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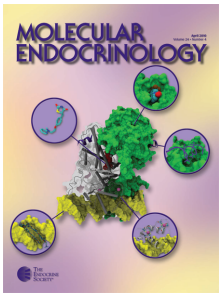

Endocrine and Nutritional Management of the Post-Bariatric Surgery Patient: An Endocrine Society Clinical Practice Guideline

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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-

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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 gastrogastric 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 anastamoses 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.

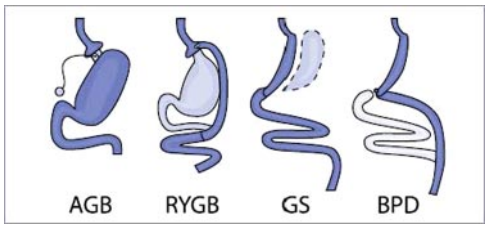

FIG. 1. Diagram of surgical options. [Adapted with permission from W. J. Pories: *J Clin Endocrinol Metab* 93:S89–S96, 2008 (248). © The Endocrine Society.]

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

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

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 Roux-limb at least 150 cm, less than 5% of patients with a Roux-limb 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 μg/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 μg/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 im 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 μg/d	1,000–2,000 μg/2–3 months im
Folic acid	Macrocytic anemia, palpitations, fatigue, eural tube defects	Cell blood count. folic acid levels, homocysteine	Oral folate, 400 μg/d (included in multivitamin)	Oral folate, 1,000 μg/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 established. Intravenous (0.25–0.5 μg/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/m^2 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 anastamoses 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 gastrogastric fistulas can also occur after gastric transection and increase the risk of marginal 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.

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