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ASMBS Guidelines

ASMBS Allied Health Nutritional Guidelines for the Surgical Weight Loss Patient

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This document is intended to provide an overview of the elements that are important to the nutritional care of the bariatric patient. It is not intended to serve as training, a statement of standardization, or scientific consensus. It should be viewed as an educational tool to increase awareness among medical professionals of the potential risk of nutritional deficiencies common to bariatric surgery patients.

The goal of this document is to provide suggestions for conducting a nutrition assessment, education, supplementation, and follow-up care. These suggestions are not mandates and should be treated with common sense. When needed, exceptions should be made according to individual variations and the evaluation findings. It is intended to present a reasonable approach to patient nutrition care and at the same time allow for flexibility among individual practice-based protocols, procedures, and policies. Amendments to this document are anticipated as more research, scientific evidence, resources, and information become available.

Nutrition care

The Dietitian's role is a vital component of the bariatric surgery process. Nutrition assessment and dietary management in surgical weight loss have been shown to be an important correlate with success [1,2]. A comprehensive nutrition assessment should be conducted preoperatively by a dietitian, physician, and/or well-informed, qualified multidisciplinary team to identify the patient's nutritional and educational needs. It is essential to determine any preexisting nutritional deficiencies, develop appropriate dietary interventions for correction, and create a plan for postoperative dietary intake that will enhance the likelihood of success.

The management of postoperative nutrition begins preoperatively with a thorough assessment of nutrient status, a strong educational program, and follow-up to reinforce important principals associated with long-term weight loss maintenance. A comprehensive nutrition evaluation goes far beyond assessing the actual dietary intake of the bariatric patient. It takes into account the whole person, encompassing several multidisciplinary facets. Not only should the practitioner review the standard assessment components (i.e., medical co-morbidities, weight history, laboratory values, and nutritional intake), it is also important to evaluate other issues that could affect nutrient status, including readiness for change, realistic goal setting, general nutrition knowledge, as well as behavioral, cultural, psychosocial, and economic issues.

The role of nutrition education and medical nutrition therapy in bariatric surgery will continue to grow as tools to enhance surgical outcome and long-term weight loss maintenance are explored further and identified. The following tables suggest the possible components of bariatric nutrition care:

Table 1: Suggested preoperative nutrition assessment
Table 2: Suggested preoperative nutrition education

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Table 1
Suggested Preoperative Nutrition Assessment

Recommended	Suggested	Other considerations
Anthropometrics		
Age, sex, race, accurate height and weight, BMI, excess body weight	Visual inspection of hair, skin, and nails	Waist circumference Other body measurements
Weight history		
Failed weight loss attempts Recent preoperative weight loss attempt (if required by program)	Life events that may have caused weight change	Personal weight loss goals
Medical history		
Current co-morbidities Current medications Vitamin/mineral/herbal supplements Food allergies/intolerances	Past medical history If available: % body fat using bioelectrical impedance; resting metabolic rate (volume of oxygen uptake); respiratory quotient	Observation of body fat distribution Consideration of patients who are athletic or muscular and BMI classifications
Available laboratory values		
Psychological history		
History of eating disorder Current/past psychiatric diagnosis		
Other		
Alcohol/tobacco/drug use Problems with eyesight Problems with dentition Literacy level Language barrier		
Dietary intake: food/fluid		
24-hr recall (weekday/weekend), Food frequency record, or Food, mood, and activity log (helps identify food group omission or dietary practices that increase nutritional risk) Restaurant meal intake Disordered eating patterns	Cultural diet influences Religious diet restrictions Meal preparation skill level Craving/trigger foods Eats while engaged in other activities	Computerized nutrient analysis (if available) Food preferences Attitudes toward food
Physical activity		
Physical conditions limiting activity Current level of activity	Types of activities enjoyed in the past Amount of time spent in daily sedentary activities	Activity preference for the future Attitude toward physical activity
Psychosocial		
Motivation/reasons for seeking surgical intervention Readiness to make behavioral, diet, exercise, and lifestyle changes Previous application of above principles listed to demonstrate ability to make lifestyle change Willingness to comply with program protocol Emotional connection with food Stress level and coping mechanisms Identify personal barriers to postoperative success	Confidence to maintain weight loss Anticipated life changes Marital status/children Support system Work schedule Financial constraints Referral to appropriate professionals for specialized physical activity instruction and/or mental health evaluation	Attitude toward lifestyle change Attitude toward taking life-long vitamin supplementation

BMI = body mass index.

Table 2
Suggested Preoperative Nutrition Education

Recommended	Suggested	Other considerations
Discuss/include		
Importance of taking personal responsibility for self-care and lifestyle choices	Realistic goal setting	Appropriate monitoring of weight loss
Techniques for self-monitoring and keeping daily food journal	Benefits of physical activity	
Preoperative diet preparation (if required by program)		
Postoperative intake	Common complaints	Long-term maintenance
Adequate hydration	Dehydration	Self-monitoring
Texture progression	Nausea/vomiting	Nutrient dense food choices for disease prevention
Vitamin/mineral supplements	Anorexia	Restaurants
Protein supplements	Effects of ketosis	Label reading
Meal planning and spacing	Return of hunger	Healthy cooking techniques
Appropriate carbohydrate, protein, and fat intake, and food/fluid choices to maximize safe weight loss, nutrient intake, and tolerance	Stomal obstruction from food	Relapse management
	Dumping syndrome	
	Reactive hypoglycemia	
Concepts of intuitive eating	Constipation	
Techniques and tips to maximize food and fluid tolerance	Diarrhea/steatorrhea	
	Flatulence/bowel sounds	
Possibility of nutrient malabsorption and importance of supplement compliance	Lactose intolerance	
	Alopecia	
Possibility of weight regain		

Table 3: Suggested postoperative follow-up
Table 4: Suggested biochemical monitoring tools for nutrition status
Table 5: Suggested postoperative vitamin supplementation

These suggestions, included in Tables 1-5, have been based on committee consensus and current research that has documented the pre- and postoperative likelihood of nutrition deficiency [1–18].

Biochemical monitoring for nutrition status (Table 4)

Deficiencies of single vitamins are less often encountered than those of multiple vitamins. Although protein–calorie undernutrition can result in concurrent vitamin deficiency, most deficiencies are associated with malabsorption and/or incomplete digestion related to negligible gastric acid and pepsin, alcoholism, medications, hemodialysis, total parenteral nutrition, food faddism, or inborn errors of metabolism. Bariatric surgery procedures specifically alter the absorption pathways and/or dietary intake. Symptoms of vitamin deficiency are commonly nonspecific, and physical examination might not be reliable for early diagnosis without laboratory confirmation. Most characteristic physical findings are seen late in the course of nutrient deficiency [17].

Laboratory markers are considered imperative for completing the initial nutrition assessment and follow-up for surgical weight loss patients. Established baseline values are important when trying to distinguish between postoperative complications, deficiencies related to surgery, noncompliance with recommended nutrient supplementation, or nutritional complications arising from preexisting deficiencies. Additional laboratory measures might be required and are defined by the presence of the existing individual co-morbid conditions. They are not included in Table 4. Table 4 is a sample of laboratory measures that programs might consider using to comprehensively monitor patients' nutrition status. It is not a mandate or guideline for laboratory testing.

Vitamin supplementation (Table 5)

Table 5 is an example of a supplementation regimen. As advances are made in the field of bariatrics and nutrition, updates regarding supplementation suggestions are expected. This information is intended for life-long daily supplementation for routine postoperative patients and is not intended to treat deficiencies. Information on treating deficiencies can be found in the Appendix "Identifying and Treating Micronutrient Deficiencies." A patient's individual co-morbid conditions or changes in health status might require adjustments to this regimen.

Rationale for recommendations

Importance of multivitamin and mineral supplementation

It is common knowledge that a comprehensive bariatric program includes nutritional supplementation guidance, routine monitoring of the patient's physical/mental well-being, laboratory values, and frequent counseling to reinforce nutrition education, behavior modification, and principles of responsible self-care. As the popularity of surgical

Table 3
Suggested Postoperative Nutrition Follow-up

Recommended	Suggested	Other considerations
Anthropometric		
Current and accurate height, weight, BMI, and percentage of excess body weight	Overall sense of well-being	Use of contraception to avoid pregnancy
Biochemical	Activity level	Psychosocial
Review laboratory findings when available	Amount, type, intensity, and frequency of activity	Changing relationship with food Changes in support system Stress management Body image
Medication review		
Encourage patients to follow-up with PCP regarding medications that treat rapidly resolving co-morbidities (e.g., hypertension, diabetes mellitus)		
Vitamin/mineral supplements		
Adherence to protocol		
Dietary intake		
Usual or actual daily intake	Estimated caloric intake of usual or actual intake	Promote anti-obesity foods containing:
Protein intake	Reinforce intuitive eating style to improve food tolerance	Omega-3 fatty acids
Fluid intake	Appropriate meal planning	High fiber
Assess intake of anti-obesity foods		Lean quality protein sources
Food texture compliance		Whole fruits and vegetables
Food tolerance issues (e.g., nausea/vomiting, "dumping")		Foods rich in phytochemicals and antioxidants
Appropriate diet advance		Low-fat dairy (calcium)
Address individual patient complaints		Discourage pro-obesity processed foods containing:
Address lifestyle and educational needs for long term weight loss maintenance		Refined carbohydrates Trans and saturated fatty acids

BMI = body mass index; PCP = primary care physician.

interventions for morbid obesity continues to grow, concern is increasing regarding the long-term effects of nutritional deficiencies. Nutritional complications that remain undiagnosed and untreated can lead to adverse health consequences and loss of productivity. The benefits of weight loss surgery must be balanced against the risk of developing nutritional deficiencies to provide appropriate identification, treatment, and prevention.

Vitamins and minerals are essential factors and co-factors in numerous biological processes that regulate body size. They include appetite, hunger, nutrient absorption, metabolic rate, fat and sugar metabolism, thyroid and adrenal function, energy storage, glucose homeostasis, neural activities, and others. Thus, micronutrient "repletion" (meaning the body has sufficient amounts of vitamins and minerals to perform these functions) is not only important for good health, but also for maximal weight loss success and long-term weight maintenance.

Obtaining micronutrients from food is the most desirable way to ensure the body has sufficient amounts of vitamins and minerals. However, some experts have suggested that most individuals in our "fast-paced, eat-out" society fail to consume sufficient amounts of unprocessed foods that are high in vitamins and minerals, such as fruits and vegetables, fish and other protein sources, dairy products, whole grains, nuts and legumes. Poor dietary selection and habits, coupled with the reduced vitamin and mineral content of foods, can lead to micronutrient deficiencies among the general public that interfere with body weight control, increasing the risk of weight gain and obesity. Therefore, a daily vitamin and mineral supplement is likely to be of value in ensuring adequate intake of micronutrients for maximal functioning of those processes that help to regulate appropriate body weight.

Taking daily micronutrient supplements and eating foods high in vitamins and minerals are important aspects of any successful weight loss program. For the morbidly obese, taking vitamin and mineral supplements is essential for appropriate micronutrient repletion both before and after bariatric surgery. Studies have found that 60–80% of morbidly obese preoperative candidates have defects in vitamin D [19–22]. Such defects would reduce dietary calcium absorption and increase a substance known as calcitriol,

Table 4
Suggested Biochemical Monitoring Tools for Nutrition Status

Vitamin/mineral	Screening	Normal range	Additional laboratory indexes	Critical range	Preoperative deficiency	Postoperative deficiency	Comments
B_1 (thiamin)	Serum thiamin	10–64 ng/mL	↓ RBC transketolase ↑ Pyruvate	Transketolase activity >20% Pyruvate >1 mg/dL	15–29%; more common in African Americans and Hispanics; often associated with poor hydration	Rare, but occurs with RYGB, AGB, and BPD/DS	Serum thiamin responds to dietary supplementation but is poor indicator of total body stores
B_6 (pyridoxine)	PLP	5–24 ng/mL	RBC glutamic pyruvate Oxaloacetic transaminase	PLP <3 ng/mL	Unknown	Rare	Consider with unresolved anemia; diabetes could influence values
B_{12} (cobalamin)	Serum B_{12}	200–1000 pg/mL	↑ Serum and urinary MMA ↑ Serum tHcy	Serum B_{12} <200 pg/mL deficiency <400 pg/mL suboptimal sMMA >0.376 μmol/L μMMA >3.6 μmol/mmol CRT tHcy >13.2 μmol/L	10–13%; may occur with older patients and those taking H_2 blockers and PPIs	Common with RYGB in absence of supplementation, 12–33%	When symptoms are present and B_{12} 200–250 pg/mL, MMA and tHcy are useful; serum B_{12} may miss 25–30% of deficiency cases
Folate	RBC folate	280–791 ng/mL	Urinary FIGLU Normal serum and urinary MMA ↑ Serum tHcy	RBC folate <305 nmol/L deficiency, <227 nmol/L anemia	Uncommon	Uncommon	Serum folate reflects recent dietary intake rather than folate status; RBC folate is a more sensitive marker Excessive supplementation can mask B_{12} deficiency in CBC; neurologic symptoms will persist
Iron	Ferritin	Males: 15–200 ng/mL Females: 12–150 ng/mL	↓ Serum iron ↑ TIBC	Ferritin <20 ng/mL Serum iron <50 μg/dL TIBC >450 μg/dL	9–16% of adult women in general population are deficient	20–49% of patients; common with RYGB for menstruating women (51%), and patients with super obesity (49–52%)	Low Hgb and Hct are consistent with iron deficiency anemia in stage 3 or stage 4 anemia; ferritin is an acute phase reactant and will be elevated with illness and/or inflammation; oral contraceptives reduce blood loss for menstruating females
Vitamin A	Plasma retinol	20–80 μg/dL	RBP	Plasma retinol <10 μg/dL	Uncommon; up to 7% in some studies	Common (50%) with BPD/DS after 1 yr, up to 70% at 4 yr; may occur with RYGB/AGB	Ocular finding may suggest diagnosis

Table 4
Continued

Vitamin/mineral	Screening	Normal range	Additional laboratory indexes	Critical range	Preoperative deficiency	Postoperative deficiency	Comments
Vitamin D	25(OH)D	25–40 ng/mL	↓ Serum phosphorus ↑ Alkaline phosphatase ↑ Serum PTH ↓ Urinary calcium	Serum 25(OH)D <20 ng/mL suggests deficiency 20–30 ng/mL suggests insufficiency	Common; 60–70%	Common with BPD/DS after 1 yr; may occur with RYGB; prevalence unknown	With deficiency, serum calcium may be low or normal; serum phosphorus may decrease, serum alkaline phosphatase increases; PTH elevated
Vitamin E	Plasma alpha tocopherol	5–20 μg/mL	Plasma lipids	<5 μg/mL	Uncommon	Uncommon	Low plasma alpha tocopherol to plasma lipids (0.8 mg/g total lipid) should be used with hyperlipidemia
Vitamin K	PT	10–13 seconds	↑ DCP ↓ Plasma phylloquinone	Variable	Uncommon	Common with BPD/DS after 1 yr	PT is not a sensitive measure of vitamin K status
Zinc	Plasma zinc	60–130 μg/dL	↓ RBC zinc	Plasma zinc <70 μg/dL	Uncommon, but increased risk of low levels associated with obesity	Common with BPD/DS after 1 yr; may occur with RYGB	Monitor albumin levels and interpret zinc accordingly, albumin is primary binding protein for zinc; no reliable method of determining zinc status is available; plasma zinc is method generally used; studies cited in this report did not adequately describe methods of zinc analysis
Protein	Serum albumin Serum total protein	4–6 g/dL 6–8 g/dL	↓ Serum prealbumin (transthyretin)	Albumin <3.0 g/dL Prealbumin <20 mg/dL	Uncommon	Rare, but can occur with RYGB, AGB, and BPD/DS if protein intake is low in total intake or indispensable amino acids	Half-life for prealbumin is 2–4 d and reflects changes in nutritional status sooner than albumin, a nonspecific protein carrier with a half-life of 22 d

RYGB = Roux-en-Y gastric bypass; AGB = adjustable gastric banding; BPD/DS = biliopancreatic diversion/duodenal switch; PLP = pyridoxal-5'-phosphate; RBC = red blood cell; MMA = methylmalonic acid; tHcy = total homocysteine; CRT = creatinine; PPIs = protein pump inhibitors; FIGLU = formiminogluatmic acid; CBC = complete blood count; TIBC = total iron binding capacity; Hgb = hemoglobin; Hct = hematocrit; RPB = retinol binding protein; PTH = parathyroid hormone; 25(OH)D = 25-hydroxyvitamin D; PT = prothrombin time; DCP = des-gamma-carboxypromthrombin.

In general, laboratory values should be reviewed annually or as indicated by clinical presentation. Laboratory normal values vary among laboratory settings and are method dependent. This chart provides a brief summary of monitoring tools. See the Appendix for additional detail and diagnostic tools.

Table 5
Suggested Postoperative Vitamin Supplementation

Supplement	AGB	RYGB	BPD/DS	Comment
Multivitamin-mineral supplement				
*A high-potency vitamin containing 100% of daily value for at least 2/3 of nutrients	100% of daily value*	200% of daily value*	200% of daily value*	Begin on day 1 after hospital discharge
Begin with chewable or liquid				
Progress to whole tablet/capsule as tolerated				
Avoid time-released supplements				
Avoid enteric coating				
Choose a complete formula with at least 18 mg iron, 400 μg folic acid, and containing selenium and zinc in each serving				
Avoid children's formulas that are incomplete				
May improve gastrointestinal tolerance when taken close to food intake				
May separate dosage				
Do not mix multivitamin containing iron with calcium supplement, take at least 2 hr apart				
Individual brands should be reviewed for absorption rate and bioavailability				
Specialized bariatric formulations are available				
Additional cobalamin (B_{12})				
Available forms include sublingual tablets, liquid drops, mouth spray, or nasal gel/spray				
Intramuscular injection	—	1000 μg/mo	—	Begin 0–3 mo after surgery
Oral tablet (crystalline form)	—	350–500 μg/d	—	
Supplementation after AGB and BPD/DS may be required				
Additional elemental calcium	1500 mg/d	1500– 2000 mg/d	1800– 2400 mg/d	May begin on day 1 after hospital discharge or within 1 mo after surgery
Choose a brand that contains calcium citrate and vitamin D_3				
Begin with chewable or liquid				
Progress to whole tablet/capsule as tolerated				
Split into 500–600 mg doses; be mindful of serving size on supplement label				
Space doses evenly throughout day				
Suggest a brand that contains magnesium, especially for BPD/DS				
Do not combine calcium with iron containing supplements: To maximize absorption To minimize gastrointestinal intolerance Wait ≥2 h after taking multivitamin or iron supplement				
Promote intake of dairy beverages and/or foods that are significant sources of dietary calcium in addition to recommended supplements, up to 3 servings daily				
Combined dietary and supplemental calcium intake >1700 mg/d may be required to prevent bone loss during rapid weight loss				
Additional elemental iron (above that provided by mvi)	—	Add a minimum of 18–27 mg/d elemental	Add a minimum of 18–27 mg/d elemental	Begin on day 1 after hospital discharge
Recommended for menstruating women and those at risk of anemia (total goal intake = 50-100 mg elemental iron/d)				
Begin with chewable or liquid				
Progress to tablet as tolerated				
Dosage may need to be adjusted based on biochemical markers				
No enteric coating				
Do not mix iron and calcium supplements, take ≥2 h apart				
Avoid excessive intake of tea due to tannin interaction				
Encourage foods rich in heme iron				
Vitamin C may enhance absorption of non-heme iron sources				

Table 5
Continued

Supplement	AGB	RYGB	BPD/DS	Comment
Fat-soluble vitamins	—	—	10,000 IU of vitamin A	May begin 2–4 weeks after surgery
With all procedures, higher maintenance doses may be required for those with a history of deficiency	—	—	2000 IU of vitamin D	
Water-soluble preparations of fat-soluble vitamins are available	—	—	300 μg of vitamin K	
Retinol sources of vitamin A should be used to calculate dosage				
Most supplements contain a high percentage of beta carotene which does not contribute to vitamin A toxicity				
Intake of 2000 IU Vitamin D_3 may be achieved with careful selection of multivitamin and calcium supplements				
No toxic effect known for vitamin K_1, phytonadione (phyloquinone)				
Vitamin K requirement varies with dietary sources and colonic production				
Caution with vitamin K supplementation for patients receiving coagulation therapy				
Vitamin E deficiency has been suggested but is not prevalent in published studies				
Optional B complex	1 serving/d	1 serving/d	1 serving/d	May begin on day 1 after hospital discharge
B-50 dosage				
Liquid form is available				
Avoid time released tablets				
No known risk of toxicity				
May provide additional prophylaxis against B-vitamin deficiencies, including thiamin, especially for BPD/DS procedures as water-soluble vitamins are absorbed in the proximal jejunum				
Note >1000 mg of supplemental folic acid, provided in combination with multivitamins, could mask B_{12} deficiency				

Abbreviations as in Table 4.

which, in turn, causes metabolic changes that favor fat accumulation [23–25].

Several of the B-complex vitamins, important for appropriate metabolism of carbohydrate and neural functions that regulate appetite, have been found to be deficient in some patients with morbid obesity [21,26,27]. Iron deficiencies, which would significantly hinder energy use, have been reported in nearly 50% of morbidly obese preoperative candidates [21]. Zinc and selenium deficits have been reported, as well as deficits in vitamins A, E, and C, all important antioxidants helpful in regulating energy production and various other processes of body weight management [19,26–28].

The risk of micronutrient depletion continues to be quite high, particularly after surgeries that affect the digestion and absorption of nutrients, such as Roux-en-Y gastric bypass (RYGB) and biliopancreatic diversion with or without duodenal switch (BPD/DS). RYGB increases the risk of vitamin B_{12} and other B vitamin deficits in addition to iron and calcium. BPD/DS procedures may also cause an increased risk of iron and calcium deficits, along with significant deficiencies in the fat-soluble vitamins A, D, E, and K, which are important for driving many of the biological processes that help to regulate body size [29–31]. As the rate of noncompliance with prophylactic multivitamin supplementation increases, the rate of postoperative deficiency may increase almost twofold [32].

In the past, it has been thought that the specific nutritional deficiencies commonly seen among malabsorptive procedures would not be present in patients choosing a purely restrictive surgery such as the adjustable gastric band (AGB). However, poor eating behavior, low nutrient-dense food choices, food intolerance, and a restricted portion size can contribute to potential nutrient deficiencies in these patients as well. Although the incidence of nutritional complications may be less frequent in this patient population, it would be detrimental to assume that they do not exist.

It is important for the bariatric patient to take vitamin and mineral supplements, not only to prevent adverse health conditions that can arise after surgery, but because some nutrients such as calcium can enhance weight loss and help prevent weight regain. The nutrient deficiency might be proportional to the length of the absorptive area bypassed during surgical procedures and, to a lesser extent, to the

percentage of weight lost. Iron, vitamin B_{12}, and vitamin D deficiencies, along with changes in calcium metabolism, are common after RYGB. Protein, fat-soluble vitamin, and other micronutrient deficiencies, as well as altered calcium metabolism, are most notably found after BPD/DS. Folate deficiency has been reported after AGB [33]. Thiamin deficiency is common among all surgical patients with frequent vomiting, regardless of the type of procedure performed. Because many nutritional deficiencies progress with time, patients should be monitored frequently and regularly to prevent malnutrition [29]. Reinforcement of supplement compliance at each patient follow-up is also important in the fight to enhance nutrition status and prevent nutritional complications.

Weight loss and nutrient deficits: restriction versus malabsorption

Malabsorptive procedures such as the BPD/DS are thought to cause weight loss primarily through the malabsorption of macronutrients, with as much as 25% of protein and 72% of fat malabsorbed. Such primary malabsorption results in concomitant malabsorption of micronutrients [34,35]. Vitamins and minerals relying on fat metabolism, including vitamins D, A, E, K, and zinc, may be affected when absorption is impaired [36]. The decrease in gastrointestinal transit time may also result in secondary malabsorption of a wide range of micronutrients related to bypassing the duodenum and jejunum or limited contact with the brush border secondary to a short common limb. Other micronutrient deficiency concerns reported for patients choosing a procedure with malabsorptive features include iron, calcium, vitamin B_{12}, and folate.

Nutrient deficiencies after RYGB can result from either primary or secondary malabsorption or from inadequate dietary intake. A minimal amount of macronutrient malabsorption is thought to occur. However, specific micronutrients appear to be malabsorbed postoperatively and present as deficiencies without adequate vitamin and mineral supplementation. Retrospective analyses of patients who have undergone gastric bypass have revealed predictable micronutrient deficiencies, including iron, vitamin B_{12} and folate [26,27–39]. Case reports have also shown that thiamin deficiency can develop, especially when persistent postoperative vomiting occurs [40–43].

Few studies that measure absorption after measured and quantified intake have been published. It can be hypothesized that the bypassed duodenum and proximal jejunum negatively affect nutrient assimilation. Bradley et al. [44] studied patients who had undergone total gastrectomy, the procedure from which the RYGB evolved. The researchers found that most nutritional status changes in patients were most likely due to changes in intake versus malabsorption. These balance studies were conducted in a controlled research setting; however, they did not measure pre- and postoperative changes nor include radioisotope labeling to measure absorption of key nutrients, and the subjects had undergone the procedure for reasons other than weight loss.

It is unclear whether intestinal adaptation occurs after combination procedures and to what degree it affects long-term weight maintenance and nutrition status. Adaptation is a compensatory response that follows an abrupt decrease in mucosal surface area and has been well studied in short-bowel patients who require bowel resection [45]. The process includes both anatomic and functional changes that increase the gut's digestive and absorptive capacity. Although these changes begin to take place in the early postoperative period, total adaptation may take up to three years to complete. Adaptation in gastric bypass has not been considered in absorption or metabolic studies. This consideration could be important even when determining early and late macro- and micronutrient intake recommendations. The effect of pancreatic enzyme replacement therapy on vitamin and mineral absorption in this population is also unknown.

Purely restrictive procedures such as the AGB can result in micronutrient deficiencies related to changes in dietary intake. It is commonly accepted that because no alteration is made in the absorptive pathway, malabsorption does not occur as a result of AGB procedures. However, nutrient deficits would be likely to occur because of the low nutrient intake and avoidance of nutrient-rich foods in the early months postoperatively and later possibly as a result of excessive band restriction. Food with high nutritional value such as meat and fibrous fresh fruits and vegetables might be poorly tolerated.

Literature has been published that addresses the effect of malabsorptive, restrictive, and combination surgical procedures on acute and long-term nutritional status. These studies have attempted, for the most part, to indirectly determine the surgical impact on nutrition status by the evaluation of metabolic and laboratory markers. Although some studies have included certain aspects of absorption, few have included the necessary components to evaluate absorption of a prescribed and/or monitored diet in a controlled metabolic setting.

The following sections examine the pre- and postoperative risks for nutritional deficiencies associated with RYGB, BPD/DS, and AGB. It is important for all members of the medical team to increase their awareness of the nutritional complications and challenges that lie ahead for the patient. Continued review of current research, by the medical team, regarding advances in nutrition science beyond the boundaries of the present report cannot be emphasized enough.

Thiamin (vitamin B_1)

Beriberi is a thiamin deficiency that can affect various organ systems, including the heart, gastrointestinal tract, and peripheral and central nervous systems. Although the condition is generally considered rare, a number of reported,

and possibly a much greater number of unreported or undiagnosed cases, of beriberi have occurred among individuals who have undergone surgery for morbid obesity. Early detection and prompt treatment of thiamin deficits in these individuals can help to prevent serious health consequences. If beriberi is misdiagnosed or goes undetected for even a short period, the bariatric patient can develop irreversible neuromuscular disorders, permanent defects in learning and short-term memory, coma, and even death. Because of the life-altering and potentially life-threatening nature of a thiamin deficiency, it is important that healthcare professionals in the field of bariatric surgery have knowledge of the etiology of the condition, and its signs and symptoms, treatment, and prevention.

Etiology of potential deficiency. Thiamin is a water-soluble vitamin that is absorbed in the proximal jejunum by an active (saturable, high-affinity) transport system [46,47]. In the body, thiamin is found in high concentrations in the brain, heart, muscle, liver, and kidneys. However, without regular and sufficient intake, these tissues become rapidly devoid of thiamin [46,47]. The total amount of thiamin in the body of an adult is approximately 30 mg, with a half-life of only 9–18 days. Persistent vomiting, a diet deficient in the vitamin, or the body's excessive utilization of thiamin use can result in a severe state of thiamin depletion within only a short period, producing symptoms of beriberi [46–48].

Bariatric surgery increases the risk of beriberi through exacerbation of pre-existing thiamin deficits, low nutrient intake, malabsorption, and episodes of nausea and vomiting [49–51]. Chronic or acute thiamin deficiencies in bariatric patients often present with symptoms of peripheral neuropathy or Wernicke's encephalopathy and Korsakoff's psychoses [21,48–54]. Early diagnosis of the signs and symptoms of these conditions is extremely important to prevent serious adverse health consequences. Even if treatment is initiated, recovery can be incomplete, with cognitive and/or neuromuscular impairments persisting long term or permanently.

Signs, symptoms, and treatment of deficiency (see Appendix, Table A1)

Preoperative risk. The risk of the development of beriberi after bariatric surgery is far greater for individuals presenting for surgery with low thiamin levels. Investigators at the Cleveland Clinic of Florida reported that 15% of their preoperative bariatric patients had deficiencies in thiamin before surgery [52]. A study by Flancbaum et al. [21] similarly found that 29% of their preoperative patients had low thiamin levels. Data obtained by that study also showed a significant difference between ethnicity and preoperative thiamin levels. Although only 6.7% of whites presented with thiamin deficiencies before surgery, nearly one third (31%) of African Americans and almost one half (47%) of Hispanics had thiamin deficits. The results of these studies suggested a need for preoperative thiamin testing, as well as thiamin repletion by diet or supplementation to reduce the risk of "bariatric" beriberi postoperatively.

Postoperative risk. Beriberi has been observed after gastric restrictive and malabsorptive procedures. A number of cases of Wernicke-Korsakoff syndrome (WKS), as well as peripheral neuropathy, have been reported for patients having undergone vertical banded gastroplasty [53–61]. A few incidences of WKS have also been reported after AGB [43,62,63]. Additionally, reports of thiamin deficiencies and WKS after RYGB [40,41,64–73] and several cases of WKS and neuropathy in patients who had undergone BPD [74] have been published. Many more cases of WKS are believed to have occurred with bariatric procedures that have either not been reported or have been misdiagnosed because of limited knowledge regarding the signs and symptoms of acute or severe thiamin deficits. Because many foods are fortified with thiamin, beriberi has been nearly eradicated throughout the world, except for patients with severe alcoholism, severe vomiting during pregnancy (hyperemesis gravidarium), or those malnourished and starved. For this reason, few healthcare professionals, until recently, have had a patient present with beriberi.

According to the published reports of thiamin deficits after bariatric procedures, most patients develop such deficiencies in the early postoperative months after an episode of intractable vomiting. Nausea and vomiting are relatively common after all bariatric procedures early in the postoperative period. Thiamin stores in the body are small and maintenance of appropriate thiamin levels requires daily replenishment. A deficiency of thiamin for only a couple of weeks or less, caused by persistent vomiting, can deplete thiamin stores. Symptoms of WKS were reported in a RYGB patient after only two weeks of persistent vomiting [67].

Bariatric beriberi can also develop in postoperative patients who are given infusate containing dextrose without thiamin and other vitamins, which is often the case for patients in critical care units, postoperative patients with complications interfering with the ingestion of food, or patients dehydrated from persistent vomiting. Malnutrition caused by a lack of appetite and dietary intake postoperatively also contributes to bariatric beriberi, as does noncompliance in taking postoperative vitamin supplements.

Although most cases of beriberi occur in the early postoperative periods, cases of patients with severe thiamin deficiency more than one year after surgery have been reported. One study reported WKS in association with alcohol abuse 13 years after RYGB [75]. Other conditions contributing to late cases of bariatric beriberi include a thiamin-poor diet, a diet high in carbohydrates, anorexia, and bulimia [46–48].

Suggested supplementation. Because of the greater likeli-

hood of low dietary thiamin intake, patients should be supplemented with thiamin. This is usually accomplished through daily intake of a multivitamin. Most multivitamins contain thiamin at 100% of the daily value. Patients having episodes of nausea and vomiting and those who are anorectic might require sublingual, intramuscular, or intravenous thiamin to avoid depletion of thiamin stores and beriberi. Caution should be used when infusing bariatric patients with solutions containing dextrose without additional vitamins and thiamin, because an increase in glucose utilization without additional thiamin can deplete thiamin stores [76].

Thiamin deficiency in bariatric patients is treated with thiamin, together with other B-complex vitamins and magnesium, for maximal thiamin absorption and appropriate neurologic function [46–48]. Early symptoms of neuropathy can often be resolved by providing the patient with oral thiamin doses of 20–30 mg/d until symptoms disappear. For more advanced signs of neuropathy or for individuals with protracted vomiting, 50–100 mg/d of intravenous or intramuscular thiamin may be necessary for resolution or improvement of symptoms or for the prevention of such. Patients with WKS generally require ≥100 mg thiamin administered intravenously for several days or longer, followed by intramuscular thiamin or high oral doses until symptoms have resolved or significantly improved. This can require months to years. Some patients might have to take thiamin for life to prevent the reoccurrence of neuropathy.

Vitamin B_{12} and folate

Vitamin B_{12} (cobalamin) and folate (folic acid) are both involved in the maturation of red blood cells and are commonly discussed in the literature together. Over time, a deficiency in either vitamin B_{12} or folate can lead to macrocytic anemia, a condition characterized by the production of fewer, but larger, red blood cells and a decreased ability to carry oxygen. Most (95%) cases of megaloblastic anemia (characterized by large, immature, abnormal, undifferentiated red blood cells in bone marrow) are attributed to vitamin B_{12} or folate deficiency [77].

Vitamin B_{12}

Etiology of potential deficiency. RYGB patients have both incomplete digestion and release of vitamin B_{12} from protein foods. With a significant decrease in hydrochloric acid, pepsinogen is not converted into pepsin, which is necessary for the release of vitamin B_{12} from protein [78]. Because AGB patients have an artificial restriction, yet complete use of the stomach, and BPD patients do not have as great a restriction in stomach capacity and parietal cells as RYGB patients, the reduction in hydrochloric acid and subsequent vitamin B_{12} deficiency is not as prevalent with these two procedures.

Intrinsic factor (IF) is produced by the parietal cells of the stomach and in certain conditions (e.g., atrophic gastritis, resected small bowel, elderly patients) can be impaired, causing an IF deficiency. Subsequent vitamin B_{12} deficiency (pernicious anemia) occurs without IF production or use, because IF is needed to absorb vitamin B_{12} in the terminal ileum [77]. Factors that increase the risk of vitamin B_{12} deficiency relevant to bariatric surgery include the following:

- An inability to release protein-bound vitamin B_{12} from food, particularly in hypochlorhydria and atrophic gastritis
- Malabsorption due to inadequate IF in pernicious anemia
- Gastrectomy and gastric bypass
- Resection or disease of terminal ileum
- Long-term vegan diet
- Medications, such as neomycin, metformin, colchicines, medications used in the management of bowel inflammation and gastroesophageal reflux and ulcers (e.g., proton pump inhibitors) and anticonvulsant agents [79]

Cobalamin stores are known to exist for long periods (3–5 yr) and are dependent on dietary repletion and daily depletion. However, gastric bypass patients have both a decreased production of stomach acid and a decreased availability of IF; thus, a vitamin B_{12} deficiency could develop without appropriate supplementation. Because the typical absorption pathway cannot be relied on, the surgical weight loss patient must rely on passive absorption of B_{12}, which occurs independent of IF.

Signs, symptoms, and treatment of deficiency (see Appendix, Table A3)

Preoperative risk. Several medications common to preoperative bariatric patients have been noted to affect preoperative vitamin B_{12} absorption and stores. Of patients taking metformin, 10–30% present with reduced vitamin B_{12} absorption [80]. Additionally, patients with obesity have a high incidence of gastroesophageal reflux disease, for which they take proton pump inhibitors, thus increasing the potential to develop a vitamin B_{12} deficiency.

Flancbaum et al. [21] conducted a retrospective study of 379 (320 women and 59 men) pre-operative patients. Vitamin B_{12} deficiency was reported as negative in all patients of various ethnic backgrounds [21]. No clinical criteria or symptoms for vitamin B_{12} deficiency were noted. In the general population, 5–10% present with neurologic symptoms with vitamin B_{12} levels of 200–400 pg/mL. Among preoperative gastric bypass patients, Madan et al. [27] found that 13% ($n = 59$) of patients were deficient in vitamin B_{12}. In a comparison of patients presenting for either RYGB or BPD, Skroubis et al. [81] recently reported that preoperative vitamin B_{12} levels were low-normal in both groups. It would be prudent to screen for, and treat, IF deficiency and/or vitamin B_{12} deficiency in all patients preoperatively,

but it is essential for RYGB patients so as not to hasten the development of a potential postoperative deficiency.

Postoperative risk. Vitamin B_{12} deficiency has been frequently reported after RYGB. Schilling et al. [82] estimated the prevalence of vitamin B_{12} deficiency to be 12–33%. Other researchers have suggested a much greater prevalence of B_{12} deficiency in up to 75% of postoperative RYGB patients; however, most reports have cited approximately 35% of postoperative RYGB patients as vitamin B_{12} deficient [82–87]. Brolin et al. [87] reported that low levels of vitamin B_{12} might be seen as soon as six months after bariatric surgery, but most often occurring more than one year postoperatively as liver stores become depleted. Skroubis et al. [88] predicted that the deficiency will most likely occur 7 months after RYGB and 7.9 months after BPD/DS, although their research did not consider compromised preoperative status and its correlation to postoperative deficiency. After the first postoperative year, the prevalence of vitamin B_{12} deficiency appears to increase yearly in RYGB patients [89].

Although the body's storage of vitamin B_{12} is significant (~2000 μg) compared with daily needs (2.4 μg/d), this particular deficiency has been found within 1–9 years of gastric bypass surgery [29]. Brolin et al. [84] reported that one third of RYGB patients are deficient at four years postoperatively. However, non-surgical variables were not explored, and many patients might have had preoperative values near the lower end of the normal range. Ocon Breton et al. [90] compared micronutrient deficiencies among two-year postoperative BPD/DS and RYGB patients and found that all nutritional deficiencies were more common among BPD/DS patients, except for vitamin B_{12}, for which the deficiency was more common among the RYGB patients studied. A lack of B_{12} deficiency among BPD/DS patients might result from a better tolerance of animal proteins in a larger pouch, greater pepsin/gastric acid production to release protein-bound B_{12}, and increased availability and interaction of IF with the pouch contents.

Experts have noted the significance of subclinical deficiency in the low-normal cobalamin range in nongastric bypass patients who do not exhibit clinical evidence of deficiency. The methylmalonic acid (MMA) assay is the preferred marker of B_{12} status because metabolic changes often precede low B_{12} levels in the progression to deficiency. Serum B_{12} assays may miss as much as 25–30% of B_{12} deficiencies, making them less reliable than the MMA assay [91]. It has been suggested that early signs of vitamin B_{12} deficiency can be detected if the serum levels of both MMA and homocysteine are measured [91]. Vitamin B_{12} deficiency after RYGB has been associated with megaloblastic anemia [92]. Some vitamin B_{12}-deficient patients develop significant symptoms, such as polyneuropathy, paresthesia, and permanent neural impairment. On occasion, some patients may experience extreme delusions, hallucinations, and, even, overt psychosis [93].

Suggested supplementation. Because of the frequent lack of symptoms of vitamin B_{12} deficiency, the suggested diligence in following up or treating these values among those asymptomatic patients has been questioned. The decision not to supplement or routinely screen patients for B_{12} deficiency should be examined very carefully, given the risk of irreversible neurologic damage if vitamin B_{12} goes untreated for long periods. At least one case report has been published of an exclusively breastfed infant with vitamin B_{12} deficiency who was born of an asymptomatic mother who had undergone gastric bypass surgery [94].

Deficiency of vitamin B_{12} is typically defined at levels $<$200 pg/mL. However, about 50% of patients with obvious signs and symptoms of deficiency have normal vitamin B_{12} levels [31]. Kaplan et al. [95] reported that vitamin B_{12} deficiency usually resolves after several weeks of treatment with 700–2000 μg/wk. Rhode et al. [96] found that a dosage of 350–600 μg/d of oral B_{12} prevented vitamin B_{12} deficiency in 95% of patients and an oral dose of 500 μg/d was sufficient to overcome an existing deficiency as reported by Brolin et al. [87] in a similar study. Therefore, supplementation of RYGB patients with 350–500 μg/d may prevent most postoperative vitamin B_{12} deficiency.

While most vitamin B_{12} in normal adults is absorbed in the ileum in the presence of IF, approximately 1% of supplemented B_{12} will be absorbed passively (by diffusion) along the entire length of the (non-bypassed) intestine by surgical weight loss patients given a high-dose oral supplement [97]. Thus, the consumption of 350–500 μg yields a 3.5–5.0-μg absorption, which is greater than the daily requirement. Although the use of monthly intramuscular injections or a weekly oral dose of vitamin B_{12} is common among practices, it relies on patient compliance. Practitioners should assess the patient's preference and the potential for compliance when considering a daily, weekly, or monthly regimen of B_{12} supplementation. In addition to oral supplements or intramuscular injections, nasal sprays and sublingual sources of vitamin B_{12} are also available. Patients should be monitored closely for their lifetime, because severe anemia can develop with or without supplementation [98].

Folate

Etiology of potential deficiency. Factors that increase the risk of folic acid deficiency relevant to bariatric surgery include the following:

- Inadequate dietary intake
- Noncompliance with multivitamin supplementation
- Malabsorption
- Medications (anticonvulsants, oral contraceptives, and cancer treating agents) [79].

Folic acid stores can be depleted within a few months postoperatively unless replenished by a multivitamin supplement and dietary sources (i.e., green leafy vegetables, fruits, organ meats, liver, dried yeast, and fortified grain products).

Signs, symptoms, and treatment of deficiency (see Appendix, Table A4)

Preoperative risk. The goals of the 1998 Food and Drug Administration policy, requiring all enriched grain products to be fortified with folate, included increasing the average American diet by 100 μg folate daily and decreasing the rate of neural tube defects in childbearing women [99]. Bentley et al. [100] reported that the proportion of women aged 15–44 years old (in the general population) who meet the recommended dietary intake of 400 μg/d folate varies between 23% and 33%. With the increasing popularity of high protein/low carbohydrate diets, one cannot assume that preoperative patients consume dietary sources of folate through fortified grain products, fruits, and vegetables. Boylan et al. [26] found folate deficiencies preoperatively in 56% of RYGB patients studied.

Postoperative risk. Although it has been observed that folate deficiency after RYGB surgery is less common than B_{12} deficiency and is thought to occur because of decreased dietary or multivitamin supplement intake, low serum folate levels have been cited from 6% to 65% among RYGB patients [26,86,101]. Additionally, folate deficiencies have occurred postoperatively, even with supplementation. Boylan et al. [26] found that 47% (n = 17) of RYGB patients had low folate levels six months postoperatively and 41% (n = 17) had low levels at one year. This deficiency occurred despite patient adherence to taking a multivitamin supplement that contained at least the daily value for folate, 400 μg [26]. Postoperative bariatric patients with rapid weight loss might have an increased risk of micronutrient deficiencies. MacLean et al. [86] reported that 65% of postoperative patients had low folate levels. These same patients exhibited additional B vitamin deficiencies: 24% vitamin B_{12} and 50% thiamin [86]. An increased risk of deficiency has also been noted among AGB patients, possibly because of decreased folate intake. Gasteyger et al. [33] found a significant decrease in serum folate levels (44.1%) between the baseline and 24-month postoperative measurements.

Dixon and O'Brien [101] reported elevated serum homocysteine levels in patients after bariatric surgery, regardless of the type of procedure (restrictive or malabsorptive). Elevated homocysteine levels can indicate, not only low folate levels and a greater risk of neural tube defects, but can also be indicative of an independent risk factor for heart disease and/or oxidative stress in the nonbariatric population [102]. However, unlike iron and vitamin B_{12}, the folate contained in the multivitamin supplementation essentially corrects the deficiency in the vast majority of postoperative bariatric patients [103]. Therefore, persistent folate deficiency might indicate a patient's lack of compliance with the prescribed vitamin protocol [32].

It is common knowledge that folic acid deficiency among pregnant women has been associated with a greater risk of neural tube defects in newborns. Consistent supplementation and monitoring among women of child-bearing age, including pre- and postoperative bariatric patients is vital in an effort to prevent the possibility of neural tube defects in the developing fetus.

Because folate does not affect the myelin of nerves, neurologic damage is not as common with folate, such as is the case for vitamin B_{12} deficiency. In contrast, patients with folate deficiency often present with forgetfulness, irritability, hostility, and even paranoid behaviors [29]. Similar to that evidenced with vitamin B_{12}, most postoperative RYGB patients who are folate deficient are asymptomatic or have subclinical symptoms; therefore, these deficient states may not be easily identified.

Suggested supplementation. Even though folate absorption occurs preferentially in the proximal portion of the small intestine, it can occur along the entire length of the small bowel with postoperative adaptation. Therefore, it is generally agreed that folate deficiency is corrected with 1000 mg/d folic acid [32] and is preventable with supplementation that provides 200% of the daily value (800 μg). This level can also benefit the fetus in a female patient unaware of her postoperative pregnancy. Folate supplementation >1000 mg/d has not been recommended because of the potential for masking vitamin B_{12} deficiency. Carmel et al. [104] suggested that homocysteine is the most sensitive marker of folic acid status, in conjunction with erythrocyte folate. Although folic acid deficiency could potentially occur among postoperative bariatric surgery patients, it has not been seen widely in recent studies, especially when patients have been compliant with postoperative multivitamin supplementation. Therefore, it is imperative to closely follow folic acid both pre- and postoperatively, especially in those patients suspected to be noncompliant with their multivitamin supplementation.

Iron

Much of the iron and surgical weight loss research conducted to date has been generated from a limited number of surgeons and scientists; however, the data have consistently pointed toward the risk of iron deficiency and anemia after bariatric procedures. Iron deficiency is defined as a decrease in the total iron body content. Iron deficiency anemia occurs when erythropoiesis is impaired as a result of the lack of iron stores. In the absence of anemia, iron deficiency is usually asymptomatic. Fatigue and a diminished capacity to exercise, however, are common symptoms of anemia.

Etiology of potential deficiency. As with other vitamins and minerals, the possible reasons for iron deficiency related to surgical weight loss are multifactorial and not fully explained in the literature. Iron deficiency can be associated with malabsorptive procedures, combination procedures, and AGB, although the etiology of the deficiency is likely to be unique with each procedure. Although the absorption of iron can occur throughout the small intestine, it is most efficient in the duodenum and proximal jejunum, which is bypassed after RYGB, leading to decreased overall absorption. Important receptors in the apical membrane of the enterocyte, including duodenal cytochrome b are involved in the reduction of ferric iron and subsequent transporting of iron into the cell [105]. The effect of bariatric surgery on these transporters has not been defined, but it is likely that, at least initially, fewer receptors are available to transport iron. With malabsorptive procedures, there is likely a decrease in transit time during which dietary iron has less contact with the lumen, in addition to the bypass of the duodenum, resulting in decreased absorption. In RYGB procedures, decreased absorption is coupled with reduced dietary intake of iron-rich foods, such as meats, enriched grains, and vegetables. Those patients who are able to tolerate meat have been shown to have a lower risk of iron deficiency [38]; however, patient tolerance varies considerably and red meat, in particular, is often cited as a poorly tolerated food source. Iron-fortified grain products are often limited because of the emphasis on protein-rich foods and restricted carbohydrate intake. Finally, decreased hydrochloric acid production in the stomach after RYGB [106] can affect the reduction of iron from the ferric (Fe^{3+}) to the absorbable ferrous state (Fe^{2+}). Notably, vitamin C, found in both dietary and supplemental sources, can enhance iron absorption of non-heme iron, making it a worthy recommendation for inclusion in the postoperative diet [39].

Signs, symptoms, and treatment of deficiency (see Appendix, Table A5)

Preoperative risk. The prevalence of iron deficiency in the United States is well documented. Premenopausal women are at increased risk of deficiency because of menstrual losses, especially when oral contraceptives are not used. The use of oral contraceptives alone decreases blood loss from menstruation by as much as 60% and decreases the recommended daily allowance to 11 mg/d (instead of 15 mg/d) [105]. Women of child-bearing age comprise a large percentage (>80%) of the bariatric surgery cases performed each year. The propensity of this population to be at risk of iron deficiency and related anemia is relatively independent of bariatric surgery and, thus, should be evaluated before the procedure to establish baseline measures of iron status and to treat a deficiency, if indicated.

Women, however, are not the only group at risk of iron deficiency, obese men and younger (<25 yr) surgical candidates have also been found to be iron deficient preoperatively. In one retrospective study of consecutive cases (n = 379), 44% of bariatric surgery candidates were iron deficient [21]. In this report, men were more likely than women to be anemic (40.7% versus 19.1%), as determined by abnormal hemoglobin values. Women, however, were more likely to have abnormal ferritin levels. Anemia and iron deficiency were more common in patients <25 years of age compared with those >60 years of age. Of these, 79% of younger patients versus 42% of older patients presented with preoperative iron deficiency, as determined by low serum iron values. Another study found iron levels to be abnormal in 16% of patients, despite a low proportion of patients for whom data were available (64%). These studies are in contrast to earlier reports of limited preoperative iron deficiency [32,107].

Postoperative risk. Iron deficiency is common after gastric bypass surgery, with reports of deficiency ranging from 20% to 49% [32,98,107,108]. Up to 51% of female patients in one series were iron deficient, confirming the high-risk nature of this population [87]. Among patients with super obesity undergoing RYGB with varying limb length, iron deficiency has been identified in 49–52% and anemia in 35–74% of subjects up to 3 years postoperatively [84].

In one study, the prevalence of iron deficiency was similar among RYGB and BPD subjects. Skroubis et al. [88] followed both RYGB and BPD subjects for five years. The ferritin levels at two years were significantly different between the two groups, with 38% of RYGB versus 15% of BPD subjects having low levels. The percentage of patients included in the follow-up data decreased considerably in both groups and must be taken into account. Although data for 70 RYGB subjects and 60 BPD subjects were recorded at one year, only eight and one subject remained in the groups, respectively, at five years. It is difficult to comment on the iron status and other clinical parameters, given the weight of the data. For example, the investigators reported that 100% of BPD subjects were deficient in hemoglobin, iron, and ferritin. However, only one subject remained, making the later results nongeneralizable. Additional studies investigating iron absorption and status are warranted for BPD/DS procedures.

Menstruating women and adolescents who undergo bariatric surgery might require additional iron. One randomized study of premenopausal RYGB women (n = 56) demonstrated that 320 mg of supplemental oral iron (ferrous sulfate) given twice daily prevented the development of iron deficiency but did not protect against the development of anemia [107]. Notably, those patients who developed anemia were not regularly taking their iron supplements (≥5 times/wk) during the period preceding diagnosis. In that study, a significant correlation was found between the resolution of iron deficiency and adhering to the prescribed oral iron supplement regimen. Ferritin levels had decreased at two years in the untreated group, but those in

the supplemented group remained within the normal range, suggesting dietary intake and one multivitamin (containing 18 mg iron) might be inadequate to maintain iron stores in RYGB patients.

A significant decline in the iron status of surgical weight loss patients can occur over time. In one series, 63% of subjects followed for two years after RYGB developed nutritional deficiencies, including iron, vitamin B_{12}, and folate [32]. In this study, those subjects who were compliant with a daily multivitamin could not prevent iron deficiency. Two thirds (67%) of those who did develop iron deficiency later became anemic. Their findings suggest that the amount of iron in a standard multivitamin alone is not adequate to prevent deficiency. Similarly, Avinoah et al. [38] found that at 6.7 years postoperatively, weight loss (56% ± 22% excess body weight) had been relatively stable for the preceding five years (n = 200); however, the iron saturation, hemoglobin, and mean corpuscular volume values had declined progressively and significantly.

Recently, case reports of a re-emergence of pica after RYGB might also be linked to iron deficiency [109]. Pica is the ingestion of nonfood substances and, historically, has been common in some parts of the world during pregnancy, with gastrointestinal blood loss, and in those with sickle cell disease. Although perhaps less detrimental than consuming starch or clay, the consumption of ice (pagophagia) is also a form of pica that might not be routinely identified. In the aforementioned case series, iron treatment was noted to resolve pica within eight weeks in five women after RYGB.

Screening for iron status can include the use of serum ferritin levels. Such levels, however, should not be used to diagnose a deficiency. Ferritin is an acute-phase reactant and can fluctuate with age, inflammation, and infection—including the common cold. Measuring serum iron, along with the total iron binding capacity is preferred to determine iron status. Hemoglobin and hematocrit changes reflect late iron-deficient anemia and are less valuable in identifying early anemia but are frequently used in the bariatric data to define anemia because of their widespread clinical availability. Serum iron, along with total iron binding capacity, should be measured at 6 months postoperatively because a deficiency can occur rapidly, and then annually, in addition to analyzing the complete blood count.

Suggested supplementation. In addition to the iron found in two multivitamins, menstruating women and adolescents of both sexes may require additional supplementation to achieve a total oral intake of 50–100 mg of elemental iron daily, although the long-term efficacy of such prophylactic treatment is unknown [87,107]. This amount of oral iron can be achieved by the addition of ferrous sulfate or other preparations, such as ferrous fumarate, gluconate, polysaccharide, or iron protein succinylate forms. Those women who use oral contraceptives have significantly less blood loss and therefore may have lower requirements for supplemental iron. This is an important consideration during the clinical interview. The use of two complete multivitamins, collectively providing 36 mg of iron (typically ferrous fumarate) is customary for low-risk patients, including men and postmenopausal women. A history of anemia or a change in laboratory values may indicate the need for additional supplementation, in conjunction with age, sex, and reproductive considerations. Treatment of iron deficiency requires additional supplementation, as noted in Appendix, Table A5.

Calcium and vitamin D

Etiology of potential deficiency. Calcium is absorbed preferentially in the duodenum and proximal jejunum, and its absorption is facilitated by vitamin D in an acid environment. Vitamin D is absorbed preferentially in the jejunum and ileum. As the malabsorptive effects of surgical procedures increase, so does the likelihood of fat-soluble vitamin malabsorption related to the bypassing of the stomach, absorption sites of the intestine, and poor mixing of bile salts. Decreased dietary intake of calcium and vitamin D-rich foods, related to intolerance, can also increase the risk of deficiency after all surgical procedures.

Low vitamin D levels are associated with a decrease in dietary calcium absorption but are not always accompanied by a reduction in serum calcium. As blood calcium ions decrease, parathyroid hormone levels increase. Secondary hyperparathyroidism allows the kidney and liver to convert 7-dehydroxycholecalciferol into the active form of vitamin D, 1,25 dihydroxycholecalciferol, and stimulates the intestine to increase absorption of calcium. If dietary calcium is not available or intestinal absorption is impaired by vitamin D deficiency, calcium homeostasis is maintained by increases in bone resorption and in conservation of calcium by way of the kidneys. Therefore, calcium deficiency (low serum calcium) would not be expected until osteoporosis has severely depleted the skeleton of calcium stores.

Signs, symptoms, and treatment of deficiency (see Appendix, Tables A6 and A7)

Preoperative risk. Although vitamin D deficiency and increased bone remodeling can be expected after malabsorptive procedures, it is very important to consider the prevalence of these conditions preoperatively. Buffington et al. [19] found that 62% of women (n = 60) had 25-hydroxyvitamin D [25(OH)D] levels at less than normal values, confirming the hypothesis that vitamin D deficiency might be associated with morbid obesity. A negative correlation between BMI and vitamin D levels was noted, suggesting that individuals with a larger BMI might be more prone to develop vitamin D deficiency [19]. In addition, a positive correlation exists between a greater BMI and increased parathyroid hormone [110]. Recently, Flancbaum et al. [21] completed a retrospective analysis of 379 preoperative gastric bypass patients and found

68.1% were deficient in 25(OH)D. Vitamin D deficiency was less common among Hispanics (56.4%) than among whites (78.8%) and African Americans (70.4%) [21]. Ybarra et al. [20] also found 80% of a screened population of patients presented with similar patterns of low vitamin D levels and secondary hyperparathyroidism.

Several mechanisms have been suggested to explain the cause of the increased risk of vitamin D deficiency among preoperative obese individuals. They include the decreased bioavailability of vitamin D because of enhanced uptake and clearance by adipose tissue, 1,25-dihydroxyvitamin D enhancement and negative feedback control on the hepatic synthesis of 25(OH)D, underexposure to solar ultraviolet radiation, and malabsorption [111–114], with the major cause being decreased availability of vitamin D secondary to the preoperative fat mass.

Considering the evidence from both observational studies and clinical trials that calcium malnutrition and hypovitaminosis D are predisposing conditions for various common chronic diseases, the need for the early identification of a deficiency is paramount to protect the preoperative patient with obesity from serious complications and adverse effects. In addition to skeletal disorders, calcium and vitamin D deficits increase the risk of malignancies (in particular, of the colon, breast, and prostate gland), of chronic inflammatory and autoimmune disease (e.g., diabetes mellitus type 1, inflammatory bowel disease, multiple sclerosis, rheumatoid arthritis), of metabolic disorders (metabolic syndrome and hypertension), as well as peripheral vascular disease [115,116].

Postoperative risk. An increased long-term risk of metabolic bone disease has been well documented after BPD/DS and RYGB. Slater et al. [36] followed BPD/DS patients four years postoperatively and found vitamin D deficiency in 63%, hypocalcemia in 48%, and a corresponding increase in parathyroid hormone (PTH) in 69% of patients. Another series found, at a median follow-up of 32 months, that 25.9% of patients were hypokalemic, 50% had low vitamin D, and 63.1% had elevated PTH, despite taking multivitamins [117]. The bypass of the duodenum and a shorter common channel in BPD/DS patients increases the risk of developing hyperparathyroidism. This could be related to reduced calcium and 25(OH)D absorption [118]. Similarly, the increased incidence of vitamin D deficiency and secondary hyperparathyroidism has also been found in RYGB patients related to the bypass of the duodenum [119].

Goode et al. [120], using urinary markers consistent with bone degradation, found that postmenopausal women showed evidence of secondary hyperparathyroidism, elevated bone resorption, and patterns of bone loss after RYGB. Dietary supplementation with vitamin D and 1200 mg calcium per day did not affect these measures, indicating the need for greater supplementation [120]. Secondary elevated PTH and urinary bone marker levels, consistent with increased bone turnover postoperatively, were also found in one small short-term series (n = 15) followed by Coates et al. [121]. The subjects were found to have significant changes in total hip, trochanter, and total body bone mineral density as a result of increased bone resorption beginning as early as three months postoperatively. These changes occurred despite increased dietary intake of calcium and vitamin D. The significance of elevated PTH is clearly an important area of investigation in postoperative patients because high PTH levels could be an early sign of bone disease in some patients. A decrease in serum estradiol levels has also been found to alter calcium metabolism after RYGB-induced weight loss [122].

Fewer studies have reported vitamin D/calcium deficits and elevated PTH in AGB patient. Pugnale et al. [123] and Giusti et al. [124] followed premenopausal women undergoing gastric banding one and two years postoperatively and found no significant decrease in vitamin D, serum calcium, or PTH levels; however, serum telopeptide-C increased by 100%, indicative of an increase in bone turnover. Furthermore, the bone mass density and bone mineral concentration decreased, especially in the femoral neck, as weight loss occurred. Despite the absence of secondary hyperparathyroidism after gastric banding, biochemical bone markers have continued to show a negative remodeling balance characterized by an increase in bone resorption [123,124]. Bone loss has also been reported in a series of men and women undergoing vertical banded gastroplasty or medical weight loss therapy. The investigators attributed the reduced estradiol levels in female patients studied to explain the increased bone turnover [125]. Reidt et al. [126] suggested that an increase in bone turnover with weight loss could occur from a decrease in estradiol secondary to a loss of adipose tissue or to other conditions associated with weight loss, such as an increase in PTH, an increase in cortisol, or to a decrease in weight-bearing bone stress. These investigators found that increasing the dosage of calcium citrate from 1000 mg to 1700 mg/d (including 400 IU vitamin D), was able to reduce bone loss with weight loss, but not stop it [126]. If left undiagnosed and untreated, it would only be a matter of time before the vitamin D/calcium deficits and hormonal mechanisms such as secondary hyperparathyroidism would result in osteopenia, osteoporosis, and ultimately osteomalacia [127].

Calcium citrate versus calcium carbonate. Supplementation with calcium and vitamin D during all weight loss modalities is critical to preventing bone resorption [121]. The preferred form of calcium supplementation is an area of debate in current clinical practice. In a low acid environment, such as occurs with the negligible secretion of acid by the pouch created with gastric bypass, absorption of calcium carbonate is poor [128]. Studies have found in nongastric bypass postmenopausal female subjects that calcium citrate (not calcium carbonate) decreased markers of bone resorp-

tion and did not increase PTH or calcium excretion [129]. A meta-analysis of calcium bioavailability suggested that calcium citrate is more effectively absorbed than calcium carbonate by 22–27%, regardless of whether it was taken on an empty stomach or with meals [130]. These findings suggest that it is appropriate to advise calcium citrate supplementation, despite the limited evidence, because of the potential benefit without additional risk.

Patients who have a history of requiring antiseizure or glucocorticoid medications, as well as long-term use of thyroid hormone, methotrexate, heparin, or cholestyramine, could also have an increased risk of metabolic bone disease; therefore, close monitoring of these individuals is also appropriate [131].

Suggested supplementation. In view of the increased risks of poor bone health in the patient with morbid obesity, all surgical candidates should be screened for vitamin D deficiency and bone density abnormalities preoperatively. If vitamin D deficiency is present, a suggested dose for correction is 50,000 IU ergocalciferol taken orally, once weekly, for 8 weeks [21]. It is no longer acceptable to assume that postoperative prophylaxis calcium and vitamin D supplementation will prevent an increase in bone turnover. Therefore, life-long screening and aggressive treatment to improve bone health needs to become integrated into postoperative patient care protocols.

It appears that 1200 mg of daily calcium supplementation and the 400–800 IU of vitamin D contained in standard multivitamins might not provide adequate protection for postoperative patients against an increase in PTH and bone resorption [120,121,132]. Riedt et al. [122] estimated that ≥50% of RYGB postoperative, postmenopausal women would have a negative calcium balance even with 1200 mg of calcium intake daily. Another study reported that increasing the dosage of vitamin D to 1600–2000 IU/d produced no appreciable change in PTH, 25(OH)D, corrected calcium, or alkaline phosphatase levels for BPD/DS patients [118]. Increasing calcium citrate to 1700 mg/d (with 400 IU vitamin D) during caloric restriction was able to ameliorate bone loss in nonoperative postmenopausal women, but did not prevent it [125]. Some investigators have suggested that although increased calcium intake can positively influence bone turnover after RYGB, it is unlikely that additional calcium supplementation beyond current recommendations (approximately 1500 mg/d for postoperative, postmenopausal patients), would attenuate the elevated levels of bone resorption [122]. It remains to be seen with additional research whether more aggressive therapy, including greater doses of calcium and vitamin D, can correct secondary hyperparathyroidism or whether perhaps a threshold of supplementation exists.

Patient supplementation compliance is often a common concern among healthcare practitioners. Weight loss can help to eliminate many co-morbid conditions associated with obesity; however, without calcium and vitamin D supplementation, it can be at the cost of bone health. The benefits of vitamin/mineral compliance and lifestyle changes offering protection need to be frequently reinforced. The promotion of physical activity such as weight-bearing exercise, increasing the dietary intake of calcium and vitamin D-rich foods, moderate sun exposure, smoking cessation, and reducing one's intake of alcohol, caffeine, and phosphoric acid are additional measures the patient can take in the pursuit of strong and healthy bones [133].

Vitamins A, E, K, and zinc

Etiology of potential deficiency. The risk of fat-soluble vitamin deficiencies among bariatric surgery patients, such as vitamins A, E, and K, has been elucidated, particularly among patients who have undergone BPD surgery [34–36,134]. BPD/DS surgery results in decreased intestinal dietary fat absorption caused by the delay in the mixing of gastric and pancreatic enzymes with bile until the final 50–100 cm of the ileum, also known as the common channel [36]. BPD surgery has been shown to decrease fat absorption by 72% [34]. It would therefore seem plausible that, particularly among postoperative BPD patients, fat-soluble vitamin absorption would be at risk. Researchers have also discovered fat-soluble vitamin deficiencies among RYGB and AGB patients [26,30,84,135] which might not have been previously suspected.

Normal absorption of fat-soluble vitamins occurs passively in the upper small intestine. The digestion of dietary fat and subsequent micellation of triacylglycerides (triglycerides) is associated with fat-soluble vitamin absorption. Additionally, the transport of fat-soluble vitamins to tissues is reliant on lipid components such as chylomicrons and lipoproteins. The changes in fat digestion produced by surgical weight loss procedures consequently alters the digestion, absorption, and transport of fat-soluble vitamins.

Signs, symptoms, and treatment of deficiency (see Appendix, Tables A8, A9, A10, A11)

Vitamin A

Slater et al. [36] evaluated the prevalence of serum fat-soluble vitamin deficiencies after malabsorptive surgery. Of the 170 subjects in their study who returned for follow up, the incidence of vitamin A deficiency was 52% at one year after BPD (n = 46) and increased annually to 69% (n = 27) by postoperative year four.

In a study comparing the nutritional consequences of conventional therapy for obesity, AGB, and RYGB, Ledoux et al. [135] found that the serum concentrations of vitamin A markedly decreased in the RYGB group (n = 40) compared with the conventional treatment group (n = 110) and the AGB group (n = 51). The prevalence of vitamin A deficiency was 52.5% in the RYGB group compared with 25.5% in the AGB group ($P < 0.01$). The follow-up period for the RYGB group

was approximately 16 ± 9 months and for the AGB group was 30 ± 12 months, a significant difference ($P < 0.001$).

Madan et al. [27] investigated vitamin and trace minerals before and after RYGB in 100 subjects and found several deficiencies before surgery and up to one year postoperatively, including vitamin B_{12}, vitamin D, iron, selenium, vitamin A, zinc, and folic acid. Vitamin A was low among 7% of preoperative patients (n = 55) and 17% (n = 30) at one year postoperatively. However, at the three- and six-month postoperative points, the subjects (n = 55) had reached a peak deficiency of 28% at six months, but only 17% were deficient at one year. The number of subjects followed up at one year is considerably less than the previous time points and the data must be viewed in this context.

Brolin et al. [84] reported, in a series comparing short and distal RYGB, that shorter limb gastric bypass patients with super obesity typically were not monitored for fat-soluble vitamin deficiency and deficiencies were not noted in this group. However, in the distal RYGB group (n = 39), 10% of subjects had vitamin A deficiency and >50% were vitamin D deficient. These findings suggest that longer Roux limb lengths result in increased nutritional risks compared with shorter limb lengths. As such, dietitians and others completing nutrition assessments should be familiar with the limb lengths of patients during their nutrition assessments to fully appreciate the risk of deficiency. Sugerman et al. [136] reported in their series comparing distal RYGB and shorter limb lengths that supplementation with 10,000 U of vitamin A, in addition to other vitamins and minerals, appeared to be adequate to prevent vitamin A deficiency. The laboratory values in this series were abnormal for other vitamins and minerals, including iron, B_{12}, vitamin D, and vitamin E [136].

Although the clinical manifestations of vitamin A deficiency are believed to be rare, case studies have demonstrated the occurrence of ophthalmologic consequences, such as night blindness [137–139]. Chae and Foroozan [140] reported on vitamin A deficiency in patients with a remote history of intestinal surgery, including bariatric surgery, using a retrospective review of all patients with vitamin A deficiency seen during one year in a neuro-ophthalmic practice. A total of four patients with vitamin A deficiency were discovered, three of whom had undergone intestinal surgery >18 years before the development of visual symptoms. It was concluded that vitamin A deficiency should be suspected among patients with an unexplained decreased vision and a history of intestinal surgery, regardless of the length of time since the surgery had been performed.

Significant unexpected consequences can occur as a result of vitamin A deficiency. Lee et al. [141], for instance, reported a case study of a 39-year-old woman, three years after RYGB, who presented with xerophthalmia, nyctalopia (night blindness), and visual deterioration to legal blindness in association with vitamin noncompliance during an 18-month period postoperatively [141]. Because vitamin A supplementation beyond that found in a multivitamin is not routine for the RYGB patient, it is likely that this individual had insufficient intake of dietary vitamin A, although this was not considered by the investigators. They concluded that the increasing prevalence of patients who have undergone gastric bypass surgery warrants patient and physician education regarding the importance of adherence to the prescribed vitamin supplementation regimen to prevent a possible epidemic of iatrogenic xerophthalmia and blindness. Purvin [142] also reported a case of nycalopia in a 43-year-old postoperative bariatric patient that was reversed with vitamin A supplementation.

Vitamin K

In the series by Slater et al. [36], the vitamin K levels were suboptimal in 51% (n = 35) of the patients one year after BPD. By the fourth year, the deficiency had increased to 68% (n = 19), with 42% of patients' serum vitamin K levels at less than the measurable range of 0.1 nmol/L compared with 14% at the end of the first year of the study. Vitamin K deficiency was also considered by Ledoux et al. [135] using the prothrombin time as an indicator of deficiency. The mean prothrombin time percentage was lower in the RYGB group versus both the AGB and conventional treatment groups, which suggests vitamin K deficiency [135].

Among the unusual and rare complications after bariatric surgery, Cone et al. [143] cited a case report in which an older woman with significant weight loss and diarrhea, that had been caused by the bacterium *Clostridium difficile*, developed Streptococcal pneumonia sepsis after gastric bypass surgery. The researchers hypothesized that malabsorption of vitamin K-dependent proteins C, S, and antithrombin, resulting from the gastric bypass, predisposed the patient to purpura fulminans and disseminated intravascular coagulation.

Vitamin E

A review of the literature revealed that most studies have examined the postoperative, and do not consider the preoperative, fat-soluble vitamin deficiencies. Boylan et al. [26] found that 23% of RYGB patients studied (n = 22) had low vitamin E levels preoperatively. Even though the food intake of all subjects was sharply decreased after surgery, most patients who were taking a multivitamin supplement that provided 100% of the daily value of vitamin E were found to maintain normal vitamin E levels. In a study of BPD/DS patients, the vitamin E levels were normal in all the patients less than one year postoperatively (n = 44) and remained normal among 96% (n = 24) of patients up to four years after surgery [36]. In a study comparing various surgical procedures, Ledoux et al. [135] found a significant prevalence of vitamin E deficiency among the RYGB com-

pared with the AGB group ($P < 0.05$), with 22.5% and 11.8% of subjects presenting with a vitamin E deficiency, respectively.

Zinc

Although zinc deficiency has not been fully explored, and its metabolic sequelae have not been clearly delineated, it is a nutrient that depends on fat absorption [36]; therefore, the likelihood of deficiency of this mineral among surgical patients appears plausible. Slater et al. [36] found that serum zinc levels were abnormally low in 51% of BPD/DS subjects (n = 43) at one year postoperatively and remained suboptimal among 50% of patients (n = 26) at four years postoperatively. In RYGB patients, Madan et al. [27] determined that zinc levels were suboptimal among 28% of preoperative patients (n = 69) and 36% of one-year postoperative patients (n = 33). In addition, Cominetti et al. [144] researched the zinc status of pre- and postoperative RYGB patients and found that the greatest changes were seen among erythrocyte and urinary zinc concentrations versus plasma zinc levels. They postulated that RYGB patients might have a lower dietary zinc intake in the postoperative period that could put them at a risk of deficiency. A lower dietary zinc intake could be related to food intolerances, especially that of red meat.

Burge [145] studied whether RYGB patients had changes in taste acuity postoperatively and whether zinc concentrations were affected. Taste acuity and serum zinc levels were measured in 14 subjects preoperatively and at 6 and 12 weeks postoperatively. Although the researchers did not find changes in zinc concentrations during the study, at six weeks postoperatively, all patients reported that foods tasted sweeter, and they had modified food selections accordingly. Finally, Scruggs et al. [146] found that after RYGB surgery, a significant upregulation in taste acuity for bitter and sour was observed, as well as a trend toward a decrease in salt and sweet detection and recognition thresholds. The researchers concluded that, among other things, taste effectors such as zinc, when in the normal range, do not alter thresholds of the four basic tastes.

Although zinc has been preliminarily investigated, the methods of analysis have not be rigorously evaluated. Many studies reporting suboptimal zinc levels did not report the method used to determine the status or how the analysis was performed. The method and accurate assessment of the dietary intake of zinc is critical to the evaluation of the reported data.

Suggested supplementation. Ledoux et al. [135] did not find a significant difference in fat-soluble vitamin deficiencies between those subjects who consumed a multivitamin (n = 24) and those who did not (n = 16). The small sample size of the study, and that the subjects were not randomized for multivitamin supplementation, versus no supplementation have made the data less meaningful. In addition, the level of dietary intake of these nutrients was not considered. It is unknown whether the two groups (with and without supplements) consumed similar diets. They proposed that all RYGB patients should be supplemented with multivitamins as complete as possible, including fat-soluble vitamins and minerals. A recommendation of 50,000 IU of vitamin A every two weeks and 500 mg of vitamin E daily, among other supplements, was suggested to correct most cases of deficiency [135].

Slater et al. [36] suggested life-long, annual measurements of fat-soluble nutrients after BPD/DS procedures, along with continued nutrition counseling and education. In addition to multivitamin/mineral recommendations, which included zinc, vitamin D, and calcium, they recommended life-long daily supplementation of a minimum of 10,000 IU of vitamin A and 300 μg of vitamin K [36].

Finally, Madan et al. [27] also concluded that water and fat-soluble vitamin and trace mineral deficiencies are common both pre- and postoperatively among RYGB patients. Routine evaluation of serum vitamin and mineral levels was recommended for this patient population.

Conclusion. The reports cited in this section illustrate that deficiencies of vitamins A, E, K, and zinc have been discovered among bariatric surgery patients. Although BPD/DS patients have been known to be at risk of fat-soluble vitamin deficiencies, the reports cited have illustrated that bariatric patients in general, including RYGB patients, may also be at risk. In addition, rare and unusual complications, such as visual and taste disturbances, might be attributable to these deficiencies. Routine supplementation, including multivitamins/minerals, along with close pre- and postoperative monitoring could attenuate the risk of these deficiencies.

Other micronutrients

Vitamin B_6. Little information is available pertaining to changes in vitamin B_6 (pyridoxal phosphate) with bariatric surgery because vitamin B_6 is not routinely measured. Boylan et al. [26] found that vitamin B_6 levels, before surgery, were adequate in only 36% of their surgical candidates. After gastric bypass, a normal status for vitamin B_6 levels was achieved in patients using supplements containing the vitamin at amounts close to the U.S. Dietary Reference Intake. Turkki et al. [147] also found normal levels of vitamin B_6 after gastroplasty for patients receiving supplementation. However, these investigators subsequently found that the serum levels might not be reflective of vitamin B_6 biologic status [148]. When co-enzyme activation of erythrocyte aminotransferase activities was used as a marker of vitamin B_6 status rather than the serum vitamin levels, supplementation of vitamin B_6 at the U.S. Dietary Reference Intake recommended amounts (1.6 mg) proved inadequate for co-enzyme activation of these enzymes in the early postoperative period. These findings suggest that

greater than the recommended amounts of vitamin B_6 might be required for normalization of vitamin B_6 status in bariatric patients.

Signs, symptoms, and treatment of deficiency (see Appendix, Table A2)

Copper. Although copper is absorbed by the stomach and proximal gut, copper is rarely measured in patients who have undergone RYGB or BPD/DS. Deficiencies in copper can not only cause anemia but also myelopathy, similar to that found with deficiencies in vitamin B_{12} [149,150]. Currently, only two cases of copper deficiency have been reported in gastric bypass patients, both of whom presented with symptoms of myelopathy (i.e., ataxia, paresthesia) [149,150]. Other cases of copper deficiency and demyelinating neuropathy, however, have been reported for individuals who have undergone gastrectomies [150]. These findings suggest that the copper status needs to be examined in RYGB and BPD/DS patients presenting with signs and symptoms of neuropathy and normal B_{12} levels. The findings would also suggest multivitamin supplementation containing adequate amounts of copper (2 mg daily value). Caution should be used when prescribing zinc supplements because copper depletion occurs when >50 mg zinc is given for a long period of time.

Other mineral deficiencies. Various other trace elements and minerals could be deficient postoperatively. Selenium, for instance, has been found to be deficient in surgical patients both pre- and postoperatively [27]. Dolan et al. [134] found that 14.5% of patients undergoing BPD/DS were selenium deficient. These investigators, as well as others [151,152], have also reported inadequate magnesium or potassium status with bariatric surgery. Dolan et al. [134] found that 5% of patients undergoing BPD/DS were deficient in magnesium. Schauer et al. [151] found that <1% (0.7%) of gastric bypass patients were magnesium deficient 1–31 months postoperatively. These same investigators, however, reported hypokalemia in 5% of their gastric bypass population. Crowley et al. [152] in a much earlier report also found low potassium levels after gastric bypass in 2.4% of patients. The findings of these studies emphasize the need for recommendations of multivitamin supplements that are complete in minerals.

Protein

Etiology of potential deficiency

Thorough mastication of food is an important first step in the overall digestion process to compensate for the reduced grinding capacity of the pouch. Breaking food into smaller particles and moistening it with saliva will facilitate a bolus of animal protein to pass from the esophagus into the pouch or through the band. In normal digestion, hydrochloric acid converts the inactive proteolytic enzyme pepsinogen (secreted in the middle of the stomach) into its active form, pepsin. This allows the digestion of collagen to begin as the contents of the stomach continues to grind and mix with gastric secretions. Cholecystokinin and enterokinase are released as the chyme comes in contact with intestinal mucosa, allowing the secretion and activation of pancreatic proteolytic enzymes, trypsin, chymotrypsin, and carboxypolypeptidase, which facilitate the breakdown of protein molecules into smaller polypeptides and amino acids. Proteolytic peptidases, located in the brush border of the intestine, allow additional breakdown into tripeptides and dipeptides. These small peptides cross the brush border intact where peptide hydrolases complete digestion into amino acids so they can be absorbed and transported to the liver by way of the portal vein.

Quite often it is thought that if a bolus of protein is not able to mix with the hydrochloric acid and pepsin, produced in the antrum of the stomach, that maldigestion will result, leading to significant protein deficiencies in patients with malabsorptive or combination procedures. However, the stomach's role in the digestion of protein is very small, with most digestion and absorption occurring in the small intestine. Strong evidence cannot be found in the literature to support the theory that the malabsorptive components of bariatric surgery alone cause deficiency. Protein malnutrition (PM) is usually associated with other contemporaneous circumstances that lead to decreased dietary intake, including anorexia, prolonged vomiting, diarrhea, food intolerance, depression, fear of weight regain, alcohol/drug abuse, socioeconomic status, or other reasons that might cause a patient to avoid protein and limit caloric intake. All postoperative patients are, therefore, at risk of developing primary PM and/or protein-energy malnutrition (PEM) related to decreased oral intake. BPD/DS patients are at risk of secondary PM/PEM because of the greater degree of malabsorption produced by this procedure.

When nutrition is withheld, for whatever reason, the body is able to metabolically adapt for survival. As a result of decreased caloric intake, hypoinsulinemia allows fat and muscle breakdown to supply the amino acids needed to preserve the visceral pool. Gluconeogenesis and fatty acid oxidation help maintain a supply of energy to vital organs (the brain, heart, and kidney). Protein sparing is eventually achieved as the body enters into ketosis. Initially, weight loss occurs as a result of water loss resulting from the metabolism of liver and muscle glycogen stores. Subsequent weight loss occurs with the breakdown of muscle mass and a reduction of adipose tissue as the body strives to maintain homeostasis. A low protein intake can be tolerated by the body, because it will adjust to the negative nitrogen balance over several days, but only to a certain extent. Eventually, without adequate intake, a deficiency will occur, characterized by decreased hepatic proteins, including albumin, muscle wasting, asthenia (weakness), and alopecia

(hair loss). Protein-energy malnutrition is typically associated with anemia related to iron, B_{12}, folate, and/or copper deficiency. Deficiencies in zinc, thiamin, and B_6 are commonly found with a deficient protein status. In addition, catabolism of lean body mass and diuresis cause electrolyte and mineral disturbances with sodium, potassium, magnesium, and phosphorus.

If the protein deficit occurs in conjunction with excessive intake of carbohydrate calories, hyperinsulinemia will inhibit fat and muscle breakdown. When the body is not able to hormonally adapt to spare protein, a decrease in visceral protein synthesis will result, along with hypoalbuminemia, anemia, and impaired immunity. If left undiagnosed, this can result in an illness in which fat stores are preserved, lean body mass is decreased, and appropriate weight loss is not seen because of the accumulation of extracellular water. This edema is associated with PM [34,153,154].

Unfortunately, loss of muscle mass is an inevitable part of the weight loss process after obesity surgery or any very-low-calorie diet. Patients, especially those undergoing BPD/DS, should be encouraged to focus on protein-rich foods, of high biologic value, in meal planning while portion size is significantly decreased during the early postoperative period. This might help compensate for the natural daily endogenous loss of protein in the gut.

It is important for the medical team members to be familiar with the process of extreme weight loss and the body's ability to metabolically adapt for survival in the semistarvation state that is commonly produced after bariatric surgery. Perhaps explaining the mechanism of weight loss and the desired outcome, in terms the patient can understand, would help to promote compliance and provide motivation to choose quality foods consisting of high biologic value protein balanced with nutrient dense complex carbohydrates and healthy food sources of essential fatty acids. In addition to consistent biochemical screening, a trained professional (typically a Registered Dietitian) should regularly complete a thorough assessment of the patient's nutritional status to ensure adequate protein intake in the midst of a calorie restriction. This might help prevent excessive wasting of lean body mass and deficiency.

Preoperative risk

Preoperative protein deficiency is rarely reported in published studies. Flancbaum et al. [21] found "abnormal" albumin levels in 4 of 357 preoperative RYGB patients. This was reported as being insignificant. They did not note other measures of protein deficiency. Rabkin et al. [155] followed 589 consecutive DS patients and found no abnormal protein metabolism preoperatively. Although it does not appear that preoperative patients are at risk of protein deficiency, it would be unwise to assume that it does not exist among the morbidly obese with today's popularity of food faddism and the well-documented disordered eating patterns among the morbidly obese. The idea that the preoperative patient is overnourished from the stand point of calories does not reflect the nutritional quality of the dietary intake. Routine preoperative screening, including laboratory measures of albumin, transferrin, and lymphocyte count, are helpful in the diagnosis of PM [76] and assessing visceral protein status. Other hepatic protein measures with shorter half-lives such as retinol binding protein and prealbumin could be useful in diagnosing acute changes in protein status. Clinical signs of deficiency might be masked by the adipose tissue, edema, and general malaise experienced by the bariatric patient. It is not appropriate to assume that the patient that appears to look well has good nutritional status and appropriate dietary intake.

Postoperative risk

Protein deficiency (albumin <3.5 g/dL) is not common after RYGB. Brolin et al. [84] reported that 13% of patients who had undergone distal RYGB as part of a prospective randomized study were found to have hypoalbuminemia two years after surgery. Those with short Roux limbs (<150 cm) were not found to have decreased albumin levels [84]. In another prospective randomized study of long-limb, super obese, gastric bypass patients, protein deficiency was not found in patients at a mean of 43 months postoperatively [156]. Two retrospective studies determined that hypoalbuminemia (defined as albumin <4.0 g/dL in one and <3.0 g/dL in the other) was negligible in both RYGB and BPD patients one to two years postoperatively [88,157]. The investigators noted the few cases of hypoalbuminemia that did occur resulted from patient "noncompliance" with nutrition instruction and were treated with protein supplementation. In addition, no evidence of protein or energy malnutrition was found by Avinnoah et al. [158], six to eight years after RYGB, despite a high prevalence of long-term meat intolerance among the subjects.

Protein deficiency is more likely to be noted in published studies in association with BPD procedures, usually during the first or second year postoperatively. Sporadic cases of recurrent late PM have been reported requiring two to three weeks of parenteral feeding for correction. The pathogenesis of this PM after BPD is multifactorial. Operation-related variables include stomach volume, intestinal limb length, individual capacity of intestinal absorption and adaptation, as well as the amount of endogenous nitrogen loss. Patient-related variables include customary eating habits, ability to adapt to the nutrition requirements, and socioeconomic status. Early occurrences of PM are typically due to patient-related factors, and recurrent late episodes of PM are more likely to be caused by excessive malabsorption as a result of surgery. Correction in these cases typically requires elongation of the common limb [34]. By varying the length of the intestinal limbs and common channel, protein malabsorption can be increased or decreased [35].

In a controlled environment (n = 15), Scopinaro et al. [159] found intestinal albumin absorption to be 73% and nitrogen absorption 57% after BPD. This indicates greater than normal loss of endogenous nitrogen. The investigators noted that the percentage of nitrogen absorption tended to remain constant when intake varied. Therefore, an increase in alimentary protein intake would result in an increased absolute amount absorbed. They postulated that the observed 30% protein malabsorption that had been identified in previous studies did not explain the PM that occurs after BPD. It was concluded that loss of endogenous nitrogen (approximately fivefold the normal value) plays a significant role in the development of PM after BPD, especially during the early postoperative period when restricted food intake might cause a negative balance in both calories and protein [159].

The incidence of PM after BPD has been reported to be approximately 7–21% [35]. However, Totte et al. [160] followed 180 BPD patients (using the method of Scopinaro et al [35] with a 50-cm common channel) and found protein deficiency developed in only two patients at 16 and 24 months postoperatively requiring parenteral nutrition, conversion of the alimentary tract, and psychiatric counseling for correction. Both cases were attributed to causes unrelated to the operation that had disrupted the patients' normal life. It was concluded that metabolic complications after BPD were the result of patient noncompliance with dietary recommendations (70–80 g/d protein) that had been explained during preoperative counseling by a Registered Dietitian [160]. Marinari et al. [161] reported mild hypoalbuminemia in 11% and severe in 2.4% of BPD patients.

Rabkin et al. [155] followed a cohort of 589 sequential DS patients (with a gastric sleeve and common channel of 100 cm) and found annual laboratory measures of serum markers for protein metabolism to slightly decrease at one year postoperatively but then stabilize at two and three years postoperatively well within normal limits. Similarly, in an earlier study, Marceau et al. [162] also reported no significant decrease in albumin levels among BPD/DS patients.

Body composition has also been studied postoperatively in malabsorptive and restrictive surgical patients. Tacchino et al. [163] studied changes in total and segmental body composition in 101 women preoperatively and at 2, 6, 12, and 24 months after BPD. A significant reduction in fat and lean body mass was observed postoperatively that stabilized between 12–24 months at levels similar to those of the controls. The investigators concluded that weight loss after BPD was achieved with an appropriate decline of lean body mass. In addition, the visceral/muscle ratio in pre-BPD patients was preserved in the post-BPD patients at 24 months [163]. Benedetti et al. [164] studied body composition and energy expenditure after BPD (n = 30) and found that after one year of weight stabilization, the subjects had a greater fat free mass and basal metabolic rate then did the controls. The postoperative patients had an increased caloric intake and physical activity expenditure. This aided in the retention of the fat free mass after weight loss and contributed to the greater basal metabolic rate [164].

Because AGB patients have weight loss due to a significant reduction in caloric intake and can experience food intolerances, leading to aversion of protein foods, it is important to consider the loss of body protein in these patients as well. A small series (n = 17) of AGB patients were followed for two years postoperatively. Total weight loss consisted of 30.1% fat mass and a 12.3% reduction in fat free mass. No significant change was found in the potassium/nitrogen ratio after surgery and the loss of fat mass was in proportion to that usually seen in weight loss [165].

When a deficiency occurs and no mechanical explanation for vomiting or food intolerance is present, patients can often be successfully treated with a high-protein liquid diet and slow progression to a regular diet [76]. Reinforcement of proper eating style (small bites of tender food, chewed well, eaten slowly) is always important to address during patient consultation in an effort to improve intake. As the protein deficiency is corrected and edema is decreased around the anastomosis, food tolerance and vomiting may resolve. Although rare, severe PM can occur, regardless of the type of surgery performed. This typically requires hospitalization, parenteral treatment to sufficiently return the total body protein to normal levels, and might warrant psychological evaluation and/or counseling. It is important to rule out all the possible underlying mechanical and behavioral causes before considering lengthening the common channel or surgery reversal.

Protein intake

Current clinical practice recommendations for protein intake after surgery without complications are consistent with those for medically supervised modified protein fasts. Experts recommend up to 70 g/d during weight loss on very-low-calorie diets [167]. The recommended dietary allowance (RDA) for protein is approximately 50 g/d for normal adults [166]. Many programs recommend a range of 60–80 g/d total protein intake or 1.0–1.5 g/kg ideal body weight (IBW), although the exact needs have yet to be defined. The use of 1.5 g of protein/kg IBW/day after the early postoperative phase is probably greater than the metabolic requirements for noncomplicated patients and might prevent the consumption of other macronutrients in the context of volume restriction. An analysis of RYGB patient's typical nutrient intake at one year post surgery found no significant changes in albumin with daily protein consumption at 1.1 g/kg IBW [168]. After BPD/DS procedures, the amount of protein should be increased by ~30% to accommodate for malabsorption, making the average protein requirement for these patients approximately 90 g/d [36].

Table 6
Indispensable and Dispensable Amino Acids [169]

Indispensable amino acids	EAR (mg/g pro)	Dispensable amino acids	Conditionally indispensable amino acids
Histidine	17	Alanine	Arginine
Isoleucine	23	Aspartic acid	Cysteine
Leucine	52	Asparagine	Glutamine
Lysine	47	Arginine	Glycine
Methionine	23	Cysteine (23)	Proline
Phenylalanine	41	Glutamic acid	Tyrosine (41)
Threonine	24	Glutamine	
Tryptophan	6	Glycine	
Valine	29	Proline	
		Serine	
		Tyrosine	

EAR = estimated average requirement.

During the early postoperative period, incorporating liquid supplements into the patient's daily oral intake provides an important source of calories and protein that help prevent the loss of lean body mass. Experts have noted that adding 100 g/d of carbohydrate decreases nitrogen loss by 40% in modified protein fasts [34]. One popular myth is that only 30 g/hr of protein can be absorbed. Although this is commonly found in both lay and some professional literature, there is no scientific basis for this claim. It is possible that, from a volume standpoint, patients might only realistically consume 30 g/meal of protein during the first year.

Modular protein supplements

It is commonly known that adequate dietary intake is required to supply the 9 indispensable (essential) amino acids (IAAs) and adequate substrate for the production of the 11 dispensable (nonessential) amino acids that compose body protein. This is referred to as the nonspecific nitrogen requirement. In the presence of physiologic stress or certain disease states, the body cannot produce enough of certain dispensable amino acids to satisfy one's need. To this end, a third category of conditionally indispensable amino acids exists, potentially increasing the body's protein requirement beyond the RDA. The Institutes of Medicine has established an estimated average requirement (EAR) for the essential amino acids that may be used as a reference value when assessing protein supplements (Table 6).

Commercially produced modular protein supplements are widely available that can be used to complement a patient's dietary intake after surgery. Clinicians are often challenged when choosing the best product to meet the patient's nutritional needs. Although convenience, taste, texture, ease in mixing, and price are important considerations that may improve intake compliance, the product's amino acid profile should be the first priority. A protein supplement that provides all the indispensable amino acids or a combination of products must be used when protein supplements are the sole source of dietary protein intake during rapid weight loss. Reputable manufacturers should be able to provide accurate information substantiating claims made about the amino acid profile of their products.

In a comprehensive review completed by Castellanos et al. [170], modular protein supplements were classified into four categories (Table 7). They noted that the amino acid content of various protein supplements differs dramatically in that a given quantity of a supplement from one category is not nutritionally equivalent to the same quantity of protein from a different category. Although peer-reviewed data do not exist to determine the quality of the various commercial products through traditional methods such as net protein use, biologic value, and protein efficiency ratio, these assessments have been conducted on the common protein sources used in commercial products (i.e., whey, casein, egg, soy). In 1991, the "protein digestibility corrected amino acid" (PDCAA) score was established as a superior method for the evaluation of protein quality. PDCAAs compare the IAA content of a protein to the EAR for each IAA, mg/g of protein. (The EAR reference values used to calculate PDCAAs are noted in Table 6.)

Table 7
Categories of Modular Protein Supplements

Protein category	Derived from	Complete	Intended use
Complete protein concentrates	Egg white, soy, or milk (casein/whey fractions)	Yes; contains all 9 IAAs relative to human requirement	Provides IAAs in dietary protein
Collagen-based concentrates	Hydrolyzed collagen; some are combined with casein or other complete proteins	No; contains low levels of 8 of 9 IAA—lacks tryptophan	Provides DAAs in dietary protein; contains high proportion of nitrogen in small volume
Amino acid dose	Large doses of ≥1 DAAs (i.e., arginine, glutamine) or amino acid precursors	No	Provides conditionally IAAs; promotes wound healing
Hybrids of protein plus an amino acid dose	Complete protein concentrate or collagen base plus ≥1 DAAs	Varies	Meets protein needs and increases intake of conditionally IAAs

IAAs = indispensable amino acids; DAAs = dispensable amino acids.
Adapted from Castellanos et al. [170], with permission.

The PDCAA score indicates the overall quality of a protein, because it represents the relative adequacy of its most limiting amino acid. The PDCAA score indicates the body's ability to use that product for protein synthesis. The PDCAA score is equal to 100 for milk, casein, whey, egg white, and soy [170].

Caution should be used when recommending any type of collagen-based protein supplement. Although some commercial collagen products can be combined with casein or other complete proteins, the resulting combination might still provide insufficient amounts of several IAAs. These types of products are typically not considered "complete." However, because collagen contains a high level of nitrogen within a small volume, it might be useful for the patient who is able to consume enough good-quality dietary protein to supply the needed IAAs, yet not be consuming enough total nitrogen to meet the nonspecific nitrogen requirement and achieve balance [171]. In this case, the patient would also have to be consuming enough calories to spare the IAAs for protein synthesis. It is very important for the practitioner to review the amino acid composition of the patient's selected commercial protein products to ensure they include adequate amounts of all the IAAs. The loss of lean body mass can occur despite meeting a daily oral intake protein goal, in the presence of IAA deficiency.

The highest quality protein products are made of whey protein, which provides high levels of branched-chain amino acids (important to prevent lean tissue breakdown), remain soluble in the stomach, and are rapidly digested. Whey concentrates can contain varying amounts of lactose, while whey protein isolates are lactose free. This can be a consideration for those individuals with a severe intolerance. Meal replacement supplements and protein bars typically contain a blend of whey, casein, and soy proteins (to improve texture and palatability), varying amounts of carbohydrate and fiber, as well as greater levels of vitamins and minerals than simple protein supplements. Many commercial protein drinks and bars are designed to supplement a mixed diet, including animal and plant sources of protein. They are not intended to provide the sole source of protein and calories for long periods of time.

Diet and texture progression

The purpose of nutrition care after surgical weight loss procedures is twofold. First, adequate energy and nutrients are required to support tissue healing after surgery and to support the preservation of lean body mass during extreme weight loss. Second, the foods and beverages consumed after surgery must minimize reflux, early satiety, and dumping syndrome while maximizing weight loss and, ultimately, weight maintenance. Many surgical weight loss programs encourage the use of a multiphase diet to accomplish these goals.

Clear liquid diet

A clear liquid diet is often used as the first step in postoperative nutrition, despite some evidence that it might not be warranted [172]. Sugar-free or low sugar bariatric clear liquid diets supply fluid, electrolytes, and a limited amount of energy and encourage the restoration of gut activity after surgery. The foods that are included in clear liquid diets are typically liquid at body temperature and leave a minimal amount of gastrointestinal residue. Gastric bypass clear liquid diets are nutritionally inadequate and should not be continued without the inclusion of commercial low-residue or clear liquid oral nutrition supplements beyond 24–48 hours.

Full liquid diet

Sugar-free or low-sugar full liquid diets often follow the clear liquid phase. Full liquid diets include milk, milk products, milk alternatives, and other liquids that contain solutes. Full liquid diets have slightly more texture and increased gastric residue compared with clear liquid diets. In addition, the calories and nutrients provided by full liquid diets that include protein supplements can closely approximate the needs of surgical weight loss patients. The liquid texture is thought to further allow healing and the caloric restriction provides energy and protein equivalent to that provided by very-low-calorie diets.

Pureed diet

The bariatric pureed diet consists of foods that have been blended or liquefied with adequate fluid, resulting in foods that range from milkshake to pudding to mash potato consistency. In addition, foods such as scrambled eggs and canned fish (tuna or salmon) can be incorporated into the diet. Fruits and vegetables may be included, although the emphasis of this phase is usually on protein-rich foods. This diet fosters additional tolerance of a gradually progressive increase in gastric residue and gut tolerance of increased solute and fiber. The protein supplements used during the full liquid phase are often continued during the puree phase to complement dietary protein intake.

Mechanically altered soft diet

The bariatric soft diet provides foods that are texture-modified, require minimal chewing, and that will theoretically pass easily from the gastric pouch through the gastrojejunostomy into the jejunum or through the adjustable gastric band. This diet is considered a transition diet that is achieved by chopping, grinding, mashing, flaking, or pureeing foods [173].

Diet and texture progression survey

Given the limited availability of research to support the use of multiphase diets after surgical weight loss procedures, dietitians who were members of the American Soci-

Table 8
Current Texture Advancement in Clinical Practice for Noncomplicated Patients

Diet phase	Duration (d)
Clear liquid	1–2
Full liquid	10–14
Puree	10–14+
Mechanically altered soft	≥14
Regular	—

ety for Metabolic and Bariatric Surgery were surveyed using an on-line survey in May 2007 to determine current clinical practice with regard to texture and diet advancement. Overall, 68 dietitians responded to the survey, representing 50% of the dietitian membership within American Society for Metabolic and Bariatric Surgery.

Demographics. Most of the respondents were from community hospitals (50%) and academic medical centers (24%). Most of the programs surveyed performed 101–300 RYGBs and <50 AGBs annually. Most of the programs (62%) reported that their institution did not perform BPD/DS procedures or performed <50/yr (31%).

Pouch size and limb lengths. The most commonly reported pouch size for RYGB was 30–39 cm^3 (54%) with a Roux limb length of 70 to <100 cm (44%). Nearly 38% of respondents reported that the limb length was 125–150 cm. The most common pouch size for AGB was 30–39 cm^3 (37%); however, almost 19% of respondents could not report the pouch size most commonly created by the surgeons in their respective programs. For those programs offering BPD/DS, the most common pouch size was 120 to <180 cm^3 (55%). The common channel was reported to be 101–200 cm (34%) by most respondents; however, 28% could not identify the length of the common channel used by their program.

Diet phases. The dietitians reported that multiple phases are used for postoperative recommendations. Most programs reported clear liquid (95%), full liquid (94%), puree (77%), ground or soft (67%), and, ultimately, regular diets with sugar, fat, and/or fiber restrictions (87%). For the purposes of the survey, it was assumed that each progressive phase allowed foods from earlier phases. Thus, items allowed on a clear liquid diet were assumed to be included in a full liquid diet and so forth. For example, protein supplements were commonly listed as being included in all phases of diet progression.

Clear liquid diets were reported by most programs to last for 1–2 days for both RYGB (60%) and AGB (40%) patients. The foods commonly reported to be included in the clear liquid diet were diet gelatin, broth, sugar-free popsicles, decaf/herbal teas, artificially sweetened beverages, and protein supplements. Although regular juices contain significant amounts of fruit sugar, 40% of respondents reported that diluted fruit juice was offered during this phase of the diet. Caution should be used when encouraging simple carbohydrate intake because this could facilitate gut adaptation.

Full liquid diets were most commonly advised for 10–14 days for both RYGB (38%) and AGB (28%) patients. Common foods included in the full liquid diet included milk and alternatives, vegetable juice, artificially sweetened yogurt, strained cream soups, cream cereals, and sugar-free puddings.

Pureed diets were most commonly reported as being given for >10 days for RYGB and AGB. The foods most commonly included in the puree phase were reported to be scrambled eggs and egg substitute, pureed meat, flaked fish and meat alternatives, pureed fruits and vegetables, soft cheeses, and hot cereal.

Most respondents reported that a traditional "ground diet" was not included in the diet progression. Instead, a soft diet that included mechanically altered meats was most commonly recommended. Traditionally a "soft diet" consists of low-residue foods; however, for surgical weight loss diets, the term "soft" most commonly relates to the texture of the food, rather than residue. Both RYGB (55%) and AGB (42%) diet progressions most commonly included >14 days of a soft diet. Foods included in the soft diet phase were ground and chopped tender cuts of meats and meat alternatives, canned fruit, soft fresh fruit, canned vegetables, soft cooked vegetables, and grains, as tolerated.

Most of the programs reported that diet advancement to a surgical weight loss regular diet occurred after eight weeks for RYGB and after six to eight weeks for AGB. The diets for RYGB and AGB were strikingly similar, as summarized in Table 8. This was perhaps a result of program administration and resource constraints or could have been related to the limited number of AGB procedures performed by the institutions responding to the survey.

Similar to AGB and RYGB, programs offering DS/BPD procedures reported that the clear liquid diet phase is employed for one to two days after surgery. The full liquid phase was most commonly noted to last >10 to 14 days, while the pureed phase was reported to be >14 days. Most

Table 9
Recommended Foods to Avoid or Delay Reintroduction

Food type	Recommendation
Sugar, sugar-containing foods, concentrated sweets	Avoid
Carbonated beverages	Avoid/delay
Fruit juice	Avoid
High-saturated fat, fried foods	Avoid
Soft "doughy" bread, pasta, rice	Avoid/delay
Tough, dry, red meat	Avoid/delay
Nuts, popcorn, other fibrous foods	Delay
Caffeine	Avoid/delay in moderation
Alcohol	Avoid/delay in moderation

programs report that a ground texture phase is not utilized. The soft diet phase was reported to last >14 days. Finally, those programs offering DS/BPD most often reported advancing patients to a regular diet five to eight weeks after surgery.

Foods commonly restricted. The American Society for Metabolic and Bariatric Surgery members reported in the survey that patients were instructed to avoid or delay the introduction of several foods as noted in Table 9. Research to support these clinical practices is limited, especially with regard to caffeine and carbonation. Practitioners might theorize that certain foods and beverages will cause gastric irritation, outlet obstruction, intolerance, delayed wound healing, or alter the weight loss course; however, much of the information is anecdotal and lacks empirical evidence. In addition, although practitioners recommend that patients avoid or delay the introduction of these foods, little information is known as to whether patients actually comply with these recommendations and whether those who do not comply have altered outcomes or clinical histories. One retrospective survey suggested that many patients are noncompliant with diet and exercise recommendations [174].

Diet advancement and nutrition intervention. Diet advancement was most commonly advised at the discretion of the dietitians (74%); however, other members of the team, including the surgeon, nurse, or midlevel provider were also noted to make recommendations for advancement. Almost 40% of respondents reported that patients followed a written protocol for diet advancement. Nutrition interventions were most commonly conducted as individual appointments at 1–2 weeks, 1, 2, 3, 6, and 9 months, and then annually [175].

Conclusion

It was the intent of this paper to serve as an educational tool for not only dietitians, but all those providers working with patients with severe obesity. Current research and expert opinion were reviewed to provide an overview of the elements that are important to the nutritional care of the bariatric patient. While the extent of this paper has been broad, the Allied Health Executive Council of the American Society for Metabolic and Bariatric Surgery realizes there are many areas for future expansion that may change the paradigm for nutritional care. The Ad Hoc Nutrition Committee sincerely hopes that this document will serve to elucidate the general nutrition knowledge necessary for the care of the pre- and postoperative patient, with consideration for the individual patient's unique medical needs, as well as the variable protocol established among surgical centers and individual practices.

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Appendix: Identifying and Treating Micronutrient Deficiencies

Tables A1 through A11 list, by micronutrient, the sources, functions, interactions, symptoms of deficiency, and recommended treatments for micronutrients discussed in this report [17,154,176]. DRI and UL are defined as "dietary reference intake" and "upper limit," respectively, for all tables in Appendix A.

Table A1

Thiamin (B_1)	
DRI and UL	DRI 1.1–1.2 mg/d (females vs males); UL not determinable
Food sources	Meat, especially pork; sunflower seeds, grains, vegetables
Functions	Co-enzyme in carbohydrate metabolism, protein metabolism central and peripheral nerve cell function, myocardial function
	Body storage limited to 30 mg; half life is 9–18 d
Food/nutrient interactions	Drinking tea/coffee or decaffeinated tea/coffee depletes thiamin in humans
	Ascorbic acid improves thiamin status
	Thiamin is poorly absorbed when folate or protein deficiency present
Early symptoms of deficiency	Anorexia
	Gait ataxia
	Paresthesia
	Muscle cramps
	Irritability
Advanced symptoms of deficiency	Wet beriberi: high output heart failure with dyspnea due to peripheral vasodilation, tachycardia, cardiomegaly, pulmonary and peripheral edema, warm extremities, mimicking cellulites
	Dry beriberi: symmetric motor and sensory neuropathy with pain, paresthesia, loss of reflexes, and
	Wernicke-Korsakoff syndrome
	Wernicke's encephalopathy classic triad includes encephalopathy, ataxic gait, and oculomotor dysfunction
	Korsakoff syndrome includes amnesia or changes in memory, confabulation, and impaired learning
Diagnosis	Decreased urinary thiamin excretion
	Decreased RBC transketolase
	Decreased serum thiamin
	Increased lactic acid
	Increased pyruvate
Treatment	With hyperemesis, parenteral doses of 100 mg/d for first 7 d, followed by daily oral doses of 50 mg/d until complete recovery; simultaneous therapeutic doses of other water-soluble vitamins; magnesium deficiency must be treated simultaneously; administration with food reduces rate of absorption

RBC = red blood cell.

Table A2

Pyridoxine (B_6)	
DRI and UL	DRI 1.3 mg; UL 100 mg/d
Food sources	Legumes, nuts, wheat bran, and meat; more bioavailable in animal sources
Function	Co-factor for >100 enzymes involved in amino acid metabolism
	Involved in heme and neurotransmitter synthesis and in metabolism of glycogen, lipids, steroids, sphingoid bases, and several vitamins, such as conversion of tryptophan to niacin
Symptoms of deficiency	Epithelial changes, atrophic glossitis
	Neuropathy with severe deficiency
	Abnormal electroencephalogram findings
	Depression, confusion
	Microcytic, hypochromic anemia (B_6 required for hemoglobin production)
	Platelet dysfunction
	Hyperhomocystinemia
Diagnosis	Decreased plasma pyridoxal phosphate
	Complete blood count (anemia)
	Increased homocysteine
Treatment	Vitamin B_6 50 mg/d; 100–200 mg if deficiency related to medication use

Table A3

Cobalamin (B_{12})	
DRI and UL	DRI 2.4 μg/d; UL not determinable
	Body stores, 4 mg; one half stored in liver
Food sources	Meat, dairy, eggs; found with animal products
Function	Maturation of RBC
	Neural function
	DNA synthesis related to folate co-enzymes
	Co-factor for methionine synthase and methylmalonyl–co-enzyme A
	B_{12} deficiency will cause folate deficiency
Symptoms of deficiency	Pernicious anemia (due to absence of intrinsic factor)/megaloblastic anemia
	Pale with slightly icteric skin and eyes
	Fatigue, light-headedness, or vertigo
	Shortness of breath
	Tinnitus (ringing in ear)
	Palpitations, rapid pulse, angina, and symptoms of congestive failure
	Numbness and paresthesia (tingling or prickly feeling) in extremities
	Demyelination and axonal degeneration, especially of peripheral nerves, spinal cord, and cerebrum
	Changes in mental status, ranging from mild irritability and forgetfulness to severe dementia or frank psychosis
	Sore tongue, smooth and beefy red appearance
	Ataxia (poor muscle coordination), change in reflexes
	Anorexia
	Diarrhea
Diagnosis	CBC: elevated MCV, high RDW, Howell-Jolly bodies, reticulocytopenia
	Low serum B_{12}
	Increased MMA and increased homocysteine
	Decreased transcobalamin II-B_{12}
	Neurologic disease can occur with normal hematocrit
Treatment	1000 μg/wk IM for 8 wk, then 1000 μg/mo IM for life or 350–500 μg/d oral crystalline B_{12}
	Neurologic defects might not reverse with supplementation

CBC = complete blood count; MCV = mean corpuscular volume; RBC = red blood cell; RDW = red blood cell distribution width; MMA = methylmalonic acid; IM = intramuscularly.

Table A4

Folate	
DRI and UL	DRI 400 μg/d; UL 1000 μg/d
	Body stores: 5–20 mg; one half stored in liver; deficiency can occur within months
Food sources	Vegetables, especially green leafy; fruit, enriched grains, including bread, pasta, and rice
Functions	Maturation of RBCs
	Synthesis of purines, pyrimidines, and methionine
	Prevention of fetal neural tube defects
Symptoms of deficiency	Megaloblastic anemia
	Diarrhea
	Cheilosis and glossitis
	Neurologic abnormalities do not occur
Diagnosis	CBC: elevated MCV, high RDW
	Decreased RBC folate (not subject to acute fluctuations in oral folate intake as seen with serum folate)
	Normal MMA with increased homocysteine
Treatment	1000 μg/d orally; up to 5 mg/d might be needed with severe malabsorption
	Correction can occur within 1–2 mo; encourage consumption of folate-rich foods and abstinence from alcohol; alcohol interferes with folate absorption and metabolism
Toxicity	>1000 μg/d can mask hematologic effects of B_{12} deficiency

RBC = red blood cell; CBC = complete blood count; MCV = mean corpuscular volume; RDW = red blood cell distribution width.

Table A5

Iron	
DRI and UL	DRI 8 mg/d for men, 18 mg/d for women 19–50 yr; UL 45 mg/d
Food sources	Meats, fish, poultry, eggs, enriched grains, dried fruit, some vegetables and legumes
Function	obin and myoglobin formation
	Cytochrome enzymes
	Iron-sulfur proteins
Symptoms of deficiency	Anemia
	Dysphagia
	Koilonychia
	Enteropathy
	Fatigue
	Rapid heart rate/palpitations
	Decreased work performance
	Impaired learning ability
Stages of deficiency	Stage 1: Serum ferritin decreases <20 ng/mL
	Stage 2: Serum iron decreases <50 μg/dL; transferrin saturation <16%
	Stage 3: Anemia with normal-appearing RBCs and indexes occurs
	Stage 4: Microcytosis and then hypochromia present
	Stage 5: Fe deficiency affects tissues, resulting in symptoms and signs
Diagnosis	CBC: low Hgb/Hct, low MCV
	Decreased serum iron
	Decreased percentage of saturation
	Increased TIBC
	Increased transferrin
	Decreased serum ferritin (affected by inflammation or infection)
Treatment	Typically, for iron replacement therapy, up to 300 mg/d elemental iron given, usually as 3 or 4 iron tablets (each containing 50–65 mg elemental iron) given during course of the day; ideally, oral iron preparations should be taken on empty stomach because food can inhibit iron absorption. When oral treatment has failed or with severe anemia, IV iron infusion should be considered.
Toxicity	Nausea, vomiting, diarrhea, constipation; iron overload with organ damage, acute systemic toxicity

RBCs = red blood cells; CBC = complete blood count; Hgb/Hct = hemoglobin/hematocrit; MCV = mean corpuscular volume; TIBC = total iron binding capacity; IV = intravenous.

Table A6

Calcium	
DRI and UL	DRI 1000–1200 mg/d; UL 2500 mg/d
Food sources	Diary products, leafy green vegetables, legumes, fortified foods, including breads and juices
Function	Bone and tooth formation
	Blood coagulation
	Muscle contraction
	Myocardial conduction
Symptoms of deficiency	Leg cramping
	Hypocalcemia and tetany
	Neuromuscular hyperexcitability
	Osteoporosis
Diagnosis	Increased parathyroid hormone
	Decreased 25-hydroxyvitamin D
	Decreased ionized calcium
	Decreased serum calcium (poor indicator of bone stores)
	Urinary cross-links and type 1 collagen telopeptides
	DEXA scan findings
Toxicity	Renal insufficiency, nephrolithiasis, impaired iron absorption

DEXA = dual-energy x-ray absorptiometry.

Table A7

Vitamin D	
DRI and UL	DRI 5 μg/d for adults, 10 μg/d for ages 50–70 yr, 15 μg/d for ages >70 yr; UL 50 μg/d 1 μg = 40 IU
Food sources	Fortified dairy products, fatty fish, eggs, fortified cereals
Function	Calcium and phosphorus absorption Resorption, mineralization, and maturation of bone Tubular resorption of calcium
Symptoms of deficiency	Osteomalacia Disorders of calcium deficiency: rachitic tetany
Diagnosis	Decreased 25-hydroxycholecalciferol Decreased serum phosphorus Increased serum alkaline phosphatase Increased parathyroid hormone Decreased urinary calcium Decreased or normal serum calcium
Treatment	50,000 IU/wk ergocalciferol (D_2) orally or intramuscularly, for 8 wk

Table A8

Vitamin A	
DRI and UL	DRI 700 μg/d females; 900 μg/d males; UL 3000 mg/d 1 RAE = 1 μg retinol, 12 μg B-carotene; for supplements, 1 RE = 1 RAE
Food sources	Liver, dairy products, fish, darkly colored fruits and leafy vegetables
Functions	Photoreceptor mechanism of the retina, format Integrity of epithelia Lysosome stability Glycoprotein synthesis Gene expression Reproduction and embryonic function Immune function
Early symptoms of deficiency	Nyctalopia, xerosis, Bitot's spots, poor wound healing
Advanced symptoms of deficiency	Corneal damage, keratomalacia, perforation, endophthalmitis, and blindness Xerosis and hyperkeratinization of the skin, loss of taste
Diagnosis	Decreased serum vitamin A Decreased plasma retinol Decreased retinal binding protein
Treatment	Without corneal changes: 10,000–25,000 IU/d vitamin A orally until clinical improvement (usually 1–2 weeks) With corneal changes: 50,000–100,000 IU vitamin A IM for 3 days followed by 50,000 IU/d IM for 2 weeks Evaluate for concurrent iron and/or copper deficiency which can impair resolution of vitamin A deficiency Potential antioxidant effects of carotene can be achieved with supplements of 25,000-50,000 IU of carotene
Toxicity	Hypercarotenosis results in staining of skin yellow-orange color but is otherwise benign; skin changes most marked on palms and soles; sclera remains white Hypervitaminosis A occurs after ingestion of daily doses of >50,000 IU/d for >3 mo; early manifestations include dry, scaly skin, hair loss, mouth sores, painful hyperostosis, anorexia, and vomiting Most serious findings include hypercalcemia, increased intracranial pressure, with papilledema, headaches and decreased cognition, and hepatomegaly, occasionally progressing to cirrhosis Excessive vitamin A has also been related to increased risk of hip fracture Diagnosis: elevated serum vitamin A levels Treatment: withdrawal of vitamin A from diet; most symptoms improve rapidly

RAE = retinol activity equivalent; RE = retinol equivalent; IM = intramuscularly.

Table A9

Vitamin E	
DRI and UL	DRI 15 mg/d; UL 1000 mg/d
Food sources	Vegetable oils, unprocessed cereal, grains, nuts, fruits, vegetables, meats
Function	Intracellular antioxidant Scavenger or free radicals in biologic membranes
Symptoms of deficiency	Hyporeflexia, disturbances of gait, decreased proprioception and vibration, ophthalmoplegia RBC hemolysis Neurologic damage Ceroid deposition in muscle Nyctalopia Muscle weakness Nystagmus
Diagnosis	Decreased plasma alpha-tocopherol Serum level of vitamin E should be interpreted in relation to circulating lipids Increased urinary creatinine Increased plasma creatine phosphokinase
Treatment	Optimal therapeutic dose of vitamin E has not been clearly defined; potential antioxidant benefits of vitamin E can be achieved with supplements of 100–400 IU/d
Toxicity	Large doses have been taken for extended periods without harm, although nausea, flatulence, and diarrhea have been reported; large doses of vitamin E can increase vitamin K requirement and can result in bleeding in patients taking oral anticoagulants

RBC = red blood count.

Table A10

Vitamin K	
DRI and UL	DRI 90 μg/d females; 120 μg/d males; UL not determinable
Food sources	Green vegetables, Brussels sprouts, cabbage, plant oils, and margarine
Function	Formation of prothrombin, other coagulation factors, and bone proteins
Symptoms of deficiency	Hemorrhage from deficiency of prothrombin and other factors Easy bruising Bleeding gums Heavy menstrual and nose bleeding Delayed blood clotting Osteoporosis
Diagnosis	Increased prothrombin time Reduced clotting factor Increased partial prothrombin time Decreased plasma phylloquinone Fibrinogen level, thrombin time, platelet count, and bleeding time are in normal range Increased des-gamma-carboxyprothrombin Also known as protein-induced in vitamin K absence Measured with antibodies
Treatment	Parenteral dose of 10 mg For chronic malabsorption, 1–2 mg/d, orally, or 1–2 mg/wk parenterally Note: if elevated prothrombin time does not improve, it is not due to vitamin K deficiency Note: Warfarin-type drugs inhibit conversion of vitamin K to its active form hydroquinone
Toxicity	High doses can impair actions of oral anticoagulants No known toxicity from dietary sources of vitamin K

Table A11

Zinc	
DRI and UL	DRI 8 mg/d females; 11 mg/d males; UL 40 mg/d
Food sources	Meat, liver, eggs, oysters, peanuts, whole grains
Function	Components of enzymes
	Synthesis of proteins, DNA, RNA
	Gene expression
	Skin and cell integrity
	Wound healing
	Reproduction and growth
Symptoms of deficiency	Hypogeusia (decreased taste sensation)
	Alterations in sense of smell
	Poor appetite
	Poor wound healing
	Irritability
	Impaired immune function
	Diarrhea
	Hair loss
	Muscle wasting
	Dermatitis
Diagnosis	Decreased plasma zinc
	Decreased serum zinc
	Decreased RBC or WBC zinc
	Decreased alkaline phosphatase
	Decreased plasma testosterone
Treatment	60 mg elemental zinc, orally twice daily
Toxicity	Acute toxicity causes nausea, vomiting, and fever
	Chronic large doses of zinc can depress immune function and cause hypochromic anemia as a result of copper deficiency

RBC = red blood cell; WBC = white blood cell.