson EH, Galanko JA, Safadi BY, Curet MJ, Morton JM 2007 Gastric bypass reduces biochemical cardiac risk factors. *Surg Obes Relat Dis* 3:8–13
 184. Dixon JB, O'Brien P 2001 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* 25:793–797
 185. Adams TD, Gress RE, Smith SC, Halverson RC, Simper SC, Rosamond WD, Lamonte MJ, Stroup AM, Hunt SC 2007 Long-term mortality following gastric bypass surgery. *N Engl J Med* 357:753–761
 186. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) 2002 Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation* 106:3143–3421
 187. Korperhoeke HW, Witteman EM, Meinardi JR, Vollaard EJ 2010 [Severe vitamin D deficiency and hypocalcaemia after bariatric surgery]. *Ned Tijdschr Geneesk* 154:A827 (Dutch)
 188. Vilarrasa N, Gómez JM, Masdevall C, Pujol J, Soler J, Elio I, Gallart L, Vendrell J 2009 Study of the relationship between adiponectin, interleukin-18, ghrelin and bone mineral density in morbidly obese women after gastric bypass. *Endocrinol Nutr* 56:355–360
 189. Nadler EP, Reddy S, Isenalumhe A, Youn HA, Peck V, Ren CJ, Fielding GA 2009 Laparoscopic adjustable gastric banding for morbidly obese adolescents affects android fat loss, resolution of comorbidities, and improved metabolic status. *J Am Coll Surg* 209:638–644
 190. Tsiftsis DD, Mylonas P, Mead N, Kalfarentzos F, Alexandrides TK 2009 Bone mass decreases in morbidly obese women after long limb-biliopancreatic diversion and marked weight loss without secondary hyperparathyroidism. A physiological adaptation to weight loss? *Obes Surg* 19:1497–1503
 191. Valderas JP, Velasco S, Solari S, Liberona Y, Viviani P, Maiz A, Escalona A, González G 2009 Increase of bone resorption and the parathyroid hormone in postmenopausal women in the long-term after Roux-en-Y gastric bypass. *Obes Surg* 19:1132–1138
 192. Al-Shoha A, Qiu S, Palnitkar S, Rao DS 2009 Osteomalacia with bone marrow fibrosis due to severe vitamin D deficiency after a gastrointestinal bypass operation for severe obesity. *Endocr Pract* 15:528–533
 193. Wang A, Powell A 2009 The effects of obesity surgery on bone metabolism: what orthopedic surgeons need to know. *Am J Orthop* 38:77–79

194. Berarducci A, Haines K, Murr MM 2009 Incidence of bone loss, falls, and fractures after Roux-en-Y gastric bypass for morbid obesity. *Appl Nurs Res* 22:35–41
195. Stein EM, Strain G, Sinha N, Ortiz D, Pomp A, Dakin G, McMahon DJ, Bockman R, Silverberg SJ 2009 Vitamin D insufficiency prior to bariatric surgery: risk factors and a pilot treatment study. *Clin Endocrinol (Oxf)* 71:176–183
196. Carlin AM, Rao DS, Yager KM, Parikh NJ, Kapke A 2009 Treatment of vitamin D depletion after Roux-en-Y gastric bypass: a randomized prospective clinical trial. *Surg Obes Relat Dis* 5:444–449
197. Fleischer J, Stein EM, Bessler M, Della Badia M, Restuccia N, Olivero-Rivera L, McMahon DJ, Silverberg SJ 2008 The decline in hip bone density after gastric bypass surgery is associated with extent of weight loss. *J Clin Endocrinol Metab* 93:3735–3740
198. Duran de Campos C, Dalcanele L, Pajecki D, Garrido Jr AB, Halpern A 2008 Calcium intake and metabolic bone disease after eight years of Roux-en-Y gastric bypass. *Obes Surg* 18:386–390
199. Schweitzer DH 2007 Mineral metabolism and bone disease after bariatric surgery and ways to optimize bone health. *Obes Surg* 17:1510–1516
200. Hamoui N, Kim K, Anthonie G, Crookes PF 2003 The significance of elevated levels of parathyroid hormone in patients with morbid obesity before and after bariatric surgery. *Arch Surg* 138:891–897
201. Newbury L, Dolan K, Hatzifotis M, Low N, Fielding G 2003 Calcium and vitamin D depletion and elevated parathyroid hormone following biliopancreatic diversion. *Obes Surg* 13:893–895
202. Sugerman HJ 2001 Bariatric surgery for severe obesity. *J Assoc Acad Minor Phys* 12:129–136
203. Balsiger BM, Kennedy FP, Abu-Lebdeh HS, Collazo-Clavell M, Jensen MD, O'Brien T, Hensrud DD, Dinneen SF, Thompson GB, Que FG, Williams DE, Clark MM, Grant JE, Frick MS, Mueller RA, Mai JL, Sarr MG 2000 Prospective evaluation of Roux-en-Y gastric bypass as primary operation for medically complicated obesity. *Mayo Clin Proc* 75:673–680
204. Favus MJ 1996 Primer on the metabolic bone diseases and disorders of mineral metabolism. Philadelphia: Lippincott-Raven
205. Ybarra J, Sánchez-Hernández J, Gich I, De Leiva A, Rius X, Rodríguez-Espinosa J, Pérez A 2005 Unchanged hypovitaminosis D and secondary hyperparathyroidism in morbid obesity after bariatric surgery. *Obes Surg* 15:330–335
206. Sánchez-Hernández J, Ybarra J, Gich I, De Leiva A, Rius X, Rodríguez-Espinosa J, Pérez A 2005 Effects of bariatric surgery on vitamin D status and secondary hyperparathyroidism: a prospective study. *Obes Surg* 15:1389–1395
207. Ott MT, Fanti P, Malluche HH, Ryo UY, Whaley FS, Strodel WE, Colacchi TA 1992 Biochemical evidence of metabolic bone disease in women following Roux-Y gastric bypass for morbid obesity. *Obes Surg* 2:341–348
208. Strauss BJ, Marks SJ, Growcott JP, Stroud DB, Lo CS, Dixon JB, O'Brien PE 2003 Body composition changes following laparoscopic gastric banding for morbid obesity. *Acta Diabetol* 40(Suppl 1):S266–S269
209. Dixon JB, Strauss BJ, Laurie C, O'Brien PE 2007 Changes in body composition with weight loss: obese subjects randomized to surgical and medical programs. *Obesity* 15:1187–1198
210. Shaker JL, Norton AJ, Woods MF, Fallon MD, Findling JW 1991 Secondary hyperparathyroidism and osteopenia in women following gastric exclusion surgery for obesity. *Osteoporos Int* 1:177–181
211. Friedman JE, Dallal RM, Lord JL 2008 Gouty attacks occur frequently in postoperative gastric bypass patients. *Surg Obes Relat Dis* 4:11–13
212. Charles P 1992 Calcium absorption and calcium bioavailability. *J Intern Med* 231:161–168
213. Pugnale N, Giusti V, Suter M, Zysset E, Héraïef E, Gaillard RC, Burckhardt P 2003 Bone metabolism and risk of secondary hyperparathyroidism 12 months after gastric banding in obese premenopausal women. *Int J Obes Relat Metab Disord* 27:110–116
214. Alborzi F, Leibowitz AB 2002 Immobilization hypercalcemia in critical illness following bariatric surgery. *Obes Surg* 12:871–873
215. Liel Y, Edwards J, Shary J, Spicer KM, Gordon L, Bell NH 1988 The effects of race and body habitus on bone mineral density of the radius, hip, and spine in premenopausal women. *J Clin Endocrinol Metab* 66:1247–1250
216. Gossain VV, Rao DS, Carella MJ, Divine G, Rovner DR 1999 Bone mineral density in obesity; effect of weight loss. *J Med* 30:367–376
217. Bell NH, Epstein S, Greene A, Shary J, Oexmann MJ, Shaw S 1985 Evidence for alteration of the vitamin D-endocrine system in obese subjects. *J Clin Invest* 76:370–373
218. De Prisco C, Levine SN 2005 Metabolic bone disease after gastric bypass surgery for obesity. *Am J Med Sci* 329:57–61
219. Fitzpatrick LA 2002 Secondary causes of osteoporosis. *Mayo Clin Proc* 77:453–468
220. Diniz Mde F, Diniz MT, Sanches SR, Salgado PP, Valadão MM, Araújo FC, Martins DS, Rocha AL 2004 Elevated serum parathormone after Roux-en-Y gastric bypass. *Obes Surg* 14:1222–1226
221. Parada P, Maruri I, Morales MJ, Otero I, Delgado C, Casal JE, Nutritional complications after bariatric surgery. Program of the 8th World Congress of the International Federation for the Surgery of Obesity, Salamanca, Spain, 2003, p 525 (Abstract 33)
222. Youssef Y, Richards WO, Sekhar N, Kaiser J, Spagnoli A, Abumrad N, Torquati A 2007 Risk of secondary hyperparathyroidism after laparoscopic gastric bypass surgery in obese women. *Surg Endosc* 21:1393–1396
223. Riedt CS, Brolin RE, Sherrell RM, Field MP, Shapses SA 2006 True fractional calcium absorption is decreased after Roux-en-Y gastric bypass surgery. *Obesity* 14:1940–1948
224. Nevitt MC 2002 Obesity outcomes in disease management: clinical outcomes for osteoarthritis. *Obes Res* 10(Suppl 1):33S–37S
225. Peltonen M, Lindroos AK, Torgerson JS 2003 Musculoskeletal pain in the obese: a comparison with the general population and long-term changes after conventional and surgical obesity treatment. *Pain* 104:549–557
226. Hooper MM, Stellato TA, Hallowell PT, Seitz BA, Moskowitz RW 2007 Musculoskeletal findings in obese subjects before and after weight loss following bariatric surgery. *Int J Obes (Lond)* 31:114–120
227. Serés L, Lopez-Ayerbe J, Coll R, Rodriguez O, Vila J, Formiguera X, Alastrue A, Rull M, Valle V 2006 Increased exercise capacity after surgically induced weight loss in morbid obesity. *Obesity (Silver Spring)* 14:273–279
228. McGoey BV, Deitel M, Saplys RJ, Kliman ME 1990 Effect of weight loss on musculoskeletal pain in the morbidly obese. *J Bone Joint Surg Br* 72:322–323
229. Mitchell JE, Lancaster KL, Burgard MA, Howell LM, Krahn DD, Crosby RD, Wonderlich SA, Gosnell BA 2001 Long-term follow-up of patients' status after gastric bypass. *Obes Surg* 11:464–468
230. Powers PS, Perez A, Boyd F, Rosemurgy A 1999 Eating pathology before and after bariatric surgery: a prospective study. *Int J Eat Disord* 25:293–300
231. Stunkard A, Foster G, Glassman J, Rosato E 1985 Retrospective exaggeration of symptoms: vomiting after gastric surgery for obesity. *Psychosom Med* 47:150–155
232. Marceau P 2000 Malabsorptive procedure in surgical treatment of morbid obesity. *Probl Gen Surg* 17:29–39
233. MacLean LD, Rhode BM, Nohr C, Katz S, McLean AP 1997 Stomal ulcer after gastric bypass. *J Am Coll Surg* 185:1–7
234. Pories WJ, MacDonald KG 1993 The surgical treatment of morbid obesity. *Curr Opin Gen Surg* 195–205
235. Lublin M, McCoy M, Waldrep DJ 2006 Perforating marginal ulcers after laparoscopic gastric bypass. *Surg Endosc* 20:51–54
236. MacLean LD, Rhode BM, Nohr CW 2000 Late outcome of gastric bypass. *Ann Surg* 231:524–528
237. Brolin RE 2000 Complications of surgery for severe obesity. *Probl Gen Surg* 17:55–61

238. Sugerman HJ, Starkey JV, Birkenhauer R 1987 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* 205:613–624
239. Collene AL, Hertzler S 2003 Metabolic outcomes of gastric bypass. *Nutr Clin Pract* 18:136–140
240. Mallory GN, Macgregor AM, Rand CS 1996 The influence of dumping on weight loss after gastric restrictive surgery for morbid obesity. *Obes Surg* 6:474–478
241. Hsu LK, Mulliken B, McDonagh B, Krupa Das S, Rand W, Fairburn CG, Rolls B, McCrory MA, Saltzman E, Shikora S, Dwyer J, Roberts S 2002 Binge eating disorder in extreme obesity. *Int J Obes Relat Metab Disord* 26:1398–1403
242. Didden P, Penning C, Masclee AA 2006 Octreotide therapy in dumping syndrome: analysis of long-term results. *Aliment Pharmacol Ther* 24:1367–1375
243. Ukleja A 2005 Dumping syndrome: pathophysiology and treatment. *Nutr Clin Pract* 20:517–525
244. Carvajal SH, Mulvihill SJ 1994 Postgastrectomy syndromes: dumping and diarrhea. *Gastroenterol Clin North Am* 23:261–279
245. Service GJ, Thompson GB, Service FJ, Andrews JC, Collazo-Clavell ML, Lloyd RV 2005 Hyperinsulinemic hypoglycemia with nesidioblastosis after gastric-bypass surgery. *N Engl J Med* 353:249–254
246. Patti ME, McMahon G, Mun EC, Bitton A, Holst JJ, Goldsmith J, Hanto DW, Callery M, Arky R, Nose V, Bonner-Weir S, Goldfine AB 2005 Severe hypoglycaemia post-gastric bypass requiring partial pancreatectomy: evidence for inappropriate insulin secretion and pancreatic islet hyperplasia. *Diabetologia* 48:2236–2240
247. Kellogg TA, Bantle JP, Leslie DB, Redmond JB, Slusarek B, Swan T, Buchwald H, Ikramuddin S 2008 Postgastric bypass hyperinsulinemic hypoglycemia syndrome: characterization and response to a modified diet. *Surg Obes Relat Dis* 4:492–499
248. Pories WJ 2008 Bariatric surgery: risks and rewards. *J Clin Endocrinol Metab* 93:S89–S96



Members have **FREE** online access
to current endocrine **Clinical Practice Guidelines.**

www.endo-society.org/guidelines