Overview of Nutritional Deficiencies After Bariatric Surgery

81

Farzin Rashti, Ekta Gupta, Timothy R. Shope, and Timothy R. Koch

Contents

Abstract

The growing worldwide epidemic of obesity has become a major concern because of its extensive medical, social, and economic consequences. As the prevalence of obesity continues to rise, it becomes increasingly important to understand the differences in medical management for those obese patients who have undergone bariatric surgery. Obese patients after bariatric surgery often have multiple medical comorbidities, making their care more complex and often requiring a higher level of acuity in the intensive care unit. The type of bariatric surgery that has been performed influences the risk of developing nutritional deficiencies. Common micronutrient deficiencies after bariatric surgery are outlined herein. Recognition of specific organ-based syndromes related to micronutrient deficiencies is important for the prevention of permanent end-organ damage. Treatment of micronutrient deficiencies after bariatric surgery is summarized.

List of Abbreviations

AGB	Adjustable gastric banding
BMI	Body mass index
BPD	Biliopancreatic diversion
ICU	Intensive care unit
RYGB	Roux-en-Y gastric bypass
SG	Vertical sleeve gastrectomy

F. Rashti (✉) • E. Gupta • T.R. Shope • T.R. Koch
Section of Gastroenterology and Hepatology and Department of Surgery, MedStar Washington Hospital Center and Georgetown University School of Medicine, Washington, DC, USA
e-mail: farzinrashti@gmail.com; Ekta.Gupta@gunet.georgetown.edu; Timothy.R.Shope@MedStar.net; timothy.r.koch@medstar.net; timkoch@live.com

R. Rajendram et al. (eds.), *Diet and Nutrition in Critical Care*,
DOI 10.1007/978-1-4614-7836-2_79

Introduction

Average body mass index (BMI; weight in kilograms/square of height in meters) has been trending upward worldwide. From 1980 to 2008, worldwide average BMI has increased by 0.4 kg/m^2 per decade, with the largest mean BMI scores in high-income countries (Finucane et al. 2011).

The National Institutes of Health classifies obesity based on BMI (see Table 1). Although there are limitations to BMI, it is a widely accepted tool which parallels body fat and provides a generalized measure of obesity. Multiple chronic medical conditions can occur in obese individuals, which can lead to a higher risk of hospitalization and admission to the intensive care unit (ICU). Obese patients have approximately two times greater risk of mortality in the ICU because of complications (Bercault et al. 2004).

Among treatment options for medically complicated obesity, bariatric surgery has consistently been shown to be superior to intensive medical programs (Sjostrom et al. 2004; Adams et al. 2012). Patients with bariatric surgery present unique challenges. An understanding of the basic underlying nutritional deficiencies for which these patients are most susceptible will assist multidisciplinary teams in providing optimal care.

Macronutrients include fat, protein, and carbohydrates; these dietary components are required for chemical energy and for tissue and cellular structure. Micronutrients are dietary factors required for biochemical and cellular processes (see Table 2). Micronutrients are required in microgram or milligram quantities in a large group of biochemical pathways and metabolic processes.

Adequate nutritional support is a vital component in the management of critically ill patients in the ICU. Nutritional support is altered by various bariatric surgical procedures which can inhibit absorption of micronutrients. This chapter will provide an overview of the most common types of bariatric surgeries, nutritional deficiencies associated with those procedures, and options for nutritional support for bariatric patients during critical illness.

Types of Bariatric Surgery and Their Effect on Micronutrients

In development of bariatric surgery, Kremen studied surgery for treatment of obesity in dogs and subsequently performed a jejunoileal bypass on an individual (Kremen et al. 1954). Since 1998, bariatric surgery has become a widely available and common treatment for morbid obesity (Scopinaro 1998). The number of bariatric surgeries performed internationally has increased to greater than 340,000 bariatric surgeries yearly (Buchwald and Oien 2013).

The anatomical configurations of bariatric surgical procedures are outlined in Fig. 1. Roux-en-Y gastric bypass (RYGB) remains the most commonly performed bariatric surgical procedure worldwide. Adjustable gastric banding (AGB) has

Table 1 Classification of obesity (Adapted from the National Institutes of Health, National Heart, Lung And Blood Institute)

	BMI (KG/M^2)	Obesity class	Disease risk relative to normal weight[a]	Weight and waist circumference
Underweight	<18.5	–	–	–
Normal	18.5–24.9	–	–	–
Overweight	25–29.9	–	Increased	High
Obesity	30–34.9	I	High	Very high
Obesity	35–39.9	II	Very high	Very high
Extreme Obesity	>40	III	Extremely high	Extremely high

BMI body mass index
[a]Disease risk for: diabetes mellitus type 2, hypertension, and cardiovascular disease

Table 2 Micronutrients required for biochemical and cellular processes

Micronutrients	Examples
Small-molecule antioxidants	Glutathione
Trace elements	Copper, zinc, manganese, selenium, chromium
Essential minerals	Iodine, iron, calcium
Water-soluble vitamins	Vitamin B1 (thiamine), vitamin B2 (riboflavin), vitamin B3 (niacin), vitamin B6 (pyridoxine), folic acid, pantothenic acid, biotin, vitamin B12, vitamin C
Fat-soluble vitamins	Vitamin A, vitamin D, vitamin E, vitamin K

been a popular procedure in Europe. However, in 2011, there was an increase in the number of patients undergoing vertical sleeve gastrectomy (SG) (Buchwald and Oien 2013).

AGB was first introduced in the 1980s. This procedure does not involve reduction or stapling of any portion of the stomach or bypass of the small intestine. After AGB, deficiencies of vitamin D, zinc, and copper should be considered (see Table 3).

SG is gaining popularity worldwide as a bariatric procedure. It involves surgical excision of the gastric fundus and body, leaving a narrow, tubular stomach along the lesser curve of the stomach. SG is irreversible and leads to restriction of caloric intake. Reported micronutrient deficiencies after SG are summarized in Table 3.

Comprising nearly 50 % of the worldwide bariatric surgeries, RYGB is most commonly performed (Buchwald and Oien 2013). A small gastric pouch is created and attached directly to the jejunum, bypassing a large portion of the stomach, duodenum, and proximal jejunum. There are three channels created: an upper continuous limb (the Roux limb), the biliopancreatic limb, and the common channel (connecting the jejunal-enteric anastomosis to the colon). Patients with a short common channel (<120 cm from the ileocecal valve) are at greater risk of developing severe malabsorption (Bal et al. 2012). Exclusion of the stomach after RYGB leads to vitamin B12 deficiency, while exclusion of the duodenum leads to iron deficiency. Micronutrient deficiencies reported after RYGB are summarized in Table 3.

Biliopancreatic diversion (BPD) was initially developed in the mid-1970s (Scopinaro et al. 1979). It involves a distal stomach resection with closure of the duodenal stump. The jejunum is divided and the distal limb is anastomosed to the proximal stomach, while the proximal limb is anastomosed to the ileum. There is a short common channel, where pancreatic secretions mix with dietary components. Due to nutritional disorders, BPD is not commonly performed.

Biliopancreatic diversion with duodenal switch was developed to preserve the pylorus and control gastric emptying. To help maintain the restrictive component of this procedure, a partial gastrectomy is performed in a sleeve configuration, involving a 70–80 % greater curve gastrectomy (Sudan and Jacobs 2011).

In a gastroplasty, a vertical pouch is created along the proximal lesser curvature of the stomach by separating the fundus with a stapled partition. The outlet of the pouch is reinforced with a narrow band of polypropylene mesh. Postoperative failure was commonly attributed to breakdown of the staple line. This procedure is now <1 % of all bariatric procedures.

Macronutrients

Bariatric surgery can cause protein malnutrition. National guidelines recommend an average of 60–120 g of daily protein intake in patients after bariatric surgery, to maintain lean body mass (Heber et al. 2010). Protein malnutrition is a potentially serious complication. Alopecia can be an early clinical manifestation of protein deficiency. Long-term manifestations include (similar to kwashiorkor): muscle mass wasting and anasarca, emaciation, depigmented hair, and findings of anemia and hypoalbuminemia (Lewandowski et al. 2007). Since albumin is an acute phase reactant, its serum levels can be affected by inflammation and inadequate protein and caloric intake (Gehring et al. 2006). In bariatric patients presenting with hypoalbuminemia, inflammatory processes, including an acute inflammatory

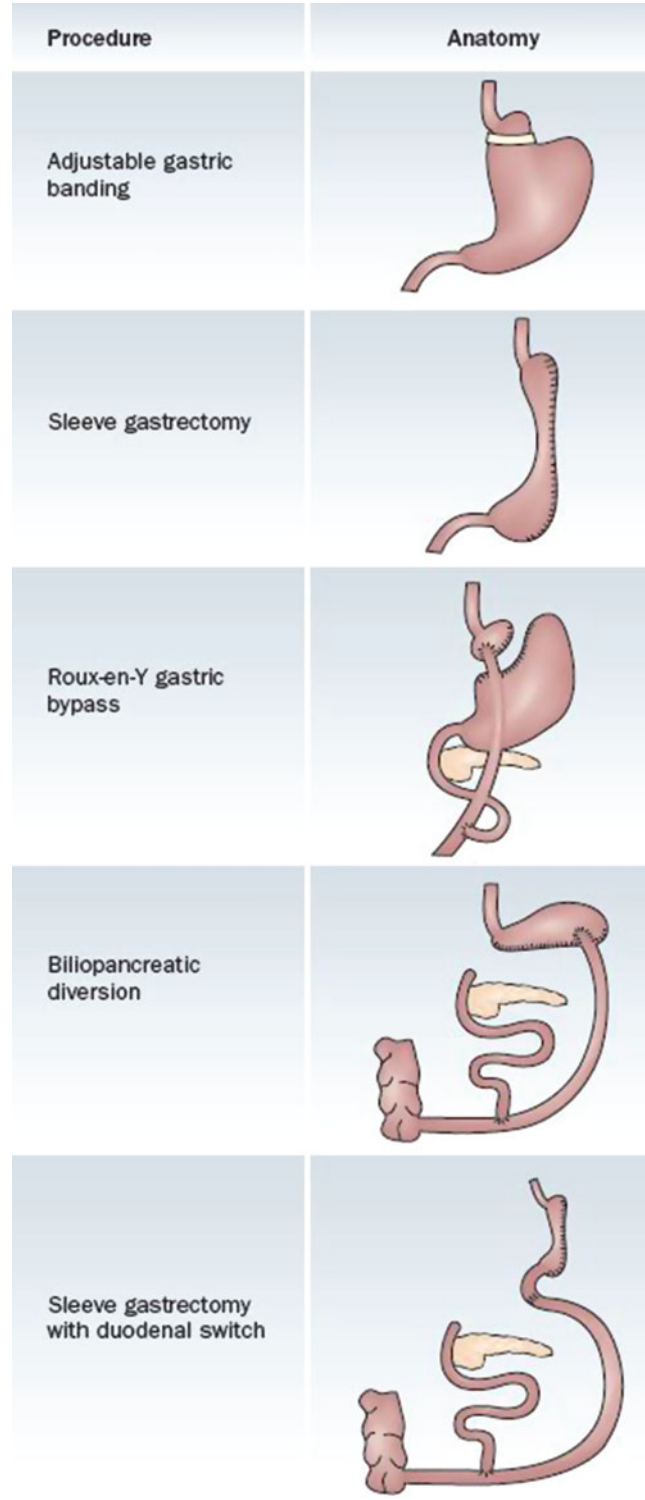

Fig. 1 Anatomical comparison of bariatric surgical procedures (Reproduced with the permission of Nature Publishing Group from Bal BS, et al. Nature Rev Endocrinol 2012; 8: 544–556)

Table 3 Common micronutrient deficiencies after bariatric surgery

Bariatric surgery	Micronutrient deficiency
Adjustable gastric banding	Vitamin D, zinc, copper
Vertical sleeve gastrectomy	Vitamin D, vitamin B12, thiamine, folic acid, vitamin B6, zinc, iron
Roux-en-Y gastric bypass	Vitamin B12, iron, vitamin D, thiamine, vitamin A, zinc, copper

disorder, chronic liver disease, and small intestinal bacterial overgrowth, are considered (Sriram et al. 2012).

The utility of liquid protein supplements to treat protein malabsorption after bariatric surgery remains unclear, because there are no adequately controlled studies. Enteral feedings with liquid protein supplements should be attempted before using total parenteral nutrition, given the overall risks. Patients experiencing severe protein malnutrition with anasarca and severe muscle wasting should consider surgical consultation for possible revision. The surgical operative report must be reviewed to evaluate the length of the common channel.

Micronutrients

Many bariatric centers and the Endocrine Society recommend daily: one or two tablets of a chewable multivitamin-mineral preparation, 1,200–2,000 mg of elemental calcium, and (after repletion of vitamin D) 1,000 IU of oral vitamin D (Heber et al. 2010).

An individual after bariatric surgery may present with multiple micronutrient deficiencies. Patients with micronutrient deficiencies may present with clinical symptom complexes as summarized in Table 4. Common clinical symptom complexes include visual symptoms or loss of vision, neurological symptoms, edema, dermatological conditions, anemia, and, uncommonly, bleeding disorders. The temporal development of symptoms may not be relevant, since patients could have had micronutrient deficiencies prior to bariatric surgery.

Table 4 Clinical symptom complexes and micronutrient investigations after bariatric surgery

Clinical symptom complex	Laboratory testing
Visual symptoms	Vitamin A; vitamin E; whole blood thiamine; copper
Neurological symptoms	Vitamin B12; whole blood thiamine; vitamin E; vitamin B2; vitamin B6; plasma niacin; copper; magnesium
Edema	Whole blood thiamine; plasma niacin; selenium
Dermatological disorders	Vitamin A; vitamin B2; vitamin B6; zinc; plasma niacin
Anemia	Ferritin; vitamin B12; folate; zinc; copper; vitamin A; vitamin E
Bleeding disorders	Complete blood count; prothrombin time

Table 5 Bariatric surgery and micronutrient deficiencies

Vitamins/ minerals/trace elements	Inadequate body stores	Dose
Thiamine (vitamin B1)	Beriberi	100 mg twice daily; in Wernicke encephalopathy or acute psychosis: 250 mg for minimum 3–5 days, intramuscularly or intravenously
Riboflavin (vitamin B2)	Ariboflavinosis	5–10 mg daily
Niacin (vitamin B3)	Pellagra	100–500 mg three times daily
Pantothenic acid (vitamin B5)	Pantothenic acid deficiency	2–4 g daily
Pyridoxine (vitamin B6)	Pyridoxine deficiency	30 mg daily
Biotin (vitamin B7)	Biotin deficiency	20 mg daily
Folic acid (vitamin B9)	Folate deficiency	1–5 mg daily
Cobalamin (vitamin B12)	Pernicious anemia	0.5–2.0 mg orally or 500 mcg

(*continued*)

Table 5 (continued)

Vitamins/ minerals/trace elements	Inadequate body stores	Dose
		sublingually, both daily; 1,000 mcg intramuscularly monthly
Ascorbic acid (vitamin C)	Scurvy	200 mg daily
Vitamin A	Vitamin A deficiency	10,000 IU daily
Vitamin D	Osteomalacia	Ergocalciferol 50,000 IU once weekly over 12 weeks, then daily cholecalciferol 1,000–5,000 IU
Vitamin E	Vitamin E deficiency	800–1,200 IU daily
Vitamin K	Vitamin K deficiency	2.5–25.0 mg daily
Calcium	Osteoporosis	1.2–2.0 g daily
Iron	Iron deficiency anemia	Ferrous sulfate 325 mg or ferrous fumarate 200 mg and vitamin C 125 mg up to four times daily
Zinc	Hypozincemia	Zinc sulfate 220 mg or zinc gluconate 30–50 mg, daily
Copper	Hypocupremia	Copper gluconate (2–4 mg) daily
Selenium	Keshan disease	100 μg sodium selenite daily

The treatment of micronutrient deficiencies is summarized in Table 5. As an aggressive approach in critically ill patients who present with a symptom complex, vitamins (water soluble and fat soluble) and trace elements can be mixed daily in 500 ml of 5 % dextrose in water and then given intravenously at 50 ml/h (Bal et al. 2012). For patients with a sudden loss of vision, a daily infusion of thiamine-HCl 250 mg with a combination of commercially available trace elements (zinc 5 mg, copper 1 mg, manganese 0.5 mg, selenium 60 mcg, and chromium 10 mcg) mixed in 500 ml of 5 % dextrose in water can be given intravenously (Bal et al. 2012).

Water-Soluble Vitamins

Most water-soluble vitamins are generally not stored in the body for long periods of time, and there is greater loss through urinary excretion. Most B-complex vitamins and vitamin C have stores of less than 2 months within the human body, but vitamin B12 may maintain adequate stores for up to 3 years.

Thiamine. Thiamine (vitamin B1) serves as an important coenzyme for the tricarboxylic acid cycle and the pentose phosphate pathway (Sriram et al. 2012). In critically ill patients, thiamine deficiency has been associated with a higher overall mortality rate, and thiamine deficiency is seen in up to 20 % of patients (Cruickshank et al. 1988).

Absorption of thiamine is mainly in the jejunum and ileum by an active, carrier-mediated, rate-limited process. Thiamine deficiency is common in patients after bariatric surgery (termed bariatric beriberi) (Carrodeguas et al. 2005; Lakhani et al. 2008) and may be found as early as 4 weeks after surgery (Sriram et al. 2012). The European Federation of Neurological Societies recommends evaluation of thiamine status in bariatric patients for at least 6 months and administration of parenteral thiamine (Galvin et al. 2010).

Thiamine deficiency (see Table 6) was originally described in patients with neuropsychiatric, neurologic, cardiac, and gastrointestinal symptoms that corrected with "beriberi factor" (e.g., thiamine). The World Health Organization supports thiamine deficiency in at risk individuals with two findings among three categories: bilateral lower extremity edema, dyspnea with exertion or at rest, or paresthesias of the extremities (tingling, itching, and burning sensations or decreased motor strength) (Prinzo 1999).

During testing, whole blood levels of thiamine reflect a small percentage of total-body thiamine. Thiamine stores may be determined through measurement of the catalytic activity of transketolase in erythrocytes (its catalytic activity is dependent

Table 6 Signs and symptoms of thiamine deficiency

Deficiency subtype	Signs and symptoms
Gastrointestinal	Nausea, vomiting, constipation
Cardiac	Lower extremity edema, tachycardia, dyspnea with exertion or at rest, L-lactic acidosis
Neurologic	Tingling, itching, and burning sensations; decreased motor strength of extremities
Neuropsychiatric	Nystagmus, ophthalmoplegia, blurred vision, psychosis, confusion, acute visual loss, ataxia

on binding to thiamine pyrophosphate) (Herve et al. 1995).

Standard therapy for deficiency includes thiamine-HCl 100 mg taken orally twice daily. If there is then no improvement, treatment of small intestinal bacterial overgrowth with adjuvant antibiotic therapy should be considered (Lakhani et al. 2008).

Symptoms of thiamine deficiency requiring emergency parenteral thiamine include psychosis or confusion, acute change in vision or visual loss, ophthalmoplegia, ataxia, respiratory distress, or L-lactic acidosis. These patients may require supportive care in the intensive care unit, as well as thiamine administration by an intravenous infusion (over 3–4 h to prevent anaphylactic reactions) or intramuscular injection of a minimum of 250 mg for at least 3–5 days (Thomson and Marshall 2006).

Riboflavin. Riboflavin (vitamin B2) is essential for flavocoenzymes, flavin adenine dinucleotide, and flavin mononucleotide. These enzymes maintain the proper functioning of glutathione peroxidase (metabolism of hydroperoxides) and (Glutathione Reductase is a flavin enzyme that catalyzes reaction of glutathione (oxidized) disulfide with NADPH to form 2 glutathiones (reduced) and NAD+).

Riboflavin is absorbed by the small intestine and produced by colonic bacteria from a high-fiber diet. Although there have been findings of biochemical riboflavin deficiency after bariatric surgery (Clements et al. 2006), this dual absorption mechanism may explain the rarity of deficiency. Riboflavin deficiency includes glossitis, cheilosis, angular stomatitis, seborrhea-like dermatitis, pruritus, alopecia, anemia, and degenerative changes of the nervous system.

Niacin. Niacin (vitamin B3) includes nicotinic acid and nicotinamide. Nicotinic acid is converted into nicotinamide, a component of nicotinamide adenine dinucleotide (involved in catabolic reactions) and nicotinamide adenine dinucleotide phosphate (involved in anabolic reactions) (Combs 2008). Reliable and sensitive measures of niacin status include urinary excretion of the methylated metabolites N-methylnicotinamide and its 2-pyridone derivative, *N*-methyl-2-pyridone-5-carboxamide (Kumar 2007). Diagnosis of niacin deficiency is supported by low plasma levels of niacin and symptomatic improvement after niacin supplementation.

Niacin deficiency (pellagra) involves neurologic, dermatologic, and gastrointestinal symptoms, including delusions or hallucinations, headaches, ataxia or myoclonus, anxiety, depression, scaly dermatitis, malabsorptive disorders, or diarrheal illnesses (colitis). Treatment of pellagra involves 100–500 mg of oral niacin, three times daily. Intramuscular injections of 100 mg of nicotinamide may be administered three times daily (Kumar 2007).

Pantothenic Acid. Pantothenic acid (vitamin B5) is required for the function of coenzyme A (Kelly 2011). Pantothenic acid deficiency leads to depression, infections, orthostatic hypotension, gait disorders, and paresthesias. Deficiency is treated with 2–4 g of oral pantothenic acid daily (Bean et al. 1955).

Vitamin B6. The biologically active form of vitamin B6, or pyridoxal phosphate, is a coenzyme in several reactions including degradation of homocysteine. Although symptoms are infrequent, patients may present with microcytic hypochromic anemia, pellagra-like dermatitis, and painful distal paresthesias (Kumar 2007). One study suggested only a modest decrease in vitamin B6 levels after laparoscopic SG and suggested the need to control and provide supplementation for deficiencies (Damms-Machado et al. 2012). Excessive intake of Vitamin B6 is associated with the development of symptoms of a peripheral neuropathy.

Biotin. Biotin (vitamin B7) is an essential coenzyme in the metabolism of fatty acids,

amino acids, and glucose. Biotin deficiency may present with symptoms including seizures, hypotonia, ataxia, hair loss, and dermatitis (Zempleni et al. 2008). There has been a report of a patient presenting with loss of taste after SG with resolution after treatment with high doses (20 mg daily) of biotin (Greenway et al. 2011).

Folic Acid. Folic acid, or vitamin B9, is a water-soluble B vitamin that serves as an enzyme cofactor necessary for the synthesis of purine and thymidine nucleotides. Patients with bariatric surgery often present with normocytic, mixed anemia with an increased red cell distribution width. Some of the symptoms may include weakness, anorexia, and weight loss. In patients presenting with folate deficiency after bariatric surgery, other small intestinal malabsorptive disorders should be considered, including celiac disease and a short common channel. Treatment of folate deficiency includes 1–5 mg of oral folic acid daily.

Vitamin B12. Surgical procedures involving an antrectomy as part of BPD and exclusion of the antrum and corpus as part of RYGB decrease physiological function by bypassing parietal cell mass. Parietal cells produce hydrochloric acid and intrinsic factor. Bariatric surgery with exclusion of the parietal cells causes a relative achlorhydria and interferes with the pH-dependent process of releasing vitamin B12 from food particles. Vitamin B12 is absorbed in the distal ileum by specific receptors that recognize the vitamin B12-intrinsic factor complex. Another cause of deficiency after bariatric surgery is bacterial utilization of vitamin B12 in proximal small intestine.

Vitamin B12 deficiency has been reported in up to 33.3 % of patients after 2 years and 61.8 % of patients greater than 5 years after RYGB (Dalcanale et al. 2010). Clinical manifestations of vitamin B12 include pernicious anemia, myelopathy, neuropathy, dementia, and depression (Saltzman and Karl 2013). A low-normal blood level of vitamin B12 suggests the presence of vitamin B12 deficiency, which is supported by a concomitant elevation of serum methylmalonic acid (Herrmann and Obeid 2008). Treatment of vitamin B12 deficiency includes oral vitamin B12 (cyanocobalamin) 500–2,000 mcg per day; intramuscular vitamin B12 1,000 mcg monthly; nasal vitamin B12 500 mcg weekly; or sublingual vitamin B12 500 mcg daily (Herrman and Obeid 2008).

Vitamin C. Humans depend on diet as a source of vitamin C (Traber and Stevens 2011). The concentrations of vitamin C in plasma and tissues are tightly controlled and regulated through absorption, tissue accumulation, and renal reabsorption.

Vitamin C deficiency is termed scurvy and is one of the oldest diseases reported in human history. Early manifestations include malaise, myalgias, edema, and petechiae and can progress to suppurative wounds, loss of teeth, neuropathy, and death (Magiorkinis et al. 2011). Vitamin C deficiency is treated with 200 mg of oral ascorbic acid taken daily.

Fat-Soluble Vitamins

Vitamin A. Vitamin A includes a group of nutritionally unsaturated hydrocarbons including retinols and carotenoids, which include β-carotenes. Excessive intake of vitamin A may cause alopecia, blurry vision, osteoporosis, muscle weakness, and hepatitis or liver damage (Chapman 2012).

Vitamin A deficiency has been demonstrated as a common finding in patients after bariatric surgery, most often after procedures with a short common channel including BPD, DS, or RYGB (Clements et al. 2006; Hatizifotis et al. 2003; Eckert et al. 2010). Mechanisms of vitamin A deficiency likely include fat-soluble vitamin malabsorption induced by a relative bile acid deficiency in the bypassed duodenojejunal segment and deconjugation of bile acids by bacterial overgrowth. Patients with vitamin A deficiency may describe reduced vision in low light, poor night vision (nyctalopia), keratomalacia, pruritus, and dry hair. Treatment of vitamin A deficiency is oral vitamin A supplementation with 10,000 IU daily as well as co-therapy for any concurrent iron deficiency. Beta-carotene, a previtamin A analogue, is a viable alternative in the treatment of vitamin A deficiency.

Vitamin D. Absorption of vitamin D is facilitated by bile salts in the small intestine with an absorption efficiency of approximately 50 %.

Vitamin D is an essential regulator of calcium metabolism in humans and helps maintain appropriate calcium and phosphate levels needed for bone formation. Vitamin D affects the functioning of parathyroid hormone by promoting absorption of calcium from the small intestine (Bell et al. 2010).

Patients with medically complicated obesity frequently have vitamin D deficiency during preoperative evaluation (DiGiorgi et al. 2008). Vitamin D deficiency is a cause of metabolic bone disease after bariatric surgery leading to long-term bone loss and a risk of fractures (Johnson et al. 2005). Patients with BPD and RYGB may have a relative bile salt deficiency in the bypassed duodenojejunal segment and a short common channel where food is allowed to mix with bile (and, therefore, bile salts) (Goldner et al. 2002; Slater et al. 2004). Finally, small intestinal bacterial overgrowth may interfere with vitamin D absorption.

Patients with vitamin D deficiency may present with bone pain and tenderness, myalgias, proximal weakness, and difficulty walking (Saltzman and Karl 2013). Vitamin D deficiency is diagnosed by measurement of serum levels of total 25-hydroxyvitamin D. Treatment consists of oral ergocalciferol 50,000 IU once weekly for 6–8 weeks with reevaluation of the serum 25-hydroxyvitamin D levels after 8–12 weeks. A maintenance dose of 1,000–2,000 IU of 1,25-dihydroxyvitamin D3 (cholecalciferol) taken with meals is then recommended. The treatment of osteomalacia requires 600,000 IU of ergocalciferol. Oral ergocalciferol 50,000 IU taken daily for 12 days is tolerated (Bhan et al. 2012).

Vitamin E. The vitamin E family includes tocopherols and tocotrienols (Borel et al. 2013). Deficiencies in vitamin E may cause hemolytic anemia, retinopathy, and neurologic abnormalities including ataxia, muscle weakness, dysarthria, and loss of vibration sensation and proprioception (Niki and Traber 2012). Vitamin E deficiency after bariatric surgery has not been well studied. Oral doses of vitamin E 400–1,000 mg per day (0.67 mg of vitamin E equals 1 IU) are well tolerated (Bendich and Machlin 1988).

Vitamin K. Vitamin K is a naturally occurring compound found in plant form (phylloquinone) and several bacterial menaquinones. With normal anatomy and health, vitamin K is moderately well absorbed (40–70 %) from the jejunum and ileum (Shearer et al. 1974). Normal bacterial flora in the human intestines synthesizes vitamin K and serves as an additional source.

The only clearly defined health risk associated with vitamin K deficiency is an increased risk of bleeding disorders. There are several reports of adverse outcomes in neonates with maternal bariatric surgery attributed to vitamin K deficiency including intracranial hemorrhage (Eerdekens et al. 2010; Kang et al. 2010). Deficiency is treated with oral vitamin K 2.5–25 mg daily or parenteral vitamin K 5–15 mg given intramuscularly or subcutaneously daily.

Essential Minerals

The most extensive data on essential minerals after bariatric surgery relates to iron and calcium. The long-term deficiencies of other minerals after bariatric surgery have not yet been fully studied.

Calcium. Calcium is the most abundant mineral in the human body and is essential for normal cell physiology. Calcium absorption may be directly impaired in the presence of excess intraluminal fat, which may be found in some patients after bariatric surgery with a short common channel (Whelton et al. 1971). Calcium deficiency may lead to osteoporosis and an increased risk of fractures due to increased bone resorption and loss from secondary hyperparathyroidism (Grant et al. 2005).

Patients with calcium and/or vitamin D deficiency after bariatric surgery may present with paresthesias, tetany, muscle cramps, back pain, and bony pain. Isolated serum calcium measurements are an unreliable measure of calcium metabolism (Dewey and Heuberger 2011). In cases of elevated alkaline phosphatase where the major component is from the bone rather than the liver, serum parathyroid levels should also be measured. Treatment of calcium deficiency requires administration of $\geq$1.2 g of oral calcium daily and also requires correction of any concurrent vitamin D deficiency.

Iodine. Bariatric surgery may provide an additional benefit of improvement or resolution of

subclinical hypothyroidism and hypothyroidism. The reduction in thyroxine requirement is likely attributable to a decrease in BMI (Chikunguwo et al. 2007).

Iron. Anemia is common in patients after bariatric surgery. Approximately 36 % of patients have been noted to have anemia ≥1 year after RYGB (Cable et al. 2011), 13 % of patients after BPD (Pata et al. 2013), 5 % of patients 1 year after SG (Hakeam et al. 2009), and 1.5 % of patients 5 years after AGB (Boza et al. 2011). Iron is absorbed mainly in the duodenum and proximal jejunum, intestinal sites that are bypassed with RYGB and BPD. Furthermore, an acidic environment helps to improve absorption of nonheme iron, but there is relative achlorhydria after RYGB.

Evaluation of iron deficiency anemia in a patient after bariatric surgery may require further examination for other causes, including gastrointestinal cancers or a malabsorption disorder. Treatment of iron deficiency in bariatric surgery patients includes either 150–200 mg daily of oral elemental iron in any preparation best tolerated by the patient (ferrous gluconate, sulfate, or fumarate) or a ferrous salt-vitamin C combination. Parenteral iron is occasionally necessary, especially in premenopausal women.

Magnesium. Magnesium plays a critical role in cellular functions. Magnesium deficiency is common in obese patients being evaluated for bariatric surgery with up to 31 % having a magnesium deficiency at baseline (Jastrzebska-Mierzynska et al. 2012).

Patients with magnesium deficiency may present with symptoms of muscle weakness, tetany, seizures, and ventricular arrhythmias. Patients with signs, symptoms, or complications of magnesium deficiency may benefit from empiric treatment with magnesium. Moderate to severe magnesium deficiency should be treated parenterally with 48 mEq of magnesium (8 mEq of elemental magnesium equals 1 g of magnesium sulfate-heptahydrate) given intravenously as a 50 % solution for 3–7 days to replete total-body stores. Patients presenting with seizures or acute cardiac dysrhythmias may be given 8–16 mEq of magnesium over 5–10 min (Tong and Rude 2005).

Trace Elements

Trace elements are vital for maintaining health and serve as cofactors in antioxidant enzymes and proteins. There is often a narrow safety range between deficiency and toxicity concentrations of trace elements, given their low daily requirements.

Chromium. Chromium is believed to be an essential trace element in humans (Moukarzel 2009). Three case reports have been published of patients developing peripheral neuropathy and glucose intolerance while on long-term total parenteral nutrition after massive bowel resection; their symptoms improved after adding chromium supplementation to their parenteral nutrition (Jeejeebhoy et al. 1977). Treatment of chromium deficiency can include an infusion of 200–250 mcg of chromium daily for up to 14 days.

Copper. Copper is a trace metal that requires an acidic environment to facilitate its release from food complexes and to support absorption in the small intestine. Hypocupremia has been reported in up to 68 % of morbidly obese patients being evaluated for bariatric surgery (de Luis et al. 2011). After bariatric surgery, serum copper levels initially decrease (de Luis et al. 2011; Gletsu-Miller et al. 2012) and then start a slow upward trend in 5-year studies (Balsa et al. 2011).

Clinical symptoms/findings reported in patients with hypocupremia include neurologic dysfunction, anemia, neutropenia, pancytopenia, and the appearance of a myelodysplastic syndrome (Halfdanarson et al. 2008; Jaiser and Winston 2010). Treatment of hypocupremia may require oral copper gluconate 4–8 mg daily with close monitoring. Neurological symptoms are not fully reversible using copper supplementation in patients with hypocupremia (Kelkar, et al. 2008), supporting the importance of evaluating other micronutrients. A RYGB patient has noted improvement in neurologic symptoms after surgical revision of the bypassed jejunum (Juhasz-Pocsine et al. 2007).

Manganese. Manganese is an important cofactor in superoxide dismutase, an antioxidant enzyme

localizing to the mitochondria. In animal models, manganese has been shown to be essential for survival (Holley et al. 2012). There are no reports of manganese deficiency after bariatric surgery.

Selenium. Selenium is a trace element required for production of selenoproteins. Serum selenium levels have been noted to be significantly reduced in morbidly obese females (Alasfar et al. 2011). Selenium deficiency is also associated with cardiomyopathy, termed Keshan disease. Selenium deficiency has been described after bariatric surgery and patients can present with life-threatening heart failure (Boldery et al. 2007). Treatment of selenium deficiency includes oral sodium selenite, 100 mcg daily.

Zinc. Zinc is the second most prevalent trace element in humans and is essential for normal cellular function and metabolism. Zinc is absorbed mainly in the duodenum and proximal jejunum (Shankar et al. 2010), making hypozincemia more common in patients with bariatric surgical procedures involving bypass of the small intestine (BPD, DS, and RYGB).

Symptoms of zinc deficiency include poor immunity and wound healing, alopecia, hypoalbuminemia, anemia, skin eruptions (acrodermatitis enteropathica), and neuropsychological disorders. Patients with acrodermatitis enteropathica may respond to treatment with zinc sulfate 1,000 mg/day for 7 days (Cunha et al. 2012). Moderate forms of zinc deficiency may be treated with 30 mg zinc to 50 mg of zinc (per 220 mg of zinc sulfate) taken by mouth 30–60 min before a meal (Bae-Harboe et al. 2012).

Applications to Critical or Intensive Care

The prevalence of obesity is rising internationally. Obesity has been associated with multiple chronic medical conditions, which predisposes these patients to a higher risk of hospitalization and admission to the intensive care unit. Obese patients have approximately two times greater risk of mortality in the intensive care unit due to a higher risk of acquired complications. Nutritional support in the intensive care unit is altered by various bariatric surgical procedures which can inhibit adequate absorption of micronutrients. This chapter summarizes the clinical symptoms and the clinical symptom complexes caused by macronutrient and micronutrient deficiencies that are seen in individuals who have undergone bariatric surgery. The treatment of both clinical symptoms and clinical symptom complexes is summarized in this chapter.

Applications to Other Conditions

Bariatric surgical procedures can inhibit adequate absorption of macronutrients and micronutrients. This chapter summarizes the frequent occurrence of malnutrition after bariatric surgery and describes reasons why individual bariatric procedure may predispose to specific micronutrient deficiencies. An approach is outlined in this chapter for the treatment of both clinical symptoms and clinical symptom complexes. This approach is applicable to any hospitalized patient who has previously undergone bariatric surgery.

Guidelines and Protocols

Postoperative guidelines for macronutrient and micronutrient support are discussed with all bariatric surgery patients. At hospital admission or transfer, a bariatric surgery patient's compliance with these macronutrient and micronutrient guidelines can therefore be assessed, and a lack of compliance may provide initial important suggestions with regard to potential sources of malnutrition.

See Fig. 1 for an overview about the type of bariatric surgery that could have been performed on the patient. Understanding the type of bariatric surgery that was performed on the patient will provide information about whether the patient had a primary restrictive surgical procedure or a combined restrictive and malabsorptive surgical procedure. Malabsorptive surgical procedures are more likely to result in malnutrition.

Table 4 can be used to evaluate the range of micronutrient deficiencies that may result in specific clinical symptom complexes, including visual

symptoms, neurological symptoms, edema, dermatological disorders, anemia, and bleeding disorders.

In Table 5, treatment protocols for specific micronutrient deficiencies are summarized.

Summary Points

- There has been an international rise in the prevalence of obesity.
- Bariatric surgery is a superior treatment option for those individuals with medically complicated obesity.
- Macronutrient and micronutrient deficiencies frequently occur after bariatric surgery.
- After bariatric surgery, specific micronutrient deficiencies are treated with the appropriate micronutrient.
- After bariatric surgery, clinical symptom complexes in critically ill individuals may require supportive care with intravenous infusion of a mixture of fat-soluble vitamins, water-soluble vitamins, and trace elements, which include copper, zinc, manganese, chromium, and selenium.

References

Adams TD, Davidson LE, Litwin SE, Hunt SC. Gastrointestinal surgery: cardiovascular risk reduction and improved long-term survival in patients with obesity and diabetes. Curr Atheroscler Rep. 2012;14(6):606–15.

Alasfar F, Ben-Nakhi M, Khoursheed M, Kehinde EO, Alsaleh M. Selenium is significantly depleted among morbidly obese female patients seeking bariatric surgery. Obes Surg. 2011;21(11):1710–3.

Bae-Harboe YS, Solky A, Masterpol KS. A case of acquired zinc deficiency. Dermatol Online J. 2012;18(5):1.

Bal BS, Finelli FC, Shope TR, Koch TR. Nutritional deficiencies after bariatric surgery. Nat Rev Endocrinol. 2012;8(9):544–56.

Balsa JA, Botella-Carretero JI, Gomez-Martin JM, et al. Copper and zinc serum levels after derivative bariatric surgery: differences between Roux-en-Y Gastric bypass and biliopancreatic diversion. Obes Surg. 2011;21(6):744–50.

Bean WB, Hodges RE, Daum K. Pantothenic acid deficiency induced in human subjects. J Clin Invest. 1955;34(7, Part 1):1073–84.

Bell TD, Demay MB, Burnett-Bowie SA. The biology and pathology of vitamin D control in bone. J Cell Biochem. 2010;111(1):7–13.

Bendich A, Machlin LJ. Safety of oral intake of vitamin E. Am J Clin Nutr. 1988;48(3):612–9.

Bercault N, Boulain T, Kuteifan K, Wolf M, Runge I, Fleury JC. Obesity-related excess mortality rate in an adult intensive care unit: a risk-adjusted matched cohort study. Crit Care Med. 2004;32(4):998–1003.

Bhan A, Rao AD, Rao DS. Osteomalacia as a result of vitamin D deficiency. Rheum Dis Clin North Am. 2012;38(1):81–91, viii–ix.

Boldery R, Fielding G, Rafter T, Pascoe AL, Scalia GM. Nutritional deficiency of selenium secondary to weight loss (bariatric) surgery associated with life-threatening cardiomyopathy. Heart Lung Circ. 2007;16(2):123–6.

Borel P, Preveraud D, Desmarchelier C. Bioavailability of vitamin E in humans: an update. Nutr Rev. 2013;71(6):319–31.

Boza C, Gamboa C, Perez G, et al. Laparoscopic adjustable gastric banding (LAGB): surgical results and 5-year follow-up. Surg Endosc. 2011;25(1):292–7.

Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2011. Obes Surg. 2013;23(4):427–36.

Cable CT, Colbert CY, Showalter T, et al. Prevalence of anemia after Roux-en-Y gastric bypass surgery: what is the right number? Surg Obes Relat Dis. 2011;7(2):134–9.

Carrodeguas L, Kaidar-Person O, Szomstein S, Antozzi P, Rosenthal R. Preoperative thiamine deficiency in obese population undergoing laparoscopic bariatric surgery. Surg Obes Relat Dis. 2005;1(6):517–22. discussion 522.

Chapman MS. Vitamin A: history, current uses, and controversies. Semin Cutan Med Surg. 2012;31(1):11–6.

Chikunguwo S, Brethauer S, Nirujogi V, et al. Influence of obesity and surgical weight loss on thyroid hormone levels. Surg Obes Relat Dis. 2007;3(6):631–5. discussion 635–6.

Clements RH, Katasani VG, Palepu R, et al. Incidence of vitamin deficiency after laparoscopic Roux-en-Y gastric bypass in a university hospital setting. Am Surg. 2006;72(12):1196–202. discussion 1203–1194.

Combs GF. The vitamins: fundamental aspects in nutrition and health. 3rd ed. Amsterdam, Boston: Elsevier Academic Press; 2008.

Cruickshank AM, Telfer AB, Shenkin A. Thiamine deficiency in the critically ill. Intensive Care Med. 1988;14(4):384–7.

Cunha SF, Goncalves GA, Marchini JS, Roselino AM. Acrodermatitis due to zinc deficiency after combined vertical gastroplasty with jejunoileal bypass: case report. Sao Paulo Med J. 2012;130(5):330–5.

Dalcanale L, Oliveira CP, Faintuch J, et al. Long-term nutritional outcome after gastric bypass. Obes Surg. 2010;20(2):181–7.

Damms-Machado A, Friedrich A, Kramer KM, et al. Pre- and postoperative nutritional deficiencies in obese patients undergoing laparoscopic sleeve gastrectomy. Obes Surg. 2012;22(6):881–9.

de Luis DA, Pacheco D, Izaola O, Terroba MC, Cuellar L, Martin T. Zinc and copper serum levels of morbidly

obese patients before and after biliopancreatic diversion: 4 years of follow-up. J Gastrointest Surg. 2011;15(12):2178–81.

Dewey M, Heuberger R. Vitamin D and calcium status and appropriate recommendations in bariatric surgery patients. Gastroenterol Nurs. 2011;34(5):367–74.

DiGiorgi M, Daud A, Inabnet WB, et al. Markers of bone and calcium metabolism following gastric bypass and laparoscopic adjustable gastric banding. Obes Surg. 2008;18(9):1144–8.

Eckert MJ, Perry JT, Sohn VY, et al. Incidence of low vitamin A levels and ocular symptoms after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2010;6(6):653–7.

Eerdekens A, Debeer A, Van Hoey G, et al. Maternal bariatric surgery: adverse outcomes in neonates. Eur J Pediatr. 2010;169(2):191–6.

Finucane MM, Stevens GA, Cowan MJ, et al. National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. Lancet. 2011;377 (9765):557–67.

Galvin R, Brathen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. Eur J Neurol. 2010;17(12):1408–18.

Gehring N, Imoberdorf R, Wegmann M, Ruhlin M, Ballmer PE. Serumalbumin–a qualified parameter to determine the nutritional status? Swiss Med Wkly. 2006;136(41–42):664–9.

Gletsu-Miller N, Broderius M, Frediani JK, et al. Incidence and prevalence of copper deficiency following roux-en-y gastric bypass surgery. Int J Obes (Lond). 2012;36 (3):328–35.

Goldner WS, O'Dorisio TM, Dillon JS, Mason EE. Severe metabolic bone disease as a long-term complication of obesity surgery. Obes Surg. 2002;12(5):685–92.

Grant AM, Avenell A, Campbell MK, et al. Oral vitamin D3 and calcium for secondary prevention of low-trauma fractures in elderly people (Randomised Evaluation of Calcium Or vitamin D, RECORD): a randomised placebo-controlled trial. Lancet. 2005;365 (9471):1621–8.

Greenway FL, Ingram DK, Ravussin E, et al. Loss of taste responds to high-dose biotin treatment. J Am Coll Nutr. 2011;30(3):178–81.

Hakeam HA, O'Regan PJ, Salem AM, Bamehriz FY, Eldali AM. Impact of laparoscopic sleeve gastrectomy on iron indices: 1 year follow-up. Obes Surg. 2009;19 (11):1491–6.

Halfdanarson TR, Kumar N, Li CY, Phyliky RL, Hogan WJ. Hematological manifestations of copper deficiency: a retrospective review. Eur J Haematol. 2008;80(6):523–31.

Hatizifotis M, Dolan K, Newbury L, Fielding G. Symptomatic vitamin A deficiency following biliopancreatic diversion. Obes Surg. 2003;13(4):655–7.

Heber D, Greenway FL, Kaplan LM, Livingston E, Salvador J, Still C. Endocrine and nutritional management of the post-bariatric surgery patient: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2010;95(11):4823–43.

Herrmann W, Obeid R. Causes and early diagnosis of vitamin B12 deficiency. Dtsch Arztebl Int. 2008;105 (40):680–5.

Herve C, Beyne P, Letteron P, Delacoux E. Comparison of erythrocyte transketolase activity with thiamine and thiamine phosphate ester levels in chronic alcoholic patients. Clin Chim Acta. 1995;234(1–2):91–100.

Holley AK, Dhar SK, Xu Y, St Clair DK. Manganese superoxide dismutase: beyond life and death. Amino Acids. 2012;42(1):139–58.

Jaiser SR, Winston GP. Copper deficiency myelopathy. J Neurol. 2010;257(6):869–81.

Jastrzebska-Mierzynska M, Ostrowska L, Hady HR, Dadan J. Assessment of dietary habits, nutritional status and blood biochemical parameters in patients prepared for bariatric surgery: a preliminary study. Wideochir Inne Tech Malo Inwazyjne. 2012;7(3):156–65.

Jeejeebhoy KN, Chu RC, Marliss EB, Greenberg GR, Bruce-Robertson A. Chromium deficiency, glucose intolerance, and neuropathy reversed by chromium supplementation, in a patient receiving long-term total parenteral nutrition. Am J Clin Nutr. 1977;30(4):531–8.

Johnson JM, Maher JW, Samuel I, Heitshusen D, Doherty C, Downs RW. Effects of gastric bypass procedures on bone mineral density, calcium, parathyroid hormone, and vitamin D. J Gastrointest Surg. 2005;9 (8):1106–10. discussion 1110–1101.

Juhasz-Pocsine K, Rudnicki SA, Archer RL, Harik SI. Neurologic complications of gastric bypass surgery for morbid obesity. Neurology. 2007;68(21):1843–50.

Kang L, Marty D, Pauli RM, Mendelsohn NJ, Prachand V, Waggoner D. Chondrodysplasia punctata associated with malabsorption from bariatric procedures. Surg Obes Relat Dis. 2010;6(1):99–101.

Kelkar P, Chang S, Muley SA. Response to oral supplementation in copper deficiency myeloneuropathy. J Clin Neuromuscul Dis. 2008;10(1):1–3.

Kelly GS. Pantothenic acid. Monograph. Altern Med Rev. 2011;16(3):263–74.

Kremen AJ, Linner JH, Nelson CH. An experimental evaluation of the nutritional importance of proximal and distal small intestine. Ann Surg. 1954;140(3):439–48.

Kumar N. Nutritional neuropathies. Neurol Clin. 2007;25 (1):209–55.

Lakhani SV, Shah HN, Alexander K, Finelli FC, Kirkpatrick JR, Koch TR. Small intestinal bacterial overgrowth and thiamine deficiency after Roux-en-Y gastric bypass surgery in obese patients. Nutr Res. 2008;28(5):293–8.

Lewandowski H, Breen TL, Huang EY. Kwashiorkor and an acrodermatitis enteropathica-like eruption after a distal gastric bypass surgical procedure. Endocr Pract. 2007;13(3):277–82.

Magiorkinis E, Beloukas A, Diamantis A. Scurvy: past, present and future. Eur J Intern Med. 2011;22(2): 147–52.

Moukarzel A. Chromium in parenteral nutrition: too little or too much? Gastroenterology. 2009;137(5 Suppl):S18–28.
Niki E, Traber MG. A history of vitamin E. Ann Nutr Metab. 2012;61(3):207–12.
Pata G, Crea N, Di Betta E, Bruni O, Vassallo C, Mittempergher F. Biliopancreatic diversion with transient gastroplasty and duodenal switch: long-term results of a multicentric study. Surgery. 2013;153 (3):413–22.
Prinzo, ZW. Thiamine deficiency and its prevention and control in major emergencies. WHO/NHD/99/13, 1999; 1–52. http://www.who.int/nutrition/publications/en/thiamine_in_emergencies_eng.pdf. Accessed 7 Nov 2011.
Saltzman E, Karl JP. Nutrient deficiencies after gastric bypass surgery. Annu Rev Nutr 2013;33:183–203.
Scopinaro N. The IFSO and obesity surgery throughout the world. International Federation for the Surgery of Obesity. Obes Surg. 1998;8(1):3–8.
Scopinaro N, Gianetta E, Civalleri D, Bonalumi U, Bachi V. Bilio-pancreatic bypass for obesity: II. Initial experience in man. Br J Surg. 1979;66(9):618–20.
Shankar P, Boylan M, Sriram K. Micronutrient deficiencies after bariatric surgery. Nutrition. 2010;26(11–12):1031–7.
Shearer MJ, McBurney A, Barkhan P. Studies on the absorption and metabolism of phylloquinone (vitamin K1) in man. Vitam Horm. 1974;32:513–42.
Sjostrom L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004;351 (26):2683–93.
Slater GH, Ren CJ, Siegel N, et al. Serum fat-soluble vitamin deficiency and abnormal calcium metabolism after malabsorptive bariatric surgery. J Gastrointest Surg. 2004;8(1):48–55. discussion 54–45.
Sriram K, Manzanares W, Joseph K. Thiamine in nutrition therapy. Nutr Clin Pract. 2012;27(1):41–50.
Sudan R, Jacobs DO. Biliopancreatic diversion with duodenal switch. Surg Clin North Am. 2011;91 (6):1281–93. ix.
Thomson AD, Marshall EJ. The treatment of patients at risk of developing Wernicke's encephalopathy in the community. Alcohol Alcohol. 2006;41(2):159–67.
Tong GM, Rude RK. Magnesium deficiency in critical illness. J Intensive Care Med. 2005;20(1):3–17.
Traber MG, Stevens JF. Vitamins C and E: beneficial effects from a mechanistic perspective. Free Radic Biol Med. 2011;51(5):1000–13.
Whelton MJ, Kehayoglou AK, Agnew JE, Turnberg LA, Sherlock S. 47 Calcium absorption in parenchymatous and biliary liver disease. Gut. 1971;12(12):978–83.
Zempleni J, Hassan YI, Wijeratne SS. Biotin and biotinidase deficiency. Expert Rev Endocrinol Metab. 2008;3(6):715–24.