Malabsorption syndromes: An Overview

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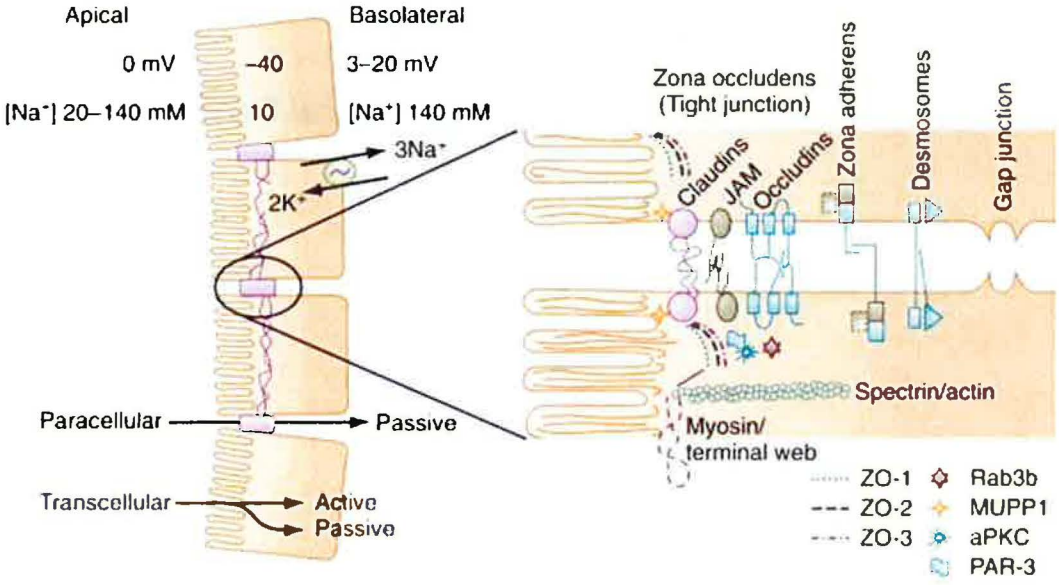
Introduction

The small intestine is optimally designed to absorb nutrients and transport fluids. It is 600- 800 cms in length and has a surface area of 2,000,000 cm^2 due to 600 fold amplification of plicae circulares, villus crypt architecture, and microvilli. The intestinal epithelium acts as first line of defense between the mucosal and serosal compartments. The intercellular tight junctions between the apical (luminal) and basolateral (serosal) membranes are relatively leaky in the small intestine to fairly tight in the large intestine.

Fig: 1



Malabsorption occurs when there is a primary transport defect without morphological changes or a secondary transport defect due to morphological changes; when the absorptive area is reduced or the transport of absorbed nutrients from the intestine is affected. Maldigestion is due to reduced hydrolysis of nutrients. These two terms are interchangeably used. It is necessary to understand the normal physiologic process of small intestinal digestion and absorption to better understand the mechanism of malabsorption and maldigestion. **The purpose of this review is to understand the pathophysiology, etiology of malabsorption; diagnostic tests. In addition, malabsorption due to bariatric surgery will be discussed in detail**.

In general, digestion and absorption of nutrients can be divided into luminal, mucosal and post absorptive states. Luminal phase is characterized by hydrolysis and solubulization of dietary fats, proteins and carbohydrates by digestive enzymes and bile. In the mucosal phase, the digested products are efficiently transported across the brush border membrane of intestinal epithelial cells. In the post absorptive phase, the lipids are transported via lymphatics and portal circulation from the epithelial cells to the other parts of the body. Thereafter, various causes of malabsorption syndromes can be classified into disorders affecting the *luminal, mucosal and post absorptive states.*

Luminal phase disorders can be divided into conditions leading to (a) impaired nutrient hydrolysis, (b) impaired micelle formation and (c) limited bioavailability of the nutrients. (a) Impaired hydrolysis of fat and protein from deficiencies of lipase and protease pancreatic enzymes leads to (1) pancreatic insufficiency which is seen in chronic pancreatitis, pancreatic resection, pancreatic cancer, or cystic fibrosis, (2) inactivation of the pancreatic enzymes by gastric hyper secretion, seen in Zollinger –Ellison syndrome and (3) inadequate mixing of the nutrients, bile, and pancreatic enzymes seen in rapid intestinal transit, gastrojejunostomy, total and partial gastrectomy, or intestinal resection after mesenteric emboli or thrombosis, also causes impaired hydrolysis.

(b) Impaired micelle formation leads to fat malabsorption from (1) decreased bile salt synthesis from severe parenchymal liver disease (eg, cirrhosis); (2) impaired bile secretion from biliary obstruction or cholestatic jaundice (eg, primary biliary cirrhosis, primary sclerosing cholangitis); (3) impaired enterohepatic bile circulation, as seen in small bowel resection or regional enteritis; or (4) bile salt deconjugation due to small bowel bacterial overgrowth from stasis of intestinal content caused by a motor abnormality (eg, scleroderma, diabetic neuropathy, intestinal obstruction), an anatomic abnormality (eg, small bowel diverticula, stricture, ischemia, blind loops), or small bowel contamination from enterocolonic fistulas.

(C) Luminal bacterial overgrowth can cause a decrease in the availability of substrates, including carbohydrates, proteins, and vitamins (eg, vitamin B-12, folate). Vitamin B-12 deficiency due to pernicious anemia is caused by a lack of intrinsic factor and by pancreatic enzyme deficiency.

Mucosal phase of malabsorption can be due to impaired brush-border hydrolase activity such as disaccharidase deficiency leading to disaccharide malabsorption seen in lactase deficiency (primary or secondary). Secondary lactase deficiency can be due to acute gastroenteritis (rotavirus and giardia infection), chronic alcoholism, celiac sprue, radiation enteritis, regional enteritis, or AIDS enteropathy. Immunoglobulin A (IgA) deficiency (most common immunodeficiency) is due to decreased or absent serum and intestinal IgA, which clinically appears similar to celiac disease and is unresponsive to a gluten-free diet. Acrodermatitis enteropathica is an autosomal recessive disease with selective inability to absorb zinc, leading to villous atrophy and acral dermatitis. Autoimmune enteropathy primarily diagnosed in children presenting with intractable secretory diarrhea and villous atrophy is due to antibodies directed against intestinal epithelial and goblet cells.

Impaired nutrient absorption can be due to inherited defects such as glucose-galactose malabsorption, abetalipoproteinemia, cystinuria, and Hartnup disease. Acquired disorders are caused by the following: (1) decreased absorptive surface area, as seen in intestinal resection of intestinal bypass; (2) damaged absorbing surface, as seen in celiac sprue, tropical sprue, Crohn's disease, AIDS enteropathy, chemotherapy, or radiation therapy; (3) infiltrating disease of the intestinal wall, such as lymphoma and amyloidosis; and (4) infections, including bacterial overgrowth, giardiasis, Whipple's disease, cryptosporidiosis, and microsporidiosis.

Postabsorptive phase of malabsorption is due to obstruction of the lymphatic system seen in congenital (eg, intestinal lymphangiectasia, Milroy disease) and acquired disease states (eg, Whipple disease, lymphoma, tuberculosis), impairs the absorption of chylomicrons and lipoproteins or a protein-losing enteropathy.

Table 1

Mechanisms of Malabsorption, Malabsorbed Substrates, and Representative Causes

MECHANISM	MALABSORBED SUBSTRATE(S)	REPRESENTATIVE CAUSES
Maldigestion		
Conjugated bile acid deficiency	Fat Fat-soluble vitamins Calcium Magnesium	Hepatic parenchymal disease Biliary obstruction Bacterial overgrowth with bile acid deconjugation Ileal bile acid malabsorption CCK deficiency
Pancreatic insufficiency	Fat Protein Carbohydrate Fat-soluble vitamins Vitamin B_{12} (cobalamin)	Congenital defects Chronic pancreatitis Pancreatic tumors Inactivation of pancreatic enzymes (e.g., Zollinger-Ellison syndrome)
Reduced mucosal digestion	Carbohydrate Protein	Congenital defects Acquired lactase deficiency Generalized mucosal disease (e.g., celiac disease, Crohn's disease)
Intraluminal consumption of nutrients	Vitamin B_{12} (cobalamin)	Small intestinal bacterial overgrowth Helminthic infections (e.g., *Diphyllobothrium latum* infection)
Malabsorption		
Reduced mucosal absorption	Fat Protein Carbohydrate Vitamins Minerals	Congenital transport defects Generalized mucosal diseases (e.g., celiac disease, Crohn's disease) Previous intestinal resection or bypass Infections Intestinal lymphoma

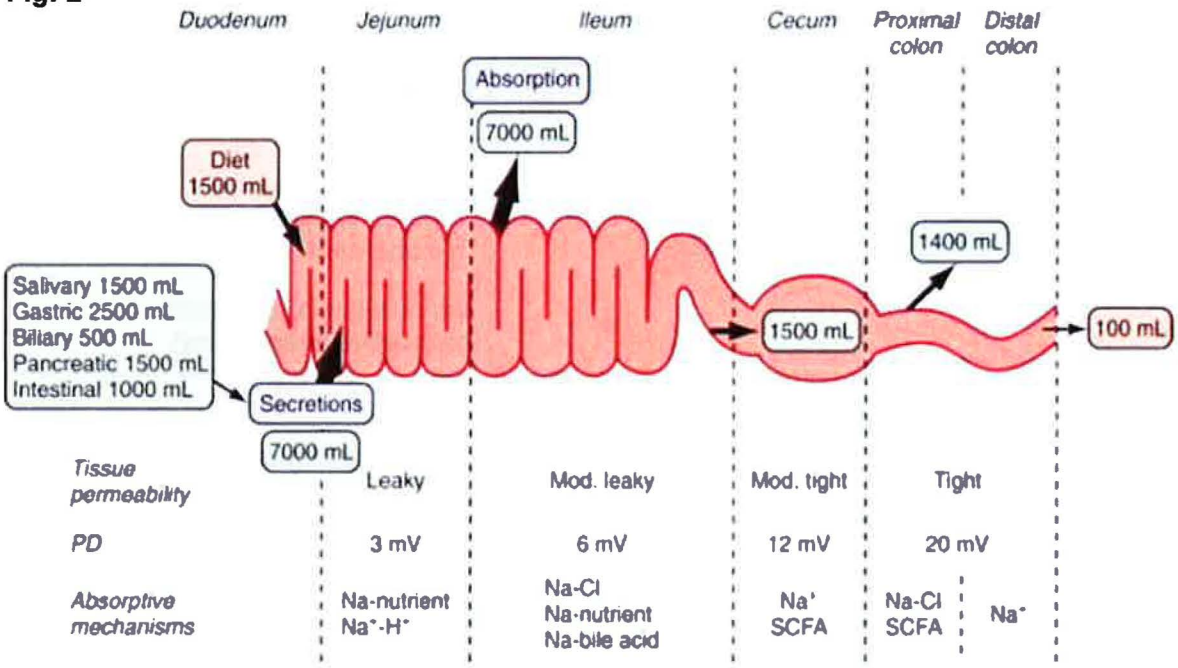
MECHANISM	MALABSORBED SUBSTRATE(S)	REPRESENTATIVE CAUSES
Decreased transport from the intestine	Fat Protein	Intestinal lymphangiectasia Primary Secondary (e.g., solid tumors, Whipple's disease, lymphomas) Venous stasis (e.g., from congestive heart failure)
Other Mechanisms		
Decreased gastric acid and/or intrinsic factor secretion	Vitamin B_{12}	Pernicious anemia Atrophic gastritis Previous gastric resection
Decreased gastric mixing and/or rapid gastric emptying	Fat Calcium Protein	Previous gastric resection Autonomic neuropathy
Rapid intestinal transit	Fat	Autonomic neuropathy Hyperthyroidism

Adapted from Feldman: Sleisenger and Fordtran's Gastrointestinal and Liver Disease, 9th ed

SIGNS AND SYMPTOMS OF MALABSORPTION

The gastrointestinal tract processes 8 to 9 L of fluid daily which is due to oral intake and endogenous exocrine secretions. Intestinal fluid absorption is 98% efficient resulting in only 100 to 200 mL to be excreted each day. The intestine also extracts nutrients, vitamins, and minerals in well orchestrated pattern and when this mechanism is disturbed, then malabsorption and diarrhea ensue. [1] Carbohydrate malabsorption causes osmotic diarrhea, fat malabsorption causes steatorrhea and protein malabsorption causes hypoalbuminemia and peripheral edema.

Fig: 2



Malabsorption syndromes can produce a myriad of signs and symptoms based on the underlying etiology. Table 2 illustrates the clinical features and underlying pathophysiology.

Table 2

Symptoms and Signs of Malabsorption and Relevant Pathophysiology

SYMPTOM OR SIGN	PATHOPHYSIOLOGY
Gastrointestinal	
Diarrhea	Osmotic activity of carbohydrates or short-chain fatty acids Secretory effect of bile acids and fatty acids Decreased absorptive surface Intestinal loss of conjugated bile acids Ileal resection Severe ileal mucosal disease

SYMPTOM OR SIGN	PATHOPHYSIOLOGY
	Congenital defects of the ileal sodium–bile acid cotransporter
Abdominal distention, flatulence	Bacterial gas production from carbohydrates in colon, small intestinal bacterial overgrowth
Foul-smelling flatulence or stool	Malabsorption of proteins or intestinal protein loss
Pain	Gaseous distention of intestine
Ascites	Protein loss or malabsorption
Musculoskeletal	
Tetany, muscle weakness, paresthesias	Malabsorption of vitamin D, calcium, magnesium, and phosphate
Bone pain, osteomalacia, fractures	Protein, calcium, or vitamin D deficiency; secondary hyperparathyroidism
Cutaneous and Mucosal	
Easy bruisability, ecchymoses, petechiae	Vitamin K deficiency and vitamin C deficiency (scurvy)
Glossitis, cheilosis, stomatitis	Vitamin B complex, vitamin B_{12}, folate, or iron deficiency
Edema	Protein loss or malabsorption
Acrodermatitis, scaly dermatitis	Zinc and essential fatty acid deficiency
Follicular hyperkeratosis	Vitamin A deficiency
Hyperpigmented dermatitis	Niacin deficiency (pellagra)
Thin nails with spoon-shaped deformity	Iron deficiency
Perifollicular hemorrhage	Malabsorption of vitamin C
Spiral or curly hair	Malabsorption of vitamin C
Other	
Weight loss, hyperphagia	Nutrient malabsorption
Growth and weight retardation, infantilism	Nutrient malabsorption in childhood and adolescence
Anemia	Iron, folate, or vitamin B_{12} deficiency
Kidney stones	Increased colonic oxalate absorption
Amenorrhea, impotence, infertility	Multifactorial (including protein malabsorption, secondary hypopituitarism, anemia)
Night blindness, xerophthalmia	Vitamin A deficiency

SYMPTOM OR SIGN	PATHOPHYSIOLOGY
Peripheral neuropathy	Vitamin B_{12} or thiamine deficiency
Fatigue, weakness	Calorie depletion, iron and folate deficiency, anemia
Neurologic symptoms, ataxia	Vitamin B_{12}, vitamin E, or folate deficiency

Adapted from Feldman: Sleisenger and Fordtran's Gastrointestinal and Liver Disease, 9th ed

Diagnosis

Diagnosis of the etiology of the various causes of the malabsorption syndrome can be done with blood work, stool studies, imaging studies such as CT scan, small bowel follow through, CT Enterography. Endoscopy with biopsies will be helpful in diagnosing conditions such as celiac disease. Whipple's disease, Collagenous sprue, etc. Capsule endoscopy and deep enteroscopy with biopsy is sometimes necessary to evaluate the entire small intestine in conditions such as refractory sprue, lymphoma, etc. The diagnostic work-up is detailed below in Table 3 and 4.

Table 3: Laboratory Tests That Are Useful in the Differential Diagnosis of Malabsorption

Hematocrit, hemoglobin	Decreased in iron, vitamin B_{12}, and folate malabsorption or with blood loss
Mean corpuscular hemoglobin or mean corpuscular volume	Decreased in iron malabsorption; increased in folate and vitamin B_{12} malabsorption
White blood cells, differential	Decreased in vitamin B_{12} and folate malabsorption; low lymphocyte count in lymphangiectasia
Biochemical Tests (Serum)	
Triglycerides	Decreased in severe fat malabsorption
Cholesterol	Decreased in bile acid malabsorption or severe fat malabsorption
Albumin	Decreased in severe malnutrition, lymphangiectasia, protein-losing enteropathy
Alkaline phosphatase	Increased in calcium and vitamin D malabsorption (severe steatorrhea); decreased in zinc deficiency
Calcium, phosphorus, magnesium	Decreased in extensive small intestinal mucosal disease, after extensive intestinal resection, or in vitamin D deficiency
Zinc	Decreased in extensive small intestinal mucosal disease or intestinal resection
Iron, ferritin	Decreased in celiac disease, in other extensive small intestinal mucosal diseases, and with chronic blood loss

Other Serum Tests	
Prothrombin time	Prolonged in vitamin K malabsorption
β-Carotene	Decreased in fat malabsorption from hepatobiliary or intestinal diseases
Immunoglobulins	Decreased in lymphangiectasia, diffuse lymphoma
Folic acid	Decreased in extensive small intestinal mucosal diseases, with anticonvulsant use, in pregnancy; may be increased in small intestinal bacterial overgrowth
Vitamin B_{12}	Decreased after gastrectomy, in pernicious anemia, terminal ileal disease, and small intestinal bacterial overgrowth
Methylmalonic acid	Markedly elevated in vitamin B_{12} deficiency
Homocysteine	Markedly elevated in vitamin B_{12} or folate deficiency
Citrulline	May be decreased in destructive small intestinal mucosal disease or intestinal resection
Stool Tests	
Fat	Qualitative or quantitative increase in fat malabsorption
Elastase, chymotrypsin	Decreased concentration and output in exocrine pancreatic insufficiency
pH	Less than 5.5 in carbohydrate malabsorption

Adapted from Feldman: Sleisenger and Fordtran's Gastrointestinal and Liver Disease, 9th ed

Laboratory Tests That Are Useful in the Differential Diagnosis of Malabsorption

TEST	COMMENT
Blood Cell Count	
Acanthocytes	Abetalipoproteinemia
Nuclear remnants in erythrocytes (Howell-Jolly bodies)	Splenic atrophy in celiac disease, inflammatory bowel disease, radiation enteritis, amyloidosis
White blood cells, differential	Eosinophilia in eosinophilic gastroenteritis and parasitic disease Low lymphocyte count in lymphangiectasia, tuberculosis, protein-losing enteropathy Low $CD4^+$ count in AIDS
Platelets	Increased in inflammatory diseases
Other Tests	
ESR, C-reactive protein	Increased in Crohn's disease, Whipple's disease, lymphoma

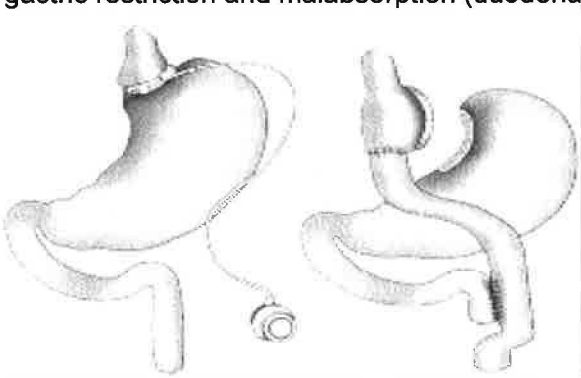
TEST	COMMENT
Ferritin	Increased in inflammatory diseases, lymphoma; decreased in iron deficiency
Iron	Decreased in celiac disease, chronic occult intestinal bleeding, chronic inflammatory diseases
Liver biochemical tests	Increased in primary biliary cirrhosis and other liver diseases, celiac disease
Immunologic Markers	
Immunoglobulins	IgA deficiency, immunodeficiency syndromes
Allergen-specific IgE	IgE-mediated hypersensitivity
Autoantibodies (e.g., ANA)	Connective tissue diseases
HLA-DQ2 or HLA-DQ8	Celiac disease, refractory sprue
Antimitochondrial autoantibodies	Primary biliary cirrhosis
HIV-ELISA/Western blot	AIDS
Neuroendocrine Markers	
ACTH, cortisol	Abnormal values in Addison's disease
Chromogranin A	Elevated in neuroendocrine tumors
5-Hydroxyindoleacetic acid in urine	Elevated in carcinoid syndrome
Gastrin	Elevated in Zollinger-Ellison syndrome
Glucagon	Elevated in glucagonoma
Serum TSH	Decreased in hyperthyroidism; increased in hypothyroidism
Somatostatin	Elevated in somatostatinoma (normal in duodenal somatostatinoma)
Tissue transglutaminase antibodies, EMA	Celiac disease
Stool Tests	
Occult blood test	Erosive or ulcerative intestinal disease or tumor
Ova and parasites	Repeated samples may be needed to detect *Giardia lamblia*
Leukocytes	Present in some inflammatory diseases of the intestine

Adapted from Feldman: Sleisenger and Fordtran's Gastrointestinal and Liver Disease, 9th ed

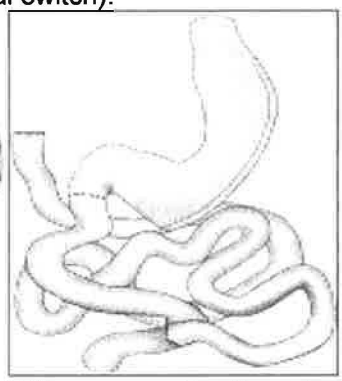
SPECIFIC DISEASES CAUSING MALABSORPTION

BARIATRIC SURGERY

The first effective weight loss surgery was jejunoileal bypass which was introduced by Kremen and Linner in 1954. In this procedure, the proximal jejunum was connected directly to the distal ileum, bypassing 90% of the small intestine out of the intestinal stream of ingested nutrients (blind loop). The procedure induced a state of malabsorption, which led to significant weight loss. However, many patients developed complications secondary to malabsorption (eg, steatorrhea, diarrhea, vitamin deficiencies, oxalosis) or due to the toxic overgrowth of bacteria in the bypassed intestine (eg, liver failure, severe arthritis, skin problems). Consequently, many patients have required reversal of the procedure, and the procedure has been abandoned. This led to a search for better operations. Modifications in the original procedures and the development of new techniques have led to 3 basic concepts for bariatric surgery: (1) gastric restriction (adjustable gastric banding, sleeve gastrectomy), (2) gastric restriction with mild malabsorption (Roux-en-Y gastric bypass), and (3) a combination of mild gastric restriction and malabsorption (duodenal switch).



Adjustable gastric banding **RY Bypass**



Bilio pancreatic diversion with duodenal switch, gastric sleeve resection

At present, the Roux-en-Y gastric bypass (RYGB) is the most widely performed weight loss procedure in the United States, followed by vertical banded gastroplasty or laparoscopic gastric banding and biliopancreatic diversion with or without duodenal switch.[2,3] Malabsorptive procedures, especially the jejunoileal bypass, produce the greatest degree of weight loss but can be associated with serious and potentially life-threatening metabolic and nutritional complications.[4,7,8] BPD and DGB-DS produce significant weight loss that persists and causes fewer metabolic complications than jejunoileal bypass. Restrictive procedures produce moderate degrees of weight loss and are associated with the lowest incidence of metabolic and nutritional complications. RYGB has consistently been shown to produce greater and more sustained weight loss

than VBG and avoids the severe metabolic and nutritional consequences of intestinal bypass. [9]

The mechanism of weight loss following vertical banded gastroplasty appears to be similar to that of other forms of caloric restriction, although the weight loss tends to be more pronounced and to persist for a longer time. The role of the regulation of central satiety mechanisms has been studied for restrictive and malabsorptive types of surgery. Ghrelin is a gastric peptide with potent orexigenic effects. Circulating ghrelin concentrations are high in obese subjects, and they increase after weight loss. In patients undergoing RYGB (compared with patients undergoing VBG or BPD), however, a decrease in ghrelin levels has been reported and appears to depend on the surgically induced bypass of the ghrelin-producing cell populations of the gastric fundus [5]. Several mechanisms have been suggested to account for the more substantial weight loss after JIB, RYGB and BPD. These can be divided into three groups: caloric restriction, changes in energy metabolism, and alterations in gastrointestinal hormones and nutrient absorption.

Caloric restriction seems to play a prominent role in jejunoileal bypass and RY gastric bypass, and numerous studies document significantly decreased caloric intake after each type of surgery. Animal studies have shown that rats subjected to intestinal bypass maintained lower weights despite caloric intakes comparable with those of sham-operated controls, suggesting an increase in energy expenditure. [6] Human data suggest a relative increase in the mean resting energy expenditure in patients undergoing RYGB. Malabsorption leading to a loss of lean body mass, diarrhea, and vitamin, mineral, and electrolyte deficiencies prominently contributes to weight loss after jejunoileal bypass, but less so after biliopancreatic diversion. After jejunoileal bypass, malabsorption results from the drastically reduced intestinal surface in contact with nutrients (common channel) and rapid transit time. It has been suggested that the enhanced weight loss after BPD is related to the combination of a short common channel (approximately 50 cm), which limits fat absorption, and a long afferent limb, which is not in direct contact with food. Therefore, the effect of the alimentary (Roux) limb, where protein and carbohydrates are absorbed, is limited.

Table -- **Advantages and Disadvantages of Bariatric Surgery Procedures for Weight Loss** [7]

PROCEDURE	WEIGHT LOSS MECHANISM	ADVANTAGES	DISADVANTAGES	REVERSIBLE?	Mean Weight Loss (kg)	
					AFTER 12 MO	AFTER 36 MO
RNYGB	Restriction and malabsorption	Good weight loss	Vitamin deficiencies; internal hernias	No	43.5	41.5
LAGB	Restriction	Relatively low risk of vitamin deficiencies; low rate of complications	Less weight loss; may require adjustments; foreign body	Band may be removed	30.2	34.8
VGB	Restriction	Ease of construction	No longer performed Staple dehiscence	Yes	32.1	32.0

PROCEDURE	WEIGHT LOSS MECHANISM	ADVANTAGES	DISADVANTAGES	REVERSIBLE?	Mean Weight Loss (kg)	
					AFTER 12 MO	AFTER 36 MO
BPD	Malabsorption	Greatest weight loss	High risk of malabsorption and vitamin deficiencies	No	51.9	53.1

Vitamin and trace element deficiencies in gastric bypass.

Though obesity is a condition with macronutrient excess, micronutrient deficiencies are commonly encountered pre operatively which can exacerbate the expected post operative deficiencies. A retrospective study of 170 patients with RY bypass between 2000 and 2005 showed 6% of the patients had nutritional deficiencies requiring pre operative supplementation. Post surgery as expected significant number of patients had vitamin and mineral deficiencies which developed within a month. [10,11]

Sites of vitamin and trace element absorption

Stomach	Copper, Iodine
Duodenum	Iron, zinc, copper, selenium, vitamin A, E, K, Thiamine, riboflavin, pyridoxine, niacin
Jejunum	Zinc, selenium, iron, copper, manganese, thiamine, riboflavin, Vitamins C, A, D, E, K, folate, pyridoxine, Niacin
Ileum	Vitamin C, D, K, folate, B12.

Iron deficiency after gastric bypass can develop for several reasons, such as intolerance to red meat, diminished gastric acid secretion, and exclusion of the duodenum. Menstruating or pregnant women may be particularly predisposed to developing iron deficiency after gastric bypass surgery. Postoperatively, oral iron and vitamin C supplementation should be prescribed, because once iron deficiency has developed it may be refractory to oral treatment.

B12 deficiency in gastric bypass is due to decreased dietary intake, decreased gastric secretions, and decreased availability of intrinsic factor as well as decreased bioavailability of B12 due to bacterial overgrowth in the defunctionalized ileal segment[14]. 350 mcg/day orally or 1000 to 2000 mcg/day for management of hematological and neurological parameters is recommended supplementation.
Folate deficiency is due to bypass of absorption site, non availability of B12, decreased oral intake. Increased homocysteine levels, glossitis, macrocytic anemia are the symptoms. 1 mg/day is recommended daily supplementation. [16,17]

Thiamine deficiency is seen in patients with persistent vomiting, rapid weight loss, inadequate intake after bariatric surgery. It is more common in duodenal switch

procedures. Symptoms: Wernicke Encephalopathy - ophthalmoplegia, ataxia, and apathetic mental confusion (Korsakoff's psychosis). Intravenous dextrose without thiamin as first line of therapy can further exacerbate cytotoxicity of the brain.[18,19] Treatment is with prophylactic parenteral thiamin administration in malnourished patients as early as 6 wk following bariatric surgery. Daily dose of oral supplementation at 25–50 mg is recommended in addition to the RDA.

Decreased levels of vitamin D in obese patients is due to inadequate intake, bypass of the primary absorption site for calcium and vitamin D. It is more seen in duodenal switch patients. Low pre- operative levels, African American status, length of the bypass limb predict the post operative risk [20,21,22,23]. Supplementation is Calcium citrate 500 mg to 1500 mg daily or Ergocalciferol 50,000 IU weekly or Cholecalciferol 800, 2000, 5000 IU per day.[24,25,26]

Vitamin A deficiency is seen in 69% of patients. 11 % had deficiency at 1 year despite taking a chewable MVI. Causes: Oxidative stress, lipid malabsorption, non alcoholic fatty liver, ↓ intake. 2500 IU per day in addition to the RDA is recommended for supplementation. [27,28,29]

Zinc deficiency is due to bypass of absorption site. Symptoms are alopecia, weight loss, diarrhea, acrodermatitis, hypogonadism in males. Supplementation: Additional 6.5 mg per day.

Selenium deficiency can cause muscle weakness, cardiomyopathy (Sheehan's cardiomyopathy). It is seen in 14 to 22% of post bariatric surgery patients. [29]

Copper is absorbed in the proximal duodenum and can cause deficiency in gastric bypass patients. It is essential for production of RBC and maintenance of nervous system. Copper deficiency can cause normocytic anemia and myeloneuropathy. [30,31]

Gastric bypass patients adhering to a set of dietary supplements had mostly stable or increased vitamin concentrations compared with both their baseline values and the changes in a non surgical control group. Pre operatively serum zinc, copper, selenium, Vitamin D, C, B 12 and folate should be checked a month before surgery and 3, 6, 12, 24 months after the surgery [32]

General recommendations for supplementation in gastric bypass patients [33, 34]

one to two multivitamin + multitrace element tablets 1200–2000 mg calcium citrate 400–800 IU vitamin D 400 µg folate 40–65 mg elemental iron 350 µg vitamin B12 (alternatively, 1000 µg/mo intramuscularly or 3000 µg/6 mo intramuscularly, or 500 µg/wk intranasally).

It is recommended that after bariatric surgery patients should have yearly measurements of a basic metabolic panel, magnesium, complete blood count, iron studies, vitamin D, parathyroid hormone, and bone density. The routine and lifelong use of multivitamins is considered necessary.

Recommendations for additional supplementation after different bariatric surgeries [35]

Micronutrients	Recommended Dietary Allowance	Banding, gastric bypass, sleeve (additional % RDA)	BPD and duodenal switch
Vitamin A	3000 IU	100	1000
B1	1-1. to 1.3 mg	150	500
B12	2.4 mcg	300	1000
Folic acid	400 mcg	150	200
Vitamin C	75-90 mg	200	400
Vitamin D	200 IU	100	750
Vitamin E	15 mg	100	1000
Vitamin K	150 mcg	25	100
Copper	900 mcg	50	200
Iron	8-18 mg	50	1000
Selenium	55 mcg	33	66
Zinc	8-11 mg	33	300

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