

Nutritional Consequences of Weight-Loss Surgery

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Malnutrition is defined as “any disorder of nutrition status including disorders resulting from a deficiency of nutrient intake, impaired nutrient metabolism, or overnutrition” [1]. Overnutrition results in obesity; therefore, obesity is a form of malnutrition. The incidence of obesity is increasing at an alarming rate. Worldwide, an estimated 1.7 billion people are overweight, with an excess of 97 million obese adults in the United States [2,3]. Obesity is also a growing problem in children, and its prevalence parallels that of adult obesity [4,5].

Weight loss in obese individuals followed by long-term maintenance of an ideal weight is difficult. Increased physical activity and caloric reduction with low-calorie or low-carbohydrate diets can reduce weight by 5% to 10% over a 6-month period, but weight regain is common [6–9]. Currently available pharmacologic therapies can be combined with dietary measures for a greater weight loss effect but dose-dependent side effects limit their use, and long-term safety and efficacy beyond 2 years have not been fully evaluated [10–12]. Surgery has been demonstrated to be the most effective treatment for weight loss and improvement in some comorbid conditions in patients with morbid obesity [13,14]. Surgical approaches to induce weight loss include the use of restriction, in which food intake is limited by a small gastric pouch or reservoir, the use of malabsorption, in which the length of intestine available for nutrient absorption is reduced, or by a combination of both techniques (Box 1). Currently, laparoscopic Roux-en-Y gastric bypass (LRYGB) and laparoscopic adjustable gastric banding

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Box 1. Surgical procedures categorized by mechanism of action

Purely restrictive

Vertical banded gastroplasty
Adjustable gastric banding
Sleeve gastrectomy
Intragastric balloon^a

Purely malabsorptive

Jejunioileal bypass^b
Jejunocolonic bypass^b

Appetite suppression

Implantable gastric stimulator^c

Malabsorption > restriction

Biliopancreatic diversion
Biliopancreatic diversion with duodenal switch
Very long limb Roux-en-Y gastric bypass

Restriction > malabsorption

Roux-en-Y gastric bypass

^a Not approved for use in the United States.

^b Abandoned techniques.

^c Not currently available for clinical use.

(LAGB) are the most commonly performed procedures in the United States. Although surgical strategies are more successful in achieving and maintaining weight loss, nutritional deficiencies with metabolic consequences can result in the short and long term [15,16]. The severity of the postoperative nutritional deficit is dependent on several factors, including the preoperative nutritional status, the type of bariatric procedure performed, the occurrence of postoperative complications, the ability to modify eating behavior, and compliance with regular follow-up and prescribed vitamin and mineral supplementation.

Absorption of micronutrients and macronutrients in the normal gastrointestinal tract

Macronutrients

Protein metabolism

When food enters the mouth, cephalic stimulation with acetylcholine release enhances acid production from gastric parietal cells [17]. Acid and acetylcholine stimulate release of chief cell pepsinogen. Pepsinogen is cleaved to

pepsin at a pH of 5 or less, and pepsin cleaves protein to peptides. Passage of peptides and amino acids into the duodenum stimulates duodenal and jejunal epithelial cells to release cholecystokinin (CCK). Food in the antrum, antral distention, and vagal stimulation increase gastrin production, and gastrin further enhances CCK release. CCK stimulates release of pancreatic acinar cell trypsinogen, which is cleaved in the duodenum by duodenal enterokinase into its active form, trypsin. Trypsin then activates pancreatic acinar cell chymotrypsin and procarboxypeptidases A and B, which hydrolyze proteins into oligopeptides and amino acids. Oligopeptides are digested by brush border peptidases and then absorbed through the sodium-dependent amino acid cotransporters located along the luminal border of the duodenal enterocyte. Fifty percent of protein absorption occurs in the duodenum, and, by the midjejunum, the majority of protein absorption is complete [17].

Carbohydrate metabolism

Salivary and pancreatic amylase hydrolyze polysaccharides into oligosaccharides. Oligosaccharides are broken down by intestinal brush border oligosaccharidases including sucrase, maltase, and lactase to the monosaccharides glucose, fructose, and galactose [17]. Glucose and galactose are actively transported into the enterocyte by a Na⁺ K⁺ ATPase pump, while fructose is transported passively through carrier-facilitated diffusion. Carbohydrate absorption begins in the duodenum and is complete within the first 100 cm of the small intestine [17].

Lipid metabolism

Dietary lipids include free fatty acids, triglycerides, phospholipids, and cholesterol. Lipids enter the duodenum and stimulate duodenal mucosal I and S cells to secrete CCK and secretin, respectively, which stimulates gallbladder contraction and pancreatic secretion of lipase, cholesterol esterase, and phospholipase A2 to create lipid by-products [17]. Bile salts emulsify the lipid by-products in bile salt–formed micelles for transport into the enterocyte. Triglycerides and cholesterol are resynthesized within the enterocyte and combine with other nonpolar lipids, phospholipids, and proteins to form chylomicrons. From the enterocyte, chylomicrons are transported into the lymphatic system. Although 93% of dietary lipids are absorbed in the proximal two thirds of the jejunum, fat absorption, including the fat-soluble vitamins A, D, E, and K, can occur throughout the length of the small intestine. Excess bile salts are absorbed in the terminal ileum [17].

Micronutrients, vitamins, essential minerals

Fat-soluble vitamins

The fat-soluble vitamins, A, D, E, and K, diffuse across the brush border plasma membrane of the intestinal epithelial cell inside micelles formed by

bile salts and lipid digestion products [17]. Although most fat-soluble vitamin absorption occurs in the proximal two thirds of the jejunum, absorption can occur throughout the small intestine. Deficiencies can occur due to reduced intake or malabsorption. In malabsorptive procedures performed for weight loss, the duodenum and varying lengths of jejunum are bypassed with resultant inadequate mixing of food with biliary and pancreatic secretions, causing steatorrhea, bile salt wasting, and malabsorption of fat-soluble vitamins.

Water-soluble vitamins

Water-soluble vitamins include B₁ (thiamine), B₂ (riboflavin), B₃ (niacin), biotin, pantothenic acid, B₆, B₁₂ (cobalamin), folate, and vitamin C (ascorbate). Most water-soluble vitamins can be absorbed by simple diffusion if taken in sufficiently high doses with the exception of vitamin B₁₂. Absorption mainly occurs in the proximal small bowel, mainly in the jejunum.

Vitamin B₁₂

Cobalamin-containing foods such as meat, eggs, and milk undergo acid and peptic hydrolysis in the stomach to release vitamin B₁₂. Once liberated, vitamin B₁₂ is avidly bound to glycoproteins known as R binders that are secreted in saliva, gastric juice, bile, and intestinal secretions. In the duodenum, pancreatic lipases degrade R binders and allow vitamin B₁₂ to bind with intrinsic factors produced by gastric parietal cells. The intrinsic factor–vitamin B₁₂ complex is bound to specific receptors in the terminal ileum and absorbed. The parietal cells that secrete acid and intrinsic factor and the chief cells that secrete pepsinogen are located primarily in the fundus and body of the stomach. Surgical procedures that produce restriction by creation of a small gastric pouch, such as RYGB, can cause significant vitamin B₁₂ deficiency by reduction of acid and pepsin digestion of protein-bound cobalamins in food, incomplete release of R binders, and decreased production of intrinsic factor.

Iron

Iron is liberated from heme proteins in foods of animal origin by exposure to acid and proteases in gastric juices. The low pH of the gastric acid solubilizes iron by reducing it from the ferric to the ferrous state for absorption in the duodenum and upper jejunum. After RYGB, reduced intake of organic heme iron, reduced conversion of the ferric to ferrous state, and reduced absorption can all contribute to low iron levels, especially in premenopausal women with additional menstrual losses. Tannins, phosphates, and phytates in food reduce iron absorption, whereas ascorbic acid increases it.

Folic acid

Dietary folate is present in food in the form of polyglutamates, which are hydrolyzed by intestinal brush border conjugases to monoglutamates.

Absorption occurs primarily from the proximal third of the small intestine but can occur along the entire length of the small intestine. Folate deficiency occurs with reduced dietary intake and reduced absorption.

Calcium

Calcium is absorbed primarily in the duodenum and proximal jejunum by an active saturation process mediated by vitamin D. Deficiency can occur with reduced intake of calcium- and vitamin D-containing food, reduced absorption due to bypass of the duodenum, and malabsorption of vitamin D. When a significant amount of the stomach is excluded, such as in RYGB, calcium citrate with vitamin D is the preferred preparation for replacement because it is more soluble than calcium carbonate in the absence of gastric acid production. Phytates, phosphates, and oxalates in food reduce calcium absorption.

Other nutrients

Essential minerals and trace elements (zinc, copper, cobalt, selenium, magnesium) are absorbed in the small intestine.

Preoperative nutritional status in bariatric patients

A morbidly obese patient is not a well-nourished patient. Although obese individuals have excess stores of energy in the form of fat, they may have clinical or subclinical nutritional deficiencies because of a poor diet over a prolonged period of time [18]. Most reported studies in the literature have concentrated on perioperative nutrient deficiencies before and after biliopancreatic diversion (BPD) and biliopancreatic diversion with duodenal switch (BPD-DS). In a review of nutritional deficiencies after BPD in 94 patients and BPD-DS in 76 patients, Slater and colleagues [19] reported a significant reduction in fat-soluble vitamin levels and abnormal calcium metabolism at 1 year following surgery. At 1 year, serum vitamin A levels were low in 52% of patients, vitamin K in 51%, and vitamin D in 57%. Serum zinc levels were also low in 51%, and hypocalcemia was present in 15% with secondary hyperparathyroidism in 31% [19]. None of the patients were symptomatic. The results suggested that fat-soluble vitamin deficiency, hypocalcemia, and low zinc levels were present in many of the patients before surgery.

Other studies have demonstrated similar findings [20–23]. Compston and colleagues [22] demonstrated low preoperative serum vitamin D levels in 16% of patients, and in a separate study, Bell and colleagues [23] confirmed these findings with a mean vitamin D level of 29 ng/mL in obese patients compared with 37 ng/mL in nonobese patients. Similar deficiencies have been reported in obese patients before RYGB, which persist and may worsen after surgery if unrecognized. In a prospective study by Sanchez-Hernandez

and colleagues [21], improvements in vitamin D deficiency and secondary hyperparathyroidism after RYGB were seen in 42% of patients; 37% of patients maintained their preoperative levels and 20% had a further reduction. Oral vitamin D supplementation and increased sunlight exposure were recommended in the morbidly obese [21]. Preoperative bone mineral density scans demonstrated osteopenia in 15 of 230 patients in a study by Johnson and colleagues [24], with further bone loss in the first year after RYGB.

Deficiencies of other essential vitamins, minerals, and trace elements are reported. Decsi and colleagues [25] demonstrated reduced serum vitamins A and E in obese boys compared with control values. Before LAGB, Gasteyer and colleagues [26] showed low iron levels in 31% and vitamin B₁₂ deficiency in 14% of their patients. At 1 year after LAGB, serum folate levels were significantly decreased [26]. Cooper and colleagues [27] also demonstrated low serum folate at 1 year following modified VBG despite low-dose multivitamin supplementation. Reduced thiamine levels are also reported, which can result in significant neurologic sequelae. Because thiamine is absent from fats, oils, and refined sugars, patients with a high carbohydrate intake derived mainly from refined sugars and milled rice are at greater risk of thiamine deficiency. The authors' group previously evaluated preoperative thiamine levels in obese patients not taking nutritional supplements, with no history of frequent alcohol consumption, other malabsorptive conditions, or previous bariatric surgery [28]. Of 303 patients, 15.5% had low preoperative thiamine levels. Female patients had lower mean preoperative thiamine levels of 2.4 µg/dL when compared with male patients (3.2 µg/dL) [28]. Deficiencies of micronutrients including vitamins A, K, C, and E, zinc, arginine, glutamine, copper, iron, essential fatty acids, bromelain, bioflavonoids, and ornithine alpha-ketoglutarate may interfere with wound healing after surgery. These findings emphasize the importance of a thorough preoperative nutritional assessment, with intervention if deficiencies exist.

Metabolic consequences of specific bariatric procedures

Restrictive procedures

Purely restrictive procedures achieve weight loss by limiting the total daily volume of food intake. Surgical options include the vertical banded gastroplasty (VBG), LAGB, or sleeve gastrectomy [29,30]. These procedures reduce the total volume and rate of food consumption. Absorption of ingested nutrients is normal because the continuity of the stomach, duodenum, and small bowel is intact. Nutritional deficiencies are uncommon unless eating habits are excessively restricted or a complication occurs. Due to a decrease in total caloric intake and selective food intolerance, particularly to meat, the ingestion of many essential nutrients, both macronutrients and micronutrients, is reduced. Due to the inability to tolerate

leafy and green vegetables, folic acid deficiency is the most common nutrient deficiency encountered after restrictive procedures. Daily multivitamin and mineral supplementation is recommended. Malnutrition can result if complications occur. Vomiting is common after VBG and LAGB and can result in significant nutritional deficiencies with dehydration and electrolyte abnormalities.

Malabsorptive procedures

Purely malabsorptive procedures include the jejunoileal (JIB) and jejuno-colonic bypass (JCB). Although it resulted in significant weight loss in over 70% of patients, the JIB was abandoned owing to serious metabolic consequences including intractable diarrhea, electrolyte disturbances, severe protein-calorie malnutrition, hypocalcemia, calcium oxalate stones, vitamin deficiencies, migratory polyarthralgias, and liver dysfunction progressing to cirrhosis and liver failure [31,32]. A high proportion of JIBs have now been revised to a less malabsorptive procedure or reversed. Nevertheless, patients continue to present with the metabolic consequences of severe malabsorption with metabolic bone, hepatic, and renal disease with hyperoxaluria and renal stones [33]. Significant hypovitaminosis D osteopathy, osteopenia, and hypocalcemia owing to vitamin D deficiency have been reported in a patient 32 years after JIB [34].

Combined malabsorptive and restrictive procedures

Procedures combining malabsorption and restriction include the RYGB, BPD, and BPD-DS. The RYGB is the most commonly performed bypass procedure. Weight loss occurs owing to a reduction in gastric volume with restricted intake, the dumping syndrome precipitated by ingestion of simple sugars, and a degree of malabsorption (Fig. 1). The dumping syndrome is precipitated by the ingestion of food with a high sugar or fat content and occurs in more than 75% of patients following RYGB [35]. It generally subsides 12 to 18 months after surgery. Patients can have disabling symptoms with food aversion and dehydration. Prevention involves consumption of small frequent meals, avoidance of high sugar content foods, chewing food thoroughly, eating slowly, and drinking liquids in between meals. A wide variety of pharmacologic therapies are available for patients with severe symptoms [35]. Low levels of vitamin D, calcium, vitamin B₁₂, and iron predominate after RYGB [36]. In a series of 41 patients who underwent RYGB, Johnson and colleagues [37] demonstrated a linear decrease in vitamin D with a linear increase in parathyroid hormone over time with increased roux limb length. Secondary hyperparathyroidism with elevated parathyroid hormone was seen in 58% of the patients after RYGB with normal vitamin D levels, suggesting selective calcium malabsorption [37]. Late development of metabolic bone disease with osteomalacia can occur due to vitamin D deficiency and secondary

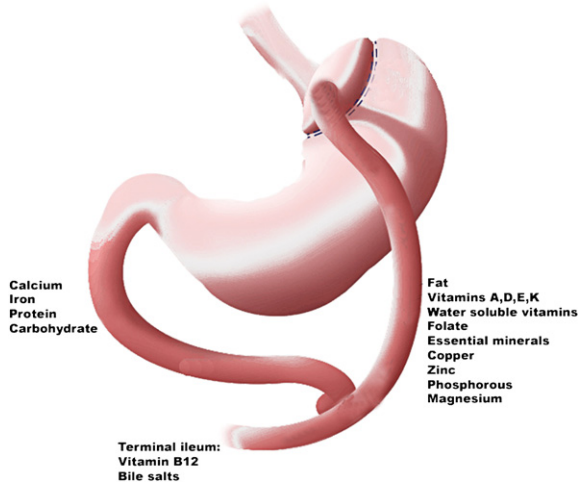


Fig. 1. Site of absorption of macronutrients and micronutrients. (Courtesy of the Cleveland Clinic Foundation, Cleveland, Ohio; with permission.)

hyperparathyroidism [38]. Postmenopausal women after RYGB demonstrate evidence of secondary hyperparathyroidism, elevated bone resorption, and reduced femoral neck and higher lumbar spine bone loss [39].

The BPD is primarily a malabsorptive procedure with minor restriction, consisting of a distal gastrectomy with a large 200- to 500-mL proximal pouch and a long Roux-en-Y reconstruction [40]. To reduce the adverse side effects of marginal ulceration, diarrhea, and protein-calorie malnutrition, the BPD was modified to the BPD-DS procedure [41,42]. The BPD-DS combines a vertical sleeve gastrectomy with a volume of approximately 100 to 150 mL and a duodenal switch with a common channel of 100 cm and an alimentary limb of 150 cm. Nutritional deficiencies are more common after the BPD and PBD-DS when compared with the RYGB. Fat-soluble vitamin deficiencies, protein-calorie malnutrition, hypocalcemia, diarrhea with dehydration and electrolyte disturbances, and deficiencies of zinc and selenium are common after BPD [43,44]. By retaining the pylorus, the BPD-DS procedure would be expected to reduce diarrhea and the risk of malnutrition when compared with BPD; however, in a study by Dolan and colleagues [45], similar weight loss and nutritional side effects were seen after BPD and PBD-DS, with hypoalbuminemia in 18%, anemia in 32%, hypocalcemia in 25%, and low levels of vitamins A, D, and K in nearly 50% of patients despite vitamin supplementation in over 80%.

Neurologic sequelae including encephalopathy, peripheral neuropathy, rhabdomyolysis, and Guillain-Barre syndrome have been reported following BPD and RYGB [46]. Of 957 patients in eight reports of neurologic complications after RYGB, 25% had vitamin B₁₂ deficiency and 11% thiamine deficiency [46]. Vitamin A deficiency has been reported after RYGB and BPD,

with ocular complications of xerophthalmia, nyctalopia, decreased visual acuity, and legal blindness [44,47].

Metabolic consequences of postoperative complications

Bariatric surgery involves an abdominal operation in a high-risk patient with significant comorbidity and the technical challenges of body habitus. None of the available surgical procedures are without complications [48–51]. Complications, whether early or late, can result in inadequate oral intake, excess gastrointestinal losses by vomiting or diarrhea, and a catabolic state with increased nutrient requirements. Early gastrointestinal complications after purely restrictive procedures are uncommon but include hemorrhage, gastric or esophageal perforation, outlet stenosis in VBG, and staple line leakage in VBG or sleeve gastrectomy. A similar range of complications can be seen after combined malabsorptive and restrictive procedures, with the addition of anastomotic leaks, fistulation, and intestinal obstruction [52]. These complications are usually detected in the immediate postoperative period and appropriate management instituted. Resultant nutritional deficiencies are rare, except in patients who have preexisting deficits. Nevertheless, acute stress and sepsis cause catabolism of lean body mass, which can be severely detrimental in an obese patient with preexisting protein depletion owing to a poor diet [53]. As is true for nonobese patients, if absence of oral intake is anticipated for 5 days or more, total parenteral nutrition or fine-bore nasojejunal feeding is recommended. These individuals are at risk for the refeeding syndrome, and serum levels of potassium, magnesium, and phosphorous should be checked daily for the first 3 days and promptly replaced if low [54].

Following recovery from the early postoperative period, other problems may arise that can contribute to nutritional deficiencies. One of the most common complaints after bariatric surgery is vomiting, occurring in 30% of patients [52]. Prolonged vomiting can result in dehydration, protein-calorie malnutrition, and thiamine deficiency resulting in neurologic sequelae [55–60]. There are multiple causes, and careful evaluation is essential (Box 2). Faintuch and colleagues [51] identified exogenous precipitating factors in 64% of 11 malnourished patients in a series of 236 patients who underwent RYGB. After VBG or LAGB, mechanical causes include outlet obstruction or stenosis, band slippage, band erosion, or cuff overdilatation with LAGB [61]. Acid reflux and dysphagia are frequent accompanying symptoms contributing to inadequate oral intake. Over time, pouch and distal esophageal dilation with megaesophagus can develop.

After RYGB, stenosis of the gastrojejunostomy is a common complication occurring in 4% to 20% of patients [62–65]. Diarrhea can occur owing to food sensitivity, the dumping syndrome, lactose intolerance, malabsorption, and bacterial overgrowth and infection, causing dehydration and electrolyte

Box 2. Causes of vomiting after bariatric surgery

- Inadequate chewing
- Overdistention of the pouch by fluid
- Large volume meals
- Food intolerance (red meat, lactose)
- Stomal outlet stenosis/obstruction
- Marginal ulceration
- Intestinal obstruction
- Gastroesophageal reflux disease
- Symptomatic gallstones
- Medications
- Dumping syndrome

imbalances. Other complications after RYGB, BPD, and BPD-DS include intestinal obstruction due to an internal, incisional, or port site hernia or adhesions (Box 3) [66,67]. Symptoms can be acute with a complete small bowel obstruction or recurrent with intermittent partial episodes of obstruction. Early intervention is important if and when complications develop to prevent ongoing symptoms. Intravenous replacement of potassium, essential vitamins including thiamine, and minerals is necessary in the presence of a history of prolonged vomiting or reduced oral intake to prevent the development of adverse nutritional and metabolic sequelae.

Modification of eating behavior

Following bariatric surgery, patients need to modify their eating behavior. After VBG, LAGB, or RYGB, patients need to reduce the food volume consumed, chew food very well, and slow the pace of eating. Failure to modify eating habits will result in vomiting and severe discomfort. The stomal size of the LAGB can be altered by filling or removing saline from the

Box 3. Late gastrointestinal complications after Roux-en-Y gastric bypass

- Intestinal obstruction
- Marginal ulcers
- Stomal stenosis
- Dumping syndrome
- Gallstones

band depending on the amount of food the patient tolerates. Red meat and poultry are often not well tolerated after restrictive procedures and RYGB [68]. Failure to chew food well may result in outlet obstruction, discomfort, and vomiting. Ground or pureed meat is easier to tolerate. Fluids should not be consumed with food but taken 30 minutes before or after eating to prevent pouch filling, vomiting, or early satiety leading to grazing between meals. Each meal volume should be measured (<45 mL), each bite should be chewed 20 times, and cessation of eating should occur once the patient starts to feel full rather than waiting for the onset of discomfort. High sugar-containing drinks and sweets should be avoided to enhance weight loss. Carbonated drinks should be avoided because the carbon dioxide can lead to discomfort. Following RYGB, the ingestion of simple sugars in concentrated form will precipitate the dumping syndrome and should be omitted from the diet. Patients should be advised to avoid sugar-containing beverages, juice, concentrated sweets, and sugar-containing condiments and sauces such as sweet and sour sauce, ketchup, and certain salad dressings. High fat-containing food should be avoided, particularly after BPD and BPD-DS. Fat-free milk is advised after BPD or BPD-DS to avoid diarrhea, bloating, and excess flatulence due to lactase deficiency.

Protein-rich food should be the major component at each meal. Many studies have demonstrated reduced protein intake below recommended levels following bariatric surgery, with reduced dietary intake of iron, folate, and calcium [69]. In a retrospective review of 69 patients who had undergone RYGB 18 months to 4 years earlier by Warde-Kamar and colleagues [70], the average daily caloric intake was variable, ranging from 624 to 3486 kcal with a mean of 733 ± 630 kcal, with 44% of calories from carbohydrate, 22% from protein, and 33% from fat. Snacks accounted for 37% of the daily intake. Following VBG, patients adversely altered their diet toward soft, high-calorie food with increased intake of milk, ice cream, and solid sweets [71,72].

The role of preoperative preparation, postoperative nutrient supplementation, and surveillance

At the authors' center, before surgery, all patients are extensively assessed by a multidisciplinary team. Preoperatively, patients are encouraged to eat three meals a day, with no snacking in between. Consumption of high-protein food including meat, fish, eggs, and poultry, and avoidance of empty calorie food is advised. Physical activity is encouraged with a goal of walking for 30 minutes daily. Patients are advised to taste different protein shakes to identify their preferred preparation for postoperative use. Daily supplementation with a multivitamin with iron, vitamin B complex, and calcium is commenced after measurement of serum levels. If serum levels are low, appropriate replacement is performed.

After surgery, all patients receive multivitamin supplementation in the form of a daily multivitamin with iron, additional vitamin B₁₂, a vitamin B complex with thiamine, and vitamin C, iron, and calcium (Table 1). Zinc and biotin supplementation are optional to minimize temporary hair thinning. Additional protein should be given in the form of protein shakes. The daily requirement following RYGB is 40 to 60 g/day. Additional levels are required after BPD-DS at 60 to 90 g/day. In the authors' unit, protein shakes are administered for 2 weeks before surgery and continued for 2 to 3 weeks. Consumption of protein-rich food in the form of cheese, fish, and meat is encouraged once oral intake is established. Patients are encouraged to continue to drink protein shakes, particularly if oral intake is poor. Additional supplementation may be required in menstruating women at risk of iron deficiency anemia, during pregnancy with increased maternal and fetal requirements, and in obese adolescents [73]. Maternal malabsorption after BPD has resulted in vitamin A deficiency with development of night blindness during the third trimester of pregnancy and in vitamin A deficiency in the newborn infant [74]. The prevalence of overweight in female children and adolescents has increased from 14% in 1999 to 2000 to 16% in 2003 to 2004, with an increase in the prevalence of overweight in male

Table 1
Postoperative nutritional supplements after bariatric surgery

Nutritional supplement	RYGB	Sleeve gastrectomy	LAGB
Calcium with vitamin D	Calcium citrate, 500 mg × 3/d	Calcium citrate, 500 mg × 3/d	Calcium citrate, 500 mg × 3/d, or carbonate, 1200 mg/d
Vitamin B ₁₂	Intramuscular, 1000 µg/month; oral, 100–300 µg/d; or sublingual, 500 µg/d	Oral, 100–300 µg/d; or sublingual, 500 µg/d	Not required unless low
Vitamin B complex with thiamine	1 tablet/d	1 tablet/d	1 tablet/d
Multivitamin tablet to replace	1 tablet/d	1 tablet/d	1 tablet/d
Vitamin A	1 mg/d	1 mg/d	1 mg/d
Vitamin D	5 µg/d	5 µg/d	5 µg/d
Vitamin E	100–300 mg/d	100–300 mg/d	100–300 mg/d
Vitamin K	65–80 µg/d	65–80 µg/d	65–80 µg/d
Iron	45–60 mg/d	45–60 mg/d	45–60 mg/d
Vitamin C	500 mg/d	500 mg/d	500 mg/d
Zinc	15 mg/d	15 mg/d	15 mg/d
Biotin	3000 µg/d	3000 µg/d	3000 µg/d

children and adolescents from 14% to 18% [5]. These patients require close monitoring and careful nutrient supplementation following surgery.

Following all bariatric surgical procedures, patients require regular and prolonged follow-up to assess weight loss, ensure compliance with diet and multivitamin and nutritional supplements, and monitor for the development of complications including nutritional deficiencies and electrolyte abnormalities. In the authors' center, all patients are seen in the outpatient clinic at 2 weeks and then every 3 months thereafter for the first year with the exception of the LAGB group, who are reviewed every 2 months. Patients are then seen every 6 months with the option of a shared medical appointment. Continuing patient education is critical to prevent noncompliance. Numerous studies have demonstrated poor compliance with vitamin and mineral supplements coupled with reduced dietary intake of below 50% of the recommended daily allowance [36,75,76].

Summary

Nutritional deficiencies are already present in many morbidly obese patients before surgery. Appropriate preoperative detection and correction is essential. Deficiencies in vitamins, minerals, proteins, lipids, carbohydrates, electrolytes, and trace elements can occur after all types of bariatric surgery. The severity and pattern is dependent on the presence of preoperative uncorrected deficiency, the type of procedure performed varying with the degree of restriction and the length of bypassed small intestine, the modification of eating behavior, the development of complications, compliance with oral multivitamin and mineral supplementation, and compliance with follow-up. Rigorous control of fluids and electrolytes with establishment of adequate oral nutrition is important in the immediate postoperative period. Regular follow-up of the metabolic and nutritional status of the patient is essential, with life-long multivitamin and mineral supplementation. Pregnancy should not be considered until at least 18 months after bariatric surgery following the completion of the period of rapid weight loss.

References

- [1] ASPEN Board of Directors and Standards Committee. Definitions of terms, style, and conventions used in ASPEN guidelines and standards. *Nutr Clin Pract* 2005;20:281–5.
- [2] Kuczmarski RJ, Flegal KM, Campbell SM, et al. Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. *JAMA* 1994;272(3):205–11.
- [3] Deitel M. The obesity epidemic. *Obes Surg* 2006;16(4):377–8.
- [4] Inge TH, Zeller M, Garcia VF, et al. Surgical approach to adolescent obesity. *Adolesc Med Clin* 2004;15(3):429–53.
- [5] Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA* 2006;295(13):1549–55.

- [6] Yanovski SZ, Yanovski JA. Obesity. *N Engl J Med* 2002;346(8):591–602.
- [7] Leser MS, Yanovski SZ, Yanovski JA, et al. A low-fat intake and greater activity level are associated with lower weight regain 3 years after completing a very-low-calorie diet. *J Am Diet Assoc* 2002;102(9):1252–6.
- [8] Solomon CG, Dluhy RG. Bariatric surgery—quick fix or long-term solution? *N Engl J Med* 2004;351(26):2751–3.
- [9] Foster GD, Wyatt HR, Hill JO, et al. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med* 2003;348(21):2082–90.
- [10] Thearle M, Aronne LJ. Obesity and pharmacologic therapy. *Endocrinol Metab Clin North Am* 2003;32(4):1005–24.
- [11] DeWald T, Khaodhilar L, Donahue MP, et al. Pharmacological and surgical treatments for obesity. *Am Heart J* 2006;151(3):604–24.
- [12] Li Z, Maglione M, Tu W, et al. Meta-analysis: pharmacologic treatment of obesity. *Ann Intern Med* 2005;142(7):532–46.
- [13] Maggard MA, Shugarman LR, Suttorp M, et al. Meta-analysis: surgical treatment of obesity. *Ann Intern Med* 2005;142(7):547–59.
- [14] Gastrointestinal surgery for severe obesity. Consensus Statement 1991;9(1):1–20.
- [15] Alvarez-Leite JI. Nutrient deficiencies secondary to bariatric surgery. *Curr Opin Clin Nutr Metab Care* 2004;7(5):569–75.
- [16] Mason ME, Jalagani H, Vinik AI. Metabolic complications of bariatric surgery: diagnosis and management issues. *Gastroenterol Clin North Am* 2005;34(1):25–33.
- [17] Ponsky TA, Brody F, Pucci E. Alterations in gastrointestinal physiology after Roux-En-Y gastric bypass. *J Am Coll Surg* 2005;201(1):125–31.
- [18] Boylan LM, Sugerman HJ, Driskell JA. Vitamin E, vitamin B-6, vitamin B-12, and folate status of gastric bypass surgery patients. *J Am Diet Assoc* 1988;88(5):579–85.
- [19] Slater GH, Ren CJ, Siegel N, et al. Serum fat-soluble vitamin deficiency and abnormal calcium metabolism after malabsorptive bariatric surgery. *J Gastrointest Surg* 2004;8(1):48–55.
- [20] Hamoui N, Kim K, Anthone G, et al. The significance of elevated levels of parathyroid hormone in patients with morbid obesity before and after bariatric surgery. *Arch Surg* 2003;138(8):891–7.
- [21] Sanchez-Hernandez J, Ybarra J, Gich I, et al. Effects of bariatric surgery on Vitamin D status and secondary hyperparathyroidism: a prospective study. *Obes Surg* 2005;15(10):1389–95.
- [22] Compston JE, Vedi S, Ledger JE, et al. Vitamin D status and bone histomorphometry in gross obesity. *Am J Clin Nutr* 1981;34(11):2359–63.
- [23] Bell NH, Epstein S, Greene A, et al. Evidence for alteration of the vitamin D–endocrine system in obese subjects. *J Clin Invest* 1985;76(1):370–3.
- [24] Johnson JM, Maher JW, Samuel I, et al. Effects of gastric bypass procedures on bone mineral density, calcium, parathyroid hormone, and vitamin D. *J Gastrointest Surg* 2005;9(8):1106–10.
- [25] Decsi T, Molnar D, Koletzko B. Reduced plasma concentrations of alpha-tocopherol and beta-carotene in obese boys. *J Pediatr* 1997;130(4):653–5.
- [26] Gasteyger C, Suter M, Calmes JM, et al. Changes in body composition, metabolic profile and nutritional status 24 months after gastric banding. *Obes Surg* 2006;16(3):243–50.
- [27] Cooper PL, Brearley LK, Jamieson AC, et al. Nutritional consequences of modified vertical gastroplasty in obese subjects. *Int J Obes Relat Metab Disord* 1999;23(4):382–8.
- [28] Carrodeguas L, Kaidar-Person O, Szomstein S, et al. Preoperative thiamine deficiency in obese population undergoing laparoscopic bariatric surgery. *Surg Obes Relat Dis* 2005;1(6):517–22.
- [29] Mason EE, Doherty C, Cullen JJ, et al. Vertical gastroplasty: evolution of vertical banded gastroplasty. *World J Surg* 1998;22(9):919–24.
- [30] Roa PE, Kaidar-Person O, Pinto D, et al. Laparoscopic sleeve gastrectomy as treatment for morbid obesity: technique and short-term outcome. *Obes Surg* 2006;16(10):1323–6.

- [31] Brown RG, O'Leary JP, Woodward ER. Hepatic effects of jejunioleal bypass for morbid obesity. *Am J Surg* 1974;127(1):53–8.
- [32] Moxley RT III, Pozefsky T, Lockwood DH. Protein nutrition and liver disease after jejunioleal bypass for morbid obesity. *N Engl J Med* 1974;290(17):921–6.
- [33] Nordenvall B, Backman L, Larsson L. Oxalate metabolism after intestinal bypass operations. *Scand J Gastroenterol* 1981;16(3):395–9.
- [34] Haria DM, Sibonga JD, Taylor HC. Hypocalcemia, hypovitaminosis d osteopathy, osteopenia, and secondary hyperparathyroidism 32 years after jejunioleal bypass. *Endocr Pract* 2005;11(5):335–40.
- [35] Ukleja A. Dumping syndrome: pathophysiology and treatment. *Nutr Clin Pract* 2005;20(5): 517–25.
- [36] Ledoux S, Msika S, Moussa F, et al. Comparison of nutritional consequences of conventional therapy of obesity, adjustable gastric banding, and gastric bypass. *Obes Surg* 2006; 16(8):1041–9.
- [37] Johnson JM, Maher JW, Demaria EJ, et al. The long-term effects of gastric bypass on vitamin D metabolism. *Ann Surg* 2006;243(5):701–4.
- [38] Collazo-Clavell ML, Jimenez A, Hodgson SF, et al. Osteomalacia after Roux-En-Y gastric bypass. *Endocr Pract* 2004;10(3):195–8.
- [39] St George LI, Lin D. Malabsorption in pregnancy after biliopancreatic diversion for morbid obesity. *Med J Aust* 2005;182(6):308–9.
- [40] Scopinaro N, Adami GF, Marinari GM, et al. Biliopancreatic diversion. *World J Surg* 1998; 22(9):936–46.
- [41] Hess DS, Hess DW. Biliopancreatic diversion with a duodenal switch. *Obes Surg* 1998;8(3): 267–82.
- [42] Marceau P, Hould FS, Simard S, et al. Biliopancreatic diversion with duodenal switch. *World J Surg* 1998;22(9):947–54.
- [43] Newbury L, Dolan K, Hatzifotis M, et al. Calcium and vitamin D depletion and elevated parathyroid hormone following biliopancreatic diversion. *Obes Surg* 2003;13(6):893–5.
- [44] Hatzifotis M, Dolan K, Newbury L, et al. Symptomatic vitamin A deficiency following biliopancreatic diversion. *Obes Surg* 2003;13(4):655–7.
- [45] Dolan K, Hatzifotis M, Newbury L, et al. A clinical and nutritional comparison of biliopancreatic diversion with and without duodenal switch. *Ann Surg* 2004;240(1):51–6.
- [46] Koffman BM, Greenfield LJ, Ali II, et al. Neurologic complications after surgery for obesity. *Muscle Nerve* 2006;33(2):166–76.
- [47] Lee WB, Hamilton SM, Harris JP, et al. Ocular complications of hypovitaminosis A after bariatric surgery. *Ophthalmology* 2005;112(6):1031–4.
- [48] Abell TL, Minocha A. Gastrointestinal complications of bariatric surgery: diagnosis and therapy. *Am J Med Sci* 2006;331(4):214–8.
- [49] Pappas PK, Caushaj PF, McCormick JT, et al. Laparoscopic management of complications following laparoscopic Roux-En-Y gastric bypass for morbid obesity. *Surg Endosc* 2003;17(4):610–4.
- [50] Podnos YD, Jimenez JC, Wilson SE, et al. Complications after laparoscopic gastric bypass: a review of 3464 cases. *Arch Surg* 2003;138(9):957–61.
- [51] Faintuch J, Matsuda M, Cruz ME, et al. Severe protein-calorie malnutrition after bariatric procedures. *Obes Surg* 2004;14(2):175–81.
- [52] Ukleja A, Stone RL. Medical and gastroenterologic management of the post-bariatric surgery patient. *J Clin Gastroenterol* 2004;38(4):312–21.
- [53] Ecklund MM. Meeting the nutritional needs of the bariatric patient in acute care. *Crit Care Nurs Clin North Am* 2004;16(4):495–9.
- [54] Elliot K. Nutritional considerations after bariatric surgery. *Crit Care Nurs Q* 2003;26(2): 133–8.
- [55] Mason EE. Starvation injury after gastric reduction for obesity. *World J Surg* 1998;22(9): 1002–7.

- [56] Worden RW, Allen HM. Wernicke's encephalopathy after gastric bypass that masqueraded as acute psychosis: a case report. *Curr Surg* 2006;63(2):114-6.
- [57] Toth C, Voll C. Wernicke's encephalopathy following gastroplasty for morbid obesity. *Can J Neurol Sci* 2001;28(1):89-92.
- [58] Sola E, Morillas C, Garzon S, et al. Rapid onset of Wernicke's encephalopathy following gastric restrictive surgery. *Obes Surg* 2003;13(4):661-2.
- [59] Albina JE, Stone WM, Bates M, et al. Catastrophic weight loss after vertical banded gastroplasty: malnutrition and neurologic alterations. *JPEN J Parenter Enteral Nutr* 1988;12(6): 619-20.
- [60] Angstadt JD, Bodziner RA. Peripheral polyneuropathy from thiamine deficiency following laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 2005;15(6):890-2.
- [61] Fobi M, Lee H, Igwe D, et al. Band erosion: incidence, etiology, management and outcome after banded vertical gastric bypass. *Obes Surg* 2001;11(6):699-707.
- [62] Carrodegua L, Szomstein S, Zundel N, et al. Gastrojejunal anastomotic strictures following laparoscopic Roux-En-Y gastric bypass surgery: analysis of 1291 patients. *Surg Obes Relat Dis* 2006;2(2):92-7.
- [63] Schwartz ML, Drew RL, Roiger RW, et al. Stenosis of the gastroenterostomy after laparoscopic gastric bypass. *Obes Surg* 2004;14(4):484-91.
- [64] Ahmad J, Martin J, Ikramuddin S, et al. Endoscopic balloon dilation of gastroenteric anastomotic stricture after laparoscopic gastric bypass. *Endoscopy* 2003;35(9):725-8.
- [65] Go MR, Muscarella P, Needleman BJ, et al. Endoscopic management of stomal stenosis after Roux-en-Y gastric bypass. *Surg Endosc* 2004;18(1):56-9.
- [66] Cho M, Carrodegua L, Pinto D, et al. Diagnosis and management of partial small bowel obstruction after laparoscopic antecolic antegastric Roux-en-Y gastric bypass for morbid obesity. *J Am Coll Surg* 2006;202(2):262-8.
- [67] Nguyen NT, Huerta S, Gelfand D, et al. Bowel obstruction after laparoscopic Roux-en-Y gastric bypass. *Obes Surg* 2004;14(2):190-6.
- [68] Balsiger BM, Kennedy FP, Abu-Lebdeh HS, et al. Prospective evaluation of Roux-en-Y gastric bypass as primary operation for medically complicated obesity. *Mayo Clin Proc* 2000; 75(7):673-80.
- [69] Blake M, Fazio V, O'Brien P. Assessment of nutrient intake in association with weight loss after gastric restrictive procedures for morbid obesity. *Aust N Z J Surg* 1991;61(3):195-9.
- [70] Warde-Kamar J, Rogers M, Flancabaum L, et al. Calorie intake and meal patterns up to 4 years after Roux-en-Y gastric bypass surgery. *Obes Surg* 2004;14(8):1070-9.
- [71] Brolin RL, Robertson LB, Kenler HA, et al. Weight loss and dietary intake after vertical banded gastroplasty and Roux-en-Y gastric bypass. *Ann Surg* 1994;220(6):782-90.
- [72] Kriwanek S, Blauensteiner W, Lebisch E, et al. Dietary changes after vertical banded gastroplasty. *Obes Surg* 2000;10(1):37-40.
- [73] Sugerma HJ. Bariatric surgery for severe obesity. *J Assoc Acad Minor Phys* 2001;12(3): 129-36.
- [74] Huerta S, Rogers LM, Li Z, et al. Vitamin A deficiency in a newborn resulting from maternal hypovitaminosis A after biliopancreatic diversion for the treatment of morbid obesity. *Am J Clin Nutr* 2002;76(2):426-9.
- [75] Bloomberg RD, Fleishman A, Nalle JE, et al. Nutritional deficiencies following bariatric surgery: what have we learned? *Obes Surg* 2005;15(2):145-54.
- [76] Trostler N, Mann A, Zilberbush N, et al. Nutrient intake following vertical banded gastroplasty or gastric bypass. *Obes Surg* 1995;5(4):403-10.