CHAPTER 30

Medical Nutrition Therapy for Lower Gastrointestinal Tract Disorders

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CHAPTER OUTLINE

- Common Intestinal Problems
- Diseases of the Small Intestine
- Intestinal Brush-Border Enzyme Deficiencies
- Inflammatory Bowel Diseases
- Disorders of the Large Intestine
- Intestinal Surgery

KEY TERMS

aerophagia—swallowing of air
blind loop syndrome—a disorder of bacterial overgrowth with resultant malabsorption secondary to alterations in the anatomy of the small intestine involving a loop that is disconnected from the main intestinal tract
borborygmus—intestinal rumbling
celiac disease—common term for gluten-sensitive enteropathy
colostomy—surgical creation of an opening into the colon through a stoma in the abdominal wall to permit defecation
constipation—a condition in which the frequency or quantity of stools is reduced
Crohn’s disease—a chronic, granulomatous inflammatory disease of unknown etiology involving the small or large intestine that results in diarrhea, strictures, fistulas, malabsorption, and the need for surgical resection
dermatitis herpetiformis—a skin disorder that is a variant of celiac disease
diarrhea—abnormal volume and liquidity of stools
diverticulitis—inflammation of diverticula
diverticulosis—presence of herniations of the mucous membrane through the muscular layers of the colonic wall
fistula—an abnormal passage between two internal organs, or from an internal organ to the surface of the body
flatulence—excessive collection and passage of gas from the gastrointestinal tract
flatus—gas in the gastrointestinal tract that is expelled through the anus
glutamine—an amino acid and the preferred fuel of the enterocyte
gluten-sensitive enteropathy (celiac disease)—a syndrome precipitated by the immunologic interaction of gluten-containing foods and intestinal cells; characterized by flattening of the villi of the small intestine
high-fiber diet—a diet containing more than 25 g of dietary fiber
hypolactasia—a decrease in the amount of the intestinal enzyme lactase
ileostomy—surgical creation of an opening into the ileum through a stoma in the abdominal wall
ileal pouch—surgical creation of a small reservoir, using folds of the distal ileum, which is then attached to the rectum
inflammatory bowel disease (IBD)—a general term for inflammatory diseases of the bowel of unknown etiology, including Crohn’s disease and ulcerative colitis
irritable bowel syndrome (IBS)—an abnormal stooling pattern associated with symptoms of intestinal dysfunction that persists for more than 3 months of the year
Dietary modifications in disorders of the intestinal tract are designed to alleviate symptoms, correct nutrient deficiencies, and, when possible, address the primary cause of difficulty. In disease, assessment of the nature and severity of the primary gastrointestinal problem precedes targeted medical, nutrition, and other forms of therapy. Increased intakes of energy, protein, vitamins, minerals, and electrolytes are frequently required to replace nutrients lost as a result of impaired digestive and absorptive capacity. Consistency, meal frequency, and other characteristics of the diet may be altered to fit the patient’s needs. Medical nutrition therapy (MNT) for all patients with diseases of the intestines must be individualized. For this reason, the principles presented in the chapter are general guidelines.

COMMON INTESTINAL PROBLEMS

Intestinal Gas and Flatulence

Pathophysiology
Intestinal gases include nitrogen (N₂), oxygen (O₂), carbon dioxide (CO₂), hydrogen (H₂), and, in some persons, methane (CH₄). About 200 ml of gas is normally present in the gastrointestinal (GI) tract, and humans excrete an average of 700 ml each day. However, the difference in the amount of intestinal gas among individuals and from day to day varies greatly (Strocchi and Levitt, 1998). Considerable amounts of gas may be swallowed or produced within the GI tract and may be absorbed across the alimentary tract into the bloodstream and expired through the lungs, expelled through eructation (belching), or passed rectally.

When patients complain about “excessive gas,” they may be referring to increased volume or frequency of passage of gas (flatulence). They may also be complaining about abdominal distention or cramping pain associated with the accumulation of gases in the upper or lower GI tract. The association between the amount of gas in the GI tract perceived by an individual and the amount actually measured is not always accurate (Levitt et al, 1996). Inactivity, decreased GI motility, aerophagia, dietary components, and GI disorders can all contribute to the amount of intestinal gas and an individual’s gas-related symptoms.

Gas in the upper intestinal tract results primarily from swallowing air (aerophagia) and, to a lesser extent, from chemical reactions that occur during the digestion of foods. Normally, only small amounts of swallowed air or gases dissolved in foods make their way as far as the colon. High N₂ and O₂ concentrations in rectal gas, both of which are substances that are present in the atmosphere in high concentrations, may indicate aerophagia. Aerophagia can be avoided to some degree by eating slowly, chewing with the mouth closed, and refraining from drinking through straws.

Increased gas production may occur in the stomach and small intestine because of bacterial fermentation, particularly with the consumption of carbohydrate, and can result in abdominal discomfort and distention. Bacterial overgrowth may occur in the stomach or small intestine with partial obstruction, with dysmotility, in immune disorders, or after some GI surgical procedures.

Increased amounts of H₂ and CO₂—and sometimes, CH₄—in rectal gas with lowered fecal pH indicate excessive colonic bacterial fermentation and suggest malabsorption of a fermentable substrate. The
amounts and types of gases produced may depend on the mix of microorganisms in the individual’s colon. Consumption of large amounts of dietary fiber (especially soluble fiber), resistant starches, lactose in persons who are lactase deficient, or modest amounts of fructose or alcohol sugars (such as sorbitol) may result in increased gas production in the colon and increased flatulence.

Consumption of fructose in the United States, especially from fruit juices and fruit drinks and high-fructose corn syrup in soft drinks and confections, has increased significantly in recent years. The average level of fructose in the daily diet is in the range of 35 to 40 g, which is sufficient in many children and adults to result in malabsorption of fructose and evoke symptoms. Sucrose is normally well tolerated but in large quantities may also result in increased amounts of fecal substrate.

**Medical Nutrition Therapy**

In the assessment of the patient, one must ask whether the problem is increased production of gas or whether the patient has difficulty with cramping and distention because the gas is not being passed. Inactivity, dysmotility, or partial obstruction may be contributing to the inability to move normal amounts of gas produced. Movement or exercise may help expel gases through eructation or rectal passage.

The primary emphasis in dietary management is the reduction of carbohydrate foods that are likely to be malabsorbed and fermented, including legumes, soluble fiber, resistant starches, and simple sugars such as fructose and alcohol sugars. When undigested carbohydrates pass into the colon, they are fermented to varying degrees to short-chain fatty acids and gases. The primary gases include H₂, CO₂, and, in about one third of individuals, CH₄. The widely recognized propensity of legumes to produce flatus (gas) is related to the presence of not only ample amounts of fiber but also stachyose and raffinose—carbohydrates that are only partially digested in the small intestine.

Excess production of gas may also be related to the dose of carbohydrate consumed. Starches such as breads, baked goods, and starchy vegetables may be almost completely digested in normal portions but when consumed in large quantities may leave a considerable fraction of undigested or unabsorbed residue for bacterial action in the colon. The properties of some so-called gas-forming foods may be explained simply by the type and amount of sugar, starch, or fiber they contain.

**Constipation**

**Pathophysiology**

Constipation is one of the most common intestinal maladies in Western societies, and it occurs in 5% to more than 25% of the population, depending on the definition of the disorder (Candelli et al, 2001). Definitions of constipation tend to be highly subjective but usually include hard stools, straining with defecation, and infrequent bowel movements. At least in older patients, hard stools, incomplete evacuation, and difficulty passing stools may be more troublesome than the infrequency of bowel movements.

Normal stool weight is about 100 to 200 g daily, and normal frequency may range from one stool every 3 days to three times per day. Normal transit time through the GI tract ranges from about 18 to about 48 hours. Persons who consume a diet that contains the recommended amounts of dietary fiber in the form of fruits, vegetables, and whole-grain breads and cereals tend to have larger, softer stools that are relatively easy to pass. Some people who believe that it is necessary to have frequent bowel movements and who ignore dietary and other health recommendations may become disturbed when this does not occur and try to compensate with the use of medications and enemas.

The most common causes of constipation in otherwise healthy persons include repeated lack of response to the urge to defecate, lack of fiber in the diet, insufficient fluid intake, inactivity, and chronic use of laxatives. Nervous strain or anxiety may aggravate the condition. Chronic constipation may also result from a variety of causes, as outlined in Box 30-1.

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**Box 30-1. Causes of Constipation**

**Systemic/Neurogenic/Metabolic**

- Side effect of medication
- Metabolic and endocrine abnormalities, such as hypothyroidism, uremia, and hypercalcemia
- Lack of exercise
- Ignoring the urge to defecate
- Vascular disease of the large bowel
- Systemic neuromuscular disease leading to deficiency of voluntary muscles
- Poor diet, low in fiber
- Pregnancy

**Gastrointestinal**

- Cancer
- Diseases of the upper gastrointestinal tract
- Diseases of the large bowel resulting in:
  - Failure of propulsion along the colon (colonic inertia)
  - Failure of passage through anorectal structures (outlet obstruction)
- Irritable bowel syndrome
- Anal fissure or hemorrhoid
- Laxative abuse

Medical Treatment for Adults

The first approach to treatment of constipation is to ensure adequate dietary fiber, fluid, and exercise and heed the urge to defecate. Patients dependent on laxatives are usually encouraged to use milder products and reduce the dose until withdrawal is complete. When constipation is not amenable to conservative treatment, that is, the patient is unable to consume an adequate amount of fibrous foods or exercise, substances that promote regular evacuation of soft stools may be prescribed. Bulking agents, such as cellulose, hemicellulose derivatives, psyllium seed, flaxseed, and ispaghula, and osmotic agents, such as lactose, magnesium hydroxide, and sorbitol, can be used. Stool softeners such as Colace may also be used. Impactions of stool may require additional evaluation, more stringent oral medications, rapid consumption of large volumes of fluids, enemas, or digital evacuation (Candelli et al, 2001). In more extreme cases, such as toxic megacolon, surgery may be advised.

Medical Treatment for Infants and Children

About 3% to 5% of all pediatric outpatient visits are related to chronic constipation. In the most severe cases, there is a flaccid colon that is insensitive to distention and encopresis develops. After initial treatment with laxatives and lubricants, fiber intake is the next focus of care. A careful history and physical examination, followed by parent and child education, behavioral intervention, and appropriate use of laxatives, often leads to dramatic improvement (Loening-Baucke, 2002) (see Chapter 10).

Medical Nutrition Therapy

Primary nutrition therapy for constipation is adequate consumption of both soluble and insoluble dietary fiber. Fiber increases colonic fecal fluid, microbial mass, stool weight and frequency and the rate of colonic transit. Fiber also softens stools and makes them easier to pass. Most adults and children in the United States chronically consume only about half the amount of fiber recommended (Institute of Medicine, 2002). These recommendations need to be written out the first time.

The recommended amount of dietary fiber is about 14 g per 1000 kcal. For adult women the diet should contain 25 g of fiber daily, and for men about 38 g (Institute of Medicine, 2002). Fiber is best provided in the form of whole grains, fruits, vegetables, legumes, seeds, and nuts (Marlett et al, 2002). These foods are high in nutrients and healthful phytochemicals and may serve as prebiotics to maintain the desired colonic microflora. Brans and powdered fiber supplements may be helpful in persons who cannot or will not eat sufficient amounts of fibrous foods. When changes in diet and activity patterns do not improve constipation, further evaluation is warranted.

High-Fiber Diet

Dietary fiber refers primarily to edible plant materials not digested by the enzymes in the upper digestive tract of humans. It generally consists of cellulose, hemicelluloses, pectins, gums, and lignin. Newer definitions may include starchy materials and oligosaccharides that are at least partially resistant to digestive enzymes. Some forms of fiber and resistant starches may also be termed prebiotics. The term residues tends to refer to vegetable matter, but it is not a quantitative term. Residue is not the same as fiber, and this term refers to the end result of digestive, secretory, absorptive, and fermentative processes. Thus, increasing dietary fiber may result in increased fecal output, but increasing dietary lactose (a fiber-free food) in a person who is a lactose malabsorber would also increase fecal weight (residue).

The high-fiber diet in Box 30-2 provides more than 25 g of dietary fiber, depending on the foods selected. A high-fiber therapeutic diet may exceed 25 to 38 g, but amounts greater than 50 g per day are not likely to be necessary and may increase abdominal distention and excessive flatulence in some persons. Appendix 42 provides a list of the fiber content of foods.

Ideally, fiber in the diet should be ingested in the form of foods such as fruits, vegetables, whole-grain breads and cereals, legumes, nuts, and seeds. These foods are not only rich in fiber but are excellent sources of vitamins, minerals, trace elements, antioxidants, and numerous protective phytochemicals. Fibrous powders or bran concentrates may be necessary to obtain the desired fiber level in some persons. Several of these concentrates available on the market are palatable and can be added to cereals, yogurts, fruit sauces, juices, or soups. The use of foods as the

Box 30-2. Guidelines for High-Fiber Diets*

1. Increase consumption of whole-grain breads, cereals, flours, and other whole-grain products (6-11 servings daily).
2. Increase consumption of vegetables, legumes, and fruits, nuts, and edible seeds (5-8 servings daily).
3. Consume high-fiber cereals, granolas, and legumes as needed to bring fiber intake to 25 g or more daily.
4. Increase consumption of fluids to at least 2 L (or about 2 qt) daily.

*May increase stool weight, fecal water, and/or gas. The amount that causes clinical symptoms varies among individuals. The age of the individual, the presence of gastrointestinal (GI) disease or malnutrition, any resection of the GI tract, and recent use of the GI tract all impact tolerance.
fiber source results in benefits from both the fiber content and the nutrients and protective phytochemicals included in the foods.

Cooking does not destroy fiber, although the structure may change. Consumption of eight 8-oz glasses (2 L) of fluids daily is recommended to facilitate the effectiveness of a high-fiber intake. Gastric obstruction and fecal impaction may occur when boluses of fibrous gels or bran are not consumed with sufficient fluid to disperse the fiber. Appropriate cautions are also warranted for persons with GI strictures or dysmotility syndromes. In these situations, the fiber content of the diet should be increased slowly, taking almost a month to reach intakes of 25 to 38 g of fiber per day (Institute of Medicine, 2002).

On initiation of a high-fiber diet, unpleasant side effects may occur, such as increased flatulence, borborygmus, cramps, or diarrhea. A gradual increase in fiber intake helps alleviate these symptoms. If fiber supplements are used, doses should be interspersed with meals, preferably in two or more small doses per day, and fluid intake may need to be increased at the same time. GI disturbances associated with initial fiber ingestion usually decrease within 4 to 5 days, but some increase in flatulence is normal with a high-fiber intake. The high-fiber diet is most effective when consumed continuously for several months.

Diarrhea
Pathophysiology
Diarrhea is characterized by the frequent evacuation of liquid stools, usually exceeding 300 ml, accompanied by an excessive loss of fluid and electrolytes, especially sodium and potassium. It occurs when there is excessively rapid transit of intestinal contents through the small intestine, decreased enzymatic digestion of foodstuffs, decreased absorption of fluids and nutrients, or increased secretion of fluids into the GI tract (Barness et al, 1996; Fine, 1998). Causes may be related to inflammatory disease; infections with fungal, bacterial, or viral agents; medications; the overconsumption of sugars; an insufficient or damaged mucosal absorptive surface; or malnutrition.

Osmotic diarrheas occur when osmotically active solutes are present in the intestinal tract and are poorly absorbed. Examples include the diarrhea that accompanies dumping syndrome and following lactose ingestion in the person with a lactase deficiency. Secretory diarrheas are the result of active secretion of electrolytes and water by the intestinal epithelium. Bacterial exotoxins, viruses, and increased intestinal hormone secretion cause acute secretory diarrheas. Unlike osmotic diarrhea, fasting does not relieve secretory diarrhea.

Exudative diarrheas are always associated with mucosal damage, which leads to an outpouring of mucus, fluid, blood, and plasma proteins, with a net accumulation of electrolytes and water in the gut. Prostaglandin and cytokine release may be involved. The diarrheas associated with Crohn’s disease, ulcerative colitis, and radiation enteritis are exudative.

Medications, especially antibiotics, can contribute to diarrhea in several ways. Antibiotics can reduce the usual “salvage” by colonic bacteria of the small amounts of foodstuffs that escape digestion and absorption. Broad-spectrum antibiotics can greatly reduce the numbers of colonic bacteria that normally convert osmotically active molecules (carbohydrate and amino acids) to gases and short-chain fatty acids (SCFAs). The SCFAs are normally absorbed from the lumen of the colon as long as the amount produced is close to normal. Absorption of the SCFAs also aids absorption of electrolytes and water from the colon (Cunha, 1998). Eradication of the bacteria from the colon results in accumulation of osmotically active molecules and reduced absorption of electrolytes and water.

If more substrates than usual are malabsorbed, as often occurs in acutely ill patients, the resulting rise in osmolality can cause considerable fluid loss. Antibiotics can also have direct effects on GI function (Bartlett, 2002; Kyne et al, 2001) (see Chapter 19). Erythromycin, for example, increases GI motility. Erythromycin, clarithromycin, and clindamycin may all increase GI secretions. Finally, some antibiotics allow opportunistic proliferation of pathogenic organisms normally suppressed by competitive organisms in the GI tract. The organisms or the toxins produced decrease absorption and increase secretion of fluid and electrolytes. Clostridium difficile is most commonly associated with antibiotic-related diarrhea and accounts for 10% to 25% of cases, but Clostridium perfringens, Salmonella, Shigella, Campylobacter, Verminia enterocolitica, and Escherichia coli organisms have also been implicated in antibiotic-associated diarrhea (Bartlett, 2002; Job and Jacobs, 1997; Kyne et al, 2001). Clindamycin, penicillins, and cephalosporins are associated most often with the development of C. difficile infection, and its occurrence depends on the number of antibiotics used, the duration of exposure to antibiotics, and the patient’s overall health.

With human immunodefi ciency virus (HIV) and other immune defi ciency states, several factors may contribute to the diarrhea, including the toxic effects of medications, proliferation of opportunistic organisms, and the GI manifestations of the disease itself (Fine, 1998; Mitra et al, 2001). Increased risk of opportunistic infection is also associated with use of anti-infective agents (Fine, 1998; Kornblau et al, 2000) and with severe malnutrition. Antacids (especially magnesium salts), histamine H2-receptor blockers, and proton pump inhibitors have also been implicated in cases of diarrhea.

Diarrhea caused by limited functional mucosa results from conditions of inadequate absorptive area or rapid transit of chyme, such as might occur in Crohn’s disease or after extensive bowel resection. This type of diarrhea is often complicated by malabsorption of lipid (steatorrhea) and other macronutrients and micronutrients.
Medical Treatment

Because diarrhea is a symptom of a disease state, the first step in medical treatment is to identify and treat the underlying problem. The next priority is to manage fluid and electrolyte replacement. Losses of electrolytes, especially potassium and sodium, should be corrected early by using oral glucose electrolyte solutions with added potassium. With intractable diarrhea, especially in an infant or young child, parenteral feeding may be required. Parenteral nutrition may even be necessary if exploratory surgery is anticipated or if the patient is not expected to resume full oral intake within 5 to 7 days (see Chapter 23).

Medical Nutrition Therapy for Adults

Nutrition therapy for adults with diarrhea includes replacing lost fluids and electrolytes by adding broths and electrolyte solutions. In most cases of diarrhea, a minimum-residue diet (Table 30-1) may be started as the acute episode resolves. Modest amounts of fat may be used if digestive mechanisms for lipid are intact. Sugar alcohols, lactose, fructose, and large amounts of sucrose may worsen osmotic diarrhea and might need to be limited. Because the activity of the disaccharidases and transport mechanisms may be decreased during inflammatory and infectious intestinal disease, sugars may need to be limited (Rumessen and Gudmand-Hoyer, 1998).

Use of modest amounts of foods or dietary supplements containing prebiotic components, such as pectin, fructose, oligosaccharides, inulin, oats, banana flakes, and chicory, may help to control or treat diarrhea. They favor the maintenance of so-called friendly lactobacillus and bifidus microbes and may prevent the overgrowth of potentially pathogenic organisms (Gibson, 1999; Van Loo et al, 1999). SCFAs in physiologic quantities serve as substrate for colonic microflora, facilitate the absorption of fluid and salts, and may help to regulate GI motility. Fibrous material and several types of prebiotic foods also tend to slow gastric emptying, moderate overall GI transit, and hold water.

Ingestion of some types of probiotics (sources of bacteria used to reestablish beneficial gut flora) in the form of cultured foods or supplements, with or without prebiotics, has been modestly successful in antibiotic-related diarrhea, traveler’s diarrhea, bacterial overgrowth, and some pediatric diarrhea. Additional study is needed, but some products appear to have promise in specific applications (Madsen, 2001; Szejewska and Mrukowicz, 2001).

Severe and chronic diarrhea is accompanied by dehydration and electrolyte depletion. If also accompanied by prolonged infectious, immunodeficiency, or inflammatory disease, malabsorption of vitamins, minerals, and protein or lipid may also occur, and the nutrients may need to be replaced parenterally or enterally. The loss of potassium alters bowel motility, encourages anorexia, and can introduce a cycle of bowel distress. Loss of iron from GI bleeding may be severe enough to cause anemia. Nutrition deficiencies themselves cause mucosal changes, such as decreased villi height and reduced enzyme secretion, further contributing to malabsorption. As the diarrhea begins to resolve, the addition of more normal amounts of fiber to the diet may help to restore normal mucosal function, increase electrolyte and water absorption, and increase the firmness of the stool.

Food in the lumen is needed to restore the compromised GI tract after disease and periods of fasting. Early refeeding after rehydration reduces stool output and shortens the duration of illness. Micronutrient replacement or supplementation may also be useful for acute diarrhea, probably because it accelerates the normal regeneration of damaged mucosal epithelial cells.

### Medical Nutrition Therapy for Infants and Children

Acute diarrhea is most dangerous in infants and small children, who are easily dehydrated by large fluid losses. In these cases, replacement of fluid and electrolytes must be aggressive and immediate. Standard oral rehydration solutions recommended by

<table>
<thead>
<tr>
<th>FOOD</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactose (in lactose malabsorbers)</td>
<td>6-12 g normally tolerated in healthy, lactase-deficient individuals.</td>
</tr>
<tr>
<td>Fiber (excess; &gt;20 g)</td>
<td>Modest amounts (10-15 g) may help maintain normal consistency of GI contents and normal colonic mucosa in healthy states and in GI disease.</td>
</tr>
<tr>
<td>Resistant starch (especially raffinose and stachyose found in legumes)</td>
<td>Well tolerated in moderate amounts; large amounts may cause hyperosmolar diarrhea or decreased fecal pH with fermentation to short-chain fatty acids.</td>
</tr>
<tr>
<td>Sorbitol, mannitol, and xylitol (excess; &gt;10 g/day)</td>
<td>Increases GI secretions, colonic motility.</td>
</tr>
<tr>
<td>Fructose (excess; 20-25 g/meal)</td>
<td>Increase GI secretions.</td>
</tr>
<tr>
<td>Sucrose (excess; &gt;25-50 g/meal)</td>
<td>Increase GI secretions.</td>
</tr>
<tr>
<td>Caffeine</td>
<td></td>
</tr>
<tr>
<td>Alcoholic beverages (especially wine and beer)</td>
<td>Increase GI secretions.</td>
</tr>
</tbody>
</table>

the World Health Organization (WHO) since 1986 and the American Academy of Pediatrics (AAP) contain a 2% concentration of glucose (20 g/L), 45 to 90 mEq/L of sodium, 20 mEq/L of potassium, and a citrate base (Table 30-2). Newer, reduced osmolarity solutions (osm ~ 130 to 200 mOsm/L) have been shown to be equally or more effective in treating persistent diarrhea in children (Hahn, Kim, and Garner, 2002).

Newer solutions such as Pedialyte, Infalyte, Lytre, Equalyte, and Rehydration typically contain less glucose and slightly less salt and are available in pharmacies, some without prescription. Oral rehydration therapy is less invasive and less expensive than intravenous rehydration and, when used with children, allows parents to assist with their children’s recovery (Goepp and Katz, 1993).

A substantial proportion of children 9 to 20 months of age can maintain adequate intake when offered either a liquid or a semisolid diet continuously during bouts of acute diarrhea. Even during acute diarrhea, the intestine can absorb up to 60% of the food eaten. Some practitioners have been slow to adopt the practice of early refeeding after severe diarrhea in infants, despite evidence that “resting the gut” is actually more damaging (Booth, 1993). A recent report from the working group report of the First World Congress of Pediatric Gastroenterology, Hepatology, and Nutrition suggests that strategies must be cohesive and uniform to address the problems of pediatric diarrhea to reduce deaths worldwide (Davidson et al, 2002). Prescription of a high-sugar, clear liquid diet is inappropriate for recovery from diarrhea.

**Steatorrhea**

**Pathophysiology**

Steatorrhea, or excessive fat in the stool, is a consequence of disease, surgical resection of organs involved in digestion, and absorption of lipid. Normally, 90% to 98% of ingested fat is absorbed; but in steatorrhea, the percent remaining in the stool may increase to 20% or more. Diagnosis is usually based on a ratio of fecal fat to ingested fat or a coefficient of absorption. A diet containing 75 to 100 g of fat is usually fed for 72 hours, the amount of fat actually consumed is recorded, and the fecal fat content is analyzed. The upper limit of normal fecal fat is usually in the range of 7%. Steatorrhea may result from (1) inadequate bile secretion secondary to liver disease or biliary obstruction; (2) blind loop syndrome; (3) pancreatic insufficiency; (4) inadequate reabsorption of bile salts due to diseases involving the distal ileum (as in sprue, Crohn’s disease, or GI irritation); or (5) decreased fat reesterification and decreased formation and transport of chylomicrons, as may be seen in abetalipoproteinemia and intestinal lymphangiectasia. Box 30-3 lists disorders associated with malabsorption.

**Medical Treatment**

Because steatorrhea is a symptom and not a disease, the underlying cause of malabsorption must be determined and treated. With pancreatic insufficiency, oral pancreatic enzymes can be used to increase lipid digestion.

**Medical Nutrition Therapy**

Steatorrhea can result in chronic weight loss and may require compensatory increased energy intake, primarily in the form of dietary protein and complex carbohydrate. Medium-chain triglycerides (MCTs) can be used in the diet because they have a short chain length, allowing easier absorption in the absence of bile acids. Medium-and short-chain fatty acids are able to enter the portal venous blood for direct transport to the liver without being resynthesized into triglycerides in the intestinal cell.

The MCTs are available in some enteral formulas and also as MCT oil (8.3 kcal/g; 1 T = 116 kcal). The oil is best used when it is incorporated into foods rather than administered by the spoonful. MCT can be used to make salad dressings, sandwich spreads, or confections, and it can be substituted for fats in most recipes. Normally, divided doses of less than 15 g of oil per feeding are better tolerated and absorbed than larger quantities. When steatorrhea is present, there is increased risk of vitamin deficiencies, especially fat-soluble vitamins and deficiencies of the minerals calcium, zinc, and magnesium.

**Gastrointestinal Strictures and Obstruction**

**Pathophysiology**

Inflammatory bowel disease (IBD), peptic ulcer disease, GI surgeries, tumors, or radiation enteritis may partially or completely obstruct the GI tract as can scarring or dysfunctional segments. Obstructions in
Box 30-3. Diseases and Conditions Associated With Malabsorption

- Inadequate digestion
- Pancreatic insufficiency
- Gastric acid hypersecretion
- Gastric resection
- Altered bile salt metabolism with impaired micelle formation
- Hepatobiliary disease
- Interrupted enterohepatic circulation of bile salts
- Bacterial overgrowth
- Drugs that precipitate bile salts
- Abnormalities of mucosal cell transport
- Biochemical or genetic abnormalities
- Disaccharidase deficiency
- Monosaccharide malabsorption
- Specific disorders of amino acid malabsorption
- Abetalipoproteinemia
- Vitamin B₁₂ malabsorption
- Celiac disease
- Inflammatory of infiltrative disorders
- Crohn’s disease
- Ulcerative colitis
- Amyloidosis
- Scleroderma
- Tropical sprue
- Gastrointestinal allergy
- Infectious enteritis
- Whipple’s disease
- Intestinal lymphoma
- Radiation enteritis
- Drug-induced enteritis
- Endocrine and metabolic disorders
- Short-bowel syndrome
- Abnormalities of intestinal lymphatics and vascular system
- Intestinal lymphangiectasia
- Mesenteric vascular insufficiency
- Chronic congestive heart failure


Medical Nutrition Therapy

Because fiber is not digested to any significant degree (except by fermentation in the colon), and because chewing is not a reliable way of reducing the size of fibrous foods, both the amount and size of fibrous material usually must be controlled. A restricted-fiber diet typically restricts fruits, vegetables, and coarse grains and usually provides less than 10 to 15 g of dietary fiber, usually in the form of particulate matter. Particularly with distal obstructions or strictures, it may be beneficial to keep the stool soft by including modest amounts of fiber but of small particle size.

Some intestinal obstruction cases may require clear liquids or total restriction of food and parenteral nutrition and fluid used as needed. Working with the physician is necessary to determine the nature, site, and duration of the obstruction so that nutrition therapy can be individualized.

DISEASES OF THE SMALL INTESTINE

Celiac Disease (Gluten-Sensitive Enteropathy)

Pathophysiology

Celiac disease, or gluten-sensitive enteropathy, results from an inappropriate T-cell–mediated immune response caused by ingested gluten by people who are genetically predisposed. The prevalence of the disease may have been underestimated in the past and now is considered to be about 1 in 133 persons in the United States. Prevalence is higher in relatives of persons with celiac disease (Fasano et al, 2003). Onset is usually from infancy to young adulthood, but about 20% of cases occur in adults over 60 years of age (Farrell and Kelley, 2002).

Gluten refers to specific peptide fractions of proteins found in wheat, rye, and barley. These peptide molecules are modified during absorption to a form that triggers a local and in many cases systemic immune response. In untreated cases, the overzealous immune and inflammatory response eventually results in damage to the intestinal mucosa, altered neuropeptide secretion, and decreased digestive and absorptive functions. Cells of the villi become deficient in the disaccharidases and peptidases needed for digestion and also in the carriers needed to transport nutrients into the bloodstream. The extent of villous changes varies greatly, but sufficient atrophy...
and flattening of the villi eventually occur to compromise micronutrient and macronutrient absorption (Figure 30-1).

Decreased release of peptide hormones from the small intestine results in reduced secretions from the gallbladder and pancreas, further contributing to malabsorption. The disease primarily affects the proximal and midportions of the small bowel, although the more distal segments may also be involved. Because the symptoms are nonspecific and vary greatly, celiac disease may be misdiagnosed for years as irritable bowel or other GI disorders.

The disease may also be associated with other inflammatory states such as dermatitis herpetiformis (a variant of celiac disease that involves the skin), muscle and joint pain, and other autoimmune diseases such as thyroiditis and type 1 diabetes. Celiac disease is normally considered chronic and requires life-long omission of gluten from the diet. Morbidity and mortality rates may be increased in persons who are undiagnosed until late or in persons who are unable to comply with the diet (Seraphin and Mobarin, 2002). Increased risk of lymphomas and other malignancies influences overall morbidity, and the risk of malignancies appears to be increased in those who continue to eat gluten-containing foods (Troncone et al, 1996). Box 30-4 lists the extraintestinal manifestations.

The diagnosis of celiac disease is made by a combination of clinical, laboratory, and histologic evaluations, but small bowel biopsy is the final diagnostic end point. The disease may become apparent when an infant begins eating gluten-containing cereals, or it may not appear until middle age, when it may be triggered or unmasked by GI surgery, stress, pregnancy, or viral infection. The presentation in young children is more likely to include the more “classic” symptoms of diarrhea and steatorrhea, malodorous stools, abdominal bloating, apathy, and poor weight gain. In late onset, the first manifestation is more varied and may include other inflammatory and autoimmune disorders, generalized fatigue, failure to gain or maintain weight, or the consequences of nutrient malabsorption, including anemias, osteoporosis, or vitamin K-related coagulopathy. Fifty percent of celiac patients, however, have few or no obvious symptoms, and some may be overweight at presentation (Fasano and Catassi, 2001; Hill et al, 2002).

Persons in whom celiac disease is suspected are further evaluated for the overall pattern of symptoms and family history and then are typically screened using serologic tests, which include the presence of antien-
Box 30-4. Extraintestinal and Associated Manifestations of Celiac Disease

<table>
<thead>
<tr>
<th>Extraintestinal</th>
<th>Associated Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia (iron or folate, rarely, B₁₂)</td>
<td>Autoimmune diseases—type 1 diabetes,</td>
</tr>
<tr>
<td>Lassitude, malaise (sometimes despite</td>
<td>thyroiditis, hepatitis, collagen</td>
</tr>
<tr>
<td>lack of anemia), arthritis, arthralgia</td>
<td>vascular disease</td>
</tr>
<tr>
<td>Dermatitis herpetiformis</td>
<td>Malignancies</td>
</tr>
<tr>
<td>Osteomalacia, osteopenia, fractures</td>
<td>IgA deficiencies</td>
</tr>
<tr>
<td>(vitamin D deficiency; inadequate</td>
<td></td>
</tr>
<tr>
<td>calcium absorption)</td>
<td></td>
</tr>
<tr>
<td>Dental enamel hypoplasia</td>
<td></td>
</tr>
<tr>
<td>Coagulopathies (vitamin K deficiency)</td>
<td></td>
</tr>
<tr>
<td>Infertility, increased risk of abortion</td>
<td></td>
</tr>
<tr>
<td>puberty, delayed growth</td>
<td></td>
</tr>
<tr>
<td>Hepatic steatosis, hepatitis</td>
<td></td>
</tr>
<tr>
<td>Neurologic symptoms (ataxia, poly-neuropathy, seizures)</td>
<td></td>
</tr>
<tr>
<td>Psychiatric syndromes</td>
<td></td>
</tr>
</tbody>
</table>


Gastrointestinal enzymes (AEAs), immunoglobulin A (IgA) or IgG-AGA antibodies (antigliadin antibodies), or the autoantigen that appears to trigger the immune response (IgA tissue transglutaminase [tTG]) (Hill et al, 2002; Mustalahi et al, 2002; Petaros et al, 2002). Some persons thought to have celiac disease may be IgA deficient, and so IgA levels are normally done first. The serologic tests may also be used for monitoring the progress of persons with confirmed celiac disease.

The serologic tests are highly specific and sensitive for celiac disease, but the gold standard for final diagnosis is still the intestinal mucosal biopsy (Farrell and Kelly, 2002). Because intestinal biopsy is relatively expensive and must be performed by upper GI endoscopy, it is not usually used for initial screening. Because dietary change would alter the diagnostic results, initial diagnostic evaluation should be done before the patient has withdrawn gluten-containing foods from the diet.

Major clinical symptoms usually abate in most patients 2 to 8 weeks after consuming a gluten-free diet, but for some patients it may take longer. With strict dietary control, levels of the specific antibodies usually become undetectable in 3 to 6 months in most persons, but in some the recovery may be slower (Farrell and Kelley, 2002; Hill et al, 2002). A small percentage of patients may be refractory to dietary therapy because of inadvertent gluten intake, pancreatic insufficiency, irritable bowel, bacterial overgrowth, fructose intolerance, or other coexisting GI maladies (Abdulkarim et al, 2002).

**Medical Treatment**

Institution of a gluten-free diet greatly diminishes the autoimmune process, and the intestinal mucosa usually reverts to normal; however, some patients may require months or even years of diet therapy for maximal recovery. The toxic peptide fractions of the respective cereals must be avoided for life. Refractory sprue may not respond entirely to the removal of gluten, or it may respond only temporarily. Many of these patients, however, do show a response to steroids, azathioprine, cyclosporine, or other medications classically used in inflammatory or immunologic reactions. For some, treatment of other underlying disease may further resolve symptoms (Figure 30-2).

A new study has identified the peptide fraction from gliadin that causes the problem (see New Directions: Bacterial Endopeptidase—A New Treatment for Celiac Disease?).

**Medical Nutrition Therapy**

Complete withdrawal of gluten results in clinical improvement. The diet requires a major life change on the part of the patient to adhere to it sufficiently to bring about remission. Insofar as possible, the diet omits all dietary wheat (gliadin), rye (secalin), and barley (hordein), sources of the prolamin fractions (Thompson, 2001) (Table 30-3).

The diet should initially be supplemented with vitamins, minerals, and extra protein to remedy deficiencies and replenish nutrient stores. Anemia should be treated with iron, folate, or vitamin B₁₂, depending on the nature of the anemia. Calcium and vitamin D administration may be necessary to correct osteoporosis or osteomalacia. Vitamins A and E may be necessary to replenish stores depleted by steatorrhea. Vitamin K may be prescribed for purpura, bleeding, or prolonged prothrombin time. Electrolyte and fluid replacement is essential for those dehydrated from severe diarrhea.

Those who continue to have malabsorption should take a multiple vitamin and mineral supplement to at least meet dietary reference intakes (DRIs). MCT may help provide calories, especially in persons with steatorrhea. Lactose and fructose intolerance sometimes occurs secondary to celiac disease. A low-lactose or low-fructose diet may be useful in controlling symptoms, at least initially. Once the GI tract returns to more normal function, lactase activity may also return and a trial of lactose ingestion can be tried.

In the traditional diet, wheat, rye, barley, and oats are normally excluded. Recently, the need for exclu-
CAUSE

MEDICAL MANAGEMENT

NUTRITIONAL MANAGEMENT

PATHOPHYSIOLOGY

CAELIC DISEASE
(GLUTEN-SENSITIVE ENTEROPATHY OR NONTROPICAL SPRUE)

GLUTEN INTOLERANCE
Alcohol-soluble component of wheat, rye, barley protein

GENETIC CONTRIBUTION

IMMUNE COMPONENT: antibodies to specific dietary protein fractions

DAMAGE TO SMALL BOWEL
Atrophy and flattening of villi
Reduced area for absorption
Cellular deficiency of disaccharidases and peptidases
Reduced nutrient transport carriers

EXTRAINTESTINAL EFFECTS
Anemia
Bone loss
Muscle weakness
Polyneuropathy
Endocrine disorders
Follicular hyperkeratosis

ELECTROLYTE AND FLUID REPLACEMENT

VITAMIN AND MINERAL SUPPLEMENTATION

CALCIUM AND VITAMIN D ADMINISTRATION

READ FOOD LABELS CAREFULLY FOR HIDDEN GLUTEN-CONTAINING INGREDIENTS

DELETE GLUTEN SOURCES (WHEAT, RYE, BARLEY) FROM DIET

SUBSTITUTE WITH CORN, POTATO, RICE, SOYBEAN, TAPIoca, AND ARROWROOT

FIGURE 30-2 • Pathophysiology algorithm: celiac disease (gluten-sensitive enteropathy or nontropical sprue). (Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Peter L. Beyer, MS, RD, LD, 2002.)
Bacterial Endopeptidase—A New Treatment for Celiac Disease?

Recently scientists identified a 33-amino acid peptide fraction from the 266-amino acid gliadin that triggers the destructive inflammatory response in celiac disease (Shan et al, 2002). The fragment appears to be the same one in other grains that contain gluten. It appears that this peptide fraction is resistant to digestion by normal human digestive enzymes, but it can be degraded by bacterial endopeptidases. In preliminary studies, destruction of the peptide fragment by the addition of the endopeptidase enzyme prevented the typical immunologic response seen with celiac disease. The hope is that oral endopeptidase enzymes can be used as an oral supplement to digest and destroy the specific fragment of gliadin in the foods that contain gluten, thus allowing their consumption.

Tropical Sprue

Pathophysiology

Tropical sprue is a diarrheal syndrome that occurs in many tropical areas. The diarrhea appears to be an infectious type, and the intestinal organism identified may differ from one region of the tropics to the next (Farthing, 1998). As in celiac disease, the intestinal villi are shortened, but the surface cell alterations are much less severe. The gastric mucosa may be atrophied and inflamed, with diminished secretion of hydrochloric acid and intrinsic factor.

Symptoms include diarrhea, anorexia, and abdominal distention as well as symptoms of nutritional deficiency, such as night blindness, glossitis, stomatitis, cheilosis, pallor, and edema. Anemia may result from iron, folic acid, and vitamin B₁₂ deficiencies.

Medical Treatment

Treatment involves restoration of fluids, electrolytes, and nutrients. Tropical sprue typically responds promptly to broad-spectrum antibiotics and folate therapy. Along with other nutrients as needed, folate is given orally, 5 mg daily, along with intramuscular vitamin B₁₂ (1000 mg/month) until symptoms subside.

The diet for celiac disease requires a major change in the types of foods normally consumed in the Western diet. Foods made from wheat, in particular (breads, cereals, pastas, and baked goods), are consumed as staples in the U.S. diet. A truly gluten-free diet requires careful scrutiny of the labels of all bakery products and packaged foods. Gluten-containing grains are not only used as a primary ingredient but may also be added during processing or preparation of foods. Hydrolyzed vegetable protein can be made from wheat, soy, corn, or mixtures of these grains (Table 30-5).

Freedom from symptoms after eating gluten does not necessarily mean that cells of the GI tract are undamaged. The precipitating condition usually continues to exist, and gluten causes mucosal changes within hours; however, overt symptoms may take 8 weeks or longer to reappear or may remain latent. Adults who start and stop a gluten-free diet numerous times eventually may reach a state at which they do not respond to the diet. Complications of chronic ulcerative jejunoileitis and extraintestinal manifestations may develop. Risk of malignant disease, especially lymphoma, is increased, and adherence to a gluten-free diet appears to reduce the risk (Saraphin and Mobarin, 2002; Troncone, 1996).
### TABLE 30-3

<table>
<thead>
<tr>
<th>FOOD PRODUCTS</th>
<th>FOODS ALLOWED</th>
<th>FOODS TO QUESTION</th>
<th>FOODS NOT ALLOWED</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Milk products</strong></td>
<td>Milk, cream, most ice cream, buttermilk, plain yogurt, cheese, cream cheese, processed cheese, processed cheese foods, cottage cheese</td>
<td>Milk drinks, flavored yogurt, frozen yogurt, sour cream, cheese sauces, cheese spreads</td>
<td>Malted milk, ice cream made with ingredients not allowed</td>
</tr>
<tr>
<td><strong>Grain products</strong></td>
<td>Breads: Bread and baked products containing amaranth, arrowroot, buckwheat, corn bran, corn flour, cornmeal, cornstarch, flax, legume flours (bean, garbanzo or chickpea, fava, lentil, pea), millet, potato flour, potato starch, quinoa, rice bran, rice flours (white, brown, sweet), sago, sorghum flour, soy flour, sweet potato flour, tapioca and teff</td>
<td>Buckwheat flour</td>
<td>Bread and baked products containing wheat, rye, triticale, barley, oats, wheat germ, wheat bran, graham flour, gluten flour, durum flour, wheat starch, oat bran, bulgur, farina, wheat-based semolina, spelt, kamut, einkorn, emmer, farro, imported foods labeled “gluten-free,” which may contain ingredients not allowed, e.g., wheat starch</td>
</tr>
<tr>
<td><strong>Cereals:</strong> Hot: Amaranth flakes, cornmeal, cream of buckwheat, cream of rice (brown, white), hominy grits, rice flaks, quinoa flakes, soy flakes and soy grits</td>
<td>Rice and corn cereals, rice and soy pabulum</td>
<td>Cereals made from wheat, rye, triticale, barley and oats; cereals with added malt extract or malt flavoring</td>
<td></td>
</tr>
<tr>
<td><strong>Pastas:</strong> Macaroni, spaghetti, and noodles from beans, corn, pea, potato, quinoa, rice, soy and wild rice flours</td>
<td>Buckwheat pasta</td>
<td>Pastas made from wheat, wheat starch and other ingredients not allowed</td>
<td></td>
</tr>
<tr>
<td><strong>Miscellaneous:</strong> Corn tacos, corn tortillas</td>
<td>Rice crackers, some rice cakes and popped corn cakes</td>
<td>Wheat flour tacos, wheat tortillas</td>
<td></td>
</tr>
<tr>
<td><strong>Meats and alternatives</strong></td>
<td>Meat, fish, poultry: Fresh</td>
<td>Deli or processed meats such as luncheon meat, ham, bacon, meat and sandwich spreads, meat loaf, frozen meat patties, sausages, pâté, vienner, bologna, salami, imitation meat or fish products, meat product extenders</td>
<td>Fish canned in vegetable broth containing HVP/HPP*</td>
</tr>
<tr>
<td><strong>Eggs:</strong> Eggs</td>
<td>Egg substitutes, dried eggs</td>
<td>Turkey basted or injected with HVP/HPP*</td>
<td></td>
</tr>
<tr>
<td><strong>Fruits and vegetables</strong></td>
<td>Fruits: Fresh, frozen, and canned fruits and juices</td>
<td>Scalloped potatoes (containing wheat flour)</td>
<td>Frozen chicken containing chicken broth (made with ingredients not allowed)</td>
</tr>
<tr>
<td><strong>Vegetables:</strong> Fresh, frozen, and dried and canned</td>
<td>French-fried potatoes (e.g., those in restaurants)</td>
<td>Battered dipped vegetables</td>
<td></td>
</tr>
<tr>
<td><strong>Soups</strong></td>
<td>Homemade broth, gluten-free bouillon cubes, cream soups and stocks made from allowed ingredients</td>
<td>Canned soups, dried soup mixes, soup bases and bouillon cubes</td>
<td>Soups made with ingredients not allowed, bouillon and bouillon cubes containing HVP or HPP* or wheat</td>
</tr>
<tr>
<td><strong>Fats</strong></td>
<td>Butter, margarine, lard, vegetable oil, cream, shortening, homemade salad dressing with allowed ingredients</td>
<td>Salad dressings, some mayonnaise</td>
<td>Packaged suet</td>
</tr>
<tr>
<td><strong>Desserts</strong></td>
<td>Ice cream, sherbet, whipped toppings, egg custards, gelatin desserts; cakes, cookies, pastries made with allowed ingredients; gluten-free ice cream cones, wafers, and waffles</td>
<td>Milk puddings, custard powder, pudding mixes</td>
<td>Ice cream made with ingredients not allowed; cakes, cookies, muffins, pies and pastries made with ingredients not allowed; ice cream cones, wafers and waffles made with ingredients not allowed</td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
<td>Beverages: Tea, instant or ground coffee (regular or decaffeinated), cocoa, soft drinks, cider; distilled alcoholic beverages such as rum, gin, whiskey, vodka, wines and pure liqueurs; some soy and rice beverages</td>
<td>Instant tea, flavored and herbal teas, flavored coffees, coffee substitutes, fruit-flavored drinks, chocolate drinks, chocolate mixes</td>
<td>Beer, ale and lager; cereal and malted beverages; soy or rice beverages made with barley or oats</td>
</tr>
</tbody>
</table>

*If the plant source in HVP/HPP (hydrolyzed vegetable protein/hydrolyzed plant protein) is not identified or if the source is from wheat protein, HVP/HPP must be avoided.


Continued
TABLE 30-3  Gluten-Free Diet by Food Groups—cont’d

<table>
<thead>
<tr>
<th>FOOD PRODUCTS</th>
<th>FOODS ALLOWED</th>
<th>FOODS TO QUESTION</th>
<th>FOODS NOT ALLOWED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miscellaneous</td>
<td>Sweets: Honey, jam, jelly, marmalade, corn syrup, maple syrup, molasses, sugar (brown and white), icing sugar (confectioner’s)</td>
<td>Spreads, candies, chocolate bars, chewing gum, and lemon curd</td>
<td>Licorice and other candies made with ingredients not allowed</td>
</tr>
<tr>
<td>Snack foods:</td>
<td>Plain popcorn, nuts, and soy nuts</td>
<td>Dry roasted nuts, flavored potato chips, tortilla or taco (corn) chips, and soy nuts</td>
<td>Pizza, unless made with ingredients allowed</td>
</tr>
<tr>
<td>Condiments:</td>
<td>Plain pickles, relish, olives, ketchup, mustard, tomato paste, pure herbs and spices, pure black pepper, vinegars (apple or cider, distilled white, grape or wine, spirit), gluten-free soy sauce</td>
<td>Seasoning mixes, Worcestershire sauce</td>
<td>Soy sauce (made from wheat), mustard pickles (made from wheat flour), malt vinegar</td>
</tr>
<tr>
<td>Other:</td>
<td>Sauces and gravies made with ingredients allowed, pure cocoa, pure baking chocolate, carob chips and powder, chocolate chips, monosodium glutamate (MSG), cream of tartar, baking soda, yeast, brewer’s yeast, aspartame, coconut, vanilla, gluten-free communion wafers</td>
<td>Baking powder</td>
<td>Sauces and gravies made from ingredients not allowed, hydrolyzed vegetable/plant protein (HVP/HPP)*, communion wafers</td>
</tr>
</tbody>
</table>


*If the plant source in HVP/HPP (hydrolyzed vegetable protein/hydrolyzed plant protein) is not identified or if the source is from wheat protein, HVP/HPP must be avoided.

TABLE 30-4  Substitutions

<table>
<thead>
<tr>
<th>Substitutions for 1 tbsp (15 ml) Wheat Flour</th>
<th>Substitutions for 1 c (250 ml) Wheat Flour*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/2 tbsp Cornstarch</td>
<td>7 ml</td>
</tr>
<tr>
<td>1/2 tbsp Potato starch</td>
<td>7 ml</td>
</tr>
<tr>
<td>1/2 tbsp White rice flour</td>
<td>7 ml</td>
</tr>
<tr>
<td>1/2 tbsp Arrowroot starch</td>
<td>7 ml</td>
</tr>
<tr>
<td>2 tsp Quick-cooking tapioca</td>
<td>10 ml</td>
</tr>
<tr>
<td>2 tsp Tapioca starch</td>
<td>10 ml</td>
</tr>
<tr>
<td>2 tbsp Uncooked rice</td>
<td>30 ml</td>
</tr>
<tr>
<td>Mix A: 2 c Brown rice flour</td>
<td>500 ml</td>
</tr>
<tr>
<td>2 c Sweet rice flour</td>
<td>500 ml</td>
</tr>
<tr>
<td>2 c Rice polish</td>
<td>500 ml</td>
</tr>
<tr>
<td>Mix B: 4 c White rice flour</td>
<td>1 L</td>
</tr>
<tr>
<td>1 c Potato starch</td>
<td>325 ml</td>
</tr>
<tr>
<td>1 c Tapioca starch</td>
<td>250 ml</td>
</tr>
<tr>
<td>Other Substitutions for 1 c (250 ml) Wheat Flour*</td>
<td></td>
</tr>
<tr>
<td>7/8 c Potato starch</td>
<td>150 ml</td>
</tr>
<tr>
<td>7/8 c White or brown rice flour</td>
<td>215 ml</td>
</tr>
<tr>
<td>1 c Cornmeal</td>
<td>250 ml</td>
</tr>
<tr>
<td>1 c Fine cornmeal</td>
<td>250 ml</td>
</tr>
<tr>
<td>5/8 c Coarse cornmeal</td>
<td>175 ml</td>
</tr>
<tr>
<td>7/8 c White or brown rice flour PLUS</td>
<td>150 ml PLUS</td>
</tr>
<tr>
<td>1/2 c Potato starch</td>
<td>75 ml</td>
</tr>
<tr>
<td>1 c Soy flour PLUS</td>
<td>250 ml PLUS</td>
</tr>
<tr>
<td>5/8 c Potato starch PLUS</td>
<td>50 ml PLUS</td>
</tr>
<tr>
<td>3/4 c Rice flour</td>
<td>175 ml</td>
</tr>
<tr>
<td>7/8 c Cornstarch</td>
<td>215 ml</td>
</tr>
<tr>
<td>7/8 c Whole bean flour</td>
<td>215 ml</td>
</tr>
</tbody>
</table>

*Store in an airtight container and use 7/8 c (215 ml) of mix A or 1 c (250 ml) of mix B for 1 c (250 ml) wheat flour. A combination of flours and starches gives a better gluten-free product. For specific flour mixes see recipes in gluten-free cookbooks using a variety of gluten-free flours.


Medical Nutrition Therapy

It is important to correct the related anemias (see Chapter 34). Nutritional deficiency may increase susceptibility to infectious agents, further aggravating the condition.

INTESTINAL BRUSH-BORDER ENZYME DEFICIENCIES

Intestinal enzyme deficiency states involve deficiencies of the brush-border disaccharidases that hydrolyze disaccharides at the mucosal cell membrane. Disaccharidase deficiencies may occur as (1) rare congenital defects, such as the sucrase, isomaltase, or lactase deficiencies seen in the newborn; (2) generalized forms secondary to diseases that damage the intestinal epithelium (e.g., Crohn’s disease or celiac disease); or, most commonly, (3) a genetically acquired form (e.g., lactase deficiency) that usually appears after childhood but can appear as early as 2 years of age. For purposes of this chapter, only lactose maldigestion is described in detail (see Chapter 45 for a discussion of metabolic disorders).

Lactose Maldigestion and Lactose Intolerance

Pathophysiology

Lactose intolerance is the most common carbohydrate intolerance, and it affects persons of all age groups. Lactose maldigestion and intolerance to lac-
Lactose is caused by a deficiency of lactase, the enzyme that digests the sugar in milk. Lactose that is not hydrolyzed into galactose and glucose in the upper small intestine passes into the colon, where bacteria ferment the lactose to SCFAs and gases, carbon dioxide, and hydrogen gas. Consumption of small amounts should be of little consequence because both the SCFAs are readily absorbed and the gases can be absorbed or passed. Larger amounts, usually greater than 12 g consumed in a single food (the amount typically found in 240 ml of milk), may result in more substrate entering the colon than can be disposed of by normal processes. As is the case with any malabsorbed sugar, the lactose may act osmotically and increase fecal water, and rapid fermentation by intestinal bacteria may result in bloating, flatulence, and cramps. In some cases loose stools or diarrhea may occur.

Seventy percent of the adult worldwide population, especially blacks, Asians, and South Americans, are lactase deficient, which implies that decline of the lactase enzyme after early childhood is the more normal state and lactase sufficiency is abnormal. Although it has been suggested that lactase persistence is induced by the continuation of milk in the diet after weaning, no evidence has been found to support this theory. It is more likely that the maintenance of lactase throughout adulthood reflects the continuation of an ancient genetic mutation (see Focus on: Lactose Tolerance—An Uncommon Anomaly?).

Typically, lactase activity declines exponentially at weaning to about 10% of the neonatal value. Even in adults who retain a high level of lactase levels (75% to 85% of white adults of Western European heritage), the quantity of lactase is about half that of other saccharides, such as sucrase, α-dextrinase, or glucoamylase.

---

**TABLE 30-5** Notes on “Foods to Question”

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>FOOD PRODUCTS</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk products</td>
<td>Milk drinks</td>
<td>Chocolate milk and other flavored drinks may contain wheat starch or barley malt.</td>
</tr>
<tr>
<td></td>
<td>Cheese spreads or sauces (e.g., nacho)</td>
<td>May be thickened or stabilized with wheat. Flavors and seasonings may contain wheat.</td>
</tr>
<tr>
<td></td>
<td>Flavored or frozen yogurt</td>
<td>May be thickened or stabilized with a gluten source. May contain granola or cookie crumbs.</td>
</tr>
<tr>
<td>Grains</td>
<td>Sour cream</td>
<td>Some low-fat or fat-free may contain modified food starch. Pure buckwheat flour is gluten-free. Sometimes buckwheat flour may be mixed with wheat flour.</td>
</tr>
<tr>
<td></td>
<td>Buckwheat flour</td>
<td>May contain barley malt extract.</td>
</tr>
<tr>
<td></td>
<td>Rice cereals</td>
<td>May contain oat syrup or barley malt extract.</td>
</tr>
<tr>
<td></td>
<td>Corn cereals</td>
<td>May contain oat syrup or barley malt extract.</td>
</tr>
<tr>
<td></td>
<td>Buckwheat pasta</td>
<td>Some “soba” pastas contain pure buckwheat flour, which is gluten-free, but others may also contain wheat flour.</td>
</tr>
<tr>
<td></td>
<td>Rice cakes, corn cakes, rice crackers</td>
<td>Multigrain often contains barley and/or oats.</td>
</tr>
<tr>
<td>Meats/alternatives</td>
<td>Baked beans</td>
<td>Some are thickened with wheat flour.</td>
</tr>
<tr>
<td></td>
<td>Imitation crab</td>
<td>May contain fillers made from wheat starch.</td>
</tr>
<tr>
<td></td>
<td>Dry-roasted nuts</td>
<td>May contain wheat.</td>
</tr>
<tr>
<td></td>
<td>Processed meat products</td>
<td>May contain fillers made from wheat. May contain HPP or HVP made from wheat.</td>
</tr>
<tr>
<td>Fruits and vegetables</td>
<td>Imitation meats</td>
<td>Dates and other dried fruits may be dusted with wheat flour to prevent sticking. Some may be thickened with flour.</td>
</tr>
<tr>
<td></td>
<td>Dried fruits</td>
<td>May contain wheat as an ingredient.</td>
</tr>
<tr>
<td></td>
<td>Fruits/vegetables with sauces</td>
<td>May contain noodles or barley. Cream soups are often thickened with flour. May contain HPP or HVP (from wheat).</td>
</tr>
<tr>
<td></td>
<td>Fruit pie fillings</td>
<td>Seasonings may contain wheat flour, wheat starch or hydrolyzed wheat protein.</td>
</tr>
<tr>
<td></td>
<td>French fries</td>
<td>May contain wheat starch or flour.</td>
</tr>
<tr>
<td>Soups</td>
<td>Canned soups, dried soup mixes, soup bases and bouillon cubes</td>
<td>Seasonings may contain wheat flour, wheat starch or hydrolyzed wheat protein. Some instant teas, herbal teas, coffee substitutes and other drinks may have grain additives. Non-dairy substitutes (e.g., rice drinks and soy drinks) may contain barley, barley malt extract or oats.</td>
</tr>
<tr>
<td>Fats/Desserts</td>
<td>Salad dressings</td>
<td>May contain wheat starch or flour.</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Milk puddings/mixes</td>
<td>Seasonings may contain wheat flour or wheat starch.</td>
</tr>
<tr>
<td></td>
<td>Beverages</td>
<td>Starch source may be from wheat.</td>
</tr>
<tr>
<td></td>
<td>Lemon curd</td>
<td>Usually thickened with flour.</td>
</tr>
<tr>
<td></td>
<td>Potato, tortilla chips, and soy nuts</td>
<td>Some potato chips contain wheat. Seasoning mixes may contain wheat flour, wheat starch or hydrolyzed wheat protein.</td>
</tr>
<tr>
<td></td>
<td>Baking powder</td>
<td>Contains starch, which may be from wheat.</td>
</tr>
<tr>
<td></td>
<td>Seasoning mixes</td>
<td>May contain wheat flour, wheat starch or hydrolyzed wheat protein.</td>
</tr>
<tr>
<td></td>
<td>Worcestershire sauce</td>
<td>May contain malt vinegar which is not gluten-free.</td>
</tr>
</tbody>
</table>

Lactase deficiency is typically diagnosed on the basis of (1) a history of GI symptoms occurring after milk ingestion, (2) a test for abnormal hydrogen levels in the breath, or (3) an abnormal lactose tolerance test. The lactose tolerance test was originally based on an oral dose of lactose equivalent to the amount in 1 quart of milk (50 g). If the patient has insufficient lactase enzyme, blood glucose produced from the lactose increases less than 25 mg/100 ml of serum above the fasting level, and GI symptoms may appear. Because hydrogen production in the colon increases significantly if lactose is not digested in the small intestine, hydrogen absorbed into the bloodstream and exhaled through the lungs can be used as another test of malabsorption. The breath hydrogen test shows increased levels 60 to 90 minutes after ingestion. Recently, doses lower than 50 g of lactose have been used to approximate more closely the usual consumption of lactose from milk products.

Medical Nutrition Therapy
Management of lactase insufficiency requires dietary changes. The symptoms of lactase intolerance are alleviated by reduced consumption of lactose-containing foods. Persons who avoid dairy products should take calcium supplements and should read ingredient labels carefully (Srinivasan and Minocha, 1998). A completely lactose-free diet is not necessary in lactase-deficient persons. Most lactose maldigesters can consume some lactose (6 to 12 g) without major symptoms, especially when taken with meals or in the form of cheeses or cultured dairy products. Many adults with intolerance to moderate amounts of milk can ultimately adapt to and tolerate 12 g or more of lactose in milk (equivalent to 240 ml of full-lactose milk) when introduced gradually, in increments, over several weeks (Srinivasan and Minocha, 1998).

Apparently, incremental or continuous exposure to increasing quantities of fermentable sugar can lead to improved tolerance, not as a consequence of increased lactase enzyme production but perhaps by altered colonic flora. This has been shown with lactulose, a nonabsorbed carbohydrate that is biochemically similar to lactose (Bezkorovainy, 2001). Individual differences in tolerance may relate to the state of saccharides.) The mutation would have selectively endured, because it would promote greater health, survival, and reproduction of those who carried the gene.

It is proposed that the mutation occurred in more than one location and then accompanied migrations of populations throughout the world. It continues primarily among whites from northern Europe and in ethnic groups in India, Africa, and Mongolia. The highest frequency (97%) of lactose tolerance occurs in Sweden and Denmark, suggesting an increased selective advantage in those able to tolerate lactose related to the limited exposure to ultraviolet light typical of northern latitudes. (Lactose favors calcium absorption, which is limited in the absence of vitamin D produced by skin exposure to sunlight.) (See Chapter 4.)

Dairying was unknown in North America until the arrival of Europeans. Thus, Native Americans and all of the non-European immigrants are among the 90% of the world’s population who tolerate milk poorly, if at all. This has practical implications with respect to group feeding programs, such as school breakfasts and lunches. Fortunately, most lactose-intolerant people are able to digest milk in small to moderate amounts.
In one objective evaluation of 375 patients with GI disease, adverse reactions to foods occurred in 32% of patients, allergies were suspected in 14.4%, and allergies were confirmed in 3.2% of patients (Bischoff et al, 1996). The permeability of the intestinal wall to molecules of food and cell fragments is likely increased in inflammatory states, allowing heightened interaction of antigens with host immune systems.

Food intolerances of various types occur more than twice as often in persons with IBD than in the population at large (Ballegaard et al, 1997). Except for a small increase in the occurrence of lactose intolerance, however, the patterns are not consistent among individuals, or even from one time to the next. Reasons for specific and nonspecific food intolerances are abundant and are likely related to the stage, location, and manifestations of the disease process. Partial GI obstructions, malabsorption, diarrhea, altered GI transit, increased secretions, food aversions, and associations are but a few of the problems experienced by persons with IBD (Reif et al, 1997). Neither food allergies nor intolerances, however, fully explain the onset or overall pathologic or clinical manifestations in all patients (see Chapter 32).

Normally, when an antigenic challenge or trauma occurs, the immune response rises to the occasion; it is then turned off (and continues to be held in check) after the challenge resolves. In IBD, either the regulatory mechanisms are defective or the factors stimulating the immune and acute-phase response are enhanced, leading to tissue fibrosis and destruction. The clinical course of the disease may be mild and episodic, or severe and unremitting.

Both Crohn’s disease and ulcerative colitis share some clinical features. For example, food intolerances, diarrhea, fever, weight loss, anemias, malnutrition, growth failure, and extraintestinal manifestations (arthritic, dermatologic, and hepatic) occur in both diseases. The diseases, however, also have distinctive features in terms of their genetic characteristics, clinical presentation, and treatment (Table 30-6).

Persons with IBD are at risk for several forms of malnutrition, and nutrition is a major consideration in each stage of the disease. Although malnutrition can occur in both forms of IBD, it is more likely to be a major and lifelong concern in patients with Crohn’s disease. In both forms of IBD, the risk of malignant disease is increased with long-standing disease. The reasons for the increased risk are not firmly established but may be associated with the increased proliferative state and nutritional factors.

**Crohn’s Disease**

**Pathophysiology**

Crohn’s disease may involve any part of the GI tract, from mouth to anus. The most common pattern of involvement (about 50% to 60% of cases) is the combination of both the distal ileum and the colon; 15% to 25% of cases involve only the small intestine or only...
INFLAMMATORY BOWEL DISEASE

CAUSE
- Genetic predisposition
- Unknown "irritant"
  - Viral?
  - Bacterial?
  - Autoimmune?
- Abnormal activation of the mucosal immune response. Secondary systemic response

PATHOPHYSIOLOGY
- Damage to the cells of the small and/or large intestine with malabsorption, ulceration, or stricture
- Diarrhea
- Weight loss
- Poor growth

MEDICAL MANAGEMENT
- Corticosteroids
- Antiinflammatory agents
- Immunosuppressants
- Antibiotics
- Anticytokine medications

NUTRITIONAL MANAGEMENT
- Oral enteral formula (tube-feed if necessary)
- Use of foods that are well tolerated
- Parenteral nutrition in patients with severe disease or obstruction

FIGURE 30-3 • Pathophysiology algorithm: inflammatory bowel disease. (Algorithm content developed by John Anderson, PhD, and Sanford C. Garner, PhD, 2000. Updated by Peter L. Beyer, MS, RD, LD, 2002.)
the colon (Kornbluth et al, 1998). In the portions of intestine involved, the inflammation may skip areas, and so these healthy segments of bowel separate inflamed portions. Mucosal involvement in Crohn’s disease is transmural in that it affects all layers of the mucosa. As inflammation, ulceration, abscesses, and fistulas resolve, fibrosis, submucosal thickening, and scarring may result, leading to narrowed segments of bowel, localized strictures, and partial or complete obstruction of the intestinal lumen.

Medical Treatment

Surgery may be necessary to repair strictures or remove portions of the bowel when medical management fails. About 50% to 70% of persons with Crohn’s disease will undergo surgery related to the disease (Hyams, 1996; Patel et al, 1997). Surgery does not cure the disease; recurrence often occurs within 1 to 3 years of surgery, and the chance of reoperation sometime in the patient’s life is about 30% to 70%, depending on the type of surgery and the age at first operation. Major resections of the intestine may result in varying degrees of malabsorption of fluid and nutrients. In extreme cases, patients may have extensive or multiple resections, resulting short-bowel syndrome, and dependence on parenteral nutrition to maintain adequate nutrient intake and hydration.

Ulcerative Colitis

Pathophysiology

Ulcerative colitis involves only the colon, and the disease always extends from the rectum. Microscopic examination shows diffusely inflamed mucosa, usually with small ulcers. Continuous disease (rather than skipped areas) is characteristic. Serosal involvement, strictures, and significant narrowing are uncommon, but rectal bleeding or bloody diarrhea is relatively common (Jenkins, 2001; Jewell, 1998) (see Table 30-6 and Figure 30-4).

Ulcerative colitis occurs most commonly in young people 15 to 30 years of age, with a secondary peak at 50 to 60 years of age, although no age group is ex-
large intestine in a segmental manner, with intervening eas, ulcerative colitis is generally a contiguous disease process. Whereas Crohn’s disease typically involves the small and large intestine in a segmental manner, with intervening “skip” areas, ulcerative colitis is generally a contiguous disease process that starts in the rectum and progresses in a retrograde fashion to involve varying lengths of the colon. (Modified from Cotran KS, Kumar V, Robbins SL: Robbins’ pathologic basis of disease, ed 4, Philadelphia, 1989, WB Saunders.)

empt. Persons with long-standing disease are at increased risk for cancer. In severe disease and with increased risk of cancer, complete removal of the colon is recommended, with creation of an ileostomy, ileal pouch, or ileoanal anastomosis.

Medical Management

The goals of treatment in IBD are to induce and maintain remission and to maintain nutritional status. Treatment of the primary GI manifestations appears to correct most of the extraintestinal features of the disease as well. The most effective medical agents during the acute stages of the disease are corticosteroids, although antiinflammatory agents (aminosalicylates), immunosuppressive agents (cyclosporine, azathioprine, mercaptopurine), and antibiotics (metronidazole) may be used for maintaining remission. Each carries the potential for medical and nutritional consequences (Regueiro, 2000). One of the newest therapeutic agents is monoclonal anti–tumor necrosis factor (anti–TNF) (infliximab), an agent that inactivates one of the primary inflammatory cytokines. It is normally used in more severe cases of Crohn’s disease and in the management of fistulas, but it has not been shown to be effective in ulcerative colitis. Figure 30-5 shows an algorithm for reversing pediatric growth failure.

Investigations of various treatment modalities for the acute and chronic stages of IBD are ongoing and include new forms of existing drugs as well as new agents targeted to regulate cytokines, eicosanoids, or other mediators of the inflammatory/acute-phase cascade response (Holtmann et al, 2001; Panes, 2001; Regueiro, 2000). Use of prebiotics and probiotic cultures have been considered plausible because each has the potential to alter the GI microflora as well as the immunologic response at the gut level (Madsen, 2001).

Medical Nutrition Therapy

Persons with IBD are at increased risk of nutrition problems for a host of reasons related to the disease and its treatment, so the primary goal is to restore and maintain the nutritional status of the individual patient (Box 30-5). Foods, dietary and micronutrient supplements, enteral and parenteral nutrition may all be used to accomplish that mission. Diet and the other means of support may change during remissions and exacerbations of the disease. Persons with IBD often have fears and misconceptions regarding the significance of minor or major GI symptoms and the role of foods and nutrition. Patients are also often confused by advice from associates, various media, and health care providers. Education is a key form of therapy.

Diet and specific nutrients may play a role in maintaining or bringing on the remission of IBD. The ability of parenteral or enteral nutrition to induce remission of IBD has been debated for several years, and the issue is still not entirely resolved. At least in theory, the use of low-residue, low-fiber liquid diets can decrease the antigenic load or reduce microbial populations in the colon. Clinical trials with parenteral and enteral nutrition and other supplements have been confounded by small numbers of patients, differences in study design, severity and location of the disease, differences in the nutritional formulas, and whether an oral diet was continued. Evaluation is further confounded by the fact that the natural course of IBD is one of exacerbations and remissions.

Results of reviews and metaanalyses of several studies have generally concluded that (1) “bowel rest” with parenteral nutrition is not a major requirement for achieving remission, (2) enteral nutrition may be the preferred means of nutritional support, and (3) at least in some cases, enteral nutrition is more successful at inducing remission than parenteral nutrition (Han et al, 1999; Messori et al, 1996). Commitment from the patient and caretaker to the sole use of oral enteral formulas must be high, or the formula must be fed by tube; however, current enteral or parenteral nutrition formulas do not appear to be as consistently effective as corticosteroids or combination medical therapy in inducing remission.

Regardless of whether or not current forms of parenteral or enteral nutrition induce remission, timely nutritional support is a vital component of therapy to restore and maintain nutritional health (Beyer, 2001). Currently available parenteral solutions are not as complete or well suited as enteral nutrition, but parenteral nutrition may be required to restore nutrition in patients with obstructions, fistulas, severe disease, and major GI resections.

Malnutrition itself compromises digestive and absorptive function and may increase the permeability of the GI tract to potential inflammatory agents (Han et al, 1999). Energy needs of patients with IBD are not greatly increased (unless weight gain is desired), but
Pediatric patient has limited nutrient intake and absorption due to Crohn's disease.

Plot the patient’s growth curves (height for age, height for weight). Perform anthropometric measurements.

Has the patient reached maturity by bone age (x-ray)?

Potential for catch-up growth is dependent on bone age. Nutritional intervention is imperative before bones mature and epiphyses fuse.

To achieve catch-up growth, calculate intake of an enteral elemental diet based on ideal weight for height.

Is growth failure severe or bone age advanced?

Is Crohn’s disease acute?

Steroids can be used to control symptoms, but dosage should be tapered under strict monitoring with initiation of elemental diet therapy.

Is the child younger than age 2?

Administer enteral elemental diet nocturnally via continuous nasogastric infusion, 60–80 kcal/kg body weight, until patient reaches maturity or normal growth levels. The patient should not be taking steroids.

Administer enteral elemental diet nocturnally via continuous nasogastric infusion, 60–80 kcal/kg body weight, for 1 month out of 4 for at least 1 year.

Does examination of growth parameters reveal growth failure?

Apply supplemental nutrition intravenously.

After 3 months, reassess the patient’s nutritional status and growth parameters.

Advise oral supplementation with a calorically dense polymeric formula.

Has the patient had acute weight loss while maintaining growth?

Yes

Yes

Yes

No

No

No

Yes

protein requirements may be increased by 50%, especially during active stages of the disease. Supplemental vitamins and minerals and trace elements may be needed to replace stores or for maintenance because of maldigestion, malabsorption, drug-nutrient interactions, or because the patient cannot eat a complete diet. Diarrhea can aggravate losses of zinc, potassium, and selenium stores in particular.

During acute and severe exacerbations of the disease, the diet is tailored to the individual patient. A minimal-residue diet that limits poorly absorbed or hyperosmolar sugars, caffeine, and excess fiber might be used initially to reduce diarrhea. During either acute or chronic stages, inflammation or scarring may result in a partially obstructed bowel; and, in that case, fiber may have to be restricted or limited to minute particles to pass through the narrowed lumen. Small, frequent feedings may be tolerated better than large meals, and small amounts of isotic, liquid, oral supplements may be valuable in restoring intake without provoking symptoms. In cases in which fat malabsorption is likely, supplementation with foods made with MCT may be valuable in adding calories and serving as a vehicle for fat-soluble nutrients.

Whether dietary factors cause exacerbations is not clear, but they certainly can aggravate symptoms. Although study results are inconsistent, the dietary factors commonly noted before exacerbations include increased sucrose intake, lack of fruits and vegetables, a low intake of dietary fiber, and altered omega-6/omega-3 fatty acid ratios (Reif et al., 1997; Shoda et al., 1996). In a small number of patients, specific food allergies may be identified. The significance of these factors is not clear, but they may simply reflect an overall poor-quality diet that may have increased the overall susceptibility of the GI tract to the disease process. Modification of oral diets and nutritional formulas with omega-3 fatty acids, specific amino acids (e.g., glutamine), and antioxidants and the use of fermentable fibers (prebiotics) or probiotics are therapeutic strategies being evaluated for management of IBD (Han et al., 1999; Jacobasch et al., 1999; Madsen, 2001; Panes, 2001).

In everyday life, patients may have intermittent “flares” of the disease characterized by partial obstructions, nausea, abdominal pain, bloating, or diarrhea. Patients can be taught to manage their disease by selecting appropriate foods and beverages. For example, patients might be taught to restrict foods during bouts of diarrhea (see Table 30-1) or to limit fiber (especially large particles) if partial obstruction is suspected. They can also be shown how to increase omega-3 fats with food choices and supplements so as to benefit from their antiinflammatory effect.

Probiotic foods or supplements may hold promise by modifying the microbial flora. In animal models of inflammatory GI disease and in preliminary studies of humans with IBD, ingestion of specific strains or mixtures of probiotic organisms has been shown to alter the GI flora and to prolong periods of remission (Linskens et al., 2001; Madsen, 2001). Prebiotic foods (e.g., oligosaccharides, fermentable fibers, and resistant starches) can serve as fuels for colonic flora and result in altered microflora and increased production of SCFAs. The altered flora and SCFAs produced may also serve to attenuate the inflammatory process, especially in ulcerative colitis. Additional study continues to identify the dose and most effective prebiotic and probiotic foods, the form in which they can be used for therapeutic and maintenance purposes, and their relative value compared with other forms of therapy (Fukuda et al., 2002; Jacobasch et al., 1999).

Food intolerances are common in patients with IBD, but the foods are variable among patients and may not be incriminated consistently from one time to the next. Patients are sometimes advised simply to eliminate the foods that they suspect are responsible for the intolerance. Often, however, the patient becomes increasingly frustrated as the diet becomes more and more limited, and symptoms may not resolve. One study (Pearson et al., 1993) revealed no significant differences in the duration of remission between patients who did or did not identify food sensitivities.
Confirming true allergic GI reactions to foods is a difficult and painstaking process (see Chapter 32). The patient must be willing to consume either an amino acid diet or a very restricted diet composed of only three or four foods, with the addition of each of the suspected foods one at a time. The allergen is identified on the basis of subjective and objective symptoms related to the repeated addition and elimination of the food. Circulating antibodies to food proteins have been considered a sign of allergy but may in fact be a sign of increased permeability rather than local GI allergy.

The same foods that are most consistently responsible for GI symptoms (gas, bloating, and diarrhea) in a normal healthy population are likely to be the triggers for at least the same symptoms in patients with mild stages of IBD or those in remission. Patients receive nutritional information from a variety of sources, including support groups, Internet news groups, the audio and printed media, well-meaning friends, and food supplement salespersons. The information is sometimes inaccurate or exaggerated, or it may pertain only to one individual’s situation. The health care provider can help patients sort out the role of foods in normal everyday GI disturbances and the role of foods in IBD and teach them how to evaluate valid nutrition information from unproved or exaggerated claims. Patients’ participation in the management of their disease may help to reduce not only the symptoms of the disease but the associated anxiety level as well.

Irritable Bowel Syndrome

Pathophysiology
Irritable bowel syndrome (IBS) is not a disease but a common syndrome involving altered intestinal motility, increased sensitivity of the GI tract, and increased awareness and responsiveness of the viscerae to enteral and external stimuli. The disorder likely involves more than the large intestine, but most of the early speculation regarding the pathology of IBS focused on the colon. In IBS no obvious tissue damage, no inflammation, and no immunologic involvement are present. In all persons, the enteric nervous system is sensitive to the presence, chemical composition, and volume of foods, and the GI tract receives various types of input from the brain and the autonomic nervous system (see Chapter 1).

Persons with IBS tend to overrespond to many of these stimuli (Mayer, 2001; Simren et al, 2001). The mediators may be abnormal secretion of peptide hormones or signaling agents (e.g., neurotransmitters secreted in response to the hormones). The syndrome typically involves one of three predominant symptom patterns (diarrhea, constipation, or abdominal pain), but patients may describe combinations of GI complaints. GI discomfort after meals or with psychosocial distress, bloating, gas, heightened gastrocolic response, lowered threshold for normal GI discomfort, and abnormal bowel movements are all common with IBS.

The diagnosis is based on international consensus criteria (ROME I or II criteria) and diagnostic algorithms that help to rule out other GI or surgical disorders that may manifest with similar symptoms (Drossman, 1997; Thompson et al, 1999). According to the criteria, symptoms of abdominal discomfort must be present for at least 12 weeks of the past year and include at least two of three features: discomfort relieved by defecation, onset associated with a change in frequency of stool, and onset associated with a change in form of the stool.

The diagnosis is usually further refined to categorize the syndrome into subtypes, such as predominant patterns of alternating constipation and diarrhea, painless diarrhea, or constipation. The most common symptoms are alternating diarrhea and constipation, abdominal pain (typically relieved by defecation), and bloating; but perception of excessive flatulence, sensation of incomplete evacuation, rectal pain, and mucus in the stool may also occur (Figure 30-6).
Symptoms typically first occur between adolescence and the fourth decade of life, but many persons do not bring the problem to the attention of a physician. In the United States, IBS occurs in about 20% of women and 10% to 15% of men. It represents about 12% of visits to family medicine clinics and 20% to 40% of visits to gastroenterologists, and it is one of the most common reasons for which patients first seek medical care (Drossman et al, 1997; Olden and Schuster, 1998). Persons with IBD often have increased absenteeism from school and work, decreased productivity, increased health care costs, and decreased quality of life as a result of their symptoms.

Persons with IBS appear to have altered enteric sensitivity and motility in response to usual GI and environmental stimuli. They react more significantly than normal persons to intestinal distention, dietary indiscretions, and psychosocial factors (Drossman et al, 1997). Normal persons may experience mild GI disturbances in response to all the situations mentioned, but they appear to have a milder response. Life stressors, such as employment changes, travel, relocation, or uncomfortable social situations, may trigger the onset or worsen symptoms and may reduce many therapeutic efforts. A history of psychosocial trauma, such as physical or sexual abuse, has been reported in 32% to 44% of cases, but the higher estimate may be somewhat exaggerated as a result of sampling.

In addition to stress and dietary patterns, factors that may worsen symptoms include (1) excessive use of laxatives and other over-the-counter medications, (2) antibiotics, (3) caffeine, (4) previous GI illness, and (5) lack of regularity in sleep, rest, and fluid intake. In patients with a strong family history of allergy, hypersensitivity to certain foods may exacerbate symptoms in some persons with IBS (Bischoff et al, 1996). A trial of food elimination and challenge may be justified under these circumstances (see Chapter 32).

Medical Management

Management includes a combination of approaches to deal with the symptoms and the factors that may trigger them. Education, medications, counseling, and diet all play a role in the care. Depending on the predominant pattern and severity of the symptoms, medications may include antispasmodic, anticholinergic, antidiarrheal, prokinetic, or antidepressive agents. Newer agents are being evaluated to target specific neurotransmitters, peptides, or other mechanisms involved in the GI motility and enteric nervous system. Biofeedback, relaxation, and stress reduction techniques may also be useful (Drossman, 1997; Villanueva et al, 2001).

Medical Nutrition Therapy

Unlike IBD, IBS is not life threatening and does not result in maldigestion or malabsorption of nutrients. However, dietary practices of persons with IBS may result in a less than complete diet, insufficient nutrient intake, and less enjoyment of foods. Dietary selections and patterns are also important in controlling symptoms.

The aim of nutritional care is to ensure adequate nutrient intake, to guide the patient toward a diet that is not likely to contribute to symptoms, and to explain the role of ordinary dietary practices in producing or avoiding GI symptoms. The recommendations made for all persons for a good-quality diet are probably even more important in IBS. Excesses of dietary fat; caffeine; sugars such as lactose, fructose, and sorbitol (Rumessen and Gudmand-Hoyer, 1998); large meals; and alcohol are less well tolerated than in normal persons. Dietary fiber intake in adolescents and adults is typically about half that recommended. Excessive use of bran or coarse fibers is not needed or recommended, but increasing dietary fiber to the recommended 25 g or so per day may help to normalize bowel habit in all types of IBS patterns. If the patient is not able to consume fiber from food sources or does not respond adequately, fiber in the form of bulk laxatives (e.g., psyllium) may be helpful. Consumption of adequate fluid is recommended, especially when powdered fiber supplements are used. Large doses of wheat bran are no longer recommended and may exacerbate symptoms in some persons with IBS.

Foods with fiber, resistant starches, and oligosaccharides may also serve as prebiotic foods, which favor the maintenance of “healthy” microflora and resistance to pathogenic infections (Madsen, 2001; Nobaek et al, 2000). Results of initial studies on the use of prebiotic and probiotic supplements have been mixed, but additional studies with different products, doses, and subtypes of IBS are needed. The nutrition practitioner can work with the person with IBS to identify his or her concerns and perceptions, review the characteristics of the disease and the potential role of foods, and teach the client how to reduce the food-related symptoms associated with the syndrome. Sometimes clients become trapped in a vicious cycle in which anxiety about food, GI distress, and social embarrassment leave them with an unnecessarily restrictive diet, worsening nutritional status, increasing anxiety, and worsening symptoms. Calming reassurance and gradual return to a good diet with limitations of only items that likely will exacerbate symptoms will often greatly improve the quality of life.

Diverticular Disease

Pathophysiology

Diverticulosis is a situation of saclike herniations (diverticula) of the colonic wall, thought to result from long-term constipation and increased colonic pressures (Figure 30-7). The incidence of diverticulosis increases with age. Thirty percent of persons older than 50 years of age, 50% of those older than 70 years, and 66% of those 85 years of age and older develop diver-
ticulosis (Simmang and Shires, 1998). Sigmoid involvement occurs in almost all cases; right-sided colonic involvement occurs in Asians, but it is rare in whites).

The cause is not known for certain, but studies in animals and humans relate the disorder to constipation and lifelong increased intracolonic pressures. The pressures result from attempts to propel small, dry, hard fecal material through the lumen of the bowel. Theoretically, circular muscles completely close around the fecal material when the stools are small and longitudinal muscles contract, attempting to push the contents distally. Increased pressures result in the opportunity for herniations of the mucosal wall to develop through weaker segments of the colon (Simmang and Shires, 1998) (see Figures 30-7 and 30-8). This theory is supported by epidemiologic studies of populations consuming high- and low-fiber diets, prospective cohort studies in men, and experimental studies in animals fed low-fiber diets throughout their lifetimes (Scheppach et al, 2001). An abnormal pattern of excitatory innervation of the colon has been associated with intraluminal pressures and the presence of diverticulosis, but it is not known whether the pattern is a consequence of the disorder or related to the cause (Tomita et al, 1999).

In general, diverticular disease is (1) relatively rare in countries where a high-fiber diet is part of the lifelong pattern, and (2) increasing where "westernization" of the diet and increased intake of refined foods of the diet have begun (Camilleri et al, 2000; Scheppach et al, 2001). Lack of exercise may also contribute to the development of diverticular disease, presumably because of the more sluggish movement of GI contents (Peters et al, 2001).

**Medical and Surgical Treatment**

Complications of diverticular disease range from painless, mild bleeding and altered bowel habits to diverticulitis, which may include its own clinical spectrum of inflammation, abscess formation, acute perforation, acute bleeding, obstruction, and sepsis. About 10% to 25% of patients with diverticulosis develop diverticulitis, and about one third of those admitted to hospitals for diverticular disease require surgery. Death rates in patients requiring surgical intervention may be as high as 10% (Deckman and Cheskin, 1993).

**Medical Nutrition Therapy**

At one time it was thought that “roughage” (dietary fiber) aggravated diverticular disease, so the classic diet therapy was one that was low in fiber. It is now recognized that a high-fiber diet promotes soft, bulky stools that pass more swiftly, require less straining with defecation, and result in lower intracolonic pressures (Scheppach et al, 2001). High-fiber intakes have been found to relieve symptoms for most patients, and exercise appears to aid in both constipation and diverticular disease (Cheek and Radley, 1999; Peters, 2001; Scheppach et al, 2001).

Patients who have followed a low-fiber diet for years may require extensive encouragement to adopt the high-fiber approach. Fiber intake should be increased gradually because it may cause bloating or gas; however, these side effects usually disappear within 2 to 3 weeks. In cases in which the patient cannot consume the necessary amount of fiber, methylcellulose and psyllium fiber supplements have been used with good results. Adequate fluid intake (e.g., 2 to 3 L daily) should accompany the high-fiber intake.

For patients with an acute flareup of diverticulitis, a low-residue diet, elemental diet, or, in complicated cases, total parenteral nutrition may be required initially, followed by a gradual return to a high-fiber diet. Colonic smooth-muscle contractions, which in-
tensify after a high-fat meal, may contribute to the discomfort felt by persons with diverticular disease (Snape, 1994). Therefore, a low-fat diet may be reasonable to suggest for these patients, at least initially.

The question of whether the consumption of seeds, nuts, or skins of plant matter should be avoided to prevent complications of diverticular disease or after bouts of diverticulitis to aid in healing remains unresolved. Common sense tends to favor avoiding consumption of very coarse materials such as husks (not necessarily seeds) like those surrounding sunflower seeds and peanuts. Whether seeds or normal fibrous materials play any role in the onset of symptoms or actually harm the diverticula has not been determined. In patients with clear cases of perforation or obstruction, large pieces of coarse plant matter might be restricted and patients should be encouraged to chew fibrous foods thoroughly.

Colon Cancer and Polyps

Pathophysiology

In the United States, colon cancer is the second most common cancer in adults (after lung cancer) and is also the second most common cause of death. The number of new cases of colorectal cancer in 2002 was estimated to be about 148,000 cases (American Cancer Society, 2002). Worldwide, it is the third most common malignant neoplasm and the second leading cause of cancer deaths. Colon cancer occurs more commonly in men than in women (52 versus 38 cases per 100,000 population, respectively). The highest rates are seen in whites of northern European origin. Rates in Africa and Asia are lower, but they tend to rise with migration and westernization.

Factors that increase the risk of colorectal cancer include family history, occurrence of IBD (both Crohn’s disease and ulcerative colitis), familial polyposis, adenomatous polyps, and several dietary components. Polyps are considered precursors of colon cancers (see Chapter 40).

Use of aspirin and nonsteroidal anti-inflammatory agents and exercise appear to be protective against colon cancer (Peters et al, 2001; Reddy, 2000; Slattery, 2000). Dietary risk factors may include increased meat or fat intake and inadequate intake of several micronutrients. Protective factors include intake of fruits, vegetables, several phytochemicals, high-fiber grains, omega-3 fatty acids, and carotenoids; maintenance of acceptable weight; the vitamins D, E, and folate; and the minerals calcium, zinc, and selenium (Garland et al, 1999; Reddy, 2000; Wollowske et al, 2001).

The role of dietary fat in colon carcinogenesis is not entirely clear because different dietary lipids may have different effects. Red meats, such as beef, pork, and lamb, along with their fats, may be incriminated more than other types of meats; poultry, fish, and dairy fats appear to have less of a role in carcinogenesis. Food preparation methods may also influence the carcinogenic potential of meats and fatty foods (Giovannucci and Goldin, 1997; Parodi, 1997) (see Chapter 40).

The use of prebiotics and probiotics alters colonic microflora, induces glutathione transferase, increases butyrate content of the stool, reduces toxic and genotoxic compounds, and in animal models reduces the development of some precancerous lesions (Brady et al, 2000; Wollowske et al, 2001).

Medical Management

Patients diagnosed with colorectal polyps or cancer may require moderate to significant interventions, including medications, radiation therapy, chemotherapy, colon surgery, and parenteral nutritional support.

Medical Nutrition Therapy

Most Americans consume far less than the recommended amounts of fruits, vegetables, legumes, whole grains, and dairy products. Therefore the best advice for many patients might be to improve their diet based on recommendations from the American Cancer Society or the National Research Council or the Dietary Guidelines for Americans. These recommendations promote the consumption of adequate amounts of whole-grain breads and cereals, calcium-containing foods, fruits and vegetables, adequate micronutrient intake, and reasonable amounts of omega-3 fatty acids (from marine sources and other sources, such as flaxseed oil) along with adequate exercise. Cancer survivors should also be encouraged to follow these same nutrition and exercise guidelines.
ulation, most cases of SBS result from congenital anomalies of the GI tract, atresia, volvulus, or necrotizing enterocolitis (Sigalet, 2001).

Obvious complications of short-bowel syndrome include malabsorption of micronutrients and macronutrients, fluid and electrolyte imbalances, weight loss, and growth failure (in children). Gastric hypersecretion, oxalate renal stones, cholesterol gallstones, and rarely d-lactic acidosis may also occur (Sundarum et al, 2002). The severity of malabsorption, the extent of complications, and the degree of dependence on parenteral nutritional support reflect the length and location of the resection, the age of the patient at the time of the operation, and the health of the remaining GI tract (Beyer, 2001; Wilmore et al, 1997) (Box 30-6).

**Jejunal Resections**

Normally most digestion and absorption of food and nutrients occurs in the first 100 cm of small intestine. What remains to be digested or fermented and absorbed are small amounts of sugars, resistant starch, fiber, lipids, dietary fiber, and fluids. After jejunal resections, the ileum is able to perform the functions of the jejunum, especially after a period of adaptation. The motility of the ileum is comparatively slow, and hormones secreted in the ileum and colon help to slow gastric emptying and secretions. Because jejunal resections result in reduced surface area and shorter intestinal transit than normal, the functional reserve for absorption of micronutrients, excess amounts of sugars (especially lactose), and lipids is reduced.

**Ileal Resections**

Significant resections of the ileum, especially the distal ileum, generally produce major nutritional and medical complications. The distal ileum is the only site for absorption of the vitamin B₁₂/intrinsic factor complex and bile salts, and the ileum normally absorbs a major portion of the several liters of fluid ingested and secreted into the GI tract (see Chapter 1). Although malabsorption of bile salts may appear to be a rather benign problem, it creates a potentially serious cascade of consequences (Beyer, 2001) (Box 30-7).

If the ileum cannot “recycle” bile salts secreted into the GI tract, hepatic production cannot maintain a sufficient bile salt pool or the secretions to emulsify lipids. The gastric and pancreatic lipases are capable of digesting some triglycerides to fatty acids and monoglycerides, but without adequate micelle formation facilitated by bile salts, lipids are poorly absorbed. This can result in significant malabsorption of fats and fat-soluble vitamins A, D, and E. In addition, malabsorption of fatty acids results in their combination with divalent cations, such as calcium, zinc, and magnesium, to form fatty acid–mineral “soaps.” This results in malabsorption of these nutrients. To compound matters, colonic absorption of oxalate, which normally is bound to divalent cations, is increased, leading to hyperoxaluria and increased renal oxalate stones. Relative dehydration and concentrated urine, which are common with ileal resections, may further increase the risk of stone formation.

If the patient has any colon left, malabsorption of what bile salts are secreted can act as irritants to the mucosa, resulting in increased fluid and electrolyte secretion and increased colonic motility rather than absorption. Consumption of high-fat diets with ileal

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**Box 30-6. Factors Affecting the Course of Short-Bowel Syndrome**

- Length of remaining small intestine
- Loss of ileum, especially distal one third
- Loss of ileocecal valve
- Loss of colon
- Disease in remaining segment(s) of gastrointestinal tract
- Radiation enteritis
- Coexisting malnutrition
- Older age at surgery

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**Box 30-7. Consequences of Ileal Resection**

- Rapid transit of intestinal contents
- Decreased fluid absorptive area
- Malabsorption of vitamin B₁₂/intrinsic factor complex
- Malabsorption of bile salts
- Inadequate bile salts for lipid solubilization, digestion, and absorption, leading to loss of fat and fat-soluble nutrients
- Loss of secreted bile salts into colon because of decreased reabsorption
- Formation of hydroxy fatty acids by colonic bacteria from malabsorbed fat, resulting in decreased fluid and electrolyte absorption
- Malabsorption of Ca²⁺, Mg²⁺, and Zn²⁺ because of formation of insoluble “soaps” with malabsorbed free fatty acids
- Increased risk of oxalate stones because of increased colonic absorption of oxalate, which normally binds to Ca²⁺, Zn²⁺, and Mg²⁺
resections and retained colon may also result in the formation of hydroxy fatty acids, which also can increase fluid secretion. Cholesterol gallstones may occur more often because the ratio of bile acid, phospholipid, and cholesterol in biliary secretions is altered as a result of ileal resections. Dependence on parenteral nutrition may further increase the risk of biliary “sludge” secondary to decreased stimulus for evacuation of the biliary tract.

Lactic acidosis is a relatively rare complication that occurs only with severe SBS and remaining colon. The problem results from excessive intake and malabsorption of carbohydrate. Metabolic acidosis and production of \( d \)-lactate result from fermentation of carbohydrate, production of SCFAs, reduced colonic pH, and proliferation of acid-resistant colonic microbes that produce \( d \)-lactate (Bongaerts et al, 1997). The problem is resolved by treating the metabolic acidosis and reducing the intake of sugars and total carbohydrates.

Medical and Surgical Management of Resections

Medications are prescribed to retard gastric emptying, decrease secretions, slow GI motility, and treat bacterial overgrowth. Recently, somatostatin and somatostatin analogs and other hormones with antisecretory, antimotility, or trophic actions have been used to retard both motility and secretions (Drucker, 2002; Sundarum et al, 2002). Surgical procedures, including reversal of segments of bowel to slow transit of GI contents, creation of reservoirs (“pouches”) to serve as a form of colon, intestinal lengthening, and intestinal transplant, have been performed to help patients with major GI resections (Pirenne et al, 2001). Intestinal transplant is still one of the most difficult organ transplants and is typically reserved for gut failure and when patients develop significant complications from TPN.

Medical Nutrition Therapy

Most patients who have significant bowel resections require TPN initially to restore and maintain nutritional status (Sundaram et al, 2002). The duration of TPN and subsequent nutrition therapy is based on the extent of the bowel resection, the health of the patient, and the condition of the remaining GI tract. In general, older patients with major ileal resections, patients who have lost the ileocecal valve, and patients with residual disease in the remaining GI tract do not fare as well. Some may require lifetime supplementation with parenteral nutrition to maintain adequate fluid and nutritional status.

The two general principles for resuming enteral nutrition after small-bowel resections are (1) to start enteral feedings early and (2) to increase feeding concentration and volume gradually over time (Beyer, 2001; Vanderhoof and Young, 2001). The role of enteral feedings is to provide a trophic stimulus to the GI tract; parenteral nutrition is used to restore and maintain nutrient status. The more severe the problem, the slower the progression. Small, frequent, mini-meals (6 to 10 per day) are likely to be better tolerated than larger feedings. If enteral feedings are used, gradual introduction of feedings stimulates GI adaptation; TPN provides the major source of fluid and nutrients. More nutrients are gradually added enterally, and the volume or concentration of TPN decreases accordingly. Because of malnutrition and disuse of the GI tract, the digestive and absorptive functions of the remaining GI tract may be compromised, and malnutrition itself will delay adaptation (Cronk et al, 2000). The transition to more normal foods may take weeks to months, and some patients may never tolerate normal concentrations or volumes of foods.

Maximal adaptation of the GI tract may take up to a year after surgery. Adaptation improves function, but it does not restore the intestine to normal length or capacity. Whole foods are some of the most important stimuli to the GI tract, but other nutritional measures have been considered as a means of hastening the adaptive process and decreasing malabsorption. Glutamine, for example, is the preferred fuel for small intestinal enterocytes and so may be valuable in enhancing adaptation. Nucleotides may also enhance mucosal adaptation, but unfortunately they are often lacking in parenteral and enteral nutritional products. SCFAs (e.g., butyrate, propionate, acetate), produced from microbial fermentation of carbohydrate and fibers, are major fuels of the colonic epithelium (Mortensen and Clausen, 1996).

Patients with jejunal resections and an intact ileum and colon will likely adapt quickly to normal diets. A normal balance of protein, fat, and carbohydrate sources is satisfactory. Six small feedings, with avoidance of lactose, large amounts of concentrated sweets, and caffeine, may help to reduce the risk of bloating, abdominal pain, and diarrhea. Because the typical American diet may be nutritionally lacking and utilization of some micronutrients may be marginal, patients should be advised that the quality of their diet is of utmost importance. A multivitamin and mineral supplement may be required to meet nutritional needs.

Patients with ileal resections require increased time and patience in the advancement from parenteral to enteral nutrition. Because of losses, fat-soluble vitamins, calcium, magnesium, and zinc may need to be supplemented. Dietary fat may need to be limited, especially in those with remaining colon. Small amounts at each feeding are more likely to be tolerated and absorbed. MCT products add to the caloric intake and serve as a vehicle for lipid-soluble nutrients.

Because boluses of MCT oil (e.g., taken as a medication in tablespoon amounts) may add to the patient’s diarrhea, it is best to divide the doses equally.
in feedings throughout the day. Fluid and electrolytes, especially sodium, should be provided in small amounts and frequently.

In patients with massive resections (e.g., when the duodenum and a few inches of jejunum are anastomosed to segments of colon), an oral diet will be able to nourish only partially. In some cases, overfeeding in an attempt to compensate for malabsorption results in further malabsorption, not only of ingested foods and liquids but also of the significant amounts of GI fluids secreted in response to food ingestion. Patients with an extremely short bowel are typically nutritionally dependent on parenteral solutions for at least part of their nutrient and fluid supply. Small, frequent snacks provide some oral gratification for these patients but typically can supply only a portion of their fluid and nutrient needs.

**Blind-Loop Syndrome (Bacterial Overgrowth)**

**Pathophysiology**

Blind-loop syndrome is a disorder characterized by bacterial overgrowth resulting from stasis of the intestinal tract as an outcome of obstructive disease, radiation enteritis, fistula formation, or surgical repair of the intestine. Bacteria deconjugate bile salts, which besides being cytotoxic in this form, are also less effective as micelle formers. Poor fat absorption and steatorrhea result. Carbohydrate malabsorption occurs because of injury to the brush border secondary to the toxic effects of the products of bacterial catabolism and consequent enzyme loss. The expanding numbers of bacteria use the available vitamin $B_{12}$ for their own growth.

**Medical Treatment**

Treatment is directed toward the removal of the blind loop or control of the bacterial growth with antibiotics.

**Medical Nutrition Therapy**

Use of a lactose-free diet, along with MCT and parenteral vitamin $B_{12}$, may be useful.

**Fistula Repair**

**Pathophysiology**

Fistulas occur as a result of prenatal developmental error, trauma, or inflammatory or malignant disease processes. Fistulas of the intestinal tract can be serious threats to nutritional status because large amounts of fluid and electrolytes are lost, and malabsorption and infection can occur.

**Medical Treatment**

Fluid and electrolyte balance must be restored; infection must be brought under control; and aggressive nutritional support is mandatory to permit spontaneous or surgical closure of the fistula and wound.

**Medical Nutrition Therapy**

Either TPN or defined liquid formula diets have been used successfully in patients with fistulas (see Chapter 23). The success rate of either method depends on the location and the cause of the fistula and the patient’s overall condition.

**Ileostomy or Colostomy**

**Pathophysiology**

Patients with severe ulcerative colitis, Crohn’s disease, colon cancer, or intestinal trauma frequently require the surgical creation of an opening from the body surface to the intestinal tract to permit defecation from the intact portion of the intestine. When the entire colon, rectum, and anus must be removed, an ileostomy, or opening into the ileum, is performed. If only the rectum and anus are removed, a colostomy can provide entrance to the colon. In some cases, a temporary opening may be made to allow surgery and healing of more distal parts of the intestinal tract.

The opening, or stoma, eventually shrinks to the size of a nickel. The output from the stoma depends on its location, as shown in Figure 30-9. The consistency of the stool from an ileostomy is liquid, whereas that from a colostomy ranges from mushy to fairly well formed. Stool from a colostomy on the left side of the colon is firmer than that from a colostomy on the right side. Odor is a major concern of the patient with an ileostomy or colostomy; however, an ileostomy stool usually has a weakly acidic odor that is not unpleasant.

**Medical Treatment**

Patients with a permanent colostomy or ileostomy require sympathetic understanding from the entire health care team. Acceptance of the condition and the problems involved in maintaining bowel regularity is usually difficult. Nursing personnel, especially ostomy specialists, can play a major role in supporting and teaching patients with ostomies. Having these patients meet other people who have undergone similar surgery may help with the adjustment. Eventually, they may be encouraged by the realization that, in the future, they will not have the multiple hospitalizations or chronic disabilities that accompanied their intestinal disease.

**Medical Nutrition Therapy**

Malodorous stool is usually caused by steatorrhea or bacteria acting on particular foodstuffs to produce odorous gas. Because an individual patient may have different flora, types and amounts of gases and odors may differ among patients and with different dietary practices. Patients learn to observe their stools to de-
Inadequate water intake can result in small urine volumes and a predisposition for renal calculi. Patients with ileostomies have an above-average need for salt and water to compensate for excessive losses. Depending on the amount of ileum resected, their ileal output may be 1.5 to 5 times greater than that of the patient who has had only a colectomy. Patients with ileostomies have an above-average need for salt and water to compensate for excessive losses in stool. Inadequate water intake can result in small urine volumes and a predisposition for renal calculi.

A normal diet provides adequate sodium, and patients should be instructed to drink at least 1 L more than their ostomy output daily.

The patient with a normal, functional ileostomy usually does not become nutritionally depleted. Surgical procedures, such as ileostomy, may require specific dietary changes but no greater energy intake; caloric expenditures in these patients are similar to those of normal subjects. Those who also undergo resection of the terminal ileum need vitamin B₁₂ supplementation or intravenous injections. Patients with an ileostomy may have low vitamin C and folate intakes because of low fresh vegetable and fruit intakes, and they require supplementation. Patients with ileostomies should be guided by physiologic reasons for intolerance of foods, not by anecdotal reports. Because it is possible for a food bolus to get caught at the point where the ileum narrows as it enters the abdominal wall, it is important to warn the patient to avoid very fibrous vegetables and to chew all food well. Other than this, patients with either an ileostomy or a colostomy should be encouraged to follow their normal diet, omitting only those foods known to cause problems.

### Ileal Pouch After Colectomy

#### Pathophysiology

As an alternative to creation of an ileostomy for persons who have had their colons removed, surgeons can create a reservoir using a portion of the distal ileum (ileal pouch). Folds of the ileum are joined together to create a small pouch, which is then connected to the rectum and ileum. This is called an ileal pouch–anal anastomosis (IPAA). The most common pouch is the J pouch, but S and W pouches are sometimes created using additional folds of ileum (Pemberton and Phillips, 1998). Like the colon, the pouch develops a microflora capable of at least partially fermenting fiber and carbohydrate (Alles et al, 1997). Because the reservoir is smaller than the colon, bowel movements are likely to occur more frequently than normal, between four and eight times daily.

#### Medical Treatment

Vitamin B₁₂ injections are usually required because, as in blind loop syndrome, the microbes may compete for and bind intraluminal vitamin B₁₂. Other problems commonly reported include obstruction, "pouchitis," and increased stool output, frequency, and gas (Thompson-Fawcett et al, 1997; Pemberton and Phillips, 1998).

The incidence of obstruction may be lessened with attention to particle size of fibrous foods, chewing thoroughly, and consuming small meals frequently throughout the day. Stool frequency and volume do not return to normal, however. The normal, intact colon absorbs 80% to 90% of the liter or so of fluid entering from the ileum, leaving only 100 to 200 ml. After surgery, the remaining ileum does adapt to a
small degree by increasing efficiency of fluid absorption, but even after adaptation, fluid output is always in the range of 300 to 600 ml.

Pouchitis, as implied by the name, is an inflammation of the mucosal tissue forming the pouch. The associated pathologic changes have been described as being somewhat similar to that of IBD (e.g., ulcerative colitis) (Goldstein et al, 1997). The cause of pouchitis is not entirely clear, but it may be related to selected bacterial overgrowth, bile salt malabsorption, or insufficient SCFA production. Antibiotics are the primary form of therapy, but experiments with different types of dietary fiber, prebiotics and probiotics, and other nutrient components are being investigated (Alles et al, 1997; Sandborn et al, 2000).

Medical Nutrition Therapy
The same dietary measures that are used by others to reduce excessive stool output (reduced caffeine, lactose avoidance in lactose-intolerant persons, avoidance of sorbitol) will likely reduce stool volume and frequency in persons with pouches. Adequate fluid and electrolyte intake are especially important because of the increase in intestinal losses.

Rectal Surgery
Nutritional care after rectal surgery, such as hemorrhoidectomy, should be directed toward maintaining an intake that will allow wound repair and prevent infection of the wound by feces. The frequency of stools is minimized by the use of constipating drugs and a minimal-residue diet (see Table 30-1). Chemically defined diets are low in residue, and their use can reduce stool volume and frequency to as little as 50 g every 6 days, making the surgical construction of a temporary colostomy unnecessary. A normal diet is resumed after healing is complete, and the patient is instructed about the benefits of eating a high-fiber diet to avoid constipation in the future (see Table 30-1).

SUMMARY
The GI tract is one of the largest sites of exposure to the outside environment. The function of the GI tract in monitoring and sealing the host interior (gut barrier) plays an important role in health maintenance. Disruptions in the gut barrier following injury from nonsteroidal antiinflammatory drugs and oxidative stress have been linked to multiorgan system failure in sepsis and immune dysregulation (DeMeo et al, 2002). Contribution of gut barrier dysfunction to GI disease is an evolving concept. As evidence for the role of gut barrier dysfunction in disorders such as Crohn’s disease, celiac disease, food allergy, and related disorders mounts, new treatments and medical nutrition therapies will be developed.

Clinical Scenario 1
Suzanne is a 33-year-old teacher with Crohn’s disease who has been referred for evaluation because of abdominal pain, bloating, and occasional nausea and diarrhea. The physician suspects a distal small-bowel stricture. Suzanne is seeking information about what to eat to prevent the problem from worsening during the 3-day period before her appointment at the clinic.

1. What information would be appropriate to gather about this patient before you advise her about a nutritional plan?
2. What, in terms of Suzanne’s symptoms, makes the physician suspect a stricture?
3. What kind of dietary advice, based solely on her presumed problem, might be warranted?

Clinical Scenario 2
Mrs. Smith has IBS with a pattern of alternating constipation and diarrhea. She comes to you requesting dietary advice for (1) day-to-day management and (2) what might be “safest” to eat when she is getting ready to present weekly to biweekly reports to the executives in her large consulting company.

1. What do you want to know about Mrs. Smith’s diet, perspectives, and lifestyle?
2. What foods or eating patterns would be best (or best to avoid) for Mrs. Smith for her day-to-day activities?
3. Why might she be asking for advice during stressful periods?

Relevant Web Sites
Celiac Disease
www.niddk.nih.gov/health/digest/pubs/celiac/
Crohn’s Colitis Foundation
www.ccfa.org/
Gastrointestinal Disorders and Treatment
www.niddk.nih.gov/health/digest/digest.htm
Gluten Enteropathy Resources
www.gluten.net
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