

Malabsorption of calcium, vitamin D, and protein can cause osteoporosis, bone pain with pathologic (compression) fractures, and skeletal deformities. In fact, 20% of osteoporosis is osteomalacia secondary to decreased absorption of vitamin D associated with malabsorption conditions.¹³³

On the other hand, excessive absorption of vitamin D and calcium through the use of calcium carbonate for acid indigestion should be evaluated by a physician; these antacids may be used by women to obtain the daily 1500-mg calcium requirement as protection against osteoporosis. The therapist can direct education and intervention toward prevention and treatment of these related conditions.

Physicians may recommend vitamin B12 or other B vitamin supplements for people with carpal tunnel syndrome. Malabsorption of these essential vitamins alters the structure and disrupts the function of the peripheral nerves, spinal cord, and brain and can cause numbness and tingling and permanent neurologic damage unresponsive to vitamin B12 therapy in extreme cases.

Vascular Diseases

Blood is supplied to the bowel by the celiac and superior and inferior mesenteric arteries. These arteries have anastomotic intercommunications at the head of the pancreas and along the transverse bowel. Obstruction of blood flow can occur as a result of atherosclerotic occlusive lesions or embolism.

Intestinal Ischemia

Acute intestinal ischemia results from embolic occlusions of the visceral branches of the abdominal aorta, generally in people with valvular heart disease, atrial fibrillation, or left ventricular thrombus. Other causes include thrombosis of one or more visceral vessels involved with arteriosclerotic occlusive changes or nonocclusive mesenteric vascular insufficiency in people with congestive heart failure receiving recently instituted digitalis therapy.

Symptoms of acute intestinal ischemia include rapid onset of crampy or steady epigastric or perumbilical abdominal pain combined with minimal or no findings on abdominal examination and a high leukocyte count. Angiography of the superior mesenteric artery is essential to early diagnosis. If occlusion is present, laparotomy is performed to reestablish blood flow to the intestine if possible and to remove necrotic bowel if some viable bowel exists.

Atherosclerotic plaque of the superior mesenteric, celiac, and inferior mesenteric arteries causes a significant decrease in blood flow to the intestines, resulting in chronic intestinal ischemia. Symptoms of epigastric or perumbilical pains lasting for 1 to 3 hours in a person over 45 years of age who has peripheral vascular disease points to a diagnosis of intestinal ischemia. Arteriography may be performed if the person is a good candidate for surgery.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-10

Intestinal Ischemia

PREFERRED PRACTICE PATTERNS

6B: Impaired Aerobic Capacity/Endurance Associated with Deconditioning (peripheral vascular disease, unspecified); see also the section on Peripheral Vascular Disease in Chapter 12.

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

Intestinal angina as a result of atherosclerotic plaque-induced ischemia can result in intermittent back pain (usually at the thoracolumbar junction) with exertion. Clinical presentation combined with past medical history, the presence of coronary artery disease risk factors (see Table 12-3), and the presence of peripheral vascular disease may alert the therapist to the need for a medical referral if the client has not been medically diagnosed.

Bacterial Infections

Foodborne Illnesses (Botulism)

Overview and Incidence. Foodborne illnesses (considered to be any illness that is related to food ingestion) and, in particular, botulism were once considered rare diseases. Today foodborne illness has become a serious public health problem. The Centers for Disease Control and Prevention reports that each year 76 million people get sick, more than 300,000 are hospitalized, and 5000 deaths occur as a result of foodborne illnesses, primarily among the very young, the older adult, and the immunocompromised.¹⁰¹ It is estimated that only a fraction of the people who experience GI tract symptoms from foodborne illnesses actually seek medical care.

Recent changes in human demographics, food preferences, changes in food production and distribution systems, microbial adaptation, and lack of support for public health resources have led to the emergence of novel in addition to traditional foodborne diseases. Increasing travel and trade opportunities have increased the risk of contracting and spreading foodborne illness locally, regionally, and globally.¹⁰²

Etiology. Most cases of foodborne illness or death are caused by bacterial agents such as *Escherichia coli* and *Campylobacter*, *Listeria*, *Shigella*, and *Salmonella* species that are linked to milk, shellfish, unpasteurized apple cider, eggs, fish, poultry, meats, and raw produce.¹⁰⁰

Clostridium botulinum is the specific bacterium responsible for foodborne botulism, often referred to as "food poisoning." *C. botulinum* spores from soil or dust can also cause wound botulism in open wounds or injured tissue. Only foodborne botulism is discussed here; see Chapter 39 for a complete discussion of the effects of botulism on the peripheral nervous system.

Pathogenesis. Once they are ingested these neurotoxins resist gastric digestion and proteolytic enzymes and are readily absorbed into the blood from the proximal small intestine. Minute amounts of circulating toxin

reach the cholinergic nerve endings at the myoneural junction and bind to the presynaptic nerve terminals. The toxin abolishes transmission at skeletal neuromuscular junctions. Inhibition of acetylcholine release from cholinergic terminals at the motor endplate results in flaccid paralysis. A predilection exists for the cranial nerves, with progression caudally and symmetrically to the trunk and the extremities.

Clinical Manifestations. Many, but not all foodborne illnesses have GI tract symptoms. Symptoms can be from mild to severe; generally, the faster the onset of symptoms, the more severe the disease.

In addition to GI symptoms of prolonged bloody diarrhea leading to dehydration, weight loss, fever, nausea, and severe abdominal pain, early neurologic involvement includes motor weakness, paresthesias, and cranial nerve palsies with visual changes (double vision or diplopia, and intolerance of light or photophobia), ptosis, dysarthria or dysphagia, difficulty breathing, and impaired swallowing.

No sensory changes occur. Motor weakness of the face and neck muscles can progress to involve the diaphragm, accessory muscles of breathing, and extremities. Vomiting, abdominal pain, back pain, headache, and constipation may occur before or after the onset of paralysis. The usual secondary effects of flaccid motor paralysis can occur, such as severe muscle wasting, pressure ulcers, and aspiration pneumonia.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Toxin identification is made by stool or serum analysis or cultures of the organism. Electromyography is confirmatory and necessary to differentiate this condition from myasthenia gravis or Guillain-Barre syndrome. Nerve conduction velocities are normal, but action potentials are decreased with a supramaximal stimulus. Facilitation is found after repetitive stimulation at high frequency.

Selection of appropriate treatment depends on identification of the responsible pathogen if possible and determining if specific therapy is available. Immediate administration of antitoxin prevents further binding of free botulinum toxin to the presynaptic endings. Many episodes of acute gastroenteritis are self-limiting and require fluid replacement and supportive care.

Untreated food botulism can be fatal within 24 hours of toxin ingestion. Intubation and mechanical ventilation may be needed. Respiratory failure caused by rapid onset of respiratory muscle weakness is a complication that is often fatal.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-11

Foodborne Illness

PREFERRED PRACTICE PATTERNS

5E: Impaired Motor Function and Sensory Integrity Associated with Progressive Disorders of the Central Nervous System

5F: Impaired Peripheral Nerve Integrity and Muscle Performance Associated with Peripheral Nerve Injury (Cranial Nerve Involvement)

7A: Primary Prevention/Risk Reduction for Integumentary Disorders (risk of pressure ulcers secondary to flaccid motor paralysis)

The sudden onset of rapidly progressive symptoms associated with botulism is most likely to be reported to a physician rather than to a therapist. However, presentation of acute symmetric cranial nerve impairment (ptosis, diplopia, dysarthria), followed by descending weakness or paralysis of the muscles in the extremities or trunk with or without back pain, and dyspnea from respiratory muscle paralysis, requires immediate medical referral.

After the acute onset and initiation of medical treatment, treatment is as for cranial nerve palsy. In mild to moderate cases, clients experience a gradual recovery of muscle strength that can take as long as a year after disease onset; in severe cases, a 40% mortality rate exists.

Inflammatory Bowel Disease

Overview and Definition

Inflammatory bowel disease (IBD) refers to two inflammatory conditions: Crohn's disease and ulcerative colitis (Table 16-6). *Crohn's disease* (CD) is a chronic lifelong inflammatory disorder that can affect any segment of the intestinal tract (intestine or ileum, large intestine or colon) and even tissues in other organs. It can affect all layers of the intestine and is characterized by diseased areas of intestine with normal intestine between ("skip" areas) with periods of exacerbation and remission. CD is also referred to as *regional enteritis*, *regional ileitis*, *terminal ileitis*, or *granulomatous colitis*, depending on the location of the inflammation.

Ulcerative colitis (UC) is a chronic inflammatory disorder of the mucosa and submucosa of the colon in a continuous manner without skips. It is characterized by chronic diarrhea and rectal bleeding with ulceration. It may be referred to as *ulcerative proctitis* (involving the rectum only) or *pancolitis* (involving the entire colon).

Incidence

Both CD and UC are bowel disorders of unknown cause involving genetic and immunologic influences on the GI tract's ability to distinguish foreign antigens from self-antigens. These two conditions can occur in all age groups and affect both genders equally; incidence peaks between the ages of 15 and 35, but CD does occur in later decades, usually after age 50. An estimated 1 million Americans are affected by IBD, with equal distribution between these two conditions.³⁷

Etiology and Pathogenesis

Inflammatory bowel conditions are considered to be autoimmune diseases in which either the antibodies or other defense mechanisms are directed against the body. Theories explaining the etiopathogenesis of IBD have been proposed ever since UC and CD were recognized as the two major forms of the disease. Although the exact causes and mechanisms of tissue damage in IBD have yet

Table 16-6 Comparison of the Characteristics of Crohn's Disease and Ulcerative Colitis

Characteristics	Crohn's Disease	Ulcerative Colitis
Incidence		
Age at onset	Any age; 10-30 yr most common	Any age; 10-40 yr most common
Family history	20% to 25%	20%
Gender (prevalence)	Equal in women and men	Equal in women and men
Cancer risk	Increased; early detection best means of prevention	Increased; preventable with bowel resection
Pathogenesis		
Location of lesions	Any segment; usually small or large intestine	Large intestine; rectum
Skip lesions	Common	Absent
Inflammation and ulceration	Entire intestinal wall involved	Mucosal/submucosal layers involved
Granulomas	Typical	Uncommon
Thickened bowel wall	Typical	Uncommon
Narrowed lumen and obstruction	Typical	Uncommon
Fissures and fistulas	Common	Absent
Clinical Manifestations		
Abdominal pain	Mild to severe; common	Mild to severe; less frequent
Diarrhea	May be absent; moderate	Typical; often severe; chronic
Bloody stools	Uncommon	Typical
Abdominal mass	Common; right lower quadrant	Uncommon
Anorexia	Can be severe	Mild or moderate
Weight loss	Can be severe	Mild to moderate
Skin rashes	Common, mild	Common, mild
Joint pain	Common, mild to moderate	Common, mild to moderate
Growth retardation (pediatric)	Often marked	Usually mild
Clinical course	Remissions and exacerbations	Remissions and exacerbations

to be completely understood, enough progress has been made to accept the following as one valid explanation.⁴¹

IBD is an inappropriate immune response that occurs in genetically susceptible individuals as the result of a complex interaction among environmental factors, microbial factors, and the intestinal immune system. Although these disorders tend to occur in families, no single genetic marker (i.e., histocompatibility antigen) has been identified yet, preventing early identification of susceptible individuals.

The onset and reactivation of disease are triggered by environmental factors that transiently break the mucosal barrier, stimulate immune responses, or alter the balance between beneficial and pathogenic enteric bacteria.¹²⁷ There is a marked overexpression of proinflammatory cytokines such as tumor necrosis factor (TNF) α and increased production of matrix-degrading enzymes by fibroblasts and macrophages, which are likely responsible for the ulceration and fistula formation that occur in CD.⁹³

A growing number of reports have demonstrated a disorder of autonomic function in subgroups of people with functional bowel disorders. Altered autonomic balance (low vagal tone, increased sympathetic activity) may alter visceral perception. Autonomic dysfunction may represent the physiologic pathway, accounting for many of the extraintestinal symptoms and frequent GI problems encountered by people with other disorders such as chronic fatigue and fibromyalgia.¹¹

Almost an endless list of environmental factors has been identified. Smoking is a risk factor for CD but a

protective factor for UC.⁴¹ A possible correlation of the onset of disease with life stresses and poor adaptation to those stresses is cited in the majority of literature regarding IBD, but this has not been proved conclusively. More likely, these factors exacerbate the illness but are not a cause of the condition. Autoimmune disorders such as systemic lupus erythematosus and fibromyalgia often accompany IBD.

There is no evidence that classic infectious agents cause IBD; rather, increasing evidence continues to point to an abnormal immune response against the normal enteric flora. Gut inflammation is mediated by cells of the innate as well as adaptive immune systems, with the additional contribution of nonimmune cells, such as epithelial, mesenchymal, and endothelial cells, and platelets.⁴¹

Improved hygiene in developed countries may be another etiologic factor in the development of inflammatory conditions such as IBD. Geographic and ethnic variations in both UC and CD suggest that parasitic worms (helminths) may be an important environmental factor. Helminths alter host mucosal and systemic immunity. They induce mucosal T cells to make regulatory cytokines that have a protective function in the intestines. Improved hygiene may preclude exposure to helminths, effectively removing immune protection against disease from dysregulated inflammation.^{48,155}

Two features distinguish CD from UC. In CD, a constitutionally weak immune response predisposes to accumulation of intestinal contents that breach the mucosal barrier of the bowel wall, resulting in granuloma formation and chronic inflammation.¹¹ The inflammation usually involves all layers of the bowel wall, referred to

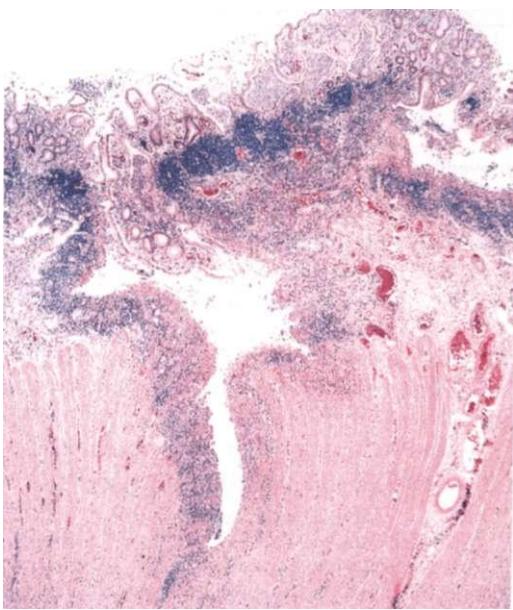


Figure 16-10

Crohn's disease of the colon. A deep fissure extending into the muscle wall and a second, shallow ulcer (upper right). Abundant lymphocyte aggregates are present, evident as blue patches of cells at the interface between the mucosa and submucosa. (From Kumar V: Robbins and Cotran: pathologic basis of disease, ed 7, Philadelphia, 2007, Saunders.)

as *transmural inflammatory disease*, and the inflammatory process is discontinuous, so that segments of inflamed areas are separated by normal tissue in a skip pattern. The transverse colon is affected in half of all cases; other common sites include the small bowel, ascending or descending colon, and anorectal region.

In UC, the large intestine (colon) is primarily affected, with involvement extending uniformly and continuously, usually starting from the distal part of the rectum.

Inflammation accompanying CD produces thickened, edematous tissue, and chronic inflammation leads to ulcerations, which produce fissures that extend the inflammation into lymphoid tissue. Mesenteric lymph nodes often are enlarged, firm, and matted together. The mesentery is a membranous peritoneal fold attaching the small intestine to the dorsal abdominal wall.

The lesions are granulomatous (epithelioid cells rimmed by lymphocytes) with projections of inflamed tissue surrounded by fibrous scarring narrowing the intestinal lumen (Fig. 16-10). A combination of the granulomas (nodular swelling), ulceration, and fibrosis results in a cobblestone appearance of the mucosal surface of the colon (Fig. 16-11).

Inflammation of the mucosa associated with UC results in small erosions and subsequent ulcerations with eventual abscess formation and necrosis (Figs. 16-12 and 16-13). Destruction of the mucosa causes bleeding, cramping pain, bowel frequency, and large volumes of watery diarrhea owing to decreased absorption and decreased transit time of intestinal contents through the colon.



Figure 16-11

Crohn's disease of the ileum, showing narrowing of the lumen, bowel wall thickening, serosal extension of mesenteric fat ("creeping fat"), and linear ulceration of the mucosal surface (arrows). (From Kumar V: Robbins and Cotran: pathologic basis of disease, ed 7, Philadelphia, 2007, Saunders.)

Clinical Manifestations

Clinically, inflammatory bowel disorders are characterized by recurrent inflammatory involvement of intestinal segments with diverse clinical manifestations, often resulting in a chronic, unpredictable course. Clinical activity associated with IBD may be rated as mild to moderate, moderate to severe, or severe to fulminant. With early and adequate treatment, asymptomatic remission is possible.

The inflammatory phase of IBD begins with low-grade fever, malaise, weight loss, diarrhea, and abdominal cramping or pain. The inflammatory phase may be followed by the obstructive phase with persistent abdominal bloating and pain and distention from the movement of gas through the system. For comparison of specific signs and symptoms, see Table 16-6.

Arthritis and Inflammatory Intestinal Diseases. The theory that an immune mechanism may be involved in the development of IBD is based in part on the presence of extraintestinal manifestations involving the hematologic, dermatologic, renal, ocular, hepatobiliary, and musculoskeletal systems.

TNF, an inflammatory cytokine produced by the cells of the immune system, may be implicated in the pathophysiology of CD and rheumatoid arthritis. It would appear that proinflammatory and immune-regulatory cytokines are up-regulated in the mucosa of individuals with IBD. This knowledge has opened up new avenues of treatment pursuing monoclonal antibodies directed against TNF.

Polyarthritides, migratory arthralgias, and redness of the skin (erythema nodosum) are the most common musculoskeletal/integument impairments. Joint involvement ranging from arthralgia only to acute arthritis is a common finding in IBD (in 25% of clients with IBD). Intestinal arthritis is usually peripheral, monoarticular, affecting the knee, ankle, or wrist, but it can affect any joint and more than one joint.

Spondylitis and sacroiliitis also can occur. Joints of the lower extremities are affected more often than those of

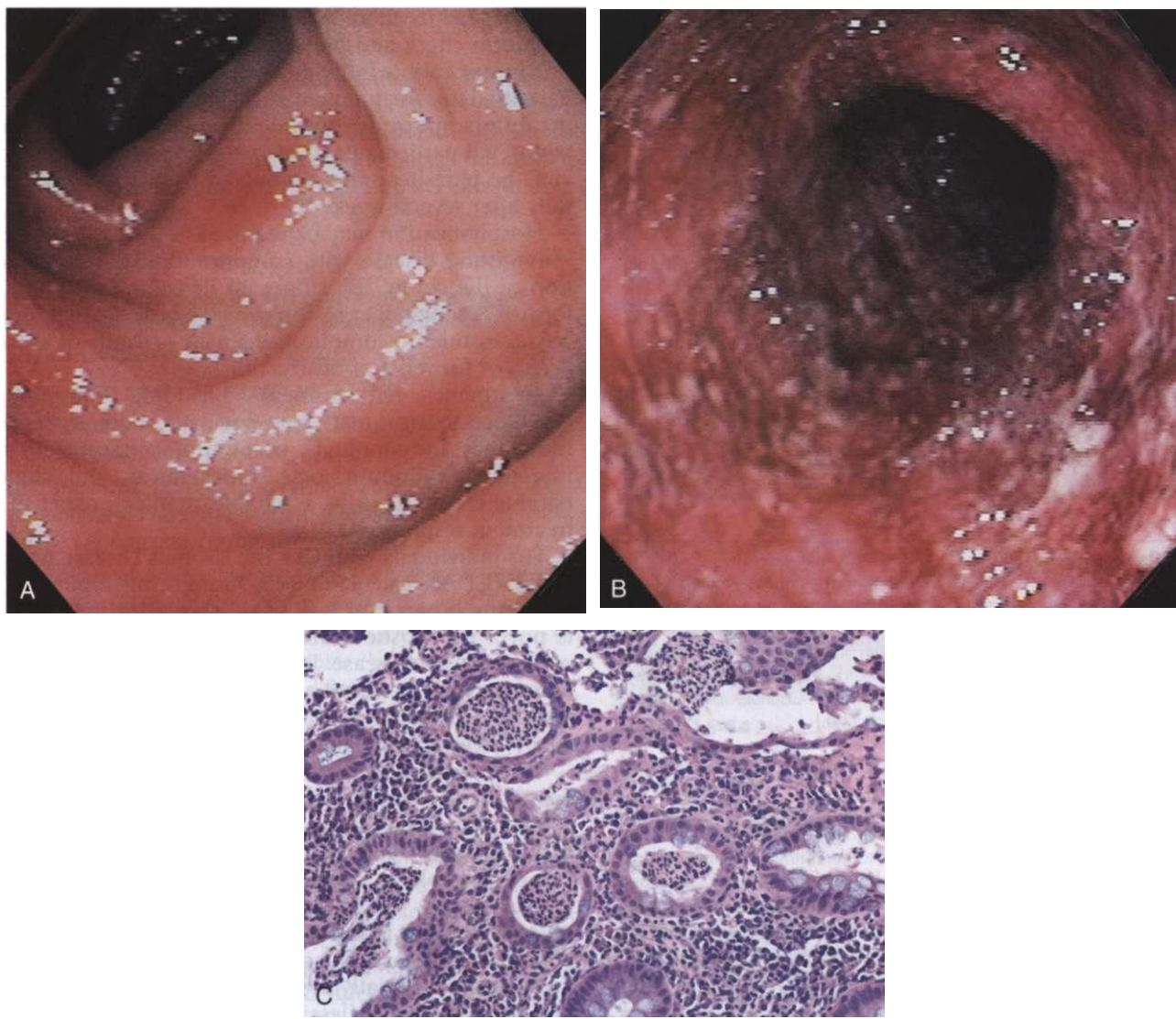


Figure 16-12

Spectrum of severity of ulcerative colitis. **A**, Colonoscopic findings in mild ulcerative colitis demonstrating edema, loss of vascularity, and patchy subepithelial hemorrhage. **B**, Colonoscopic findings in severe ulcerative colitis with loss of vascularity, hemorrhage, and mucopus. **C**, Histologic specimen showing a severe acute and chronic inflammatory process with multiple abscesses. (From Feldman M: *Sleisenger and Fordtran's gastrointestinal and liver disease*, ed 8, Philadelphia, 2006, Saunders.)

the upper extremities, and asymmetric inflammation of the proximal interphalangeal joints of the toes is particularly suggestive of enteropathic arthritis.^{3,8}

Acute arthritis associated with chronic IBD comes and goes, occurring during the course of the bowel disease or preceding repeat episodes of bowel symptoms by 1 to 2 weeks. The onset of arthritis occurs abruptly and reaches a peak within 48 to 72 hours, manifesting as pain, erythema, swelling, and limited range of motion.

Peripheral arthritis is usually self-limited, resolving within several months as the underlying disease is treated and without permanent sequelae or joint deformities. Spinal disease runs a course independent of the bowel disease and does not necessarily improve with medical treatment of the underlying IBD.

Pathologically, the synovitis is nonspecific without crystals or evidence of infection. Tests for specific forms

of arthritis (e.g., rheumatoid factor and antinuclear antibody) usually give negative results; test results for HLA-B27 are positive (see explanation of HLA in Chapter 7; see also Table 40-20). Treatment of intestinal arthritis is toward control of the underlying intestinal inflammation.

MEDICAL MANAGEMENT

DIAGNOSIS. Because no specifically distinctive characteristic features or specific diagnostic tests exist, the diagnosis of CD and UC remains one of exclusion based on medical history and clinical presentation.

Diagnostic and monitoring procedures may require a combination of radiographic modalities and other tests such as colonoscopy, barium enema x-ray, fecal occult blood test, and blood testing (e.g., low hemoglobin level may indicate intestinal blood loss). Microscopically,



Figure 16-13

Total colectomy specimen from an individual with ulcerative colitis. The colon shows diffuse mucosal inflammation that extends proximally from the rectum without interruption to the transverse colon. The mucosal pattern in the terminal ileum and cecum (arrow) is normal. The distal mucosa is erythematous and friable with many ulcers and erosions. (From Feldman M; Sleisenger and Fordtran's gastrointestinal and liver disease, ed 8, Philadelphia, 2006, Saunders. Courtesy of Feldman's GastroAtlas online, Current Medicine Group Ltd.)

granulomas present in CD (but not present in UC) distinguish CD from UC.

TREATMENT. Current treatment is directed toward symptomatic relief and control of the disease process on an individual basis. Treatment is directed to minimize toxicity, delay or decrease the likelihood of recurrence, and optimize quality of life. Some treatment measures may include diet and nutrition, symptomatic medications such as antidiarrheals or antispasmodics, or specific drug therapy (e.g., immune modifiers, antibiotics, corticosteroids, aminosalicylates to reduce intestinal or systemic inflammation).

NSAIDs help reduce pain and stiffness associated with enteropathic arthritis but may cause exacerbation of the IBD. Cyclooxygenase-2 (COX-2) inhibitors may be used instead.

People with CD often have relapses; current investigations are looking for better treatments to maintain remission. A broad range of cytokine-based (biologic) therapies directed at the mucosa and enteric flora have been approved by the Food and Drug Administration and are breaking ground in the management of enteropathic arthropathies (e.g., TNF antagonist adalimumab [Humira]) in addition to growth hormone, a new glucocorticoid (budesonide), methotrexate (MTX), and other agents.^{135,140}

Experimental treatment with intestinal parasitic worms for IBD is under investigation. Helminthic parasites such as *Trichuris suis* have the ability to downregulate host immunity, protecting themselves from elimination.^{103,119}

Exposure to these parasites may keep the immune system in check and prevent it from attacking the intestine in susceptible individuals. Individuals with IBD who have tried this treatment method have been able to reduce the number of or discontinue the medications they were taking. All participants improved without adverse effects, and the parasites were eliminated from the body naturally after several weeks.⁴⁸

Hospitalization may be required in the case of severe, unremitting disease with complications. Surgical resection of the colon, colostomy, or ileostomy may be performed for toxic megacolon or if medical intervention is unsuccessful and complications such as fistula or abscess occur, or for relief of obstruction.

Treatment refractoriness including physiologic resistance to treatment may have its origin in specific individual immunologic peculiarities; researchers are investigating immunologic, biochemical, and clinical parameters to identify a reliable marker predictive of treatment response.⁵⁷

For individuals with CD, the Crohn's Disease Activity Index (CDAI) calculator is a monitoring tool used to gauge the progress or lack of progress with treatment. It is not a prognostic indicator and does not predict the outcome of the disease. It is designed to help individuals compare their personal situation from one week to the next. It is available on-line at: <http://www.ibdjohn.com/cda/> (accessed May 26, 2007).

Chemoprevention and routine screening colonoscopy are advised for the prevention of colorectal cancer associated with longstanding IBD. Chemoprevention can include aspirin and other NSAIDs, 5-aminosalicylates, ursodeoxycholic acid, and folate supplementation.⁸⁴

PROGNOSIS. CD is an incurable, chronic, and sometimes debilitating disease with a known increased risk of intestinal cancer, especially in people who develop this condition at a young age (less than 30 years of age). The risk for colorectal cancer is increased with duration of disease, with a 2% incidence of cancer after 10 years, a 9% incidence after 20 years, and a 19% incidence after 30 years of disease. The development of cancer accounts for one third of deaths related to UC.⁸⁴ Remission is possible for individuals who respond well to treatment; some people are able to discontinue the use of ongoing corticosteroids.

Surgical removal of the diseased bowel does not prevent bowel cancer in CD, so screening of stool specimens and periodic colonoscopic examination and biopsy are required for early detection. With proper medical and surgical treatment, the majority of people are able to cope with this disease and its complications. Few people die as a direct consequence of CD. The mortality rate (5% to 10%) increases with the duration of the disease.

Like CD, UC is a chronic, occasionally debilitating disease. However, it can be cured by colon resection, although this is not always the best course of action. The clinical course is variable but recurrent with long periods of remission possible.

Approximately 85% of clients with UC have mild to moderate intermittent disease managed without hospitalization. The remaining 15% demonstrate a full-blown

course involving the entire colon, severe diarrhea, and systemic signs and symptoms.

A 20% mortality rate exists during the first 10 years of UC when complications occur. Also as in CD, 10 years of chronic attacks of UC can predispose the colon to metaplastic changes leading to colon cancer, but unlike in CD, in UC removal of the affected bowel can prevent bowel cancer.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-12

Inflammatory Bowel Disease

PREFERRED PRACTICE PATTERNS

4A: Primary Prevention/Risk Reduction for Skeletal Demineralization

4D: Impaired Joint Mobility, Motor Function, Muscle Performance, and Range of Motion Associated with Connective Tissue Dysfunction (arthritic component)

4E: Impaired Joint Mobility, Motor Function, Muscle Performance, and Range of Motion Associated with Localized Inflammation (ankylosing spondylitis; psoas abscess)

4F: Impaired Joint Mobility, Motor Function, Muscle Performance, Range of Motion, and Reflex Integrity Associated with Spinal Disorders (ankylosing spondylitis)

Musculoskeletal Involvement

When terminal ileum involvement in CD produces periumbilical pain, referred pain to the corresponding low back is possible. Pain of the ileum is intermittent and perceived in the lower right quadrant with possible associated iliopsoas abscess or ureteral obstruction from an inflammatory mass, causing buttock, hip, thigh, or knee pain, often with an antalgic gait. Specific objective tests are available to rule out systemic origin of hip, thigh, or knee pain (Figs. 16-14 and 16-15; see also Fig. 16-22).

Psoas abscesses most commonly result from direct extension of intraabdominal infections such as appendicitis, diverticulitis, and CD. Clinical manifestations of a psoas abscess include fever, lower abdominal pain, or referred pain as described. Flexion deformity of the hip may develop from reflex spasm with a positive psoas sign as shown. Symptoms are exacerbated by hip extension. A tender or painful mass may be palpated in the groin.

Approximately 25% of all clients with IBD present with migratory arthralgias, monarthritis, polyarthritis, or sacroiliitis. The joint problems and GI disorders may appear simultaneously, the joint problems may manifest first (sometimes even years before bowel symptoms), or intestinal symptoms present along with articular symptoms but are disregarded as part of the whole picture by the client.

Any time a client presents with low back, hip, or sacroiliac pain of unknown origin, the therapist must screen for medical disease by asking a few simple questions about the presence of accompanying intestinal symptoms, known personal or family history of IBD, and possible relief of symptoms after passing stool or

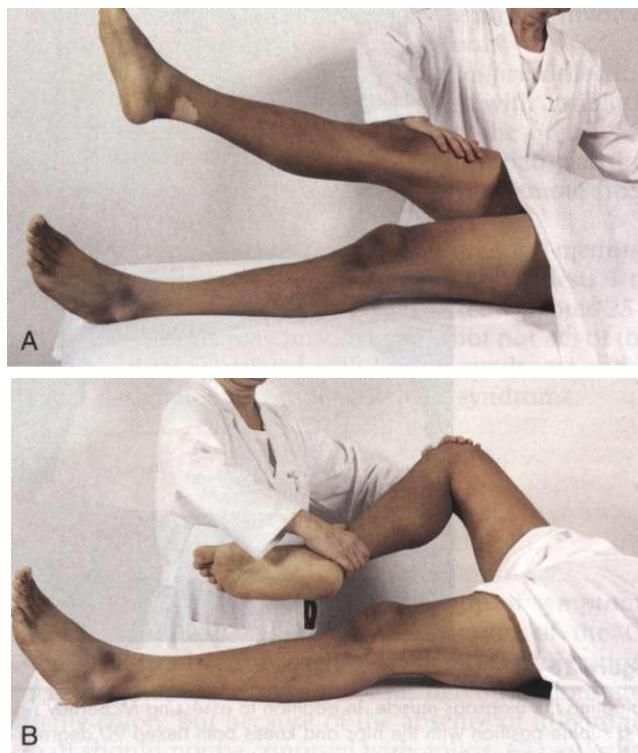


Figure 16-14

Muscle tests. **A**, Iliopsoas muscle test. With the client supine, instruct the client to lift the right leg straight up; apply resistance to the distal thigh as the client tries to hold the leg up. When the test result is negative, the client feels no change; when the test result is positive (i.e., the iliopsoas muscle is inflamed or abscessed), pain is felt in the right lower quadrant. **B**, Obturator muscle test. With the client supine, perform active assisted motion, flexing at the hip and knee; hold the ankle and rotate the leg internally and externally. A negative or normal response is no pain; a positive test result for inflamed obturator muscle is right lower quadrant (abdominal) pain. (From Jarvis C: Physical examination and health assessment, ed 4, Philadelphia, 2004, Saunders, p 586.)

gas.⁶⁰ Joint problems usually respond to treatment of the underlying bowel disease but in some cases require separate management. Interventions for the musculoskeletal involvement follow the usual protocols for each area affected.

People with IBD are known to have low bone mineral content and a high prevalence of osteoporosis. The pathogenesis is not completely understood but is considered multifactorial at this time, including possible genetic factors,¹³² malabsorption, corticosteroid use, and deficiency of fat-soluble vitamins, among them vitamin K necessary for calcium binding to bone.¹³¹

Low bone mineral density may be more characteristic of CD than of UC, but no consistent differentiation has been made between CD and UC in this regard.¹³⁰ The therapist can provide osteoporosis education and prevention for clients with IBD. See the section on Osteoporosis in Chapter 24.

The therapist must always know what medications clients are taking so that the first sign of possible side

Continued.

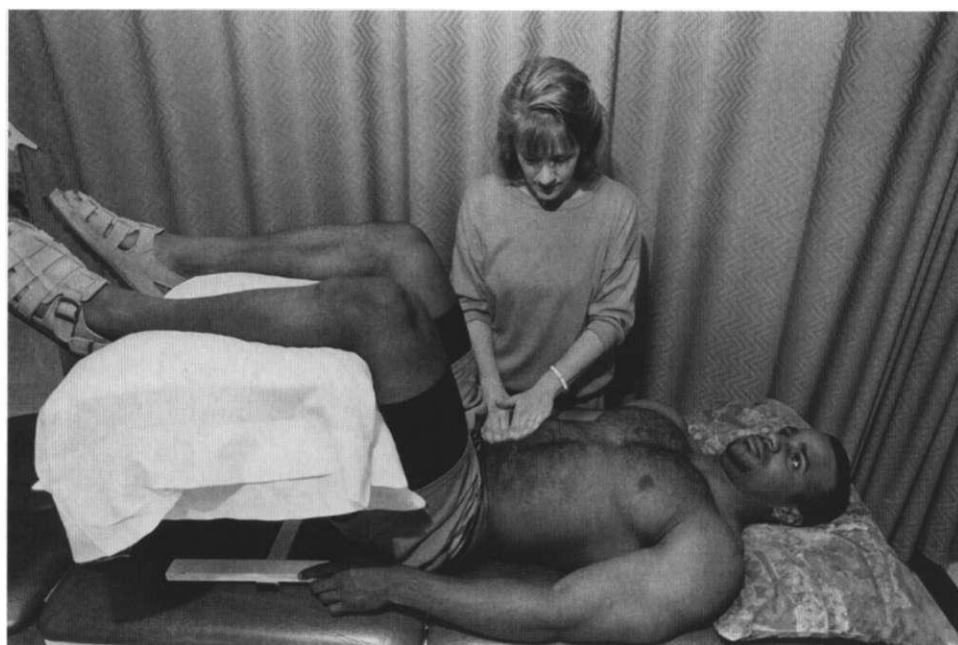


Figure 16-15

Palpating the iliopsoas muscle. In addition to assessing McBurney's point, the examiner may also palpate the iliopsoas muscle by placing the client in a supine position with the hips and knees both flexed 90 degrees and with the lower legs resting on a firm surface (a traction table stool works well for this; pillows under the lower legs may be necessary to obtain the full position). Palpate approximately one third the distance from the anterior superior iliac spine toward the umbilicus; it may be necessary to ask the client to initiate hip flexion on that side to help isolate the muscle and avoid palpating the bowel. A positive test result for iliopsoas abscess is right lower quadrant (abdominal) pain; alternatively, palpation may produce back pain or local muscular pain from a shortened or tight muscle, indicating the need for soft tissue mobilization (STM) and stretching of the iliopsoas muscle. STM in this area would be contraindicated in the presence of any abdominal or pelvic inflammatory process involving the iliopsoas muscle until the infection has been treated with complete resolution.

effects will be recognized and the physician alerted. Corticosteroids are an important and effective drug for treating moderate and severe IBD but carry with them all of the complications of prolonged high-dose steroid therapy (see Table 5-4).

Dehydration

Hydration and nutrition are always long-term concerns with clients who have UC or CD. The client must be observed for any signs of dehydration (e.g., dry lips, dry hands, headache, brittle hair, incoordination, disorientation; see Box 5-8), in addition to any increase or pathologic change in symptoms. Any increase in painful symptoms or increased stool output or stool frequency must be reported to the physician.

Psychologic Issues

People with IBD may have a characteristic personality susceptible to emotional stresses, which precipitate or exacerbate their symptoms. Studies have investigated the effects of emotional words on the digestive tract in cases of irritable bowel syndrome (IBS, a separate entity from IBD; see later discussion of IBS), and the same response may be present in IBD, but this has not been substantiated yet. Preliminary data (in cases of IBS) demonstrate a change in rectal tone during exposure to angry, sad, or anxious words.^{13,14}

Additionally, the chronic nature of IBD affecting persons in the prime of life often results in feelings of anger, anxiety, and possibly depression. These emotions are important factors in the client's response to treatment and in modifying the overall course of the disease.

The therapist can be instrumental in acknowledging the client's feelings, validating the effects of the disease, and prescribing an exercise program to match the needs of the individual. Exercise can help moderate depression, boost immune system function, combat the effects of long-term corticosteroid use, and help improve body image.

The therapist can help individuals develop positive coping strategies and management techniques to deal with the disruptions that can occur with intermittent and unexpected bouts of diarrhea and abdominal pain. Stress management, relaxation techniques such as Physiologic Quieting and autogenic breathing, or guided imagery can help with visceral pain and discomfort. The therapist can help individuals find ways to improve activity and participation, not just deal with impaired body structures and functions.

Antibiotic-Associated Colitis

Antibiotics can suppress normal GI tract flora, the bacteria usually residing within the lumen of the intestine, thus allowing yeasts and molds to flourish. Other kinds of microorganisms can replace normal GI tract flora suppressed by antibiotic therapy, such as *Clostridium difficile*, the major cause of colitis in people with antibiotic-associated diarrhea. Although nearly all antibiotics have been associated with this syndrome, drugs such as clindamycin, ampicillin, and the cephalosporins commonly are implicated.

C. difficile is not invasive but replaces normal GI tract flora by producing toxins that damage the colonic mucosa. *C. difficile* toxins compromise the epithelial cell barrier by at least two pathophysiologic pathways involving complex interactions between immune and inflammatory cells.¹¹⁶

The overgrowth of *C. difficile* causes lesions described as raised, exudative, necrotic, and inflammatory plaques. These plaques attach to the mucosal surface of the small intestine or colon, or both, giving this condition the name *pseudomembranous enterocolitis* (PMC). When the lesions are restricted to the small intestine, the term *pseudomembranous enteritis* is applied.

Onset of symptoms (primarily voluminous, watery diarrhea, but also abdominal cramps and tenderness and fever) occurs during early administration or within 4 weeks after the drug has been discontinued. Complications of untreated illness include dehydration with accompanying electrolyte imbalance, perforation, toxic megacolon, and death.

Diagnosis is made by a stool test for *C. difficile* toxin or other laboratory tests. Discontinuation of the antibiotics is usually enough to relieve symptoms, but antimicrobial agents such as metronidazole or the more expensive vancomycin are prescribed to treat the *C. difficile* overgrowth.

Supportive measures may include administration of intravenous fluids to correct fluid losses, electrolyte imbalance, and hypoalbuminemia. Recurrence of symptoms is common when treatment is discontinued or in the case of vancomycin-resistant organisms, requiring retreatment, or in some cases, careful medical observation. In severe disease, mortality rates can be as high as 30%.

For further discussion of this topic, see Chapter 8.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-13

Antibiotic-Associated Colitis

The primary concern with any client experiencing excessive watery diarrhea is fluid and electrolyte imbalance. See the implications previously discussed in Chapter 5. Because the onset of this condition may occur up to 1 month after the antibiotic has been discontinued, the client may not recognize the association between current GI symptoms and previous medications. Anytime someone taking antibiotics or recently completing a course of antibiotics develops GI

symptoms, especially with joint and/or muscle involvement, encourage physician notification.

Reactive arthritis occurring after *C. difficile* infection is most common with colitis associated with antibiotic therapy. *Reactive arthritis* is defined as the occurrence of an acute, aseptic, inflammatory arthropathy arising after an infectious process, but at a site remote from the primary infection.

The arthritis typically involves the large and medium joints of the lower extremities and first manifests 1 to 4 weeks after the infectious insult (see Chapter 25). Reactive arthritis may include some, but not all, of the three features associated with Reiter's syndrome and is often designated incomplete Reiter's syndrome.

Irritable Bowel Syndrome

Definition and Incidence

Irritable bowel syndrome (IBS) is a group of symptoms that represent the most common disorder of the GI system. IBS has been referred to as nervous indigestion, functional dyspepsia, spastic colon, nervous colon, and irritable colon, but because of the absence of inflammation, it should not be confused with colitis, CD, or other inflammatory diseases of the intestinal tract.

IBS is a chronic condition and is not limited to the colon but can occur anywhere in the small and large intestines. It is one of the most common GI disorders diagnosed in the United States, affecting up to 20% of the population.^{34,125}

Women are affected much more often than men, especially in early adulthood, with a second peak after age 50. However, IBS can occur in either gender at any age. Approximately 45 million Americans have been identified with this condition; it is likely that many more are affected but remain undiagnosed.

Extra-GI conditions associated with IBS are numerous, such as fibromyalgia, chronic fatigue syndrome, temporomandibular joint disorder, and chronic pelvic pain.^{67,157}

Etiologic Factors and Pathogenesis

IBS is considered a "functional" disorder because the symptoms cannot be attributed to any identifiable abnormality of the bowel (structural or biochemical). It has been suggested that IBS may involve three main abnormalities of gut function: altered GI motor activity, visceral hypersensitivity, and/or altered processing of information by the nervous system.²⁰

IBS is characterized by abnormal intestinal contractions, presumably as a result of the digestive tract's reaction to emotions, stress, and certain chemicals in particular foods. People with IBS have an exaggerated gastrocolic reflex, the signal the stomach sends to the colon to stimulate contractions after food arrives.

In some cases of IBS, pain and discomfort are not accompanied by changes in GI motility, suggesting an increased internal sensitivity, that is, enhanced sensation and perception of what is happening in the digestive tract referred to as "enhanced visceral nociception."

In such cases it may be that the internal pain threshold is lowered for reasons that remain unclear. Individuals with IBS experience pain and bloating at much lower pressures than people without IBS. Serotonin, a neurotransmitter produced in the gut and located inside enteric nerve cells, may also play a role in the disorder. And an imbalance in the beneficial bacteria normally present in the gut may also be a predisposing factor.

It is well documented that individuals with IBS report a greater number of symptoms compatible with a history of psychopathologic disorders, abnormal personality traits, psychologic distress, and sexual abuse.^{82,154} Episodes of emotional or psychologic stress, fatigue, smoking, alcohol intake, or eating (especially a large meal with high fat content, roughage, or fruit) do not cause but rather trigger symptoms. Intolerance of lactose and other sugars may account for IBS in some people.

Scientists continue to explore the brain (nervous system)-gut connection to better understand IBS and other functional GI disorders. The enteric nervous system is composed of a vast network of neurons located throughout the GI tract. This neuronal network communicates directly with the brain through the spinal cord. There are as many neurons in the small intestine as in the spinal cord, and the same hormones and chemicals that transmit signals in the brain have been found in the gut, including serotonin, norepinephrine, nitric oxide, and acetylcholine.

The GI tract is very sensitive to changes in serotonin levels; it may be that IBS occurs as a result of abnormalities in serotonin levels responsible for digestive functions. Increased levels of serotonin in the gut result in diarrhea, while decreased levels may account for individuals who have IBS-associated constipation.

Studies investigating the effects of emotional words on the digestive tract substantiate the close interaction among mind, brain, and gut. Preliminary data demonstrate an increase in intestinal contractions and change in rectal tone during exposure to angry, sad, or anxious words. These changes in intestinal motor function may influence brain perception.^{13,14}

Clinical Manifestations

Symptoms of IBS usually begin in young adulthood and persist intermittently throughout life with variable periods of remission. A generally accepted definition of IBS requires at least 3 months of abdominal pain that is relieved by a bowel movement and at least three of the following symptoms (present at least 25% of the time): abdominal bloating or distention, passage of mucus, changes in stool form (hard or loose and watery), alterations in stool frequency, or difficulty in passing a movement.

For some people, IBS symptoms are annoying but manageable. For others, IBS significantly affects quality of life and daily function. Diarrhea, constipation, or alternating diarrhea and constipation with abdominal cramps and pain is common. Rapid alterations in the speed of bowel movement create an obstruction to the natural flow of stool and gas. The resultant pressure buildup in the bowel produces the pain and spasm reported.

Pain may be steady or intermittent, and there may be a dull, deep discomfort with sharp cramps in the morning or after eating. The typical pain pattern consists of lower left quadrant abdominal pain accompanied by constipation and diarrhea. Upper abdominal pain that extends up under the ribs can occur when the sigmoid colon in the left lower abdomen contracts and gas rises into the transverse colon.

Other symptoms may include nausea and vomiting, anorexia, foul breath, sour stomach, and flatulence. Symptoms of IBS tend to disappear at night when the affected individual is asleep. Nocturnal GI symptoms suggest a diagnosis other than IBS.⁹¹

MEDICAL MANAGEMENT

DIAGNOSIS. Diagnosis is based on a classic history, as there is no definite objective blood test, x-ray, or other indicator of IBS. Symptom-based criteria have been developed for the diagnosis of IBS and require the presence of abdominal pain or discomfort for 12 or more weeks (they do not have to be consecutive) within the last 12 months, accompanied by at least two of these symptoms:

- Relief of abdominal discomfort with defecation
- Change in the frequency of bowel movements
- Change in the appearance or form of stool

Other symptoms such as abnormal stool frequency (more than three times per day or less than three times per week); straining, urgency, or feeling of incomplete evacuation; passage of mucus; and a bloated feeling or abdominal distention are also taken into consideration when making the diagnosis.

Sigmoidoscopy often reveals marked spasm and mucus in the colonic lumen and frequently provokes a spontaneous exacerbation of symptoms. Laboratory studies include a complete blood count and stool examination to rule out lactose intolerance and the presence of occult blood, parasites, and pathogenic bacteria. GI tract films may show altered motility without other evidence of abnormalities. Other radiologic modalities may be employed to diagnose this condition.

TREATMENT. Treatment is aimed at relieving abdominal discomfort, stabilizing bowel habits, and altering underlying causes of the syndrome. Lifestyle changes (especially dietary changes), medications, behavioral counseling, and psychotherapy have been advocated.

Dietary exclusion of milk and milk products may be helpful for those people with lactose intolerance. Increased dietary fiber, use of bulking agents such as psyllium preparations, and avoidance of alcohol, tobacco, gas-producing foods (e.g., cauliflower, cabbage, baked beans, broccoli), and GI stimulants such as caffeine-containing beverages often are recommended.

Since people with IBS can have a slower intestinal transit time, resulting in constipation, maintaining regular bowel movements is an important part of the management of IBS. Once constipation occurs, getting rid of painful symptoms is difficult. The use of fiber supplements such as polycarbophil (FiberCon), psyllium seed (Metamucil), and increased intake of water and other fluids is advised.

A stress reduction program with a regular program of relaxation techniques and exercise in conjunction with psychotherapy and biofeedback training may be effective for some people. Behavioral therapy is focused on identifying and reducing or eliminating triggers and reducing negative self-talk. Hypnotherapy (hypnosis) can give some control over the muscle activity of the GI tract and the gut's sensitivity to stress and other influences.¹⁵⁹

Medications may include antianxiety or antidepressant drugs, and anticholinergic agents before meals to help control symptoms. Antidepressants may reduce visceral hypersensitivity at the level of the visceral afferent fibers. The fact that the enteric nervous system and the brain use the same chemicals and hormones may explain why low doses of antidepressants designed to affect the brain can improve certain digestive diseases.

Newer drugs used in IBS management include serotonin-modulating agents that inhibit the action of serotonin (5-hydroxytryptamine or 5-HT) in the gut. Serotonin, a neurotransmitter found in the gut (and in the brain), appears to be a common link involved in GI motility, intestinal secretion, and pain perception.

The GI tract contains approximately 90% to 95% of the body's serotonin. Serotonin release in the bowel subsequent to bowel distention has been associated with changes in GI motility, secretion, and possibly pain transmission.³⁸ Research continues to search for targeted medications that can be individualized to each person based on his or her particular manifestation of this condition.

Alternative therapy, including peppermint oil (capsule form) and other natural substances (e.g., chamomile, rosemary, valerian, ginger, turmeric), has antispasmodic effects and may relieve cramping. Probiotic treatment with *Lactobacillus* and *Bifidobacterium* may help to alter the microbial flora of the intestinal tract and ease the symptoms of IBS.

PROGNOSIS. IBS is not a life-threatening disorder, and prognosis is good for controlling symptoms through diet, medication, regular physical activity, and stress management. No known relationship exists between IBS and malignancy of the bowel.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-14

Irritable Bowel Syndrome

Regular physical activity helps relieve stress and assists in bowel function, particularly in people who experience constipation. The therapist should encourage anyone with IBS to continue with the prescribed rehabilitation intervention program during symptomatic periods.

Therapists must be alert to the person with IBS who has developed breath-holding patterns or hyperventilation in response to stress. Teaching proper breathing is important for all daily activities, especially during exercise and relaxation techniques.

For therapists working in the field of women's health, a known correlation exists between a history of (childhood or adult) emotional or sexual abuse and neurologic and functional (or organic) GI disorders.^{5,120}

The first step in effective intervention is recognizing these individuals. Many articles are available to help the therapist incorporate this type of evaluation, including the American Physical Therapy Association *Guidelines for Recognizing and Providing Care for Victims of Domestic Violence*.^{7,29,75} More must be done in this area to offer therapists guidelines and intervention management skills when sexual abuse has been part of the history.

Diverticular Disease

See also the section on Meckel's diverticulum.

Definition and Incidence

Diverticular disease is the term used to describe diverticulosis (uncomplicated disease) and diverticulitis (disease complicated by inflammation). Diverticulosis refers to the presence of outpouchings (diverticula) in the wall of the colon or small intestine, a condition in which the mucosa and submucosa herniate through the muscular layers of the colon to form outpouchings containing feces (Fig. 16-16).

When food particles or feces become trapped in the diverticula and the pockets become infected and inflamed, then diverticulitis can develop. This acquired deformity of the colon is rarely reversible and usually asymptomatic. The most common site is the sigmoid colon (95% of cases) because of the high pressures in this area required to move stool into the rectum, but any segment of the colon may be involved.

Diverticular disease is common and increasing in incidence in westernized countries because of low-fiber diets, and is present in approximately 10% of people in the



Figure 16-16

Multiple diverticula in resected section of the colon. Weak spots in the muscle layers of the intestinal wall permit the mucosa to bulge outward (herniate) into the pelvic cavity. (From Rosai J: Ackerman's surgical pathology, ed 7, St Louis, 1989, Mosby.)

United States. Incidence increases after age 60 and in obese individuals. The disease is present in as many as half of all adults over 65 years in the United States.

Etiologic and Risk Factors

Causes of diverticular disease include atrophy or weakness of the bowel muscle, increased intraluminal pressure, obesity, and chronic constipation. Disturbances of the pelvic floor in women are particular risk factors for constipation leading to diverticular disease.

Some people are born with diverticula, probably resulting from an inherited defect in the muscular wall of the intestines, but the majority of people who have diverticulosis develop it with age, indicating that both heredity and lifestyle play a role. Use of NSAIDs and acetaminophen, the active ingredient in Tylenol, may be associated with diverticular disease. Further studies are under way to verify this potential link.⁴

The current hypothesis as to the primary cause (and major risk factor) of diverticular disease is a low-fiber diet, which decreases stool bulk and predisposes individuals to constipation. The subsequent increased intraluminal pressure pushes the mucosa through connective tissue, weakening bowel muscle.

In addition to a low-fiber diet as a major risk factor, ingestion of poorly chewed or poorly digested foods that can block the opening of the diverticulum and cause inflammation also may contribute to the development of diverticular disease. Such foods as corn and popcorn, and foods with tiny seeds (e.g., cucumbers, tomatoes, berries) once considered part of the problem are now actually part of the solution. Fruits and vegetables, including those with small seeds, are good sources of fiber and should not be avoided.¹⁴⁴

Pathogenesis

Diverticula form at weak points in the colon wall, usually where arteries penetrate the muscularis to nourish the mucosal layer. Changes in the connective tissue of the gut wall contribute to the diminished resistance of the intestinal wall.

The circular and longitudinal muscles (*taeniae coli*) surrounding the diverticula become thickened and hypertrophy as a result of age-related changes in collagen and progressive deposition of elastin in longitudinal muscle. They function to propel luminal contents in an oral-to-anal direction and are not designed to pump out the contents of side pockets. If anything, when they contract, the colonic muscles may act as valves on the mouth of the diverticula, holding the contents in rather than evacuating them out.

Increased contraction of these muscles is required when hard, compact stools form in the absence of adequate fiber, but decreased neurons in the distal colon associated with aging result in disorders of neuromuscular function and impaired evacuation, compounding the problem. Increased intraluminal pressure then increases the herniation. Over a person's lifetime, diverticula may increase in number and size but only rarely extend to other portions of the colon.

Diverticulitis occurs when undigested food blocks the diverticulum (blind outpouching), decreasing blood

supply to the blood vessels penetrating the internal circular layer of bowel muscularis.

The inflamed area becomes congested with blood and may bleed. Diverticulitis can lead to perforation when the trapped mass in the diverticulum erodes the bowel wall. Chronic diverticulitis can result in increased scarring and narrowing of the bowel lumen, potentially leading to obstruction. Perforated diverticula provide an opening through which bacteria can enter, leaving the bowel at risk for a bacterial invasion into the diverticulum with subsequent inflammation and infection.

Clinical Manifestations

Diverticular disease is asymptomatic in 80% of people affected. More commonly, these individuals report passing fresh blood and clots and experience a sense of urgency to defecate.

When diverticula become inflamed, diverticulitis develops, and the person experiences episodic or constant, severe abdominal pain located in the left quadrant or midabdominal region, often with extension into the back. The mechanism of pain is probably increased tension in the colonic wall with an associated rise in intraluminal pressure.

Other symptoms may include pelvic pain in women, constipation alternating with diarrhea, increased flatus, fever, sudden onset of painless rectal bleeding, and anemia in the presence of chronic blood loss. Eating and increased intraabdominal pressure increase pain (see Box 16-1), whereas temporary partial or complete relief may follow a bowel movement or passage of flatus.

MEDICAL MANAGEMENT

DIAGNOSIS. Barium enema studies show the characteristic diverticula; colonoscopy and sigmoidoscopy also may provide diagnostic information, and a CT scan may sometimes reveal the inflamed colon segment. No specific laboratory tests exist, but results of fecal examination for occult blood may be positive, and anemia may be identified. Stool cultures may be used to exclude bacterial or parasitic infections.

TREATMENT. Insights on the pathophysiology and mechanisms of neural injury may lead to more specific treatment in the future (e.g., serotonergic agents and neurotrophins), but for now, treatment is directed at relieving symptoms and preventing diverticulitis. This is accomplished primarily through dietary changes with adherence to a high-fiber diet; prevention of constipation with adequate fluid intake, bran, and bulk laxatives; and exercise during periods of remission.

Acute diverticulitis may require antibiotics and complete rest of the colon accomplished by nasogastric tube feedings and parenteral fluid administration until the inflammatory process has been resolved. Bleeding is a rare complication of diverticulosis and usually is self-limited; but in approximately 10% of people with bleeding diverticula, the hemorrhage is severe enough to require hospitalization, blood transfusion, and possible surgical removal of the affected part of the colon and possible temporary colostomy.

PROGNOSIS. Prognosis is good for the person with known diverticular disease, especially when prevention of diverticulitis is possible by consuming a high-fiber diet, chewing food carefully, and avoiding indigestible foods. If the diverticulum is not blocked and infected or inflamed (diverticulitis), the person may be asymptomatic. If the trapped fecal material (fecaliths) does not liquefy and drain from the diverticulum, diverticulitis develops.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-15

Diverticular Disease

Exercise is an important treatment component during periods of remission. The therapist is instrumental in helping establish an appropriate exercise program. Throughout all activity and exercise, clients with diverticular disease must be careful to avoid activities that increase intraabdominal pressure (see Box 16-1) to avoid further herniation. The therapist can provide valuable information regarding appropriate body mechanics and techniques to reduce intraabdominal pressure for all activities.

Back pain can occur as a symptom of this disease. Anyone with back pain of nontraumatic or unknown origin must be screened for medical disease, including possible GI tract involvement. If infection occurs and penetrates the pelvic floor or retroperitoneal tissues (i.e., those organs outside the peritoneum such as the kidneys, pancreas, colon, and rectum), abscesses may result, causing isolated referred hip or thigh pain.

A variety of objective test procedures may be employed by the therapist to assess for iliopsoas abscess formation, including palpation of McBurney's point (see Fig. 16-22), assessment of the pinch-an-inch test (see Fig. 16-24), the iliopsoas muscle test, the obturator test, and palpation of the iliopsoas muscle (see Figs. 16-14 and 16-15).

Neoplasms **Intestinal Polyps**

A growth or mass protruding into the intestinal lumen from any area of mucous membrane can be termed a *polyp*. Polyps are either *neoplastic* or *nonneoplastic*. Adenomatous (benign neoplastic) polyps of the intestine usually develop during middle age, and more than two thirds of the population over 65 years old has at least one polyp. Until a polyp becomes large enough to obstruct the intestine, no symptoms are discernible. Early symptoms may be lower abdominal cramping pain, diarrhea with rectal bleeding, and passage of mucus.

Nonneoplastic polyps include adenomatous and hyperplastic and are usually asymptomatic, although inflammatory polyps may present with symptoms of the underlying IBD usually present (e.g., UC or CD). Treatment is not always required; polyps associated with rectal bleeding may be removed using a proctoscope.

Adenomatous polyps may be a risk factor for the development of adenocarcinomas (colorectal cancer); therefore, regardless of the clinical manifestations, adenomatous polyps are removed (by polypectomy, usually performed through a sigmoidoscope or colonoscope; large polyps may require removal by laparotomy). Hyperplastic polyps do not progress to become cancerous.

Contrary to previous beliefs, studies have now shown that high-fiber diets do not reduce the risk of recurrent colorectal adenomas in people who have had at least one precancerous polyp already removed. Evidence suggests that high-fiber diets can slow the development of adenomas.^{88,128,129}

Benign Tumors

The most common benign tumors of the small intestine are adenomas, leiomyomas, and lipomas. Benign tumors of the small intestine rarely become malignant and may be symptomatic or may be incidental findings at operation or autopsy.

Adenomas account for 25% of all benign bowel tumors and are usually asymptomatic, although bleeding and intussusception (see the section on Mechanical Obstruction in this chapter) are occasional complications.

Leiomyomas are smooth muscle tumors that can occur at any location in the intestine but are most common in the jejunum. They usually are associated with bleeding when they protrude into the lumen, where necrosis of tumor tissue and ulceration of the mucosa occur. Obstruction is uncommon, but intussusception or volvulus (Figs. 16-17 and 16-18) may occur. Surgical removal of large leiomyomas is recommended because of the bleeding and the increased risk of malignancy with increasing size.

Lipomas are fatty tumors that occur throughout the length of the small intestine but occur most frequently in the distal ileum. When symptomatic, the presenting symptom is obstruction resulting from tumor size, causing intussusception. Other complications may include ulceration and bleeding of the overlying mucosa and subsequent sequelae.

Malignant Tumors

The most common malignant tumors of the small intestine are metastatic through direct extension from adjacent organs (e.g., stomach, pancreas, colon). Adenocarcinoma

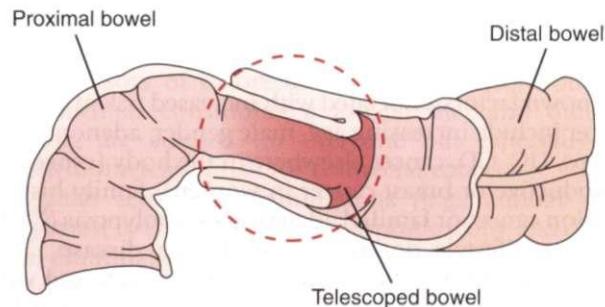


Figure 16-17

Intussusception. A portion of the bowel telescopes into adjacent (usually distal) bowel.

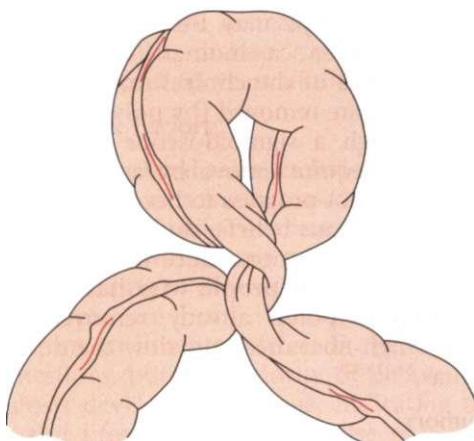


Figure 16-18

Volvulus. The intestine twists, causing obstruction and ischemia.

and primary lymphoma account for the majority of bowel malignancies. Other types of colorectal cancer, including melanoma, fibrosarcoma, and other types of sarcoma, are rare and are not discussed further in this book.

Adenocarcinoma

Overview and Incidence. Adenocarcinoma of the colon and rectum (colorectal cancer) is the second leading cause of cancer death (after lung cancer) among American men and women combined.⁷³ It is the second leading cause of cancer death among men in the United States and third in women after lung and breast cancer, except after age 75 when colorectal cancer is responsible for more deaths than breast cancer.

Incidence increases with age starting at 40 years, and the disease occurs slightly more often in men and in populations of high socioeconomic status, possibly owing to dietary factors. African Americans have the highest incidence of colorectal cancer among all racial groups, with death rates about 30% higher than for Caucasians. This disparity is most likely due to differences between African Americans and whites in screening rates, early detection, and intervention.^{6,117,121}

Colorectal tumors can be staged using the Dukes classification (Box 16-2). Overall incidence and mortality rates are on the decline, possibly indicating that advances in diagnosis and treatment are making an impact.

Etiologic and Risk Factors. The cause of colon cancer is unknown, although a number of environmental and familial factors have been considered. Genetic syndromes are more likely to occur before age 40 and make up less than 6% of all colorectal cancers.

Known factors associated with increased risk of colonic cancer include increasing age, male gender, adenomatous polyps, UC, CD, cancer elsewhere in the body (especially reproductive or breast cancer in women), family history of colon cancer or familial adenomatous polyposis (FAP), sedentary lifestyle, and immunodeficiency disease.

Anyone who has first-degree relatives diagnosed with colon or rectal adenoma is twice as likely to develop colon cancer as those with no history of such cancer in the immediate family. The risk is even higher if the relative was under 50 years of age at the time of diagnosis.

Box 16-2

DUKES' STAGING OF COLORECTAL TUMORS

- Stage A: Cancer limited to the bowel wall; mucosal involvement only
- Stage B: Cancer extending through the bowel wall; local invasion without penetration of the serous membrane
- Stage C: Involvement of regional lymph nodes, with or without extension into the bowel wall
- Stage D: Distant metastases regardless of primary tumor size

Cigarette smoking may increase the risk of colorectal cancer, but data collected suggest a lag time of at least 35 years for tobacco-induced tumors to develop. Excessive alcohol consumption may possibly increase risk.⁸⁶

Geographic distributions of highest incidence coincide with regional diets low in fiber and high in animal fat and protein; people who emigrate tend to acquire the risk characteristics of their new environment. Eating large amounts of red or processed meat over a long period of time can increase colorectal cancer risk, but the risk from obesity and lack of exercise (inactivity) is even greater.²⁴

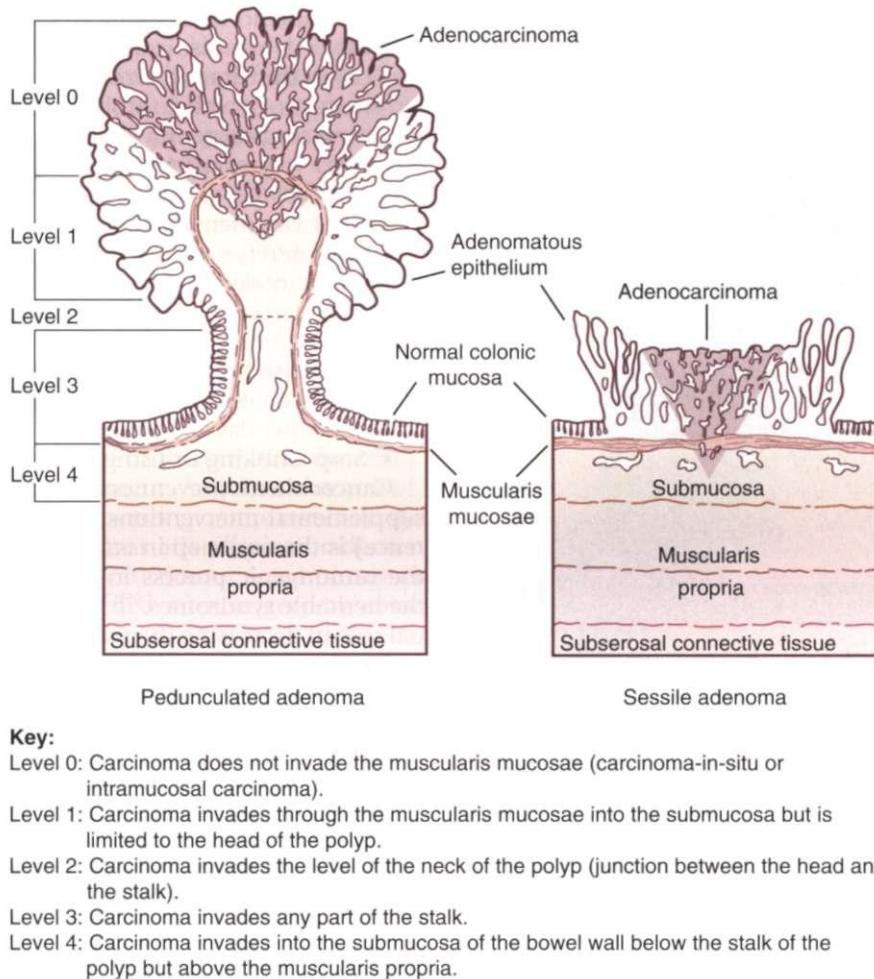
Calcium from dairy products or supplements may help reduce the risk of colon cancer, but scientists do not know what the mechanism of this action is yet. About 75% of all colorectal cancer occurs in people with no known predisposing factors; for such individuals, the lifetime risk of developing this type of cancer is about 5%.

Colon cancer incidence and mortality rates are lower in females compared with males, and numerous epidemiologic studies suggest that estrogen replacement therapy reduces cancer risk in postmenopausal women.²¹ It is not clear how estrogen acts to reduce fatal colon cancer risk. One theory is that it lowers the concentration of bile acids, perhaps creating an environment that is hostile to the growth of cancer cells in the colon; another is that estrogen acts directly on the lining of the colon to suppress tumor growth.

Pathogenesis. Most colorectal cancers have a long pre-invasive phase, growing slowly during invasion. More than 95% of colorectal cancers are adenocarcinomas that arise from glandular cells that line the mucosa of the colon and rectum (Fig. 16-19).

This disease has a well-defined sequence of events. The stepwise progression of this cancer often occurs over many years and begins with a polyp, a collection of cells on the lining of the large intestine. At first, there is an aberrant proliferation or hyperplasia of cells, which leads to a particular type of polyp called benign adenoma, containing abnormal but not malignant cells. Then the cells transform to carcinoma in situ, and finally to metastatic carcinoma. This process is thought to be due to a series of genetic mutations within the cells.⁴² Some benign polyps may regress and disappear over time, but most continue to undergo changes that transform them into malignant tumors (Fig. 16-20).

The accumulation of molecular genetic alterations involves activation of oncogenes, inactivation of tumor suppressor genes, and abnormalities in genes involved in

**Figure 16-19**

Adenomatous polyps (adenomas), growths on the inner surface of the colon and rectum. Most colorectal cancers probably arise from adenomatous polyps, which may be tubular, called *pedunculated* (the lesion is on a stalk), or *sessile* (without a stalk). Adenomatous polyps should be considered precursors of cancer and can be classified according to the depth of invasion. [From Haggitt RC, Glotzbach RE, Soffer EE: Prognostic factors in colorectal carcinomas arising in adenomas: implications for lesions removed by endoscopic polypectomy, *Gastroenterology* 89:328-336, 1985.]

DNA repair (see Chapter 9). Hormones stimulating the rate of cell turnover and excessive free radical formation and oxidation also may play a role in colon carcinogenesis.⁸³

Untreated tumors can grow into the wall of the colon. The lymphatic channels are located underneath the muscularis mucosae so that the lesions must extend across this layer before metastasis can occur.

Clinical Manifestations. Colon carcinoma has few early warning signs, as is the case with esophageal and stomach cancers. When symptoms do present, they present according to whether the lesion is in the ascending colon, descending colon, or transverse colon.

A persistent change in bowel habits is the single most consistent symptom for either side. Bright red blood from the rectum is a cardinal sign of colon cancer, but the latter must be differentiated from diverticulosis, which is also a common cause of bright red blood without pain.

Other symptoms may include persistent stomach pain, gas, diarrhea, or constipation. Many cases of colon cancer are asymptomatic until metastasis has occurred. Compli-

cations include intestinal obstruction, bleeding, perforation, anemia, ascites, and distant metastases, to the liver most commonly but also to the lungs, bone, and brain.

MEDICAL MANAGEMENT

PREVENTION. Evidence exists that reductions in colorectal cancer morbidity and mortality can be achieved through detection and treatment of early-stage cancer and the identification and removal of adenomatous polyps, the precursors of colorectal cancer. Lifestyle modifications such as consuming a low-fat diet and quitting smoking are advised for everyone but especially for anyone at increased risk for colorectal cancer.

Regular screening examinations (e.g., annual stool sample test to check for blood, sigmoidoscopy and digital rectal examination every 5 to 10 years) are recommended for adults over 50 years and for younger people with a family history of the disease. Unfortunately, less than half of U.S. adults 50 years old or older have been tested. Current guidelines for screening colonoscopy do not specify an age limit. Questions have been raised about

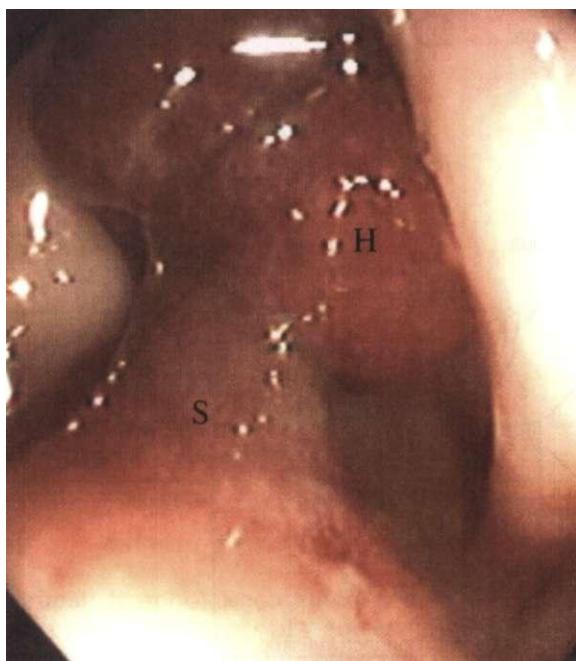


Figure 16-20

Large pedunculated polyp in the rectum. The stalk (*S*) itself is benign, with the head (*H*) containing the adenomatous tissue. The polyp was removed safely in a one-step endoscopic procedure. (From Goldman L: *Cecil textbook of medicine*, ed 22, Philadelphia, 2004, Saunders. Courtesy of Pankaj Jay Pasricha.)

the cost effectiveness and gains in life expectancy for screening people 80 years of age and older.⁹⁰

The American Cancer Society recommends more intensive surveillance for individuals at higher risk for colorectal cancer. These individuals include anyone with a history of adenomatous polyps, personal previous history of curative-intent resection of colorectal cancer, family history (first-degree relative) of colorectal cancer before age 60, personal history of IBD of significant duration, and family history or genetic testing indicating hereditary syndromes such as nonpolyposis colorectal cancer or FAP.¹³⁹

Colonoscopy is the primary screening test, which involves inserting a snakelike scope into the rectum and large bowel to detect tiny polyps before they develop into cancer. The invasive nature of this test prevents participation in the colorectal cancer screening process so that investigators are working to develop alternative screening tests.

Advances in CT technology and computer capabilities have contributed to the development of a new imaging modality for colorectal lesions called *CT colonography* or *virtual colonoscopy*. This is a rapid, minimally invasive, and painless procedure in which a tiny probe is inserted just 4 cm into the rectum. This screening technique may not be sensitive enough and does not identify flat lesions at all, requiring continued development of the technology before it can be applied as a standard procedure.^{124,141}

Alternatively, technology has made it possible to use a tiny camera that can be swallowed for a virtual endoscopy that is less invasive but may not be as complete, since the camera's field of view is only 140 degrees,

leaving some portions of the GI tract in blind spots. Food and other debris also can obscure lesions from view. In rare cases, the vitamin-sized capsule may get obstructed by strictures or other problems within the intestines, requiring surgical removal.

Other preventive strategies include the following^{31,86,138}:

- Increase intensity and duration of physical activity and exercise
- Limit intake of red and processed meat
- Take recommended levels of calcium with vitamin D
- Eat more fruits and vegetables
- Avoid excess alcohol consumption (no more than 1 drink per day in women or 2 drinks per day in men)
- Stop smoking or using tobacco products

Cancer chemoprevention (pharmacologic, nutritional supplemental interventions applied before cancer occurrence) is the next step in attempting to inhibit or reverse the tumorigenic process in colorectal cancer, especially the heritable syndromes.^{65,126} Nutrients that play a potential role in decreasing the risk of colorectal cancer include antioxidants such as vitamins A, C, and E, which block the formation of oxygen free radicals that damage DNA and trigger malignant transformation of cells. Calcium intake may reduce the colon's mucosal exposure to carcinogens by binding with bile salts and fatty acids.

Antioxidant supplementation reduces the risk of colonic polyps, a risk factor for colorectal cancer. Folate needed for DNA and ribonucleic acid (RNA) synthesis has been shown to reduce the risk of colon polyps and colon cancer. Understanding the neoplastic events at the molecular level may provide more definitive preventative information in the coming decade.

Individuals at increased risk for colorectal cancer should talk with their physicians about the preventive use of aspirin and NSAIDs, which has shown some promise in preventing colorectal cancer. The mechanism by which these drugs reduce the risk of colorectal cancer is the ability to block angiogenesis in tumors and enhance the effects of chemotherapy and radiation. Another possible mechanism of this benefit is decreased prostaglandin production, achieved through inhibition of COX activity.^{92,150}

DIAGNOSIS. Carcinoma of the colon should be suspected in anyone over the age of 40 who presents with occult blood in the stool, iron deficiency anemia, overt rectal bleeding, or alteration in bowel habits, especially if associated with abdominal discomfort or any of the risk factors mentioned earlier. Physical examination of the abdomen to detect liver enlargement and ascites is followed by palpation of appropriate lymph nodes.

Diagnostic procedures include rectal examination, sigmoidoscopy, proctoscopy, colonoscopy with biopsy of lesions, CT scan, and barium enema studies. Virtual colonoscopy (also known as *CT colonography*), a series of CT images of the intestine, has advanced enough to become sensitive and accurate enough for use with many but not all people.¹¹⁴

Laboratory diagnostic tests may include a screening test for occult fecal blood and a blood test for carcinoem-

bryonic antigen (CEA), detected in some individuals with colorectal carcinoma. CEA is one of the most widely used tumor markers worldwide, primarily in GI cancers, especially colorectal malignancy. It is of little use in detecting early colorectal cancer, but high preoperative concentrations of CEA correlate with adverse prognosis, and serial CEA measurements can detect recurrent cancer in asymptomatic clients.⁴⁵

Staging. The tumor-node-metastasis (TNM) staging system of the American Joint Committee on Cancer (AJCC) is the standard for colorectal cancer. The TNM system incorporates both clinical and pathologic staging approaches and can be applied to the preoperative evaluation of affected individuals.³³

TREATMENT. Surgical removal of the tumor is the mainstay of colorectal cancer treatment. Adjuvant chemotherapy may be administered, depending on the results of the staging process, to treat metastatic disease; radiation therapy is helpful for rectal carcinoma. A temporary or permanent colostomy is needed after surgery by some people.

Using the AJCC/TNM staging, stage 0 disease requires local or regional excision of the polyp with wide margins. Stage 1 cancer is treated the same way, but the individual may need bowel resection and reanastomosis. Adjuvant therapy has not been proven effective in improving survival rates compared with surgical excision alone.

Stage 3 disease requires surgical excision and removal and biopsy of regional lymph nodes. Regional metastasis has occurred at this stage, requiring additional regimens of chemotherapy and/or radiation therapy. Stage 4 cancer is accompanied by systemically metastasized disease requiring a more comprehensive treatment regimen to address local, regional, and systemic disease.⁵⁸

Radiation therapy may be given before surgery to reduce the tumor size or alter the malignant cells to prevent tumor survival after surgery. Tumors in the distal rectum may require resection of the entire rectum with subsequent permanent colostomy. Given the link between adenomatous polyps and cancer, emphasis is on prevention through screening for colonic polyps and carcinomas.

The use of monoclonal antibodies (MABs such as cetuximab [Erbitux], bevacizumab [Avastin], panitumumab [Vectibix]) for the treatment of colorectal cancer is under investigation. These MABs bind to vascular endothelial growth factor receptors and prevent the formation of new blood vessels (angiogenesis) supplying a tumor, thus effectively starving tumor cells. MABs may enhance the effects of chemotherapy and enhance radiation-induced apoptosis. Studies are underway to determine the benefits and optimal timing for use in metastatic and nonmetastatic colorectal cancer.^{55,87,96} Use of bevacizumab for early-stage colon cancer in phase III clinical trials was stopped in February 2006 after the deaths of four people.

PROGNOSIS. Colorectal cancer survival is related closely to the clinical and pathologic stage of the disease at diagnosis. Colorectal cancer detected at an asymptomatic phase has a more favorable prognosis compared to later

detection. Polyps containing invasive carcinoma represent about 5% of all adenomas. Malignant polyps constitute a form of early carcinoma that can be cured by endoscopic removal. The risk of an unfavorable outcome increases with the presence of lymph node or local metastases or local recurrence.³³

Approximately 65% of cases present with advanced disease. The 5-year survival rate for cancer limited to the bowel wall at the time of diagnosis approaches 90% but drops to 35% to 60% when lymph nodes are involved and less than 10% with metastatic disease to distant organs such as the liver.

Local recurrence can occur if special operative precautions to prevent implantation of malignant cells are not followed. CEA is a marker for recurrent tumor and should be monitored every 6 months to improve survival. Routine follow-up colonoscopy also is recommended.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-16

Adenocarcinoma of the Colon and Rectum

Preferred Practice Patterns may vary depending on individual comorbidities and clinical presentation. For example, the client who has a history of corticosteroid therapy or the woman who has had surgically induced menopause prematurely may be at risk for skeletal demineralization (Pattern 4A: Primary Prevention/Risk Reduction for Skeletal Demineralization).⁵⁸

Impaired posture (Pattern 4B) can occur as a result of adaptive shortening of the abdominal musculature due to extensive surgical disruption and pain contributing to a stooped posture and inability to lie completely supine. Such a condition can place increased stress on the muscles of the lower back and alter body mechanics, placing the individual at increased risk for injury during lifting and risk of decreased function in other activities.⁵⁸

The removal of lymph nodes from the abdominal/pelvic area can put a client at increased risk of lymphedema. The therapist can be instrumental in providing education regarding lymphedema risk and prevention. Many individuals are deconditioned before beginning cancer treatment. The benefits of exercise in general and especially for those recovering from cancer treatment have been reported in the literature and are summarized in Chapter 9.

Metastases

Tumors of the rectum can spread through the rectal wall to the prostate in men and the vagina in women. Prostate involvement can cause dull, vague, aching pain in the sacral or lumbar spine regions. See Special Implications for the Therapist: Prostate Cancer in Chapter 19. Pelvic, hip, or low back pain may occur with extension to the vagina in women.

Systemic and pulmonary metastases occur through the hemorrhoidal plexus, which drains into the vena cava. Subsequent chest, shoulder, arm, or back pain can occur, usually accompanied by pulmonary symp-

Continued.

toms; see also Special Implications for the Therapist: Lung Cancer in Chapter 15.

Liver metastasis occurs after invasion of the mesenteric veins from the left colon or the superior veins from the right colon, which empty into the portal circulation; see also the section on Hepatic Encephalopathy in Chapter 17. Any time a person with low back pain reports simultaneous or alternating abdominal pain at the same level as the back pain, or GI symptoms associated with back pain, a medical referral is required.

Anemia caused by intestinal bleeding associated with colon cancer requires special consideration. See Special Implications for the Therapist: The Anemias in Chapter 14.

Rehabilitation

Many factors can influence whether an individual with colorectal cancer will be referred to rehabilitation services. The client's tolerance to pain, ability to recover, presence of other comorbidities, and even the physician's perception of physical debilitation may play a role in the decision to refer.³⁶

Complications and side effects arising from comprehensive treatment can produce impairments for which physical therapy intervention can improve quality of life, tolerance of cancer treatment, and recovery of function.³⁸

The therapist may be involved in the rehabilitation of some clients with (colorectal) cancer to improve function and mobility after abdominal surgery or other medical treatments.³²

Individuals who have had laparoscopic surgery may be able to tolerate a more aggressive approach to rehabilitation and recovery of function.⁷⁷

Movement to stimulate the gastric system and help the flow of the abdominal contents may begin as early as the same day as surgery, with ambulation every 4 to 6 hours on the first day.⁴² The therapist may be called upon to evaluate the client's needs and to instruct the staff and family in ambulation if assistance is needed.

Many people who have a colectomy without a colostomy bag have to relearn bowel control, since the pelvic floor muscles are often manipulated and weakened during surgery. A program of pelvic floor rehabilitation, possibly including biofeedback and electrical stimulation, can help restore pelvic floor function.¹⁶

development of primary intestinal lymphoma of the MALT type.¹⁰⁴

An apparent causal relationship exists between lymphoma and chronic inflammatory intestinal conditions such as celiac disease, possibly because of the persistent activation of lymphocytes in the bowel. The risk of intestinal lymphoma is also increased in conditions of immunodeficiency after treatment with immunosuppressive drugs. The mechanisms by which malignant transformation occurs remain under investigation.

Signs and symptoms may include chronic abdominal pain, diarrhea, clubbing of the fingers, weight loss, and occult bleeding. Intestinal obstruction, intussusception, and perforation with massive hemorrhage and peritonitis are possible complications.

Clinical diagnosis is by radiographic studies of the small intestine, CT or magnetic resonance imaging (MRI) scans, and endoscopic ultrasonography.

Combined-modality therapy with chemotherapy followed by radiation therapy is still used in many centers for large cell lymphoma, and radiation therapy alone is used for later stages of MALT lymphoma. But chemotherapy alone may be sufficient to treat early-stage (1 and 2) high-grade MALT.¹⁰

With these improved treatment modalities, primary lymphoma has become a highly curable disease. Surgical resection for small, local involvement is possible, but the role of surgery has come into question with increasing knowledge of intestinal lymphoma pathogenesis and with the new therapeutic approaches described.

The overall prognosis is much less favorable when extraintestinal spread occurs (5-year survival rate is less than 10%). When the disease is localized and confined to the small intestine, it does not recur after surgical removal in more than half the cases, but the disease is usually too diffuse to permit surgery.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-17

Primary Intestinal Lymphoma

Special considerations relate to any complications present with this condition, such as anemia from intestinal bleeding or complications associated with chemotherapy and radiation therapy. See Special Implications for the Therapist: Oncology in Chapter 9.

Obstructive Disease

Anything that reduces the size of the gastric outlet, preventing the normal flow of chyme and delaying gastric emptying, can cause an obstruction of the bowel. Delayed gastric emptying may be secondary to obstruction of the stomach or secondary to an inability to generate effective propulsive forces (peristalsis). Obstruction of the intestines can occur as a result of (1) organic disease, (2) mechanical obstruction, or (3) functional obstruction (Table 16-7).

Primary Intestinal Lymphoma. Primary intestinal lymphoma originates in nodules of lymphoid tissue within the bowel wall and accounts for 15% of small bowel cancers in the United States and two thirds of such cancers in undeveloped countries.

Primary lymphoma is the most common form of presentation for GI lymphomas, and the stomach is one of the most frequent sites of extranodal lymphoma. The cause of primary lymphoma is unknown, although chronic *H. pylori* infection is associated strongly with the

Table 16-7 Causes of Intestinal Obstruction

Organic Disease	Mechanical	Functional
Ulcers	Postoperative adhesions	Postoperative Drugs
Strictures	Intussusception	Anticholinergics
Gallstones	Volvulus	Opiates
Neoplasms		Electrolyte imbalance
Granulomatous processes	External hernia	Metabolic conditions
Viral infections		Diabetes mellitus
Scleroderma		Hypoparathyroidism
Polymyositis		Pregnancy
Brainstem tumors		Vagotomy
Gastroesophageal reflux		Anorexia nervosa
Vascular occlusion		Spinal cord injury
Mesenteric infarction		Vertebral fracture
Abdominal angina		Pneumonitis

Many of the organic and functional classes do not cause obstruction as much as they cause ileus with reduced or absent peristalsis. The presentation is the same but the cause and treatment are different.

Organic Disease

Clinical Manifestations. Distention develops accompanied by colicky, cramping pain and tenderness in the perumbilical area progressively becoming constant. Vomiting occurs as a reflex associated with the waves of pain. Constipation develops into obstipation (intractable constipation) as fecal obstruction builds up in the distal bowel.

Propulsion of gas through the intestines causes a rumbling noise called *borborygmus*, and the person is aware of intestinal movement. Constitutional symptoms such as low-grade fever, perspiration, tachycardia, and dehydration may accompany this condition. The affected individual is restless, changing position frequently because of the constant pain.

Signs of dehydration, hypovolemia, and metabolic acidosis may be seen within 24 hours of complete obstruction. Impaired blood supply to the bowel results in necrosis and strangulation. Strangulation is characterized by fever, leukocytosis, peritoneal signs, or blood in the feces. Further complications may develop, such as perforation, peritonitis, and sepsis. In debilitated persons, distention of the abdomen can be severe enough to compress the diaphragm, decreasing lung compliance and resulting in atelectasis and pneumonia.

Pathogenesis. Gases and fluids accumulate proximal to the obstruction, causing abdominal distention. The body's response to distention is temporarily increased peristalsis as the bowel attempts to force the material through the obstructed area. The distention also decreases the intestine's ability to absorb water and electrolytes, which are further imbalanced by vomiting.

If the obstruction is at the pylorus or high in the small intestine, metabolic alkalosis develops as a result of vomiting and excessive loss of hydrogen ions. With prolonged obstruction or obstruction lower in the intestine, metabolic acidosis is more likely to occur, because bicarbonate from pancreatic secretions and bile cannot be reabsorbed.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Diagnosis requires differentiation of obstruction from other acute abdominal conditions such as inflammation and perforation of a viscus, renal or gallbladder colic, obstruction from other causes, vascular disease, and torsion of an organ, as occurs with an ovarian cyst. Abdominal radiography is the most useful diagnostic tool, and laboratory findings may reveal electrolyte disturbances associated with vomiting and dehydration.

Supportive care to alleviate pain and symptoms and to facilitate passage of flatus and feces is instituted toward the goals of restoration of bowel function and prevention of surgical intervention. Intestinal intubation (insertion of a tube into the intestinal lumen to decompress the lumen and break up the obstruction) may relieve obstruction without surgery. Complete obstruction of the intestine is surgically resected; immediate surgery is required in the case of intestinal strangulation. Prognosis varies with the underlying cause; strangulation increases the mortality rate to 25%.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-18

Organic Obstructive Disease

The therapist may see this client in an acute care setting for ambulation after the obstructive incident has been treated. Dehydration is the primary concern, requiring monitoring of vital signs and symptoms (e.g., dry lips, dry hands, headache, brittle hair, incoordination, disorientation; see Box 5-8) along with encouragement of fluid intake throughout therapy. Movement and activity, along with deep-breathing exercises, help promote abdominal relaxation and restore bowel function.

Mechanical Obstruction

Postsurgical adhesions are by far the most common cause of mechanical small bowel obstruction, followed by strangulated hernia, malignancy, CD, and, more rarely, volvulus. In small bowel obstruction, the intestine dilates above the blockage due to an accumulation of GI secretions and swallowed air. Vomiting is often the first symptom of proximal small bowel obstruction.

Small bowel distention can lead to lymphatic compression and bowel wall lymphedema. The increasing intraluminal pressure can result in reduced venous and arterial blood flow and severe fluid loss, dehydration, electrolyte imbalance, hypovolemic shock, and even death.

Adhesion. Adhesions are the most common cause of small and large intestine obstruction caused by fibrous scars formed after abdominal surgery. These fibrous bands of scar tissue can loop over the bowel, either mechanically obstructing the bowel by constricting it or becoming an axis around which the bowel can twist (volvulus). Peritonitis may cause obstruction by kinking or angulating the bowel or by directly compressing the lumen.

Intussusception. Intussusception is a telescoping of the bowel on itself; that is, one part of the intestine prolapses into the lumen of an immediately adjacent section (see Fig. 16-17). A reported link between the rotavirus vaccine and intussusception in infants and young children is under investigation.¹⁰⁶

In adults the leading point of an intussusception is often a lesion in the bowel wall, such as Meckel's diverticulum or a tumor. Once the leading point is entrapped, peristalsis drives it forward, dragging the mesentery into the enveloping lumen. As the two walls of the intestine press against each other, inflammation, edema, and decreased venous return and venous stasis occur. Untreated, necrosis and gangrene develop.

Clinical manifestations in adults are as listed for obstruction; complications in children include prolonged ischemia and subsequent necrosis with eventual perforation, peritonitis, and sepsis. Usually, diagnostic testing includes rectal examination, abdominal radiography, and barium enema. The barium enema may be part of the diagnosis and treatment, as the force of the flowing barium is usually enough to push the invaginated bowel into its normal position. If the hydrostatic reduction is unsuccessful (in 30% to 40% of cases), surgical reduction of the intussusception and resection of any nonviable intestine are performed. The prognosis for children and adults with this condition is good if treated.

Volvulus. Volvulus is a torsion of a loop of intestine twisted on its mesentery, kinking the bowel and interrupting the blood supply (see Fig. 16-18). The cause of this phenomenon is usually a congenital abnormality such as a malrotation of the bowel that allows excess mobility of the bowel loops and predisposes the intestine to volvulus. Other focal points serving as a stationary object about which the bowel twists are tumors or Meckel's diverticulum.

Treatment to decompress the bowel by inserting a long tube to release the pressure against the proximal end of the loop is tried first. If successful, the bowel volvulus relaxes. If unsuccessful, surgical intervention is sometimes required.

Hernia

Definition and Incidence. A hernia is a protrusion of part of an organ or tissue in the groin, abdomen, and navel (often the intestine) through a weakness in the connective tissue structure normally containing it. About 5 million Americans of all ages have some type of abdominal hernia. Hernias can occur at any age in men or women, and most frequently occur in the abdominal cavity as a result of a congenital or acquired weakness of abdominal musculature.

Sports hernia or *athletic hernia* is a term used to describe weakness of the posterior wall of the inguinal canal (transversalis fascia) resulting in chronic activity-related groin pain but without a clinically detectable hernia. Athletes who participate in sports requiring twisting and turning at high speeds (e.g., soccer, rugby, ice hockey, tennis) are at greatest risk. Insidious onset of unilateral groin pain is the most common symptom of this type of hernia.^{78,80}

Weakness can occur as part of the aging process, contributing to acquired hernias. As people age, muscular

tissues become infiltrated by adipose and connective tissues, resulting in weakness. The most common types of hernias are inguinal (direct and indirect), femoral, umbilical, and incisional or ventral (Fig. 16-21). Hiatal hernia is discussed earlier in this chapter.

Etiologic and Risk Factors. When muscular weakness (congenital or acquired) is accompanied by obesity, pregnancy, heavy lifting, coughing, surgical incision, or traumatic injuries from blunt pressure, the risk of developing a hernia increases. Often, herniation is the result of a multifactorial process involving one or more of these factors.

For example, herniation can occur when increased abdominal pressure in a postoperative or posttraumatic injury is aggravated by nutritional or metabolic factors that result in poor wound healing and defective or poor collagen synthesis. Many other possible combinations of risk factors exist (Table 16-8).

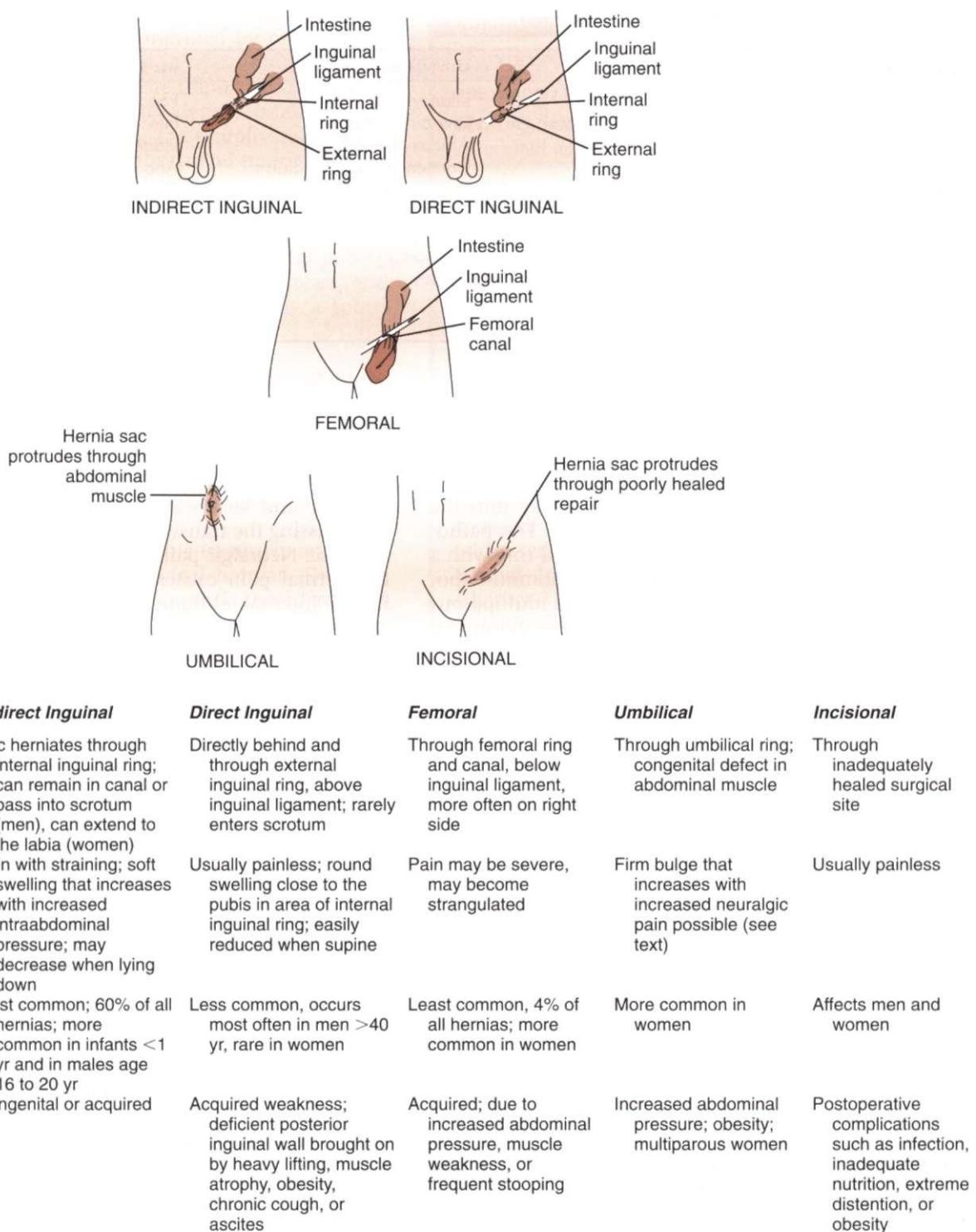
Structural abnormalities account for most congenital hernias, but congenital factors do not explain the increased incidence of hernias (e.g., the direct inguinal type) in advancing age groups. Sudden stress, as occurs in abdominal trauma or industrial accidents, also may contribute to the development of a hernia with or without an underlying congenital defect.

Predisposing factors are equally important, such as situational stress (e.g., repetitive local trauma, strenuous physical activities), degenerative changes associated with increased abdominal pressure, the wear and tear of living, multiparity (women), and altered collagen synthesis in middle age.

Pathogenesis. Structural and biochemical abnormalities and abnormalities of local collagen metabolism have been proposed as factors in the eventual appearance of a hernia. Other biologic factors can affect the balance between collagen synthesis and lysis, eventually leading to the development of herniation. For example, any condition such as renal failure, diabetes mellitus, malnutrition, vitamin or mineral deficiencies, underlying systemic disease, altered immunity, or resistance to infection that can impair a person's ability to generate the proteinaceous constituents of collagen can alter collagen metabolism.

Inguinal hernias account for about 75% of all hernias and affect about 2% of men in the United States. Women can have inguinal hernias too. They occur in the groin when a sac formed from the peritoneum and containing a portion of the intestine pushes either directly outward through the weakest point in the abdominal wall (direct hernia) or downward through the internal inguinal ring into the inguinal canal through which the testes descend into the scrotum during infancy (males) or to the labia (females) (indirect hernia).

Sports hernias occur because adductor contraction during sporting activities creates a shearing force across the pubic symphysis that can stress the posterior inguinal wall. Consequent repetitive stretching of (or a more intense force to) the transversalis fascia and the internal oblique can lead to their separation from the inguinal ligament. This mechanism may also explain the osteitis pubis and adductor tenoperiostitis that often accompany sports hernias.⁷⁸ The most common operative finding

**Figure 16-21**

Hernias. (Modified from Jarvis C: *Physical examination and health assessment*, ed 3, Philadelphia, 2000, Saunders, p 779.)

with a sports hernia is a deficient posterior wall of the inguinal canal, although other abdominal wall abnormalities are frequently found.¹⁴⁸

Direct hernias occur most often as a result of a deficient number of transversus abdominis aponeurotic fibers at a site called *Hesselbach's triangle*, the area between the pubic ramus and the musculofascial components in the lower

abdominal wall. The direct inguinal hernia is more common in older adults, especially in an area that is congenitally weak because of a deficient number of muscle fibers. The indirect inguinal hernia is most common in infants, young people, and males, in the last because it follows the tract that develops when the testes descend into the scrotum before birth. A wide space at

Table 16-8 Risk Factors for Hernias

Inguinal	Femoral	Umbilical	Incisional
Advanced age	Pregnancy (multiparous)	Infancy	Poor wound healing
Prematurity		Low birth weight	Infection
Positive family history		African descent	Inadequate nutrition
Abdominal wall defects		Congenital hypothyroidism	Abdominal distention
Undescended testis		Mucopolysaccharidoses	Obesity
Connective tissue disorders		Down syndrome	Prolonged use of steroids
Cystic fibrosis		Obesity	Advanced age
Shunt for hydrocephalus		Pregnancy	Immunosuppression
Ascites		Ascites	Postoperative pulmonary complications (coughing, straining)
			Type of incision (vertical)

the inguinal ligament also can contribute to the development of an inguinal hernia.

Femoral hernia is a protrusion of a loop of intestine into the femoral canal, a tubular passageway into the thigh that carries nerves and blood vessels. The pathologic anatomy present is an enlarged femoral ring with a correspondingly narrowed transversus abdominis aponeurosis. This type occurs more often in multiparous women, acquired as a result of increased intraabdominal pressure gradually forcing more and more preperitoneal fat into the femoral canal, enlarging the femoral ring.

The pathology of *umbilical hernias* is caused by increased abdominal pressure (see discussion of risk factors) exerted against a thinning of the umbilical ring and fascia. *Incisional hernia* occurs postoperatively (see discussion of risk factors) when the transected fibers are unable to form collagen links strong enough to hold the edges of the wound together.

Clinical Manifestations. The most common manifestation of a hernia of any type is an intermittent or persistent bulge, accompanied by intermittent or persistent pain. Inguinal hernia usually begins as a small, marble-sized soft lump under the skin. At first it is painless and can be reduced by pushing it back in place. As pressure from the abdominal contents pushes against the weak abdominal wall, the size of the lump formed by the hernia increases, requiring surgical repair (herniorrhaphy).

The pain associated with simple hernias depends on the involved structures and whether these are compressed or irritated. The pain usually is localized and sharp, aggravated by changes in position, by physical exertion, during a bowel movement, or by any activity causing the Valsalva maneuver (bearing down with increased intraabdominal pressure such as during coughing or sneezing), and relieved by cessation of the physical activity that precipitated it.

Inguinal hernias are often more noticeable after a heavy meal or long period of standing. Sports hernias are aggravated by sudden movements, acceleration, twisting and turning, cutting, kicking, resisted sit-ups, or any activities that stretch or stress the abdominal muscles.

Pain may radiate from the groin to the testicles (males), ipsilateral thigh, flank, or hypogastrium (lowest middle abdominal region). In the female, painful symptoms may be aggravated by the onset of menstruation. Sports hernia

in females is often characterized by tenderness at the site of the superficial inguinal ring.⁷⁸

The ilioinguinal nerve penetrates the abdominal wall cranially and somewhat laterally to the deep inguinal ring, passing the transverse and internal oblique muscles stepwise. Neuralgic pain may occur when the dull inguinal hernial pain causes a local reflex increase of tone in the internal oblique and transverse muscles of the abdomen. As the nerve passes these muscles in steps, it may be exposed to pressure, giving rise to pain of the neuralgic type.

Ilioinguinal or femoral neuritis caused by nerve entrapment from sutures, adhesions, or the actual formation of a symptomatic neuroma after section of a nerve in this region can occur. These conditions usually resolve spontaneously without specific treatment, but therapy in conjunction with local nerve blocks may be indicated if symptoms persist after the first postoperative month.

Genitofemoral neuralgia (causalgia) occurs less commonly but results in severe pain and paresthesia (or hyperesthesia) in the distribution of the genitofemoral nerve. Radiation of pain to the genitalia and upper thigh may occur, and pain is aggravated by walking, stooping, or hyperextending the hip. Recumbency and flexion of the thigh may relieve painful symptoms. This condition requires neurectomy for pain relief.

When the contents of the hernial sac can be replaced into the abdominal cavity by manipulation, the hernia is said to be *reducible*. Hernias that cannot be reduced or replaced by manipulation are referred to as *irreducible* and *incarcerated*.

Complications occur when the protruding organ is constricted to the extent that circulation is impaired (strangulated hernia) or when the protruding organs encroach on and impair the function of other structures. When a hernia contains incarcerated or strangulated structures, the pain becomes persistent and often is associated with systemic signs or symptoms such as elevated temperature, tachycardia, vomiting, and abdominal distention.

MEDICAL MANAGEMENT

DIAGNOSIS. History and physical examination remain the most important aspects of diagnosis for all types of hernia; there may be a past history of hernia. The diag-

nosis of umbilical hernia is usually obvious because of protrusion of the umbilicus confirmed by palpation of the involved structures.

Radiographic investigations are important in diagnosing sports hernias, especially to identify osteitis pubis, adductor tenoperiosteal lesions, and symphyseal instability, and to rule out hip osteoarthritis and tumors. A bone scan may be needed to visualize active osteitis pubis, stress fractures, and tenoperiosteal lesions.⁷⁸

MRI may be used to diagnose bone marrow edema about the pubic symphysis (a sign of osteitis pubis), stress fractures, avascular necrosis, labral hip tears, and articular cartilage defects that can accompany a sports hernia.¹⁴⁸

TREATMENT. Various supports and trusses are available to contain hernias, but these offer only a temporary solution and may not prevent the hernia from getting bigger with associated complications. The use of strapping techniques is not recommended, because the tape used may lead to ulceration of the thin skin covering the hernia and eventual rupture.

Watchful waiting is an acceptable treatment approach for minimally symptomatic *inguinal* hernias. Delaying surgical repair until symptoms increase is considered safe because acute hernia incarcerations rarely occur.

Surgical repair is the only curative treatment, but it is no longer recommended as preventive repair in all cases. Complications of herniorrhaphy such as cutaneous nerve injury, bleeding, wound infection, chronic pain, and recurrence have led to a rethinking of surgical correction for asymptomatic hernias. Further studies are needed to identify the natural history of inguinal hernias and to identify the best treatment plan.^{54,146}

When surgical repair is indicated, there are two main methods used: the traditional approach, known as tension repair, and a newer method, the mesh repair. The tension repair uses an open incision to realign the soft tissues and stitch them closed. A mesh patch is stitched over the hernia to strengthen the repair. There is major discomfort postoperatively, and recovery (return to normal function) takes approximately 6 to 8 weeks for the average person.

The mesh repair uses a plastic mesh hernia repair patch that can be inserted laparoscopically. One side of the patch is positioned to lie against the inner abdominal wall and is held in place by pressure from the abdomen. The other (smaller) side patches the outer abdominal wall. Using this three-dimensional patch system distributes the pressure over a larger area, making the repair stronger. The patch also provides a matrix for tissue ingrowth. Recovery is much faster; many individuals are back to normal activity within 10 days.

PROGNOSIS. Prognosis varies with the type of hernia and accompanying complications. The incidence of incarceration is about 10% in indirect inguinal hernia and 20% in femoral hernia. Umbilical hernia in adults has a high morbidity and mortality associated with incarceration. Open and laparoscopic repairs produce excellent results; the laparoscopic procedure allows earlier return to play for athletes.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-19

Hernia

PREFERRED PRACTICE PATTERN

6H: Impaired Circulation and Anthropometric Dimensions Associated with Lymphatic System Disorders

Prevention, Screening, and Referral for Hernia

Congenital muscle weakness complicated by the additional risk factors of obesity and increased intraabdominal pressure should be identified and treated. Educate clients in proper lifting techniques and precautions to avoid heavy lifting and straining, which reduce intraabdominal pressure as an additional risk factor for the development of hernias and aid in preventing worsening of an already existing hernia. The mouth-open position as a reminder to breathe properly and to prevent increased intraabdominal pressure is essential during all lifting procedures. Obesity as a cause of increased intraabdominal pressure may be addressed by weight control through exercise (see Special Implications for the Therapist: Obesity in Chapter 2).

Early diagnosis is important in preventing bowel incarceration and strangulation. Any client experiencing chronic cough, pregnancy, or back, hip, groin, or sacroiliac pain should be asked, "Have you ever been told you have a hernia, or do you think you have a hernia now?"

Any person (especially an older client) with a known hernia complaining of pain, nausea, vomiting, or other new symptom in the anatomic vicinity of the hernia should report these symptoms to the physician to rule out a systemic condition unrelated to the herniation.

Any time a person chooses to wear a truss without prior physician evaluation, the therapist is advised to encourage that client to seek medical advice. The therapist should be aware of two complications that may occur in a client wearing a truss. In the client with a small hernia the pressure of the overlying truss on a protruding hernial mass enhances the chances of strangulation by obstructing lymphatic and venous drainage. In a person with a large direct inguinal hernia, the constant overlying pressure of the truss pad on the margins of the hernial defect can lead to atrophy of the fascial aponeurotic structures, enlarging the hernial opening and promoting growth of the hernia, thus making subsequent surgical repair more difficult.

Although uncommon, psoas abscess still can be confused with a hernia. The therapist may perform evaluative tests to rule out a psoas abscess, but the physician must differentiate between an abscess and a hernia; see the iliopsoas and obturator muscle tests (see Fig. 16-14), iliopsoas palpation (see Fig. 16-15), and McBurney's point (see Fig. 16-22). Psoas abscess is often softer than a femoral hernia and has ill-defined borders, in contrast to the more sharply defined margins of the hernia. The major differentiating feature

Continued.

is the fact that a psoas abscess lies lateral to the femoral artery, not medial to it, as is the case for the femoral *hernia*.

Postoperative Recovery

For most clients recovering from surgical repair of a hernia, heavy lifting and straining should be avoided for 4 to 6 weeks after surgery. This guideline may vary depending on the specific type of hernia and surgical procedure used. The therapist should read the medical record to identify these two features before beginning a postoperative rehabilitation program.

Substance abusers and cigarette smokers have an additional risk factor for delayed wound healing and dehiscence. Transient anesthesia of the skin beneath the hernial incision is a possible postoperative phenomenon. Whether in the presence of an uncorrected hernia or postoperatively, the client should avoid activities and positions that produce painful symptoms associated with the hernia.

Whereas most people do well after surgical repair, some have persistent postoperative pain or discomfort. If a person has had a previous inguinal hernial repair and now presents with painful groin or thigh pain, the physician must differentiate between ilioinguinal nerve entrapment and neuroma of a branch of the nerve severed previously.

Although most hernia surgeries are done laparoscopically, for those individuals who have an open procedure, special care must be taken when there is a vertical incision. When a vertical incision transects fascial aponeurotic fibers, the incision is made perpendicular to the direction of those fibers. Simple muscle contraction, as in coughing, straining, or turning over in bed, tends to distract the wound edges. Laparoscopic repair will likely eliminate this type of surgical incision.

Postoperative Rehabilitation

For the most part, the guidelines for postoperative rehabilitation of herniorrhaphy presented in the literature are not evidence based.¹⁴⁸ Research in this area is needed. It has been suggested that postoperative rehabilitation of the athlete with an uncomplicated sports hernia can begin with isometric abdominal and adductor exercises as early as the first day after minimally invasive surgery. Progression to concentric and eccentric strengthening progresses after the end of the first week. Walking can begin during the first week after surgery.⁷⁸ The safety of these recommendations has not been verified by randomized controlled trials.

The same authors suggest that the client can be progressed to jogging by day 10 if approved by the surgeon. Straight-line sprinting begins around day 21, with sport-specific exercises introduced gradually after that. Athletes may expect to return to full sports participation 6 to 8 weeks after open surgery (much sooner after minimally invasive surgery) unless comorbid muscle strains or unrepaired tears exist; recovery from complicated injuries can take 3 months or more. The therapist should address all aspects of pelvic flexibility, strength, and stability.⁷⁸

Other retrospective studies report that most athletes return to their normal sports level within 3 months after surgery.¹⁵¹ A structured rehabilitation program including client education on proper posture and body mechanics, core and pelvic stabilization strengthening exercises, eventually progressing to sport-specific drills is suggested as a means of maximizing the benefits of surgery, but there are no empirical data to support this recommendation.⁸⁵

Functional Obstruction

Adynamic or Paralytic Ileus. Adynamic or paralytic ileus is a neurogenic or muscular impairment of peristalsis that can cause functional intestinal obstruction. The term *functional obstruction* is somewhat of a misnomer in that the intestine is not blocked or plugged so much as peristalsis stops and movement of intestinal contents stops or is slowed down considerably. This condition has a variety of causes (see Table 16-7), most commonly occurring after abdominal surgery when the bowel ceases to function for a limited period of time (several hours to several days). Paralytic ileus is a common sequela of spinal cord injury. Neurogenic impairment also may occur after abdominal procedures in which the surgeon handles the bowel extensively or after surgery involving the retroperitoneal area (e.g., anterior spinal fusion with cages).

Clinical manifestations include mild to moderate abdominal pain that tends to be continuous rather than colicky, as with mechanical obstruction. Borborygmus and bowel sounds are absent. Abdominal distention is often the first sign of ileus. Dehydration with prolonged vomiting along with massive generalized abdominal distention may occur. The diagnosis is suspected in the presence of a precipitating condition and signs and symptoms of obstruction in the absence of bowel sounds. Diagnosis is confirmed by radiography of the abdomen and barium enema to rule out organic obstruction.

Most cases of adynamic ileus respond to restricted oral intake with gradual reintroduction of foods as bowel function returns. Severe and prolonged ileus may require complete elimination of food and fluids and aspiration of gastric secretions by suctioning until the bowel begins to function again. In such cases, parenteral nutrition is utilized to reintroduce fluids and electrolytes. Prognosis depends on the underlying cause of adynamic ileus. Removal of the cause may result in resolution of the ileus. Intubation with a rectal tube or colonoscope to decompress a dilated colon may be successful in returning bowel function but is not a commonly performed procedure.

Ogilvie's Syndrome. In a small number of individuals with trauma to the hip and pelvis or after elective hip or pelvic surgery (e.g., total hip replacement), acute colonic pseudo-obstruction can occur in the early postoperative period. The condition may be referred to as Ogilvie's syndrome or adynamic ileus of the colon.

It is characterized by colonic dilatation and functional obstruction but with no obvious mechanical cause.

In contrast to the involvement of the large and small bowel seen with adynamic ileus, acute colonic pseudo-obstruction affects the colon but not the small bowel. Signs and symptoms include abdominal distention and discomfort, loss of appetite, nausea and vomiting, diarrhea, and excessive flatus (passing gas).¹⁵

The exact etiology of Ogilvie's syndrome is unknown; it is suspected that altered autonomic activity occurs as a result of trauma or surgical intervention in people who are at risk. Dysfunction of the sacral parasympathetic nerves S2 to S4 supplying the left colon and rectum results in atonic or spastic large bowel leading to a functional obstruction.¹⁵

Previous publications hypothesized that acetabular trauma (reaming and acetabular preparation) could lead to retroperitoneal swelling (edema and hematoma) disturbing and inhibiting the pelvic splanchnic nerves, resulting in sympathetic overflow leading to dilation and atony of the distal large bowel.¹⁵ Heat generation from bone cement also could lead to an imbalance of the autonomic nervous supply of the colon, resulting in this syndrome.¹⁰⁹

Risk factors for this condition include male gender, increasing age, immobility, and patient-controlled analgesia. Older adults have overall poorer health, use more medications that decrease gut motility, and have more episodes of previous GI surgery, all of which increase the risk for developing this condition.

Prompt recognition and early consultation are needed to avoid perforation of the colon and significant morbidity and mortality.^{49,109} The therapist can expect a slower rehabilitation to preoperative functional levels due to the medical complications associated with this disorder.³⁰

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-20

Adynamic or Paralytic Ileus

Anterior lumbar fusion procedures may indirectly cause a functional ileus when the client is unable to ambulate early or remains immobile and inactive for any reason. The therapist may use transcutaneous electrical nerve stimulation (TENS) in the acute care setting for the short term to assist in pain control and to encourage mobility. Increased activity stimulates movement of air out of the bowel and helps prevent constipation and the subsequent development of a functional ileus.

Congenital Conditions

Intestinal atresia and stenosis, although rare, are the most frequent causes of neonatal intestinal obstruction. Either condition is diagnosed on the basis of persistent vomiting of bile-containing fluid during the first 24 hours after birth. Surgical correction is usually successful, but coexistent anomalies often are seen and complicate treatment.

Stenosis and Atresia. *Stenosis*, is a narrowing of a canal, in this case the small intestine. Intestinal atresia

refers to defects caused by the incomplete formation of a lumen, in this case the tubular portion of the intestine. Many hollow organs originate as strands and cords of cells, the centers of which are programmed to die, thus forming a central cavity or lumen.

Most cases of intestinal atresia are characterized by complete occlusion of the lumen, which was not fully established in embryogenesis. Meconium ileus (accumulation of meconium in the small intestine causing neonatal intestinal obstruction) accounts for 25% of all cases, and cystic fibrosis accounts for another 10%. Intestinal atresia may have several forms: multiple intestinal occlusions giving the appearance of a string of sausages, disconnected blind ends, blind proximal and distal sacs joined by a cord, or a thin transluminal diaphragm across the opening. All forms require surgical intervention with good prognosis for recovery.

Meckel's Diverticulum. Meckel's diverticulum is an outpouching of the bowel located at the ileum of the small intestine, near the ileocecal valve. It occurs because of failure of destruction of the vitelline duct, an embryonic communication between the midgut and the yolk sac. Meckel's diverticulum is the most common congenital malformation of the GI tract, present in 2% of the population. Males are affected more often than females in a 2:1 ratio, with accompanying complications in the same ratio.

Meckel's diverticulum may be asymptomatic or produce symptoms that include abdominal pain similar to that in other conditions such as appendicitis, CD, and peptic ulcer disease. Common complications associated with Meckel's diverticulum include bleeding or hemorrhage from peptic ulceration of the ileum adjacent to the ectopic gastric mucosa, intestinal obstruction, diverticulitis that mimics appendicitis, and perforation caused by peptic ulceration in the diverticulum or in the ileum.

Diagnosis is made usually during the first 2 years, but the condition may go undetected into adulthood when it is discovered on autopsy or during laparotomy for an unrelated condition. The diagnosis is made usually based on history, physical examination, and radionuclide scan. Prognosis is good with surgical resection to remove the diverticulum. Severe hemorrhage must be treated before surgery to correct hypovolemic shock through the administration of blood transfusion, intravenous fluids, and oxygen.

THE APPENDIX

Appendicitis

Definition and Incidence

Appendicitis is an inflammation of the veriform appendix that often results in necrosis and perforation with subsequent localized or generalized peritonitis. On the basis of operative findings and histologic appearance, acute appendicitis is classified as simple, gangrenous, or perforated.

It is the most common disease of the appendix, occurring at any age, with the peak incidence among men in their second and third decades. The lifetime risk of acute appendicitis in the United States is about 9% for males

and 7% for females.⁷⁰ The overall incidence is declining for unknown reasons, possibly as a result of increased dietary fiber intake in recent years or improved hygiene and fewer intestinal infections associated with indoor plumbing.

Etiologic Factors

Approximately half of all cases of acute appendicitis have no known cause. At least one third are caused by obstruction that prevents normal drainage. Obstruction may occur as a result of tumor, foreign body such as fecal material (fecolith) lodged in the lumen of the appendix, parasites (e.g., intestinal worms), or lymphoid hyperplasia.

Because the appendix is chiefly lymphatic tissue, an infection that produces enlarged lymph nodes elsewhere in the body also can increase the glandular tissue in the appendix and obstruct its lumen. Other causes include CD of the terminal ileum, UC when it spreads to the mucosa of the appendix, and tuberculous enteritis.

Pathogenesis

Classically, appendicitis is believed to develop primarily from obstruction of the lumen and secondarily from bacterial infection. When the long, narrow appendiceal lumen becomes obstructed, inflammation begins in the mucosa, with swelling and hyperemia of the veriform appendix. As secretions distend the obstructed appendix, the intraluminal pressure rises and eventually exceeds the venous pressure, causing venous stasis and ischemia.

The accumulation of neutrophils produces microabscesses, and arterial thromboses aggravate the ischemia. The infected necrotic wall becomes gangrenous and may perforate, often in 24 to 48 hours. The mucosa ulcerates and permits invasion by intestinal bacteria. *E. coli* and other bacteria multiply and cause inflammation and infection that spread to the peritoneal cavity unless the body's defenses are able to overcome the infection or the appendix is removed before it ruptures.

Clinical Manifestations

The presenting symptoms of acute appendicitis occur in a classic sequence of abdominal (epigastric, periumbilical, or right lower quadrant) pain accompanied by anorexia, nausea, vomiting, and low-grade fever in adults (children tend to have higher fevers). Infants and children often seem withdrawn with a nonspecific presentation.⁷⁰ Women may experience acute pelvic pain that must be differentiated from other causes of pelvic pain (e.g., ectopic pregnancy, diverticulitis, incarcerated hernia, kidney stones).

Pain associated with appendicitis is constant and may shift within 12 hours of symptom onset to the right lower quadrant with point tenderness over the site of the appendix at McBurney's point, a point between 1½ and 2 inches superomedial to the anterior superior iliac spine, on a line joining that process and the umbilicus (Fig. 16-22). Signs and symptoms of perforation include a white blood cell count of 20,000/mm³ or greater; a tense, rigid abdomen; and elevated temperature (102° F [39° C]).

Aggravating factors include anything that increases intraabdominal pressure (see Box 16-1), such as cough-

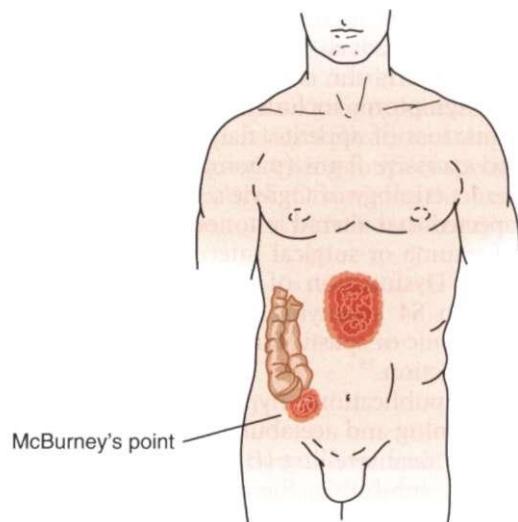


Figure 16-22

Pain areas associated with appendicitis, including McBurney's point. Tenderness upon palpation of this point is indicative of appendicitis. With the client supine and legs extended, isolate the anterior superior iliac spine and the umbilicus, then gently palpate approximately one half the distance between those two points. A positive test produces painful symptoms in the right lower quadrant. (Modified from O'Toole M, ed: *Miller-Keane encyclopedia and dictionary of medicine, nursing, and allied health*, ed 6, Philadelphia, 1997, Saunders, p 119.)

ing, walking, laughing, and bending over. Older adults frequently have few or no symptoms with minimal fever and only slight tenderness until perforation occurs. Confusion or increased confusion may be the first and only presenting symptom among older adults. While appendicitis is rare in older adults, half of all people who die from a ruptured appendix are 70 years old or older.¹⁴³

Atypical Appendicitis. Many cases of appendicitis are atypical because of the position of the appendix (Fig. 16-23), the person's age, or the presence of associated conditions, such as pregnancy. The person may not recognize the need for medical attention but may report symptoms to the therapist. Early recognition of the need for medical evaluation is imperative.

Retrocecal appendicitis and *retroileal appendicitis* may occur when the inflamed appendix is shielded from the anterior abdominal wall by the overlying cecum and ileum. The pain seems less intense and less localized, and less discomfort occurs with walking or coughing. The pain may not shift as expected from the epigastrium to the right lower quadrant.

Pelvic appendicitis may begin with pain in the epigastrium but quickly settles in the lower abdomen, commonly localized to the left side for an unknown reason. The absence of abdominal tenderness may be deceptive, but the physician will elicit this symptom on pelvic examination. *Appendicitis in the immunosuppressed individual* presents as abdominal pain and fever without leukocytosis, but concern about other causes usually delays recognition.

Appendicitis in the aging adult is usually vague with minimal pain and only slight temperature elevation. Abdominal tenderness is present and localized to the

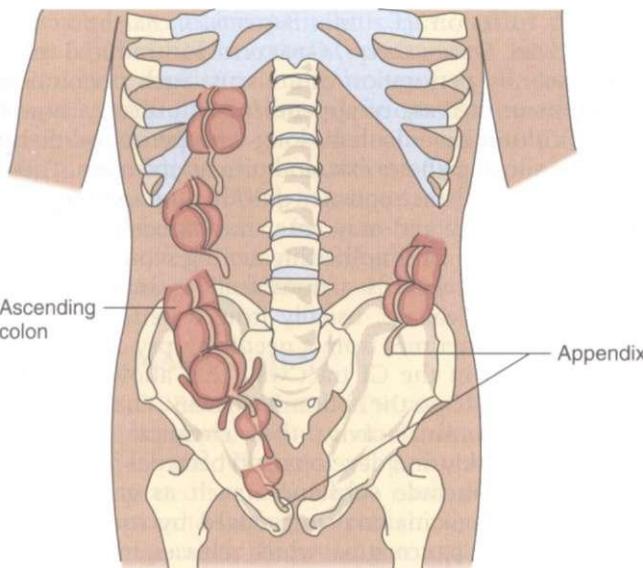


Figure 16-23

Variations in the location of the veriform appendix. Negative test results for appendicitis using McBurney's point may occur when the appendix is located somewhere other than at the end of the cecum. (From Goodman CC, Snyder TE: Differential diagnosis for physical therapists: screening for referral, ed 4, Philadelphia, 2007, Saunders.)

right lower quadrant but is deceptively mild. *Appendicitis in pregnancy* does not present a diagnostic problem in the first trimester but later in gestation may be confused with an obstetric condition. Displacement of the appendix by the enlarged uterus may result in tenderness in the right subcostal area or adjacent to the umbilicus.

MEDICAL MANAGEMENT

DIAGNOSIS. Acute appendicitis must be diagnosed early to prevent perforation, abscess formation, and postoperative complications, but the diagnosis is not always easy to make. Although the clinical diagnosis may be straightforward in people who present with classic signs and symptoms, atypical presentations require the physician to differentiate appendicitis from a large variety of GI, genitourinary, and gynecologic conditions.

A careful history and thorough physical examination are the primary diagnostic tools. Rebound tenderness is a widely used physical examination test for clients with suspected appendicitis, but the test can be very uncomfortable for the individual. Some experts no longer advise its use with clients who have abdominal pain but prefer the "pinch-an-inch" test, which is a form of the rebound test in reverse.

To perform the pinch-an-inch test, a fold of abdominal skin over McBurney's point is grasped and elevated away from the peritoneum. The skin is then allowed to recoil back against the peritoneum. The test is considered positive for peritonitis if there is increased pain when the skin fold strikes the peritoneum (Fig. 16-24).¹

An elevated white blood cell count (more than 20,000/mm³; leukocytosis) suggests ruptured appendix and peritonitis. Urinalysis reveals abnormalities in up to 40% of individuals tested.⁷⁰ Abdominal CT is more diagnostic than ultrasound but is usually only done when a diagno-

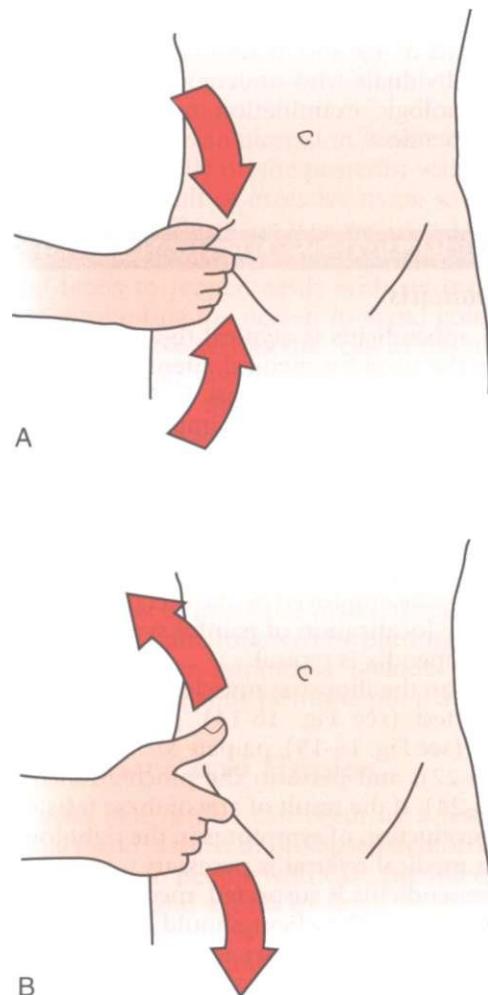


Figure 16-24

Pinch-an-inch test.¹ To avoid the discomfort of the traditionally used (and sometimes inaccurate) rebound test, the pinch-an-inch test has been developed as an alternative test for peritonitis associated with appendicitis. **A**, Gently grasp and lift 2 to 3 inches of skin from over McBurney's point on the left side of the abdomen. The individual with a low threshold of pain may report pain during the initial pinch. The clinician must assess this response accordingly. **B**, Quickly release the skin. A positive sign occurs if increased pain is reported when the skin returns to its position against the abdominal wall.

sis cannot be made.⁷⁰ Histologic examination of the resected appendix is used to confirm the diagnosis.

TREATMENT. Appendectomy, or surgical removal of the veriform appendix, is performed as soon as possible. Antibiotics are administered preoperatively to decrease the incidence of postoperative wound infection and intraabdominal abscess.⁷⁰ With accurate diagnosis and early surgical removal, mortality and morbidity rates are less than 1%.

PROGNOSIS. Prognosis is good unless diagnosis is delayed and perforation occurs (rarely during the first 8 hours of symptomatic presentation). Perforation with complications such as peritonitis, hypovolemia, and septic shock

has a poor prognosis. Perforation is more likely in infants under 2 years of age and in adults over 60 years. In up to 20% of individuals who undergo emergency appendectomy, pathologic examination of the tissue shows a normal appendix.⁶⁹

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-21

Appendicitis

When appendicitis is atypical the client may not recognize the need for medical attention but may report the symptoms to the therapist. Early recognition of the need for medical referral is important. In an athletic training or physical therapy setting, appendicitis may present with symptoms of right thigh pain, groin (testicular) pain, pelvic pain, or referred pain in the hip.

In addition to screening for the presence of constitutional symptoms, a variety of objective test procedures may be employed by the therapist. Ask the client to cough: localization of painful symptoms to the site of the appendix is typical.

Perform the iliopsoas muscle test and the obturator muscle test (see Fig. 16-14), palpate the iliopsoas muscle (see Fig. 16-15), palpate McBurney's point (see Fig. 16-22), and perform the pinch-an-inch test (see Fig. 16-24). If the result of any of these tests is positive for reproduction of symptoms in the right lower quadrant, a medical referral is necessary.

If appendicitis is suspected, medical attention must be immediate. The client should be instructed to lie down and remain as quiet as possible, taking nothing by mouth (including water); heat is contraindicated.

bacterial infection (*E. coli*, *Bacteroides*, *Staphylococcus*, *Streptococcus*, *Pneumococcus*, *Gonococcus*) introduced most commonly by perforation of a viscus. Such perforation can occur in the case of appendicitis, an ulcer, a bowel infarct, colonic diverticulum, long-term peritoneal dialysis at the site of catheter exit, and urinary infection. These bacterial organisms spread quickly throughout the abdominal cavity and may enter the bloodstream from the peritoneum, causing life-threatening septicemia.

Chemical peritonitis is a noninfectious inflammation caused by bile leakage, usually from a perforated gallbladder but sometimes from a needle biopsy of the liver, or any breach in the GI tract wall that allows GI tract contents to spill into the abdominal cavity. Once bacteria enter the abdominal cavity, then chemical peritonitis progresses quickly and develops into bacterial peritonitis. Other causes include substances such as gastric acid, blood, or foreign material introduced by surgery (e.g., talc), and acute pancreatitis, which releases and activates lipolytic and proteolytic enzymes. *Metastatic peritonitis* occurs when neoplasm perforates the viscus of the stomach and tumor cells infiltrate the peritoneum.

Pathogenesis

The GI tract normally contains bacteria, but the peritoneum is sterile. Inflammation and perforation of the GI tract from appendicitis, diverticulitis, perforated gallbladder, or a peptic ulcer allow bacteria to invade the peritoneum.

Once the inflammatory process has begun, a fibrinous-purulent exudate covers the peritoneal surface. The exudate becomes organized and fibrotic, forming adhesions and causing obstruction. Usually infection in the peritoneal cavity is localized as an abscess.

When a perforation drains contaminants into the peritoneal cavity, however, the ability of the peritoneum to combat the inflammatory process can be overpowered. The entire surface of the peritoneum may be involved (generalized peritonitis) or only specific sites (localized). When the pelvic peritoneum is involved, pelvic peritonitis (also called *pelvic inflammatory disease*) occurs.

Peritonitis creates severe systemic effects. Circulatory alterations, fluid shifts, and respiratory problems can cause critical fluid and electrolyte imbalances. The circulatory system undergoes great stress from several sources. The inflammatory response shunts extra blood to the inflamed area of the bowel to combat the infection. Peristaltic activity of the bowel ceases, leading to bowel obstruction. Fluids and air are retained within its lumen, raising pressure and increasing fluid secretion into the bowel; circulating volume diminishes.

Clinical Manifestations

Peritonitis commonly decreases intestinal motility and causes intestinal distention with gas. At first the affected individual may feel vague, generalized abdominal pain. As the peritonitis progresses, the client presents with an acute abdomen and severe abdominal pain. The abdomen becomes rigid (involuntary guarding) and sensitive to touch. Pain is severe, increasing with movement and respirations, and can be referred to the shoulder or thoracic area. Nausea, vomiting, and high fever follow.

THE PERITONEUM

Peritonitis

Overview

Peritonitis, or inflammation of the serous membrane lining the walls of the abdominal cavity, is caused by a number of situations that introduce microorganisms into the peritoneal cavity. Peritonitis that occurs spontaneously is called *primary peritonitis*. Peritonitis as a consequence of trauma, surgery, or peritoneal contamination by bowel contents (e.g., perforated duodenal ulcer or appendix) is referred to as *secondary peritonitis*.

Etiologic Factors

Specific causes of peritonitis are many and varied. Primary peritonitis is associated with ascites and chronic liver disease or the nephrotic syndrome. Secondary peritonitis occurs as a result of inflammation of abdominal organs, irritating substances from a perforated gallbladder or gastric ulcer, rupture of a cyst, or irritation from blood, as in cases of internal bleeding.

Secondary peritonitis may be classified as bacterial, chemical, or metastatic. *Bacterial peritonitis* is caused by

Without treatment, peritonitis can lead to paralytic ileus (diminished to absent peristalsis), fatal bowel obstruction, sepsis, or multiple organ dysfunction syndrome. A peritoneal abscess develops if the perforation becomes self-encased or walled off. Antibiotic therapy may mask or delay the recognition of signs of abscess.

In persons with underlying ascites, the signs and symptoms of peritonitis may be more subtle, with fever as the only manifestation of infection, or possibly nausea, vomiting, nonspecific abdominal pain, or altered mental status.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Abdominal films, barium enema, and an abdominal tap may be used in the differential diagnosis of peritonitis. Peritonitis should be treated immediately to control infection; preserve the barrier to further microbial invasion; minimize the effects of paralytic ileus; and correct fluid, electrolyte, and nutritional disorders.

If peritonitis is advanced and surgery is contraindicated because of shock and circulatory failure, oral fluids are prohibited and intravenous fluids are necessary for replacement of electrolyte and protein losses. A long intestinal tube is inserted through the nose into the intestine to reduce pressure within the bowel. Once the infection has become walled off and the client's condition improves, surgical drainage and repair can be attempted.

Despite treatment with antibiotics, surgical drainage and debridement, and supportive measures, generalized peritonitis is still associated with a mortality rate of 50% and is especially dangerous in the older adult. Individuals recovering from an episode of bacterial peritonitis should be considered as potential candidates for liver transplantation.

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-22

Peritonitis

Special considerations associated with peritonitis are related to the underlying cause (e.g., liver or kidney disease, postoperative state, cancer) and resultant complications (e.g., fluid and electrolyte imbalance, pulmonary compromise). The client with peritonitis usually is hospitalized and undergoing medical treatment. The therapist should be familiar with implications associated with the underlying cause and any complications present.

Vital signs should be regularly monitored (see Appendix B) and a semi-Fowler position used to help the client breathe deeply with less pain to prevent pulmonary complications. Position changes must be accomplished with extreme caution, because the slightest movement intensifies the pain. Watch for signs of dehiscence (separation of layers of a surgical wound), such as the person's reporting that something broke loose or gave way inside. Follow all safety measures such as keeping the side rails up on the bed if fever and pain disorient the client.

THE RECTUM

Rectal Fissure

A rectal or anal fissure is an ulceration or tear of the lining of the anal canal, usually on the posterior wall. An acute fissure occurs as a result of excessive tissue stretching or tearing, such as childbirth or passage of a large, hard bowel movement through the area. The skin tear is very fragile and tends to reopen easily with the next bowel movement, prompting the person to avoid going to the bathroom for days. Neglecting the "call of the stool" can result in constipation and further exacerbation of the problem, especially in the presence of risk factors for constipation (see the section on Constipation in this chapter).

Anal fissures frequently heal within a month or two when treated with a combination of bran and bulk laxatives or stool softeners, sitz baths, and emollient suppositories. Chronic fissures are usually secondary to a tight rectal sphincter or infectious material retained in the anal sinuses. Sharp pain, followed by burning, accompanies defecation. Other symptoms include mucus, an external skin tag at the anus (sentinel pile), and itching.

Rectal Abscesses and Fistulas

Anal abscesses and fistulas can occur as a result of an infected anal gland, fissure, or prolapsed hemorrhoid and are most common in people with CD. The infection can cause pus, mucus, and blood to drain from the anus. Fistulas (abnormal channels to the body's surface) may form spontaneously to drain the abscesses. Swelling, pain, and throbbing exacerbated by sitting or walking are the primary symptoms of these conditions, which may heal with time or may be treated with surgical drainage with or without antibiotics.

Hemorrhoids

Hemorrhoids, or piles, are varicose veins of a pillowlike cluster of veins that lie just beneath the mucous membranes lining the lowest part of the rectum and anus. They can be internal or external. Hemorrhoids are fairly common, affecting as many as half of all adults more than 50 years of age. It is hypothesized that the connective tissues that support and hold hemorrhoids in place can weaken with age, causing hemorrhoids to bulge and prolapse.

This condition is associated especially with anything that increases intraabdominal pressure (see Box 16-1), such as chronic constipation, pregnancy (the enlarged uterus presses on the veins), pelvic congestion or pelvic venous disease, congestive heart failure, prolonged sitting or standing, low-fiber diet, obesity, diarrhea, and delaying a bowel movement when the urge presents itself.

Higher resting anal canal tone may also contribute to the development of hemorrhoids. The smooth muscle of the anal canal tends to be tighter than average, even when not straining. Constipation and straining add to the pressure in the anal canal.

Internal hemorrhoids occur in the lower rectum and usually are noticed first when a small amount of bleeding occurs during passage of stool, especially if straining occurs during a bowel movement. Internal hemorrhoids are asymptomatic (rectal tissue lacks nerve fibers) except in the presence of an anal fissure, thrombosis, or strangulation of the varicose vein. In the case of strangulation, straining can cause an internal hemorrhoid to protrude (prolapse) from the anus. The blood supply is cut off by the anal sphincter, causing local discomfort and possible itching. This can result in thrombosis when blood within the hemorrhoid clots.

Internal hemorrhoids may require ligation, sclerosing, laser surgery, or cryosurgery to destroy the affected tissue. In the case of advanced chronic hemorrhoids, recurrent bleeding and anemia may necessitate surgery (hemorrhoidectomy, stapled hemorrhoidopexy).

External hemorrhoids located under the skin around the anus bleed (bright red blood) if the hemorrhoid is injured or ulcerated and are very painful because they form in nerve-rich tissue outside the anal canal. Other manifestations include pressure, rectal itching, irritation, and a palpable mass. Severe bleeding or mild bleeding repeatedly from prolonged trauma to the vein during defecation can cause iron-deficiency anemia.

External hemorrhoids can be treated with local application of topical medications, sitz baths, high-fiber diet, and avoidance of constipation and other causes of increased intraabdominal pressure. A stool softener or psyllium preparation may be used when a modified diet is unsuccessful in eliminating constipation. Local topical preparations for hemorrhoids are used to reduce pain or itching. In addition, moderate aerobic exercise such as brisk walking 20 to 30 minutes daily can help stimulate bowel function,

SPECIAL IMPLICATIONS FOR THE THERAPIST 16-23

The Rectum

Therapists may be involved with clients with pelvic pain and/or pelvic floor dysfunction accompanied by chronic rectal conditions. The client may have a history of sexual assault/abuse and/or anal intercourse. These individuals experience severe muscle spasm of the sphincter, resulting in groin or pelvic pain and trigger points in the pelvic floor and gluteal muscles. Any anorectal symptoms (e.g., change in bowel habits, rectal pain, bleeding) that have not been reported to the physician or are changing in pattern must be evaluated by a physician.

Clients involved in any activity requiring increased abdominal support or causing increased intraabdominal pressure should be questioned as to the presence of hemorrhoids. For clients with hemorrhoids postoperatively, prone positioning or side lying supported with pillows between the knees and ankles is preferred. Supine positioning and sitting for brief periods can be accomplished with a rubber air ring under the buttocks for support. Movement, exercise, drinking plenty of fluids, and heeding the call of nature are important in the prevention of constipation-induced hemorrhoids.

References

To enhance this text and add value for the reader, all references are included on the companion Evolve site that accompanies this textbook. The reader can view the reference source and access it on-line whenever possible. There are a total of 161 cited references and other general references for this chapter.

CHAPTER 17

The Hepatic, Pancreatic, and Biliary Systems

CATHERINE C. GOODMAN • CELESTE PETERSON

The *liver* has more than 500 separate functions such as the conversion and excretion of bilirubin (red bile pigment, which is an end-product of heme from hemoglobin in red blood cells [RBCs]). The liver is the sole source of albumin and other plasma proteins and also produces 500 to 1500 ml of bile each day. Other important functions of the liver include production of clotting factors and storage of vitamins. The liver and gut are the key organs in nutrient absorption and metabolism; nutrients bind to toxins in this pathway and aid in eliminating these toxins from the body.

The liver contributes to a functional immune system by reducing the amount of toxins that could impair the gut lining, which in turn helps prevent the entry of bacteria and viruses into the system. Bile acids, drugs, chemicals, and toxins undergo extensive enterohepatic circulation during the processes of metabolism. The liver also filters all of the blood from the gastrointestinal (GI) system and is therefore the primary organ for metastasis of intestinal cancer.

The *pancreas* is both an exocrine and an endocrine gland. Its primary function in digestion is exocrine secretion of digestive enzymes and pancreatic juices, transported through the pancreatic duct to the duodenum. Proteins, carbohydrates, and fats are broken down in the duodenum, aided by pancreatic and other secretions, which also help to neutralize the acidic substances passed from the stomach to the duodenum. The endocrine function involves the secretion of glucagon and insulin by islet of Langerhans cells for the regulation of carbohydrate metabolism. Pancreatic disease may result in a variety of clinical presentations, depending on whether the exocrine or endocrine function has been impaired.

The *gallbladder*, acting as a reservoir for bile, stores and concentrates the bile during fasting periods and then contracts to expel the bile into the duodenum in response to the arrival of food. Bile helps in alkalinizing the intestinal contents and plays a role in the emulsification, absorption, and digestion of fat. The signal for the gallbladder to contract comes from the release of cholecystokinin, a hormone released into the bloodstream from the wall of the duodenum and upper small intestine.

SIGNS AND SYMPTOMS OF HEPATIC DISEASE

Primary signs and symptoms of liver diseases vary and can include GI symptoms, edema/ascites, dark urine, light-colored or clay-colored feces, and right upper abdominal pain (Box 17-1). Impairment of the liver can result in *hepatic failure* when either the mass of liver cells is sufficiently diminished or their function is impaired as a result of cirrhosis, liver cancer, or infection and/or inflammation. Hepatic failure does not refer to one specific morphologic change but rather to a clinical syndrome that includes hepatic encephalopathy, renal failure (hepatorenal syndrome), endocrine changes, and jaundice.

Dark urine and *light stools* occur in association with jaundice (yellow pigmentation of skin, sclerae, and mucous membranes) (see the section on Jaundice in this chapter) when the serum bilirubin level increases from normal (0.1 to 1.0 mg/dl) to a value of 2 or 3 mg/dl. Any damage to the liver impairs bilirubin metabolism from the blood. Normally, bile converted from bilirubin causes brown coloration of the stool. Light-colored (almost white) stools and urine the color of tea or cola indicate an inability of the liver or biliary system to excrete bilirubin properly.

Skin changes associated with the hepatic system include jaundice, pallor, and orange or green skin. When bilirubin reaches levels of 2 to 3 mg/dl, the sclera of the eye takes on a yellow hue. When bilirubin level reaches 5 to 6 mg/dl, the skin becomes yellow. The changes described here in urine, stool, or skin color may be caused by hepatitis, gallbladder disease, pancreatic cancer blocking the bile duct, hepatotoxic medications, or cirrhosis. Other skin changes may include bruising, spider angiomas, and palmar erythema.

Spider angiomas (arterial spider, spider telangiectasis, or vascular spider) are branched dilations of the superficial capillaries, which may be vascular manifestations of increased estrogen levels (hyperestrogenism) (see Fig. 10-3). Spider angiomas and palmar erythema both occur in the presence of liver impairment as a result of

Box 17-1**MOST COMMON SIGNS AND SYMPTOMS OF HEPATIC DISEASE**

Gastrointestinal symptoms (see Table 16-1)

Edema/ascites

Dark urine

Light- or clay-colored stools

Right upper quadrant abdominal pain

Skin changes

Jaundice

Bruising

Spider angioma

Palmar erythema

Neurologic involvement

Confusion

Sleep disturbances

Muscle tremors

Asterixis

Musculoskeletal pain (see text for sites)

Hepatic osteodystrophy

Jaundice

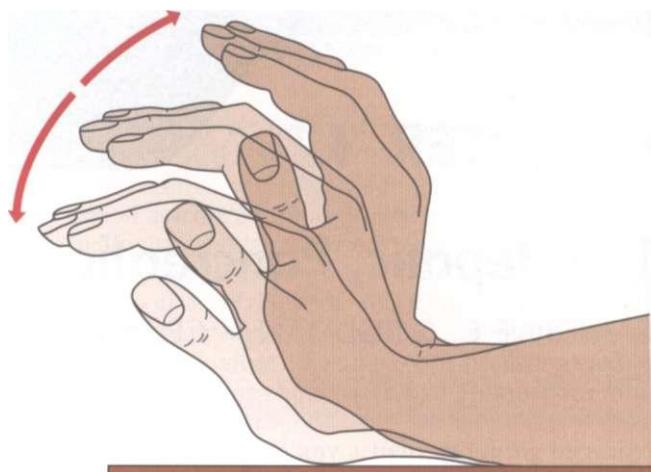


Figure 17-1

Flapping tremor. The flapping tremor elicited by attempted wrist extension while the forearm is fixed is the most common neurologic abnormality associated with liver failure. It can also be observed in uremia, respiratory failure, and severe heart failure. The tremor is absent at rest, decreased by intentional movement, and maximal on sustained posture. It is usually bilateral, although one side may be affected more than the other. (From Sherlock S, Dooley J: *Diseases of the liver and biliary system*, ed 9, Oxford, 1993, Blackwell Scientific Publications.)

increased estrogen levels normally metabolized by the liver. *Palmar erythema* (warm redness of the skin over the palms, also called *liver palms*) especially affects the hypothenar and thenar eminences and pulps of the finger. The soles of the feet may be similarly affected. The person may complain of throbbing, tingling palms.

Neurologic symptoms, such as confusion, sleep disturbances, muscle tremors, hyperreactive reflexes, and asterixis (see following discussion), may occur. When liver dysfunction results in increased serum ammonia and urea levels, peripheral nerve function can be impaired. Ammonia from the intestine (produced by protein breakdown) is normally transformed by the liver to urea, glutamine, and asparagine, which are then excreted by the renal system. When the liver does not metabolize and detoxify ammonia, ammonia is transported to the brain, where it reacts with glutamate (excitatory neurotransmitter) to produce glutamine. The reduction of brain glutamate impairs neurotransmission, leading to altered central nervous system (CNS) metabolism and function.

Asterixis and numbness or tingling (misinterpreted as carpal tunnel syndrome) can occur as a result of this ammonia abnormality, causing intrinsic nerve pathology. Asterixis, also called *flapping tremors* or *liver flap*, is a motor disturbance; specifically, it is the inability to maintain wrist extension with forward flexion of the upper extremities. A test for asterixis is asking the client to extend the wrist and hand with the rest of the arm supported on a firm surface or with the arms held out in front of the body. Observe for quick, irregular extensions and flexions of the wrist and fingers (Fig. 17-1). Altered neurotransmission, in the form of impaired inflow of joint and other afferent information to the brainstem reticular formation, causes the movement dysfunction.

Musculoskeletal locations of pain associated with the hepatic and biliary systems include thoracic pain between

scapulae, right shoulder, right upper trapezius, right interscapular, or right subscapular areas. Sympathetic fibers from the biliary system are connected through the celiac and splanchnic (visceral) plexuses to the hepatic fibers in the region of the dorsal spine. These connections account for the intercostal and radiating interscapular pain that accompanies gallbladder disease. Although the innervation is bilateral, most of the biliary fibers reach the cord through the right splanchnic nerves, producing pain in the right shoulder.

Hepatic osteodystrophy, abnormal development of bone, can occur in all forms of cholestasis (bile flow suppression) and hepatocellular disease, especially in the alcoholic. Bone pain may occur especially in the presence of osteomalacia or more often, osteoporosis. Hepatic osteoporosis is secondary to osteoblastic dysfunction rather than to excessive bone resorption. The pathogenesis is probably complex, and factors include calcium malabsorption, alcohol, corticosteroid therapy, estrogen deficiency in the postmenopausal woman, and vitamin D deficiency with secondary hyperparathyroidism.

Vertebral wedging, vertebral crush fractures, and kyphosis can be severe; decalcification of the rib cage and pseudofractures occur frequently. Pseudofractures or Looser's zones are narrow lines of radiolucency (areas of darkness on x-ray film) usually oriented perpendicular to the bone surface. This may represent a stress fracture that has been repaired by the laying down of inadequately mineralized osteoid, or these sites may occur as a result of mechanical erosion caused by arterial pulsations, as arteries frequently overlie sites of pseudofractures.

Osteoporosis associated with primary biliary cirrhosis and primary sclerosing cholangitis parallels the severity of liver disease rather than the duration. Painful

osteoarthropathy may develop in the wrists and ankles as a nonspecific complication of chronic liver disease.

Portal hypertension, ascites, and hepatic encephalopathy are three other major complications of liver disease that are discussed in greater depth in this chapter as distinct clinical conditions.

SPECIAL IMPLICATIONS FOR THE THERAPIST 17-1

Signs and Symptoms of Hepatic Disease

Any client presenting with undiagnosed or untreated jaundice must be referred to the physician for follow-up. Active, intense exercise should be avoided when the liver is compromised (i.e., during jaundice or any other active liver disease) because the cornerstone of medical treatment and promotion of healing of the liver is rest. (See also Special Implications for the Therapist: Jaundice, Portal Hypertension, Hepatic Encephalopathy, and Ascites in this chapter.)

An increased risk of coagulopathy (decreased clotting ability) also occurs with liver disease, necessitating precautions. Easy bruising and bleeding under the skin or into the joints in response to the slightest trauma can occur when coagulation is impaired. This condition necessitates extreme care in the therapy setting, especially with any intervention requiring manual therapy or the use of any equipment, including modalities, resistive exercise or weight-training devices, and potentially the use of gait belts.

The most common neurologic abnormality associated with liver failure (liver flap or asterixis) (see Fig. 17-1) also can be observed in uremia, respiratory failure, and severe heart failure. The rapid flexion extension movements at the metacarpophalangeal and wrist joints often are accompanied by lateral movements of the digits. Sometimes movement of arms, neck, and jaws; protruding tongue; retracted mouth; and tightly closed eyelids are involved, and the gait is ataxic. The tremor is absent at rest, decreased by intentional movement, and maximal on sustained posture. It is usually bilateral, although not bilaterally synchronous, and one side may be affected more than the other. It may be observed by gentle elevation of a limb or by the client's gripping the therapist's hand. In coma, the tremor disappears.

The decreased liver weight is accompanied by diminished blood flow. This combination of decreased liver mass and blood flow may account for some changes in drug elimination observed in the older adult. Many factors may influence drug metabolism in the older adult, including changes in body composition, decline in renal function, alterations in serum albumin levels (drugs such as sulfonamides and salicylates compete with bilirubin for binding sites on albumin, a transport plasma protein), individual variability, poor compliance, lack of understanding of drug therapy, presence of other systemic illnesses, and taking multiple drugs simultaneously (drug-drug interaction).

In the past, adjusting dosage downward based on hepatic function related to aging has not been considered necessary. However, emerging data on the use of pharmacologic agents in the older population suggest that the administration of many agents (especially chemotherapeutic agents) may be affected by physiologic changes occurring with age. Dose adjustments to compensate for pharmacokinetic and pharmacodynamic changes that occur in the older adult are being studied and adjustments for specific drugs presented.⁸⁰

The liver itself becomes more fibrotic, but this change is not synonymous with pathologic cirrhotic changes. On autopsy, a buildup of brown pigment is seen, which is a result of accumulated unexcreted metabolic residue (end-stage metabolic products of lipids and proteins) acquired over a lifetime. No apparent physiologic significance has been identified with this pigment. Changes in liver oxidative status and function and the activity of the enzymatic antioxidant defense system, as well as the influence of aging on the susceptibility of the hepatic system to hepatotoxins, remain the focus of ongoing animal studies.¹¹⁸

Other studies are bringing to light the effects of physiologic age-related changes in the hepatic system such as the significance of growth hormone synthesis by the hepatic system responsible for maintaining various body tissues (e.g., bones, muscles, cardiovascular system, immune system, and CNS). Researchers are particularly interested in examining whether treatment with exogenous growth hormone can retard or reverse hepatic age-related changes in body structure and function.¹¹

The pancreas undergoes structural changes, such as fibrosis, fatty acid deposits, and atrophy, but the pancreas has a large reserve capacity, and 90% of its function would have to be lost before any observable dysfunction occurs. Much remains unknown about the gallbladder in the aging process, but aging apparently has little effect on gallbladder size, contractility, or function. The gallbladder releases less bile into the liver, allowing more time for gallstones to develop. There is some evidence that moderate and vigorous physical activity enhances the function of the gallbladder as measured by reduced risk of gallbladder removal in physically active women.⁷⁵

AGING AND THE HEPATIC SYSTEM

The liver decreases in size and weight with advancing age, requiring more time to process substances, medications, and alcohol. Liver function test results (see Chapter 40), such as levels of aspartate transaminase (AST), alanine transaminase (ALT), γ -glutamyl transpeptidase (GGTP), alkaline phosphatase (ALP), and total serum bilirubin remain unchanged and within normal limits established for the adult. However, these tests often measure hepatic damage rather than overall function; abnormal values for these tests in older adults reflect disease rather than the effects of aging.

HEALING IN THE HEPATIC SYSTEM

In general, older organs may not adapt to injury as well as younger organs, so severe hepatic illnesses (e.g., severe

hepatitis) may not be tolerated as well by the older person. Delayed or impaired tissue repair may require longer time for recovery of homeostasis. Chronic liver disease is associated with intrapulmonary shunting and reduced pulmonary vascular function. Vascular changes in the lungs have been well documented in people with chronic liver disease and reflect the importance of the liver in the control of vasoactive substances that regulate normal fluid balance, including lung fluids.²⁸

Liver injury is followed by complete parenchymal regeneration, the formation of scars, or a combination of both. The outcome depends on the extent and chronicity of the insult. Chronic hepatic injury, such as chronic viral hepatitis or alcoholic liver injury, destroys the extracellular matrix framework. This type of destruction results in a combination of regenerated nodules separated by bands of fibrous connective tissue (fibrosis), which is termed *cirrhosis*.

Orthotopic liver transplantation has become an established therapy for end-stage liver disease (e.g., cirrhosis caused by alcohol abuse or hepatitis C [HCV]), acute liver failure, and primary biliary cirrhosis or primary sclerosing cholangitis, as well as for nonalcoholic cirrhosis and hepatic or biliary malignancy. Biliary atresia is the most common indication for pediatric liver transplantation. Theoretically, anyone with advanced, irreversible liver disease with certain mortality may be considered for a liver transplant provided the disease could be corrected by liver transplantation (see Table 21-5).

Current animal research is centered on identifying and harvesting specific stem cells from the bone marrow that under special conditions will convert into functioning liver tissue. In human research, a new procedure called *hepatocyte transplantation* is being pioneered. In this procedure, billions of donor liver cells are injected by intravenous infusion into the blood with the hope that the cells will correct life-threatening liver problems that would otherwise require a liver transplant. Other research efforts are working toward the development of bioartificial devices for liver support. An effective temporary liver support system could improve the chance of survival with or without a transplant as the final treatment (see Chapter 21).

LIVER

Liver Disease Complications

As a result of the extraordinary number of vital functions the liver performs, severe complications result when the liver has been damaged or is no longer functioning. Jaundice is a symptom that occurs with many types of diseases and disorders (both acute and chronic). End-stage complications occur most often because of cirrhosis and include portal hypertension, hepatic encephalopathy, ascites, and the hepatorenal syndrome. Any illness, toxin, or infection that leads to end-stage liver disease can display these complications.

Jaundice (Icterus)

Jaundice or icterus is not a disease but is rather a common symptom of many different diseases and disorders

Box 17-2

CLASSIFICATION OF JAUNDICE

Diseases Associated with Overproduction of Bilirubin

- Hemolysis
 - Thalassemia, sickle cell anemia
 - Autoimmune hemolytic anemia
- Reabsorption of hematoma
- Blood transfusion

Decreased Uptake or Conjugation in Bilirubin Metabolism

- Gilbert's syndrome
- Jaundice of newborns
- Drugs

Hepatocyte Dysfunction

- Hepatitis
 - Viral
 - Alcohol-related
 - Autoimmune
 - Toxic/drug-induced
 - Ischemia
- Chronic hepatic disease
 - Wilson's disease
 - Hemochromatosis

Impaired Bile Flow

- Cholelithiasis
- Primary sclerosing cholangitis
- Pancreatic cancer
- Pancreatitis

(Box 17-2). It is clinically characterized by yellow discoloration of the skin, sclerae, and mucous membranes. Jaundice occurs either as a result of an overproduction of bilirubin, defects in bilirubin metabolism (in uptake by the liver or conjugation), the presence of liver disease, or obstruction of bile flow.

In the normal breakdown of hemoglobin, the end product is bilirubin. In this metabolic process, the heme portion of hemoglobin is converted into biliverdin and then to bilirubin in the bone marrow and the spleen. Bilirubin is released into the bloodstream, where it binds to albumin, and is then taken up by hepatocytes to be conjugated with glucuronic acid. Once it is conjugated, it is then released into the bile. A small percentage of conjugated bile returns to the plasma and is excreted into the kidneys. In the terminal ileum and colon, conjugated bilirubin is deconjugated and excreted as colorless urobilinogen. Diseases that result in ineffective erythropoiesis (abnormal formation of erythrocytes) produce large amounts of bilirubin due to chronic hemolysis or destruction of cells.

Some diseases, such as Gilbert's syndrome, have defects in the liver's ability to conjugate bilirubin. Drugs, such as rifampin, may compete with bilirubin for uptake by the liver, decreasing the quantity of bilirubin the liver can process. Diseases, toxins, infections, and ischemia can cause generalized liver disease (acute and chronic), which reduces the capability of the liver to function normally and process bilirubin. Finally, bile ducts can be obstructed

by diseases, tumors, and stones, leading to an elevation in bilirubin that has been conjugated.

As mentioned, jaundice is not clinically evident (particularly in the sclera of the eyes) until the plasma level reaches 3 mg/dl. Once the level reaches 5 to 6 mg/dl, the skin becomes a yellow color. Urine turns a darker color and stool is light in color. Signs and symptoms of liver disease may also be present.

Laboratory testing can aid in the specific diagnosis of jaundice. Bilirubin can be reported as conjugated or unconjugated, which provides a more accurate measurement than direct and indirect. Direct refers to the capability of the laboratory to directly measure conjugated bilirubin, while the indirect bilirubin refers to the unconjugated portion of bilirubin, which cannot be directly measured in the laboratory, and must be subtracted from the total bilirubin present in the blood.

Conjugated and direct are not always equivalent, particularly in disorders of bilirubin metabolism. In clients with jaundice, an elevation in the conjugated bilirubin is more common than unconjugated. Elevations in liver transaminases (AST and ALT) suggest that liver disease is involved. Many other tests are available for the specific diagnosis of jaundice, depending on the suspected process, and are included in the specific disease sections in this chapter.

SPECIAL IMPLICATIONS FOR THE THERAPIST 17-2

Jaundice (Icterus)

With successful treatment of the underlying cause, jaundice usually begins to resolve within 4 to 6 weeks. After this time, activity and exercise can be resumed or increased per individual tolerance, depending on the overall medical condition and presence of any complications. The return of normal stool and urine colors is an indication of resolution. (See also Special Implications for the Therapist: Signs and Symptoms of Hepatic Disease in this chapter.)

Cirrhosis

Cirrhosis is the final common pathway of chronic, progressive inflammation of the liver. It is characterized pathologically by a progressive loss of normal tissue that is replaced with fibrosis and nodular regeneration. There are many diseases, medications, and toxins that can damage the liver and ultimately lead to cirrhosis, but the most common in the United States include alcohol abuse and HCV.

Overall, in the United States, cirrhosis is the twelfth leading cause of death, accounting for about 28,000 deaths a year.⁷ Cirrhosis of the liver occurs when inflammation (from disease or toxin) causes liver tissue damage and/or necrosis. With continued cycles of inflammation and healing, fibrous bands of connective tissue replace normal liver cells. These fibrous bands eventually constrict and partition the liver into irregular nodules. Once 80% to 90% of the liver is replaced with scar tissue, there

is also significant loss of function, associated with decompensation of homeostasis.

The signs and symptoms of cirrhosis (Figs. 17-2 and 17-3) are multiple and varied, representing interference with major functions of the liver, and include processing dietary amino acids, carbohydrates, lipids, and vitamins; metabolizing cholesterol, hormones, vitamins, medications, and toxins; producing clotting factors and other plasma proteins; and storing glycogen.

Clients with cirrhosis exhibit fatigue, weight loss, jaundice, coagulopathies, loss of ability to metabolize drugs, and hypoalbuminemia (the remaining serious complications are discussed later). History, physical examination, laboratory tests, and imaging tests aid in diagnosing the specific cause. Once cirrhosis has developed, it is usually not reversible, although each disease may have a specific therapy to reduce the risk of developing cirrhosis. Typically, complications are treated on an individual basis and transplantation provides the best therapy for long-term survival.

SPECIAL IMPLICATIONS FOR THE THERAPIST

17-3

Cirrhosis

PREFERRED PRACTICE PATTERNS

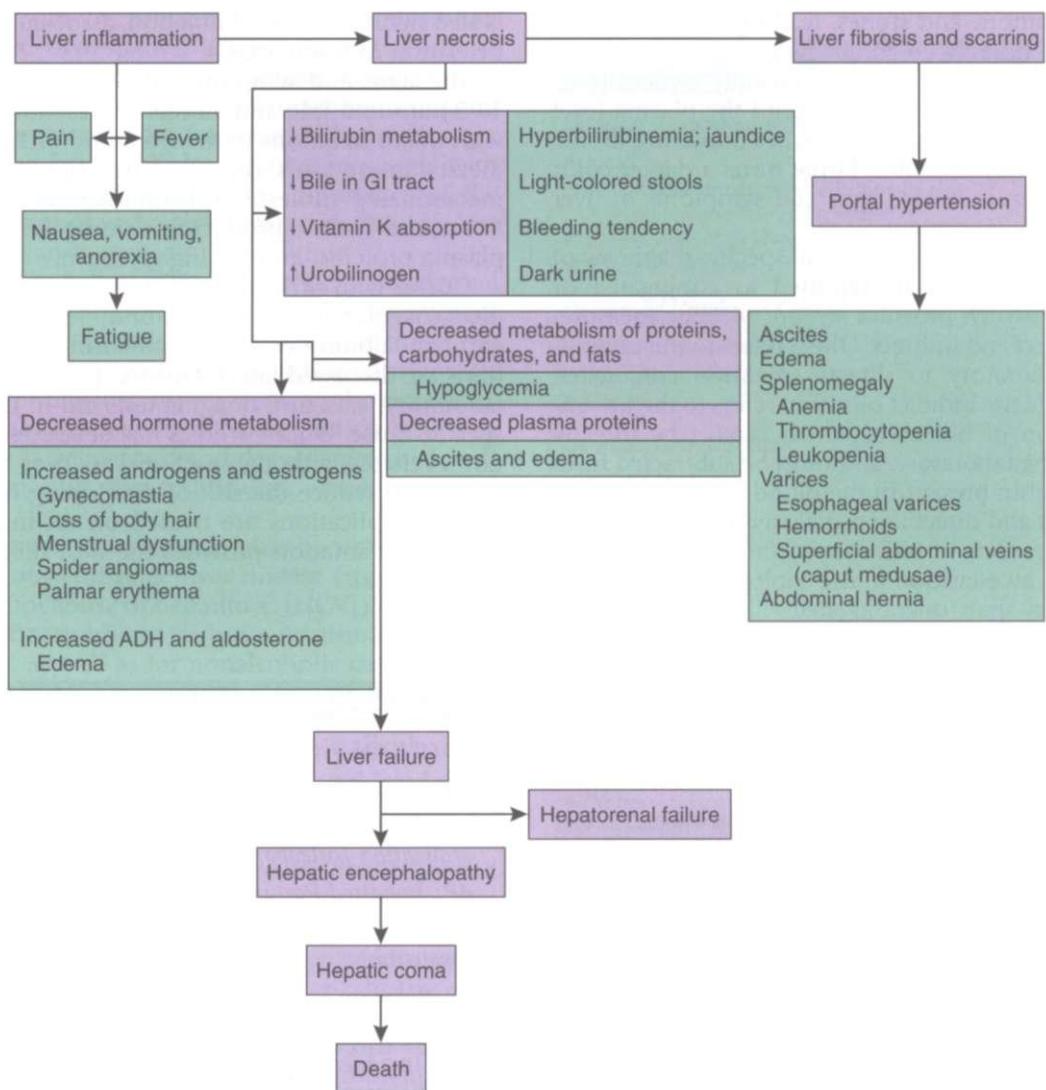
- 4A: Primary Prevention/Risk Reduction for Skeletal Demineralization (*osteoporosis*)
- 4B: Impaired Posture (*osteoporosis*)
- 4C: Impaired Muscle Performance (*osteoporosis; alcoholic myopathy*)
- 5A: Primary Prevention/Risk Reduction for Loss of Balance and Falling
- 6D: Impaired Aerobic Capacity/Endurance Associated with Cardiovascular Pump Dysfunction or Failure (*chronic alcoholic myopathy: cardiomyopathy*)

(See also Special Implications for Portal Hypertension, Ascites, Hepatitis, Encephalopathy, Anemia, Splenomegaly, Esophageal Varices, Renal Failure, or Liver Transplantation according to the client's condition.)

One of the most common symptoms associated with cirrhosis is ascites, an accumulation of fluid in the peritoneal cavity surrounding the intestines. The distention often occurs very slowly over a number of weeks or months and may be associated with bilateral edema of the feet and ankles. The client may be unable to put on a pair of shoes, preferring to leave the shoes unlaced or to wear slippers. In a home health or inpatient hospital setting, this change in dress may not be as noticeable as it would be in a private practice or outpatient clinic. It is always important to remain alert to these potential signs of fluid retention and to ask about any changes in health status or weight gain.

Detection of blood loss in the form of hematemesis, tarry stools, bleeding gums, frequent and heavy nosebleeds, or excessive bruising must be reported to the physician. Preventing increased intraabdominal pressure (see Box 16-1) and preventing injury owing to

Continued.

**Figure 17-2**

Pathologic basis (purple boxes) and resultant clinical manifestations (green boxes) associated with cirrhosis of the liver.

falls require client education regarding safety precautions.

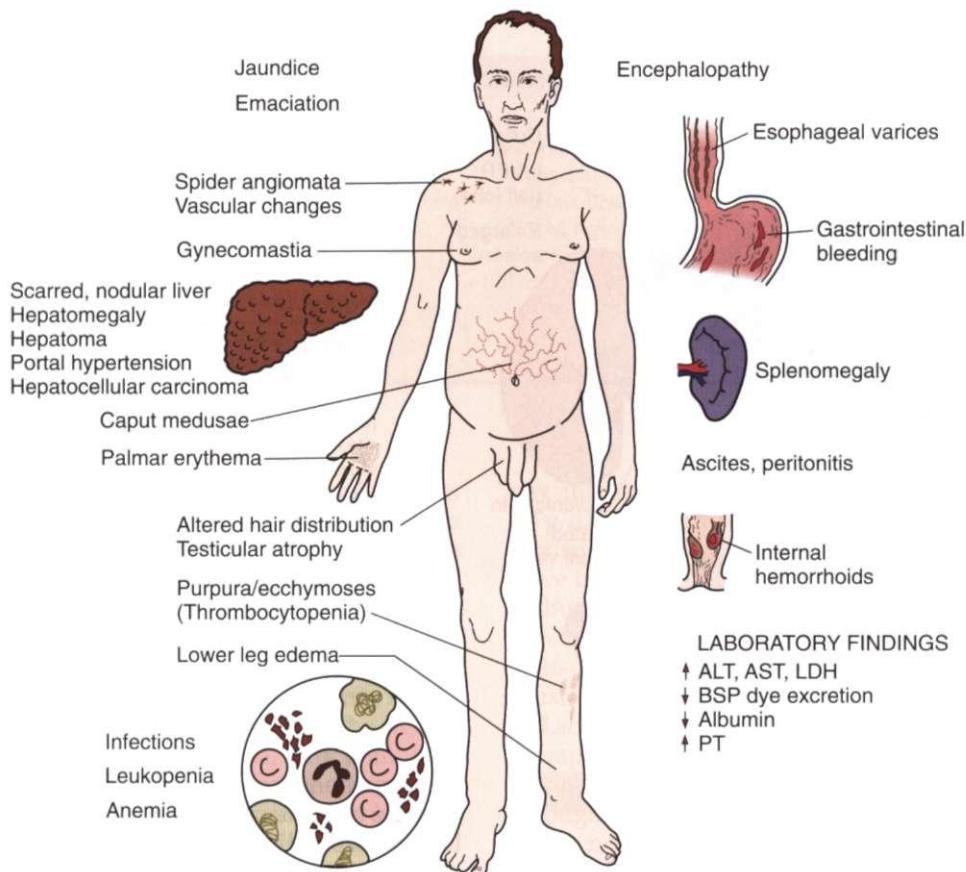
Alcohol causes whole body and tissue-specific changes in protein metabolism. Chronic alcohol use increases nitrogen excretion and reduces skeletal muscle protein synthesis with concomitant loss of lean tissue mass. Loss of skeletal collagen contributes to alcohol-related osteoporosis. The loss of skeletal muscle protein (i.e., chronic alcoholic myopathy) occurs in up to two-thirds of all chronic alcohol users. Protein turnover changes in organs such as the heart have important implications for cardiovascular function and morbidity. Most clients with cirrhosis have significantly reduced aerobic capacity, although the exact mechanism for this has not been proved.^{39,131}

Rest to reduce metabolic demands on the liver and to increase circulation often is recommended for clients with cirrhosis. Frequent rests during therapy and avoiding unnecessary fatigue are also important.

Exercise limitation in cirrhosis is typically attributed to cirrhotic myopathy without impaired oxygen utilization. Chronic alcoholic myopathy affecting the proximal muscles is usually mild and results in muscle atrophy and measurable decrease in muscle strength. The therapist must remain alert to any potential medical complications in any client, regardless of the physical therapy diagnosis.

Portal Hypertension

Portal hypertension is defined as an increase in hepatic sinusoidal (sinuses in the liver where blood flows) pressure over 6 mm Hg. Portal refers to the area where blood vessels enter into the liver. Venous blood returning from the stomach, large and small intestine, pancreas, and spleen is transported via the portal vein to the liver (the

**Figure 17-3**

Liver cirrhosis. Clinical presentation and laboratory findings associated with liver cirrhosis. ALT, Alanine aminotransferase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; BSP, sulfobromophthalein; PT, prothrombin time. (From Black JM, Matassarin-Jacobs E, eds: *Medical-surgical nursing: clinical management for continuity of care*, ed 5, Philadelphia, 1997, WB Saunders.)

splanchnic circulation). Most cases of portal hypertension are related to cirrhosis. Other causes include thrombus, tumor, infection, or may be idiopathic. With the development of cirrhosis, hyperdynamic vascular responses and mechanical factors prevent the flow of blood, resulting in increased pressure and resistance in the portal circulatory system (Fig. 17-4).

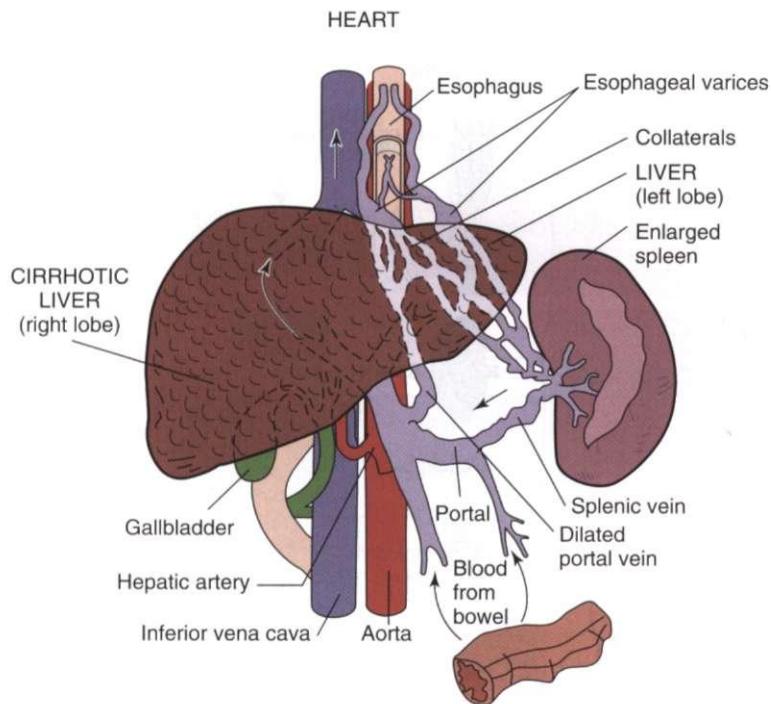
A reduction in nitric oxide release from liver endothelial cells and production of endothelin-1 leads to intrahepatic vasoconstriction, while increased blood flow from the portal vein and splanchnic circulation further increases the pressure. Fibrosis, nodularity, and abnormal liver architecture combine to form mechanical barriers to blood flow and increase the resistance.

As a result of this increased portal pressure, blood that normally flows to the portal vein is reversed and blood begins to flow back to the stomach, esophagus, umbilicus, and rectum. This system is normally small with modest blood flow. However, in the cirrhotic state, blood flow increases in these vessels, causing dilation and expansion. These engorged vessels give rise to rectal varices, prominent vessels around the umbilicus (caput medusae), and gastroesophageal varices. The collateral veins of the stomach and esophagus are the most likely to bleed because of a lack of communicating vessels.

Gastroesophageal varices are one of the most serious complications of portal hypertension, occurring in 40% of people with cirrhosis. Endoscopy should be performed in all clients with cirrhosis to screen for varices. Follow-up endoscopy is scheduled, depending on the presence and severity of varices. Clinical manifestations of gastroesophageal bleeding include hematemesis or melena (or both). The blood is usually dark red in color. Over half of bleeds stop spontaneously and over 90% of bleeds can be controlled with therapy. But serious bleeding can quickly result in hypovolemia, shock, and death. Treatment is aimed at preventing bleeding by decreasing portal blood flow and intrahepatic pressure.

Nonselective β -blockers aid in preventing bleeding and rebleeding, whereas vasopressin, octreotide, and somatostatin are used to decrease splanchnic flow. Nitrates may relieve intrahepatic vasoconstriction. Endoscopic sclerotherapy can be used prophylactically or in clients who do not tolerate medications; variceal band ligation can be utilized in acute esophageal bleeding and to prevent rebleeding. Surgical shunts or more commonly, placement of a transjugular intrahepatic portacaval shunt (TIPS) can divert blood around the liver and away from the collateral systems.

Prognosis is poor for clients with repeated esophageal varices, and liver transplantation should be pursued.

**Figure 17-4**

Portal hypertension. Normally, in the portal venous system (consisting of the portal veins, sinusoids, and hepatic veins), the portal veins carry blood from the GI tract, gallbladder, pancreas, and spleen to the liver. Veins collecting from these sites form the splenic vein and superior and inferior mesenteric veins, which in turn merge to create the portal vein. Portal hypertension occurs when portal venous pressure exceeds the pressure in the nonportal abdominal veins (e.g., inferior vena cava) by at least 6 mm Hg. As portal pressure rises, increased resistance to blood flow causes blood pooling in the spleen and the development of collateral channels formed in an effort to equalize pressures between these two venous systems. These collateral vessels or varices bypass the liver and cause large, tortuous veins, especially in the esophagus (*esophageal varices*).

SPECIAL IMPLICATIONS FOR THE THERAPIST 17-4

Portal Hypertension

PREFERRED PRACTICE PATTERNS

Patterns will depend on complications associated with portal hypertension. For example, some people with varices develop anemia or toxic neuropathy, whereas others may develop ascites and encephalopathy. The therapist should refer to the practice patterns for each of those (or other) conditions on an individual basis.

Portal pressure in individuals is dynamic, with highest pressures during the night, after eating, and in response to coughing, sneezing, and exercise. Such variations may combine with local factors in vessel walls to contribute to a pressure surge that can lead to a variceal bleed. The therapist can teach the individual how to modify and reduce pressure, especially anything that increases intraabdominal pressure (see Box 16-1) such as coughing, straining at stool, or improper lifting. Any therapy program for a client with known varices must take this factor into account when presenting active or active-assisted exercises, or unsupported gait training. (See also the section in this chapter on Esophageal Varices and Anemia.)⁴⁴

Hepatic Encephalopathy

Hepatic encephalopathy or portosystemic encephalopathy refers to a potentially reversible, decreased level of consciousness in people with severe liver disease. This complication can occur with both acute and chronic liver disease. People with chronic end-stage liver disease often have an insidious onset; initially there are mild changes in ability to concentrate and complete complex tasks. As

the hepatic encephalopathy progresses, mental status changes become more obvious and are classified in stages I to IV, depending on the severity of neurologic involvement (Table 17-1).

Stage I is characterized by impaired attention, depression, and some personality changes; neurologic signs include tremor and incoordination. Stage II displays drowsiness, sleep disorders, changes in behavior, and poor short-term memory; accompanying signs are asterixis (flapping tremor; see Fig. 17-1), ataxia, and slurred speech. The motor apraxia in stage II can be best observed by keeping a record of the client's handwriting and drawings of simple shapes such as a circle, square, triangle, and rectangle. Progressive deterioration is apparent in these handwriting samples.

Confusion and somnolence are indicative of stage III encephalopathy associated with nystagmus, clonus, muscular rigidity, and hypoactive reflexes. Stage IV reflects severe encephalopathy; the person is in a comatose state and exhibits abnormal reflexes, such as the oculocephalic ("doll's eye") reflex; decerebrate posturing may be present; pupils may be dilated; and there is no response to stimuli. There are characteristic electroencephalogram (EEG) findings at each stage.

The cause of hepatic encephalopathy has not been completely elucidated, although several key elements are known. First, laboratory animal models and clinical experience have shown that high-protein meals can lead to encephalopathy. It is felt that the liver is unable to process nitrogenous metabolites, leading to elevated levels of ammonia and other toxins (although these "other toxins" have not been well described).

The second element is the shunting of blood away from the hepatic portal system (because of cirrhosis) to the vena cava, which also worsens encephalopathy. Ammonia is one cause of encephalopathy that is fairly

Table 17-1 Stages of Hepatic Encephalopathy

Stage I (Prodromal)	Stage II (Impending)	Stage III (Arousal)	Stage IV (Comatose)
Slight personality changes	Tremor progresses to asterixis	Hyperventilation	No asterixis
Slight tremor	Resistance to passive movement	Marked confusion	Positive Babinski's reflex
Bilateral numbness/tingling	Myoclonus	Incoherent speech	Oculocephalic (doll's eye) reflex
Muscular incoordination	Lethargy	Asterixis (liver flap)	Decerebrate posturing
Apraxia	Unusual behavior (abusive, violent, noisy)	Muscle rigidity	Dilated pupils
	Apraxia	Hyporeactive deep tendon reflexes	Lack of response to stimuli
	Ataxia	Sleeps most of the time	
	Sleep disorders		
	Slow or slurred speech		

well documented but is certainly not the sole cause. Research is beginning to show how elevated ammonia and other metabolic abnormalities (including changes in neurotransmitters in the brain and circulating amino acid levels) combine to result in encephalopathy.

Ammonia is created by bacteria in the colon from the metabolism of protein and urea. Ammonia is absorbed into the portal blood system and is 5 to 10 times higher there than in the general circulatory system. The liver is typically able to metabolize ammonia, but with liver disease and shunting of blood away from the liver (particularly to the brain), ammonia levels rise. The kidneys are also a source of ammonia, which is increased in the face of diuretic use and hypokalemia. Muscles aid in ammonia removal but cirrhosis is often accompanied by muscle wasting. In the brain, ammonia appears to directly alter the function and signaling of nerve cells.

Ammonia also appears to affect glutamate and γ -aminobutyric acid (GABA) signaling. The GABA receptor complex is the principal nerve network, which inhibits the CNS. When GABA or benzodiazepines bind to this complex, there is an inhibition in the functioning of the brain. The liver contains high concentrations of GABA, and dysfunction of the liver may contribute to higher levels of GABA.

Ammonia is known to combine with α -ketoglutarate in the CNS to form glutamate, which is in turn used to produce GABA. Ammonia production is therefore tied to glutamate and GABA production. Although serum ammonia levels are used to monitor therapy, the level does not correspond well with the severity of encephalopathy, reinforcing the fact that other mechanisms are involved in the development of encephalopathy.

The development of hepatic encephalopathy warrants a careful evaluation and correction of the cause. Serious and common causes include bleeding, infection (particularly spontaneous bacterial peritonitis), hypovolemia, or electrolyte abnormalities (hypokalemia). Other common factors that may precipitate or severely aggravate hepatic encephalopathy include constipation, diuretics, increased dietary protein, and CNS-depressant drugs, such as alcohol, benzodiazepines (e.g., Librium, Valium, Dalmane, and Tranxene), and opiates.

Because protein can precipitate or worsen encephalopathy, many of the symptoms can be improved by eliminating or reducing sources of protein (i.e., stopping

any internal bleeding and restricting dietary protein to 60 g/day). Health care providers must also be aware of any subtle changes in mental status since the ability to drive is often impaired. In one study, clients with cirrhosis but no evidence of hepatic encephalopathy underwent psychomotor testing; 60% were found unfit to drive and 25% displayed questionable driving skills.¹⁴⁷ Driving must be assessed on a case-by-case basis.

Lactulose (or Lactitol) is a first-line pharmacologic therapy that decreases nitrogenous compounds from being absorbed from the gut and increases transit time through the intestine (the goal is 2 to 4 bowel movements a day). Ammonia-lowering therapy in suspected encephalopathy cases can be beneficial even when the ammonia level is normal since the production is tied to other toxins.

Neomycin is an antibiotic that decreases the bacterial count in the intestine but carries nephrotoxicities and ototoxicities as a result of systemic absorption. The new medication rifaximin (a nonabsorbable antibiotic) has been shown in preliminary studies to be effective in hepatic encephalopathy.⁹³ Flumazenil is a benzodiazepine-receptor antagonist that may block the GABA receptor complex, reversing inhibition. It has been shown to improve hepatic encephalopathy in the short term but has no effect on survival.³

Reversal of hepatic encephalopathy is typically successful when a source is identified, corrected, and treated appropriately. However, without intervention, mortality is high, as the person's condition progresses into coma. Similar to most complications of end-stage liver disease, liver transplantation provides the best long-term treatment.

SPECIAL IMPLICATIONS FOR THE THERAPIST

17-5

Hepatic Encephalopathy

PREFERRED PRACTICE PATTERNS

See also Anemia and Ascites.

5A: Primary Prevention/Risk Reduction for Loss of Balance and Falling

5E: Impaired Motor Function and Sensory Integrity Associated with Progressive Disorders of the Central Nervous System (alcoholic ataxia)

Continued.

5G: Impaired Motor Function and Sensory Integrity Associated with Acute or Chronic Polyneuropathies

5I: Impaired Arousal, Range of Motion, and Motor Control Associated with Coma, Near Coma, or Vegetative State

7A: Primary Prevention/Risk Reduction for Integumentary Disorders (pressure ulcer secondary to malnutrition, immobility, edema)

The inpatient or homebound client with hepatic encephalopathy has difficulty ambulating and is extremely unsteady. Protective measures must be taken against falls. The home health therapist must be alert for any report of GI bleeding that will result in protein accumulation in the GI tract, exacerbating this condition (e.g., blood in stools or black or tarry stools). The client should be following a low-protein diet.

The physician may prescribe lactulose to decrease ammonia in the bowel, but diarrhea is a side effect. The client experiencing prolonged diarrhea should be encouraged to report this information to the physician for possible follow-up. A reduced dosage may be required to prevent further electrolyte imbalance. (See the section on Electrolyte Imbalance in Chapter 5.)

The immobile client who lacks reflexes is vulnerable to numerous complications requiring attention to the prevention of pneumonia and skin breakdown. Skin breakdown in a client who is malnourished from liver disease and is immobile, jaundiced, and edematous can occur in less than 24 hours. Careful attention to skin care, passive exercise, and frequent changes in position are required.

Rest between activities is advocated, and strenuous exercise is to be avoided. The therapist should watch for (and immediately report) signs of anemia (e.g., reduced hemoglobin, weakness, dyspnea on exertion, easy fatigability, skin pallor, or tachycardia; see the section on Anemia in Chapter 14), infection (see Box 8-1), and GI bleeding (e.g., melena, hematemesis, easy bruising).

Ascites

Ascites is the abnormal accumulation of fluid within the peritoneal cavity. Ascites is most often caused by cirrhosis (85% of cases), but other diseases associated with ascites include heart failure, abdominal malignancies, nephrotic syndrome, infection, and malnutrition.

The mechanism for the accumulation of fluid in the case of cirrhosis is principally a result of portal hypertension. High pressure in the vessels attempting to pass blood through the cirrhotic liver leads to vasodilatation of the splanchnic vessels (vessels to the gut or viscera), which in turn decreases the filling of the vessels going to the kidney. The renin-angiotensin-aldosterone system is activated, resulting in sodium and water retention. However, because of the high pressure in the liver and splanchnic vessels, excessive lymph is produced that leaks into the tissues and eventually into the abdominal cavity.¹⁰

Ascites becomes clinically detectable when more than 500 ml has accumulated, causing weight gain, abdominal

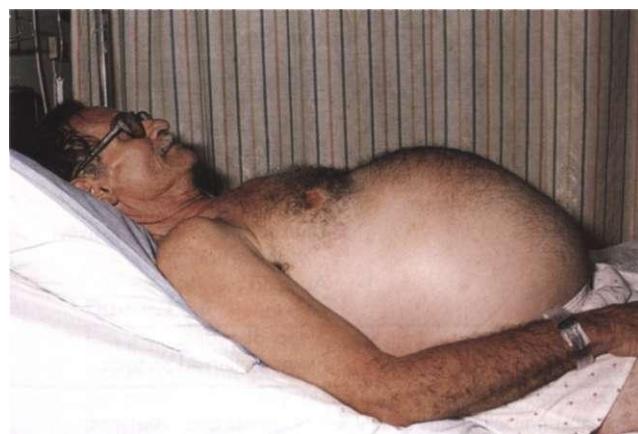


Figure 17-5

Ascites in an individual with cirrhosis. Distended abdomen, dilated upper abdominal veins, and inverted umbilicus are classic manifestations. Peripheral edema associated with developing ascites may be observed first by the therapist. (From Swartz MH: *Textbook of physical diagnosis*, ed 5, Philadelphia, 2006, WB Saunders.)

distention, increased abdominal girth, and eventually, peripheral edema (Fig. 17-5). Dyspnea with increased respiratory rate occurs when the fluid displaces the diaphragm.

Diagnosis of ascites is usually based on clinical manifestations in the presence of liver disease. Paracentesis is used as the initial test in people with new-onset ascites to determine the cause. Fluid is sent to the laboratory for chemical and microscopic evaluation. Abdominal ultrasonography can aid in locating pockets of ascitic fluid that may be loculated (formed or divided into small cavities or compartments).

In people with established cirrhosis, paracentesis can be diagnostic and therapeutic. Large volume paracentesis with administration of albumin is the treatment of choice for tense ascites (i.e., when a person is no longer able to breathe or eat comfortably), followed by the use of diuretics to reduce reaccumulation of fluid.

Treatment of mild-to-moderate ascites includes sodium restriction accompanied by diuretic use.¹⁴⁰ Fluid restriction is appropriate when the serum sodium decreases to less than 120 to 125 mEq/L. True refractory ascites (i.e., meaning not responsive to therapy) is uncommon and can be treated with serial large-volume paracentesis or TIPS. This alleviates pressure in the portal area but may induce hepatic encephalopathy caused by bypassing the liver (see Hepatic Encephalopathy section).

Prophylactic antibiotics may be used in some clients with a history of *spontaneous bacterial peritonitis* (SBP) and are often administered to people with a GI bleed.¹⁷² The development of refractory ascites is associated with a poor prognosis, with a 12-month survival of 25%. Liver transplantation provides the best treatment option but is not always readily available.

Complications include hepatorenal syndrome (discussed later) and SBP. The microbial source of SBP (infection of the ascitic fluid) is the gut, where organisms (typically *Escherichia coli*, streptococci [mostly pneumococci], and *Klebsiella*) are translocated into lymph nodes

and then into the ascitic fluid. Bacterial peritonitis is symptomatic in 87% of cases (fever, chills, abdominal pain, mental status changes, and tenderness), but symptoms can often be subtle. Without diagnosis by paracentesis and antibiotic treatment, the infection can be fatal.

SPECIAL IMPLICATIONS FOR THE THERAPIST 17-6

Ascites

PREFERRED PRACTICE PATTERNS

See *Cirrhosis*.

6A: Primary Prevention/Risk Reduction for Cardiovascular/Pulmonary Disorders (prevention of pneumonia/atelectasis)

6E: Impaired Ventilation and Respiration/Gas Exchange Associated with Ventilatory Pump Dysfunction or Failure (decreased lung capacity; elevated diaphragm)

6H: Impaired Circulation and Anthropometric Dimensions Associated with Lymphatic System Disorders (lymphedema)

7A: Primary Prevention/Risk Reduction for Integumentary Disorders (peripheral edema; lymphedema)

Most people with ascites are more comfortable in a high Fowler's position (head of the bed raised 18 to 20 inches above the level with the knees elevated). Breathing techniques are important to maintain adequate respiratory function and to prevent the development of atelectasis or pneumonia. The homebound person who has ascites should be monitored for the possible development of bacterial peritonitis. Onset of fever, chills, abdominal pain, and tenderness should be reported to the physician.

Decreases in serum albumin associated with the development of ascites are accompanied by a parallel decrease in oncotic pressure in blood vessels causing peripheral edema. Persons with serum albumin levels between 2.5 and 3.5 g/dl are at moderate risk of malnutrition, and those whose levels are 2.5 g/dl or less are at great risk (see Table 40-5). Because albumin binds to calcium, serum albumin levels drop when serum calcium levels are low.

The edema associated with ascites may mask muscle wasting that occurs when the body does not have an adequate intake of protein to maintain structure and facilitate wound healing. The client must be encouraged to change position to maintain integrity of the skin and promote circulation. Small pillows or folded towels can be used to support the rib cage and the bulging flank while the client is lying on his or her side.

The abdominal distention associated with ascites may develop very slowly over a number of weeks or months and may be accompanied by bilateral edema of the feet and ankles. The client may be unable to put on a pair of shoes, preferring to leave the shoes unlaced or to wear slippers. In a home health, inpatient hospital, or nursing home setting, this change in dress may not be as noticeable as it would be in a private practice or outpatient clinic. It is always important to remain alert to these potential signs of fluid retention and to

ask about any changes in health status or weight gain.

Fluid intake and output are usually carefully measured and restricted, so in any setting the therapist is encouraged to know the individual's limits and to participate in reporting measurements as well. This is especially important because clients frequently ask the therapist for fluids in response to perceived exertion or increased exertion after exercise or ambulation. The person who is noncompliant or in denial requests fluids because of the false belief that fluids provided but not recorded do not count. For the homebound client who is receiving diuretics, the bedroom should be close to the bathroom.

Hepatorenal Syndrome

Hepatorenal syndrome is characterized by renal dysfunction in people with portal hypertension and advanced liver failure, but with normal renal tubular function (i.e., the kidney tubular cells function). About 7% to 15% of people with end-stage cirrhosis will develop this syndrome, which portends a poor prognosis. This syndrome is hypothesized to occur as a result of portal hypertension in which increased vascular pressure from liver disease causes vasodilation of the blood vessels in the splanchnic circulation (the body's attempt to reduce this pressure). This vasodilation leads to underfilling of the arteries and a low blood pressure elsewhere in the body, which particularly affects the blood pressure in the kidney (which is very sensitive to blood pressure). Because the blood pressure the kidney "sees" is low, there is an activation of the renin-angiotensin-aldosterone system in an attempt to constrict the blood vessels. This leads to constriction of the vessels in the limbs and to the brain, as well as the kidneys. The total effect is that the vasoconstrictors have a greater effect than the vasodilators, and kidney dysfunction develops.⁴⁶

Criteria defining the diagnosis of hepatorenal syndrome were established by the International Ascites Club and include the presence of advanced liver disease with portal hypertension; serum creatinine of greater than 1.5 mg/dl; urine protein of less than 500 mg/dl; and most importantly, the absence of other causes for kidney involvement.

Common illnesses found in people with cirrhosis that can cause renal insufficiency include infection (particularly spontaneous bacterial peritonitis), shock, medications, bleeding, and fluid losses. Renal obstruction should be "ruled out" by ultrasonography. With the presence of these features, along with the failure to improve after diuretics are removed and 1 to 1.5 liters of saline given, suggests the diagnosis of hepatorenal syndrome.

Hepatorenal syndrome is classified into two types. Type 1 is rapid both in onset and progression to renal failure and carries a poor short-term prognosis. Type 2 is more insidious in onset with progression over months; ascites is often the key feature of this type. Because of the intense vasoconstriction, treatment centers around the

use of vasodilators and albumin, which aid in increasing blood flow to the kidneys (such as vasopressin analogues or proportional variant-adrenergic agonists). Transjugular intrahepatic portosystemic shunts (TIPS) may also be of benefit.⁹

Optimal treatment consists of liver transplantation, which provides a 5-year posttransplant survival rate of 70%. Although many people will improve with medical treatment, liver transplantation should be pursued because of poor long-term prognosis. Hemodialysis may be required to bridge treatment until a transplant is available.

Hepatitis

Hepatitis is an acute or chronic inflammation of the liver caused by a virus, a chemical, a drug reaction, or alcohol abuse. Classifications of hepatitis discussed in this text are listed in Box 17-2. Six different identifiable hepatitis viruses (A, B, C, D, E, and G) are responsible for more than 95% of all viral-induced cases worldwide. The letter F was not skipped; it represents the fulminant form of hepatitis, the generic term for any rapidly progressing form of liver inflammation resulting in hepatic encephalopathy within a few weeks of developing infection. Fulminant hepatitis is not strictly viral induced but can develop as a result of any form of hepatitis.

Other viral causes of hepatitis include Epstein-Barr virus (mononucleosis), herpes simplex virus types I and II, varicella-zoster virus, measles, or cytomegalovirus (CMV). Hepatitis from any cause produces very similar symptoms and usually requires a careful client history to establish the diagnosis.

People with mild-to-moderate acute hepatitis rarely require hospitalization. The emphasis is on preventing the spread of infectious agents and avoiding further liver damage when the underlying cause is drug-induced or toxic hepatitis. Persons with fulminant hepatitis (which has a severe, sudden intensity and is sometimes fatal) require special management because of the rapid progression of the disease and the potential need for urgent liver transplantation.

Chronic Hepatitis

Chronic hepatitis comprises several diseases that are grouped together because they have common clinical manifestations and are all marked by chronic necroinflammatory injury that can lead insidiously to cirrhosis and end-stage liver disease. The disease is defined as chronic with evidence of ongoing injury for 6 months or more.

Previously, chronic hepatitis was classified as either chronic persistent hepatitis (CPH) or chronic active hepatitis (CAH), but recent advances in understanding of the causes and natural history of this type of liver injury have led to the elimination of these terms. Now chronic hepatitis is described in diagnostic terms that include the etiology, degree of active inflammation and injury (i.e., grade: mild, moderate, severe, or I, II, III), and the degree of scarring or how advanced the process is (i.e., stage: I, II, or III; IV represents cirrhosis). Stages of disease are usually irreversible.

Chronic hepatitis has multiple causes, including viruses, medications, metabolic abnormalities, and autoimmune disorders. Despite extensive testing, some cases cannot be attributed to any known cause and are probably the result of as yet unidentified viruses. Hepatitis B (HBV), with or without hepatitis D (HDV); HCV; and hepatitis G (HGV) can progress to chronic hepatitis.

Most people with chronic hepatitis are asymptomatic, and when symptoms occur, these are nonspecific and mild, with fatigue, malaise, loss of appetite, polyarthralgias, and intermittent right upper quadrant discomfort. Some people report sleep disturbances or difficulty in concentrating. Symptoms of advanced disease or an acute exacerbation include nausea, poor appetite, weight loss, muscle weakness, itching, dark urine, and jaundice. Once cirrhosis is present, weakness, weight loss, abdominal swelling, edema, easy bruising, muscle wasting and weakness, gastrointestinal bleeding, and hepatic encephalopathy with mental confusion may arise.

The diagnosis of chronic viral hepatitis is based on serologic testing. The strict definition of chronic hepatitis (from any cause) is based on histologic features of hepatocellular necrosis and chronic inflammatory cell infiltration in the liver, but the diagnosis can usually be made from clinical features and blood test results alone.

Liver biopsy is important to assess the severity of underlying liver disease (grade and stage) and to determine the need for antiviral treatment. The treatment for chronic viral hepatitis has improved substantially in the last decade and depends on the underlying cause and grade and stage of disease. With the advances currently being made in the fields of antiviral and immunomodulatory therapeutics, it is anticipated that the considerable progress made in treating these diseases over the past decade will continue in the future.

The prognosis in chronic hepatitis is variable depending on the development of cirrhosis and other complications such as hepatocellular carcinoma (HCC). Male gender, moderate-to-severe alcohol consumption, and other coexistent liver disorders are the factors that increase the rate of progression to cirrhosis; HCV is the major risk factor of development of liver cancer. The 5-year survival rate for compensated cirrhosis is greater than 90%, but the prognosis and survival rate for decompensation (characterized by development of variceal bleeding, ascites, and hepatic encephalopathy) are extremely poor. Progression of chronic hepatitis to decompensated cirrhosis is an indication for liver transplantation.

Fulminant Hepatitis

Fulminant hepatitis is the generic term for any rapidly progressing form of liver inflammation that results in hepatic encephalopathy (confusion, stupor, and coma) within a few weeks of developing infection. This type of hepatitis is rare, occurring in less than 1% of persons with acute viral hepatitis, but can be fatal. The most common causes are acetaminophen hepatotoxicity (20% to 25% of all cases of fulminant hepatitis),¹⁰ idiosyncratic drug reaction, hepatitis A (HAV) and HBV, and hepatic ischemia. Encephalopathy may progress to cerebral edema, which is the most common cause of death. In addition to liver failure, numerous complications can occur,

including infection, hypoglycemia, coagulation defects, lactic acidosis, GI hemorrhage, electrolyte disturbances, and renal insufficiency.

Diagnosis is made in the presence of a combination of hepatic encephalopathy, acute liver disease (elevated serum bilirubin and transaminase levels), and liver failure. Treatment is supportive because the underlying etiologic factors of liver failure are rarely treatable short of liver transplantation.

Prognosis is determined in part by the cause of the condition. If the prognosis is deemed poor and no contraindications to transplantation are present, the person should be immediately considered for transplantation. Short-term prognosis without liver transplantation is very poor, and the mortality rate is high (80%). Despite the poor prognosis, complete recovery can occur as a result of liver cell regeneration with recovery of liver function. The development of artificial liver support devices has not been shown to improve outcome nor are they widely available.

Viral Hepatitis²¹

Overview. Each of the recognized hepatitis viruses belongs to a different virus family, and each has a unique epidemiology. Characteristics of these strains of viruses are presented in Table 17-2. The identification of the specific virus is made difficult by the fact that a long incubation period often occurs between acquisition of the infection and development of the first symptoms. The incubation period for HAV is 15 to 50 days, 1 to 6 months for HBV, and 1 week to 6 months for HCV. Not all causative agents have been identified, and because hepatitis can be easily spread before symptoms appear, morbidity is high in terms of loss of time from school or work. More than half and possibly as many as 90% of all cases go unreported because symptoms are mild or even subclinical.

Hepatitis A virus (HAV), formerly known as *infectious hepatitis*, is transmitted by the oral-fecal route. The oral-fecal route of transmission is primarily from poor or improper handwashing and personal hygiene, particularly after using the bathroom and then handling food for public consumption. This route of transmission also may occur through the shared used of oral utensils such as straws, silverware, and toothbrushes.

Major outbreaks of HAV occur when people consume contaminated water or food. HAV most commonly affects children, men who have sex with men (MSM), and people who live or travel in underdeveloped countries (Table 17-3). HAV is rarely transmitted through transfused blood, and little placental transmission occurs, although the antibody is often detected in infants of infected mothers.

HAV is highly contagious, with the peak time of viral excretion and contamination occurring during the 2-week period *before* the onset of jaundice. Thus the greatest danger of infection is during the incubation period, when a person is unaware that the virus is present. The illness can last from 4 to 8 weeks; it generally lasts longer and is more severe in persons older than 50 years or in people with chronic, underlying liver disease.⁴²

Hepatitis B virus (HBV) is transmitted percutaneously (i.e., puncture of the skin) or through mucosal contact.

HBV is highly infectious: 100 times more infectious than human immunodeficiency virus (HIV) and 10 times more infectious than HCV. Because HBV can be transmitted through heterosexual or homosexual intercourse, it is considered a sexually transmitted disease. The average incubation period is 90 days (with a range of 60 to 150 days) with symptoms occurring around 60 days.⁹⁴

Hepatitis C virus (HCV), formerly posttransfusion non-A, non-B hepatitis associated with blood transfusion, is now most commonly associated with injection-drug use. As with HAV, the period of infectivity begins before the onset of symptoms, and the person may become a lifetime carrier of this virus. Clinically, HCV is very similar to HBV and often is asymptomatic; the acute HCV infection is usually mild. Chronic HCV varies greatly in its course and outcome from asymptomatic with normal liver function to mild degree of liver injury and overall good prognosis to severe symptomatic HCV with complications of cirrhosis and end-stage liver disease.

Hepatitis D virus (HDV), or delta virus, is a defective single-stranded RNA that presents as a coinfection or superinfection of HBV. This virus requires hepatitis B surface antigen (HBsAg) for its replication, so only individuals with HBV are at risk for hepatitis D. Risk factors and transmission mode are the same as for HBV; parenteral drug users have a high incidence of HDV. The symptoms of HDV are similar to those of HBV except that clients are more likely to have fulminant hepatitis and to develop chronic active hepatitis and cirrhosis.

Hepatitis E virus (HEV), previously known as enteric non-A, non-B hepatitis, is transmitted by contaminated water via the oral-fecal route and clinically resembles HAV. It is believed to be nonfatal, although it has been clearly associated with liver damage. A 20% to 25% mortality rate exists in pregnant women from fulminant hepatitis.² This virus tends to occur in poor socioeconomic conditions, primarily occurs in developing countries (contaminated waste water and sewage), and is rare in the United States. No specific treatment is available for HEV, but ensuring clean drinking water remains the best preventive strategy.

Hepatitis G virus (HGV) is most prevalent in African countries. It is parenterally transmitted, although vertical and sexual transmissions are well documented. Parenteral refers to transmission in some other mode than via the alimentary canal (enteric system) such as by subcutaneous, intramuscular, intraorbital, intracapsular, intraspinal, intrasternal, or intravenous injection. It may cause acute or chronic infection, but little information about this viral cause of hepatitis is known.¹⁴⁶ HGV has been identified as the causative agent of approximately 20% of posttransfusion hepatitis cases and approximately 15% of community-acquired hepatitis cases that are not caused by HAV. Although present in liver tissue, pathogenicity to the liver has not been proved.

Incidence and Risk Factors. Each year, approximately 500,000 Americans are infected with some form of hepatitis virus; annually about 15,000 persons die from its complications. HAV is the predominant type of hepatitis, causing 40% to 60% of acute viral hepatitis cases, although the number of affected people in the United States has been on the decline, with about 4500

Table 17-2 Types of Viral Hepatitis

	Hepatitis A (HAV)	Hepatitis B (HBV)	Hepatitis C (HCV)	Hepatitis D (HDV)	Hepatitis E (HEV)
Incidence	Less than 4500 cases reported to CDC in 2005; reduced incidence with introduction of hepatitis A vaccine (approximately 42,000 new cases occurred)	Reduced incidence; 51,000 new acute cases in the US (2005); 1.5 million carriers	Transfusion-related cases decreasing with blood screening but increased incidence expected related to risk behaviors in the 1960s and 1970s; 3.2 million chronically infected	Uncommon in US; most common in drug addicts, sexually active young adults, individuals receiving multiple transfusions	Epidemic in developing countries; rare in US; risk greatest to persons traveling to endemic regions
Morbidity	Results in acute infection only; does not progress to chronic hepatitis or cirrhosis; small risk of fulminant hepatitis; lifetime immunity	Most common cause of chronic hepatitis and liver cancer; second major cause of cirrhosis in the United States after alcohol abuse	Accounts for 60% to 70% of all chronic hepatitis; 30% of chronic cases progress to cirrhosis; associated with liver cancer	Coinfection of HBV and HDV leads to more severe acute disease (fulminant hepatitis 2%-20%) but low risk of chronic disease Superinfection (acquire HDV after HBV) has high risk of severe chronic disease (70%-80%)	Causes acute self-limiting infection; does not progress to chronic hepatitis; high mortality in pregnant women, 10% mortality
Transmission	Fecal-oral route; spread by feces, saliva, and contaminated food and water	Parenteral Sexual contact Vertical Unidentified exposure	Parenteral Unidentified exposure	Parenteral Sexual contact Same as B; perinatal rare requires coinfection with HBV to reproduce	Same as A; fecal-oral (contamination of water)
Treatment	Immune globulin before or within 2 weeks of exposure; supportive; most people recover within 4-8 wks	Alpha interferon and antiviral agents for chronic HBV; HBIG for exposed, unvaccinated persons	Combination therapy (interferon, ribavirin) in select cases	Interferon α -2b can inhibit HDV replication, but effect ends when therapy ends	None; preventive measures
Diagnosis	Blood test to identify antibody; IgM; anti-HAV	Blood tests to identify antigen and antibodies; HBsAg; HBeAg; HBcAg; see text under diagnosis	Blood test to identify antibody; does not distinguish between current and past infection; anti-HCV Limited use of nucleic acid test (polymerase chain reaction)	Blood test to detect antigen and antibody; anti-HDV	Blood test to detect anti-HEV IgM antibodies
Vaccine	Vaccine available; combined HAV and HBV vaccine available (Twinrix)	Vaccines available; see text	None available	Immunization against HBV can prevent HDV infection	Recombinant vaccine (rpHEV vaccine); IgM antibodies under investigation

From Centers for Disease Control and Prevention (CDC); National Center for Infectious Diseases (online); <http://www.cdc.gov/diseases/hepatitis>. 2007. HBsAg, Hepatitis B surface antigen; HBeAg, hepatitis B "e" antigen; HBcAg, hepatitis B core antigen; IgM, immunoglobulin M.

acute cases reported in 2005 (probably 42,000 new cases occurred when taking into account asymptomatic and unreported cases).¹⁶⁷

Since HAV is transmitted via the fecal-oral route, one risk factor for acquiring the virus is working at a daycare center; children who attend daycare centers are also at

higher risk. Because of the HAV vaccine the infection rate in this population has improved; this may change the prevalence of the disease, with more cases being reported in adults (adults display a more severe clinical course than children).¹⁶⁸ Another risk factor for HAV is visiting or living in an underdeveloped country where the rate is high.

Table 17-3 Risk Factors for Hepatitis

Hepatitis A (HAV)	Hepatitis B (HBV)	Hepatitis C (HCV)	Hepatitis D (HDV)	Hepatitis E (HEV)
Household contacts or sexual contacts of infected persons	Injection-drug use	Current or previously used injected illegal drugs (even if only 1 or 2 times years ago); intranasal cocaine use with shared equipment	Same as B	Same as A
Unprotected homosexual/bisexual activity	Unprotected homosexual/bisexual activity; persons with multiple sex partners or diagnosis of sexually transmitted disease			
Injection/non-injection illegal drug users (regional outbreaks reported)	Incarceration in correctional facilities—adults and youth (drug use, unsafe sexual practices)	Received blood transfusion or organ transplant before July 1992 or blood clotting products made before 1987		
Living in areas with increased rates of HAV (children at greatest risk)	Certain ethnic groups and adoptive families with adoptees from these areas: Asia, South America, South Africa, Mexico, Eastern and Mediterranean Europe			
Travel in areas where HAV is epidemic	Travel to high-risk areas			
Tattoo inscription or removal; body or ear piercing with shared or unsterile needles	Occupational risk*: morticians, dental workers, emergency medical technicians, firefighters, health care workers in contact with body fluid or blood	Tattooing/ body piercing as a risk factor for HCV has not been completely evaluated in the US but does not appear likely		
	Liver transplant recipient	Evidence of liver disease, liver transplant recipient		
	Infants born to mothers with HBV	Infants born to HCV-infected mothers (low risk: 5%)		
	Immunocompromised individuals; receiving/administering chronic kidney dialysis (clients/staff)	Long-term kidney dialysis (clients/staff)		
Blood clotting factor disorder (no new cases between 1998 and 2002)	Multiple blood product or blood transfusions before July 1992			

From Centers for Disease Control and Prevention (CDC): National Centers for Infectious Diseases (online): <http://www.cdc.gov/ncidod/diseases/hepatitis.2007>.

*HBV can also survive in dried blood at least 1 week.

The incidence of HBV has also declined, with only 5400 acute cases reported in 2005 and a total of about 51,000 new cases (if asymptomatic and reported cases are taken into account).¹⁶⁷ Prevalence of HBV infection has significantly decreased in most ethnic populations except blacks, who continue to demonstrate an elevated prevalence of three times that of other racial/ethnic populations.⁹⁴ Prevalence varies most according to risk factors for acquisition, so for parenterally transmitted hepatitis (B, C, D, and G), the highest rates are among persons with direct percutaneous blood exposures such as injection-drug users.

Common risk factors for HBV include sexual relations, injection-drug use, sharing needles, needlesticks, and perinatal (vertical) transmission from mother to child. Injection-drug use and intimate contact with another person with HBV are the two most frequent sources of HBV in the United States. Transfusion-related HBV is rare, since the initiation of donor screening for HBV and the HBV vaccine have improved the incidence in dialysis clients and workers. Only 670 new cases of HCV were reported in 2005; including asymptomatic cases, the actual number of new cases was probably close to 20,000 cases. Men were more likely to contract HCV than women,

but the incidence was fairly equal among various ethnic groups/populations. HCV deceased 5.8% among the age group of 20 to 39 years, where it had been the highest.

In the past, HCV infection was commonly acquired through blood transfusion. Currently, because of donor screening, the incidence of HCV from transfusion is uncommon. The number of people with chronic HCV is significant: about 3.2 to 4 million people are chronically infected with HCV. Most of these cases were acquired during the 1970s and 1980s when the rates were the highest.

Risk factors for HCV are similar to those for HBV. In 2005, injection-drug use was the most common risk factor and having multiple sex partners second (although transmitting HCV by sexual contact is inefficient). Currently, 68% of the new cases of HCV occur among injection-drug users sharing needles. Up to one third of individuals with a bleeding disorder are coinfecte with HIV and persons with a bleeding disorder who were infected with HIV also contracted HCV (before screening for HCV was made available). Vertical transmission is not common in HCV. See Table 17-3 for other risk factors.

The number of cases of people who developed HBV/HCV infection from dialysis or work-related exposure has become low. People undergoing chronic hemodialysis are at risk for HBV/HCV infection because the process of hemodialysis requires vascular access for prolonged periods. In an environment where multiple clients receive dialysis concurrently, repeated opportunities exist for person-to-person transmission of infectious agents, directly or indirectly via contaminated devices, equipment and supplies, environmental surfaces, or hands of personnel. Furthermore, individuals receiving hemodialysis are immunosuppressed, which increases their susceptibility to infection, and they require frequent hospitalizations and surgery, which increases their opportunities for exposure to nosocomial infections.¹⁰⁶

HBV is relatively stable in the environment and remains viable for at least 7 days on environmental surfaces at room temperature. HBsAg has been detected in dialysis centers on clamps, scissors, dialysis machine control knobs, and doorknobs. Thus blood-contaminated surfaces that are not routinely cleaned and disinfected represent a reservoir for HBV transmission. Dialysis staff members can transfer virus to clients from contaminated surfaces by their hands or gloves or through use of contaminated equipment and supplies.¹⁰⁶

HEV is most common in developing countries. On the basis of serological tests, an estimated one-third of the world's population has been infected with HEV. In India, the lifetime prevalence is more than 60%.¹⁵³

Pathogenesis. The viruses associated with hepatitis are not typically cytopathic (destroy cells), yet the body's reaction to the virus often creates significant inflammation; the intensity of the disease depends on the degree of immune response.^{18,135} Initially, cytokines (interferons) and natural killer cells are employed to remove virus from the body. Later, antigen-specific T cells (matured in the lymph tissue) enter the liver to aid in the removal of virus; antibodies prevent spread of virus and provide immunity against further infection. In adults with intact immunity, this response is able to clear the virus.

However, in infants, young children, and the immunosuppressed, the immune system is unable to mount an adequate defense and the virus continues to replicate and reside in the liver, leading to a chronic state. In these people, there is a weak response with few antigen-specific T cells.

In HCV, the virus is able to bypass the immune system in most cases. Current theories include T-cell exhaustion, T-cell dysfunction, viral escape mutations, or rapid T-cell deletion in the liver.¹¹⁴ Antibodies are not produced soon after infection with HCV and in some cases do not develop at all.

Clinical Manifestations. Most cases of acute viral hepatitis are asymptomatic and never reported (for HAV, HBV, and HCV). Up to 70% of children under the age of 6 years remain asymptomatic with HAV infection.⁴² Infants, children under the age of 5, and immunosuppressed adults typically have no symptoms associated with acute HBV infection, while most cases of HCV infection are subclinical and not reported.

Classic symptoms of acute hepatitis are often the same, regardless of the responsible virus. Most individuals present with malaise, fatigue, mild fever, nausea, vomiting, anorexia, right upper quadrant discomfort, and occasionally diarrhea. Jaundice, dark urine, and clay-colored stools may also be observed, particularly with acute HAV, HDV, HEV, and frequently in HBV (30% of cases).

Acute HCV usually does not present with jaundice. In over 95% of adults with normal immunity, infection with HBV is self-limited; but in 5% of adults, 30% of children (under the age of 5), 90% of infants, and those with immunodeficiencies the infection becomes chronic. Most people who acquire HCV become chronic carriers of the disease (60% to 85%).

Some people may develop extrahepatic manifestations (more frequent in HCV than in HBV) such as essential mixed cryoglobulinemia, porphyria cutanea tarda, lichen planus, rheumatoid arthritis, Hodgkin's lymphoma, and diabetes mellitus. Rheumatologic and skin manifestations are the most common. Individuals with acute HBV may also demonstrate extrahepatic symptoms including rash, arthralgias, and arthritis.

MEDICAL MANAGEMENT

PREVENTION. Prevention takes place at three levels: primary, secondary, and tertiary. Primary prevention involves primary immunization (HAV, HBV, and HEV), education regarding food preparation and proper hand-washing, avoiding needle punctures by contaminated needles (or other similar infective material), and practicing protective sex or avoiding sexual contact during the period of HBsAg positivity.

Secondary prevention involves passive immunization following exposure to HAV or HBV, travel precautions when visiting areas where hepatitis is endemic (e.g., avoid drinking unbottled water or beverages served with ice; avoid eating foods rinsed in contaminated water, such as fruits and vegetables; and avoid eating shellfish).

Tertiary prevention involves education to those infected about preventing possible infectivity to others and self-care during active infection (e.g., avoid strenuous activity and ingestion of hepatotoxins, such as alcohol and

acetaminophen; some advocate alternative treatment such as herbs, acupuncture, and dietary measures).

Hepatitis A. Preventive measures include HAV vaccine, standard precautions, and immune globulin (IG), which is a preparation of antibodies against HAV. IG can be used before infection and provides passive immunity for 3 to 5 months or can be given to household and intimate contacts of people with HAV. When given after exposure, IG is 80% to 90% effective in preventing HAV infection. The sooner IG is given after exposure, the more efficacious it is.⁴² People who have received the HAV vaccine at least 1 month before exposure do not require IG.

HAV vaccine is recommended (before exposure) for persons 1 year of age and older (as a standard childhood vaccination), adults wishing to obtain immunity, or anyone who is at risk for infection (e.g., persons traveling to areas with intermediate to high rates of endemic HAV, persons living in communities with high endemic rates or periodic outbreaks of HAV infection, men who have sex with men or who are bisexual and their partners of either gender, and clients with chronic liver disease). HAV vaccine confers 97% to 100% protection in children and 94% to 100% in adults.

Hepatitis B. Hepatitis B is a preventable disease achieved by administering a HBV vaccine. Currently, two vaccines are available as single-antigen vaccines: Recombivax HB and Engerix-B. Three are available as combination vaccines: Twinrix (for HAV and HBV in adults), Comvax (for HBV and *Haemophilus influenzae* type B in children), and Pediarix (for HBV, diphtheria and tetanus toxoids, pertussis, and poliovirus in children). The vaccine is given in 3 doses over a period of 6 months.

Life-long protective immunity develops in more than 90% of healthy adults. In the unvaccinated person, hepatitis B IG (HBIG) is used for postexposure prophylaxis (PEP) and should be administered soon after the exposure, followed by the HBV series at 0, 1, and 6 months. Effectiveness of PEP decreases with time between exposure and treatment. PEP should be given in less than 7 days from exposure for needlesticks and less than 14 for sexual exposure. Immunity to HBV also confers immunity to HDV; therefore HBV vaccination is recommended for preexposure immunization prophylaxis for HDV.

Once individuals begin to engage in behaviors associated with high-risk groups, they may become infected before vaccine can be given. A major obstacle in eliminating HBV is identifying persons and vaccinating them before they become infected. For this reason, in the United States, it is now recommended that all infants, health care workers, and persons in the high-risk category for HBV receive the HBV vaccine.

According to the Occupational Safety and Health Administration (OSHA) Bloodborne Pathogen Standard (1991), HBV vaccination must be offered to all employees within 10 days of employment. Records related to this vaccination must be maintained. Employees who decline vaccination must sign a standardized declination form.

Children younger than 5 years of age, if infected, have a high risk of becoming HBV carriers. Testing to identify pregnant women who are HBsAg positive and providing their infants with immunoprophylaxis effectively prevents HBV transmission during the perinatal period. The

HBsAg is a core protein antigen of the HBV present in the nuclei of infected cells. Presence of HBsAg in the blood usually indicates the individual is infectious. HCb antibodies appear during the acute infection but do not protect against reinfection.

The World Health Organization (WHO) has recommended that all countries integrate HBV vaccination into their national immunization programs, and much progress has been made toward the goal to control, eliminate, and eradicate hepatitis in the coming generations.¹⁶³

Hepatitis C. Currently, no vaccine is available to prevent HCV because of the rapidity of HCV mutations in adaptation to the environment⁷² and no immunoglobulin (Ig) is effective in treating exposure. The only means of preventing new cases of HCV are to screen the blood supply, encourage health care professionals to take precautions when handling blood and body fluids, and educate people about high-risk behaviors (particularly injection-drug users and those involved with multiple sex partners).

Hepatitis E. A new recombinant protein vaccine (rpHEV) has been developed and shown effective in preventing HEV in high-risk populations.¹⁵³

DIAGNOSIS. In addition to the history and clinical examination, serologic and molecular testing help provide an accurate diagnosis. Serology is the standard for diagnosis of viral hepatitis (see Table 17-2). People with a positive IgM HAV have acute disease, whereas IgG HAV is present at the onset of disease and remains in the blood for life. IgG HAV signifies previous exposure, whereas IgM HAV signifies only acute infection.

Serology relating to HBV changes as the disease progresses or is contained. HBV has an acute phase, a convalescent phase, and a chronic phase (Table 17-4). Early in HBV infection, the serologic marker HBsAg is positive, but the antibodies are negative until an immune response has been mounted by the body. Acute infection is demonstrated by a positive HBsAg and the presence of antibodies total anti-HBc (antibody to the hepatitis B core antigen) and IgM anti-HBc (IgM is an acute antibody). Resolving infection has eliminated the HBsAg, and antibodies remain present (anti-HBsAg, or the antibody to the surface antigen, may also be present). A serologic pattern positive for anti-HBc and anti-HBs demonstrates past infection and existing immunity.

Chronic infection is marked by the presence of HBsAg and anti-HBc. Vaccinated people display a positive anti-HBsAg. HBeAg appears early in the acute infection and continued presence in the chronic state suggests increased infectivity, greater number of virus replication, and the need for antiviral therapy.

HBV deoxyribonucleic acid (DNA) can be detected in persons with chronic infection by polymerase chain reaction (PCR) techniques⁸⁹; this information is useful in treatment and prognosis. None of the serodiagnostic tests are able to determine the current extent of liver damage; a liver biopsy provides information about the severity of disease and establishes grading and degree of fibrosis but is only necessary in the case of chronic hepatitis.

Testing for HCV includes an initial screening for anti-HCV, which, if positive, is followed by a more specific

Table 17-4 Interpretation of Serologic Results of Hepatitis B Testing

	HBsAg	Anti-HBc	Anti-HBs	HBeAg
Early infection	Present	Negative	Negative	Present
Acute infection	Present	Present	Negative	Present
Resolving infection	Negative	Present	Present/negative	Negative
Past infection	Negative	Present	Present	Negative
Chronic infection	Present	Present	Negative	Present
Vaccine immune	Negative	Negative	Present	Negative

HBsAg, Hepatitis B surface antigen; *anti-HBc*, antibody to hepatitis B core antigen; *anti-HBs*, antibody to hepatitis B surface antigen; *HBeAg*, hepatitis B e antigen.

serologic test (such as RIBA) or a nucleic acid test (NAT). The best method is the detection of HCV ribonucleic acid (RNA) in the serum. Liver biopsy is not required but is often helpful in determining severity and making treatment decisions.

Transaminases (enzymes normally present in hepatocytes) are released from the acutely damaged hepatocytes, and serum transaminase levels rise, often to levels exceeding normal levels by twentyfold. An elevated serum bilirubin level is usually found. Liver function tests (see Table 40-5) can indicate other viral liver diseases, drug toxicity, or alcoholic hepatitis. Researchers are working to develop improved molecular techniques for increased diagnostic precision for all the hepatitis viruses.¹⁰⁴

TREATMENT. Treatment options have expanded and prevention methods are available for two of the six viruses although no cure is available for the viruses that are responsible for chronic liver disease (HBV, HDV, and HGV). The development of new antiviral agents that inhibit steps in the viral replication process is under investigation.¹¹⁰ A potential cure for HCV has been announced (see further discussion later). Any hepatic irritants, such as alcohol, medications, or chemicals (e.g., occupational exposure to carbon tetrachloride), must be avoided in all types of hepatitis.

Hepatitis A. Treatment for HAV is primarily symptomatic and supportive. Good sanitation and hygiene practices should be followed. Hospitalization may be required for excessive vomiting and subsequent fluid and electrolyte imbalance.

Hepatitis B. Treatment for acute HBV is symptomatic. Only clients with chronic disease receive treatment, which depends on the severity of the disease. People with normal liver enzyme values, an HBV DNA level of less than 10^{21} copies/ml, and findings on biopsy that are normal are considered "inactive carriers." Their prognosis is good and compares favorably with people who are not infected with HBV. People with abnormal liver enzymes (elevated AST), HBV DNA levels greater than 10^{21} copies/ml, and architecture on liver biopsy consistent with active disease require treatment. Pegylated interferon and lamivudine, adefovir, and entecavir (nucleoside analogues) are current therapies available. Interferon is expensive and has many side effects, but therapy duration is limited and maintenance therapy is not required.

Nucleoside analogues can be given orally, cost less than interferon, and have few side effects but require

continued treatment to maintain response. The newest of these drugs for chronic HBV infection, telbivudine (Tyzeka) is an oral medication that reduces the viral load by inhibiting HBV reverse transcriptase and HBV replication. It is indicated for adults with evidence of viral replication and either persistent elevations in serum aminotransferases or histologically active HBV. Combination therapy is frequently used. Since people with cirrhosis and HBV are at high risk for the development of HCC, ultrasonography of the liver every 6 to 12 months is advisable.

Hepatitis C. Since the majority of complications occur in clients who develop cirrhosis from HCV, treatment is provided to those at risk for developing cirrhosis. This includes people with a history of alcohol abuse, people who contracted HCV at a young age, or those with active disease by liver biopsy. Because people with genotypes 2 and 3 (subtypes of HCV) have a high likelihood of responding to therapy, a liver biopsy is often not required before therapy.

Researchers reported at the 38th annual Digestive Disease Week conference in Washington, D.C., that HCV is curable and should be treated (with peginterferon alfa-2a alone or with ribavirin) even if the person has no symptoms.¹⁴⁹ A study using combination therapy consisting of pegylated interferon and ribavirin (Virazole, a nucleoside analogue) for 3 to 12 months (depending on genotype and response) provided a sustained clearance of HCV in about 55% of clients.¹⁵²

The treatment involves a new "pegylated" form of the commonly used HCV drug interferon. By attaching polyethylene glycol (PEG) molecules to the interferon, its half-life is lengthened, allowing it to stay in the body much longer. The new interferon formulation is given as a weekly injection rather than three times a week. PEG interferon has been reported to reverse infection-related liver damage previously believed impossible. The study showed reductions in liver fibrosis, or scar tissue, in three out of four people given the new treatment. Half of those with cirrhosis also showed improvement.¹⁵⁰ Many potential side effects and contraindications exist for the use of interferon that prevents this treatment from being applied to all individuals with HCV infection.

Surveillance by ultrasonography every 6 to 12 months is recommended to detect HCC. Administration of HAV and HBV vaccine is recommended for anyone with chronic HCV because of the potential for increased severity of acute hepatitis superimposed on existing liver disease.¹⁵⁴

PROGNOSIS. Prognosis varies with each type. A substantial proportion of HBV morbidity and mortality that occurs in the health care setting can be prevented by vaccinating health care workers against HBV. In addition, health care workers must practice infection control measures (see Appendix A). Other prophylactic strategies are based on avoidance of high-risk behavior and use of IG.

Hepatitis A. HAV is almost always a self-limited disease and rarely leads to fulminant hepatitis requiring transplantation (about 0.3% to 0.6% of cases). It does not lead to chronic hepatitis or cirrhosis. Most people recover fully and become immune to HAV.

Hepatitis B. In adults with normal immune status, most (94% to 98%) recover completely from newly acquired HBV infections, eliminating virus from the blood and producing neutralizing antibody that creates immunity from future infection. In immunosuppressed persons (including hemodialysis clients), infants, and young children, most newly acquired HBV infections result in chronic infection.

Although the consequences of acute HBV can be severe (1% die from acute hepatitis), most of the serious sequelae associated with the disease occur in persons in whom chronic infection develops. Approximately 15% of people who acquire the disease as adults and 25% of people who acquired HBV as a child die prematurely from cirrhosis or liver cancer.⁹⁴

Despite reinfection rates, orthotopic liver transplantation for cirrhosis caused by HBV provides the best treatment. Continued treatment of the virus with the administration of HBIG (intravenously or intramuscularly) used in combination with lamivudine has been found to be efficacious, making liver transplantation outcomes successful.

Hepatitis C. Acute HCV infection only leads to death in 1.3% of cases. The majority of disease sequelae occurs from chronic disease, particularly cirrhosis. Approximately 20% to 30% of all cases of HCV progress to cirrhosis. Between 1% and 5% of people with HCV-related cirrhosis develop HCC each year.³¹

HCV-related cirrhosis is the most common reason for liver transplantation and between 8000 and 12,000 people die each year from HCV-related disease.⁴ The number of annual deaths is most likely to triple as people with chronic disease develop end-stage liver disease.⁸

The progression of HCV is accelerated if a person is also coinfected with the acquired immunodeficiency syndrome (AIDS) virus, causing cirrhosis more quickly and twice as often.¹⁵⁶ HIV/HCV coinfection among individuals with bleeding disorders results in mortality; more than 5000 people have died, and the numbers continue to climb.¹¹¹

Hepatitis D. HDV by itself appears to be relatively harmless, but it has a high morbidity and mortality rapidly leading to hepatic failure and cirrhosis when it accompanies HBV.

Hepatitis E. HEV is typically a self-limited acute hepatitis, usually lasting 1 to 4 weeks. Severity of symptoms increases with age. It does not progress to chronic disease. When it occurs in epidemics, it can cause substantial rates of death and complications, especially in pregnant women. The overall fatality rate is estimated to be 3%;

pregnant women who develop the infection have the highest risk of acute hepatic failure.¹⁵³

SPECIAL IMPLICATIONS FOR THE THERAPIST

17-7

Viral Hepatitis

PREFERRED PRACTICE PATTERNS

Practice patterns depend on clinical presentation. Although extrahepatic manifestations, such as neuropathy and skin, joint, and/or bone involvement, occur, these are usually unresponsive to physical therapy intervention and resolve gradually with medical intervention for the underlying cause (hepatitis). Exceptions occur such as in the case of required wound care or management of rheumatic diseases or fibromyalgia.

Any direct contact with blood or body fluids of clients with HBV or HCV requires the administration of IG, a preparation of antibodies, in the early incubation period. Therapists at risk for contact with HBV because of their close contact with the blood or body fluids of carriers should receive active immunization against HBV. All therapists should follow standard precautions at all times to protect themselves and must wear personal protective equipment whenever appropriate. Such gear should never be worn in the car or laundered at home to avoid contamination of those sites.

Enteric precautions are required when caring for individuals with type A or E hepatitis. Any therapist working in dialysis units or providing wound care should review infectious control guidelines available.¹⁰⁶ For the therapist who has been diagnosed with viral hepatitis, recommendations and work restrictions for health care workers are discussed in Chapter 8; see Table 8-5.

Studies dealing with the natural history of acute hepatitis have provided perspective on the frequency of skin and joint manifestations. More than a third of the adults studied had joint pains during the course of the illness. The frequency of arthralgia as a symptom associated with hepatitis increases with age. Joint pains affected only 18% of children, compared with 45% of adults older than 30 years.

No known studies have been published regarding the benefit of physical therapy in providing symptomatic joint relief until these symptoms resolve as the person recovers from the underlying pathology. In the case of a client with undiagnosed hepatitis presenting with joint symptoms, the systemically derived arthralgia will not respond to therapy. Any time intervention fails to provide symptomatic relief or resolution of symptoms, the results must be reported to the physician for further follow-up evaluation.

Overall, in the recovery process, adequate rest to conserve energy is important. The affected individual is encouraged to gradually return to levels of activity before illness. Fatigue associated with the anicteric phase of hepatitis may interfere with activities of daily living and may persist even after the jaundice resolves. A careful balance of activity is important to avoid weakness secondary to prolonged bed rest; a

Continued.

reasonable activity level is more conducive to recovery than is enforced bed rest. Whenever possible, rehabilitation intervention or increased activity should not be scheduled right after meals.

Watch for signs of fluid shift, such as weight gain and orthostasis; dehydration; pneumonia; vascular problems; and pressure ulcers and any signs of recurrence. After the diagnosis of viral hepatitis has been established, the affected individual should have regular medical checkups for at least 1 year and should avoid using any alcohol or over-the-counter (OTC) drugs during this period.

α -Interferon (antiviral used in the treatment of some hepatitis) has bone marrow suppressive effects requiring careful monitoring of platelet or neutrophil count (see Tables 40-8 and 40-9). Other side effects of combination therapy may include increased fatigue, increased muscle pain and potential inflammatory myopathy, headaches, local skin irritation (site of injection), irritability and depression, hair loss, itching, sinusitis, and cough. These symptoms usually subside in the first few weeks of treatment; prolonged or intolerable side effects must be reported to the physician.

Hepatitis B and the Athlete

Great concern is often expressed over the possibility of contagion among athletes in competitive sports, particularly sports with person-to-person contact. Infectious agents, such as HIV and other viruses, bacteria, and even fungi, have been examined. No cases of HIV or HCV transmission resulting from sports participation have been reported, but two cases of HBV transmission through exposure to blood during sports participation have been documented.

For most of the infections considered, the athlete is more at risk during activities off the playing field than while competing. Inclusion of immunizations against measles and HBV as a prerequisite to participation would eliminate these two diseases from the list of dangers to athletes (and all individuals). Education, rather than regulations, is the best approach in considering the risks to athletes from contagious diseases.³⁶

Although the risk of bloodborne pathogen infection during sports is exceedingly small, good hygiene practices concerning blood are still important. The American Academy of Pediatrics (AAP) has made recommendations to minimize the risk of bloodborne pathogen transmission in the context of athletic events and has issued safety precautions. The therapist in this type of setting is encouraged to review these guidelines.⁶

No evidence has been reported that intense, highly competitive training is harmful for the asymptomatic HBV-infected person, whether the disease is acute or chronic. Therefore the presence of HBV infection does not contraindicate participation in sports or athletic activities; decisions regarding play are made according to clinical signs and symptoms such as fatigue, fever, or organomegaly. Chronic HBV infection with evidence of organ impairment requires reduction in intensity and duration of activity.

Drug-Related Hepatotoxicity Overview and Incidence

Injury to the liver can be caused by many drugs or toxins. More than 600 medicinal agents, chemicals, and herbal remedies are recognized as producing hepatic injury.⁷⁹ For example, herbal products, such as chaparral, comfrey, germander, Jin Bu Huan, mistletoe, nutmeg, ragwort, sassafras, senna, or tansy, can cause liver-related complications (ranging from acute hepatitis and jaundice to cirrhosis and death from liver failure) in anyone with an underlying liver disease.

Although OTC and prescription medications are often thought to be the only agents to cause liver injury, complementary or alternative medications, such as chaparral, germander, pennyroyal oil, Jin Bu Huan, and Sho-saiko-to, are also known to be hepatotoxic.¹⁵⁹

Chemicals, such as carbon tetrachloride, trichloroethylene, derivatives of benzene and toluene, vinyl chloride, and organic pesticides, can also lead to liver injury. Carbon tetrachloride was the most common industrial chemical considered an occupational inhalation poison until it was banned in most countries. Ingestion of poisonous mushrooms, including *Amanita phalloides* and related species (rare in the United States but more common in Europe), can lead to fulminant liver failure.

Although uncommon, drugs are currently the most common cause of acute liver failure.¹¹⁷ A recent study of the WHO database demonstrated acetaminophen, troglitazone, valproate, stavudine, and halothane to be the five drugs most frequently associated with hepatotoxicity and death (particularly acetaminophen).^{16,70}

The incidence of hepatotoxicity as a result of these noninfectious agents is difficult to determine since many cases may be subclinical (i.e., mild symptoms) or misdiagnosed, leading to underreporting. One French study demonstrated a rate of 14 cases/100,000 people in the general population; 12% required hospitalization, and 6% died as a result of acute liver failure.¹⁴⁸ Hepatotoxicity has also been the principal reason for removing several new drugs from the market.⁴⁹

Drugs or chemicals typically cause either predictable (or dose-related) or unpredictable (or idiosyncratic) liver injury (Box 17-3). Drugs that cause predictable liver damage are dose-related (i.e., a specific toxic dose most likely will cause damage) and result in injury soon after ingestion. Acetaminophen is the most common example of predictable drug-related liver disease. Unpredictable reactions occur without warning, are unrelated to dose, and may occur days to months after ingestion.

Etiologic Risk Factors

A host of factors may enhance susceptibility to drug-induced liver disease, including age (adults more so than children), gender (women have a higher risk than men), obesity, malnutrition, pregnancy, concurrent medication use, history of drug reactions, and genetic variability (probably the most important factor). Preexisting liver diseases and comorbidities appear to alter the rate of recovery rather than affect the risk of developing hepatotoxicity.¹⁴¹ Alcohol use and fasting may increase the likelihood of acetaminophen-related hepatotoxicity.¹⁷¹