CHAPTER 24 Nursing Care of Clients with Gallbladder, Liver, and Pancreatic Disorders

LEARNING OUTCOMES

- Describe the pathophysiology of commonly occurring disorders of the gallbladder, liver, and exocrine pancreas.
- Use knowledge of normal anatomy and physiology to understand the manifestations and effects of biliary, hepatic, and pancreatic disorders.
- Relate changes in normal assessment data to the pathophysiology and manifestations of gallbladder, liver, and exocrine pancreatic disorders.

CLINICAL COMPETENCIES

- Assess functional health status of clients with gallbladder, liver, or pancreatic disease.
- Monitor for, document, and report expected and unexpected manifestations in clients with gallbladder, liver, or pancreatic disease.
- Prepare clients for and understand the purpose and significance of diagnostic tests for gallbladder, liver, and pancreatic disorders.
- Integrate appropriate dietary, pharmacologic, and other interdisciplinary measures into nursing care and teaching of the client with a gallbladder, liver, or pancreatic disorder.
- Provide appropriate nursing care for the client who has surgery of the gallbladder, liver, or pancreas.
- Integrate psychosocial, cultural, and spiritual considerations into the plan of care for a client with a gallbladder, liver, or pancreatic disorder.
- Use evidence-based practice to develop, implement, evaluate, and, as needed, revise the plan of care for clients with disorders of the gallbladder, liver, or pancreas.
- Provide appropriate client and family teaching to promote, maintain, and restore functional health status for clients with gallbladder, liver, and pancreatic disorders.

MEDIALINK

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone

KEY TERMS

alcoholic cirrhosis, *710*
ascites, *704*
balloon tamponade, *718*
biliary colic, *698*
cholecystitis, *698*
cholelithiasis, *697*
chronic hepatitis, *706*
cirrhosis, *710*
esophageal varices, *704*
fulminant hepatitis, *706*
gastric lavage, *718*
hematochezia, *722*
hepatitis, *705*
hepatorenal syndrome, *704*
jaundice, *704*
laparoscopic cholecystectomy, *699*
liver transplantation, *719*
pancreatitis, *726*
paracentesis, *717*
portal hypertension, *704*
portal systemic encephalopathy, *704*
steatorrhea, *727*
transjugular intrahepatic portosystemic shunt (TIPS), *719*

Gallbladder, liver, and exocrine pancreatic disorders may occur as primary disorders, or develop secondarily to other disease processes. One organ's functioning frequently affects that of another. Duct inflammation or obstruction, and changes in the multiple functions of these organs, can cause significant health effects.

Clients with a gallbladder, liver, or pancreatic disorder may experience pain, multiple metabolic and nutritional disturbances, and altered body image. Nursing care addresses physiologic and psychosocial needs of the client and family.

GALLBLADDER DISORDERS

Altered bile flow through the hepatic, cystic, or common bile duct is a common problem. It often leads to inflammation and other complications. Gallstones are the most common cause of obstructed flow. Tumors and abscesses also can obstruct bile flow.

THE CLIENT WITH GALLSTONES

Cholelithiasis is the formation of stones (*calculi* or *gallstones*) within the gallbladder or biliary duct system. Cholelithiasis is a common problem in the United States, affecting more than 10% of men and 20% of women by age 65 (Tierney et al., 2005). Box 24–1 lists risk factors for gallstones. The incidence of gallstones varies among people of different ethnic backgrounds; see the accompanying Focus on Cultural Diversity box.

Physiology Review

Normally, bile is formed by the liver and stored in the gallbladder. Bile contains bile salts, bilirubin, water, electrolytes, cholesterol, fatty acids, and lecithin. In the gallbladder, some of the water and electrolytes are absorbed, further concentrating the bile. Food entering the intestine stimulates the gallbladder to contract and release bile through the common bile duct and sphincter of Oddi into the intestine. The bile salts in bile increase the solubility and absorption of dietary fats.

BOX 24–1 Risk Factors for Gallstones

- Age
- Family history of gallstones
- Race or ethnicity: Native American (either Northern or Southern Hemisphere); Northern European heritage
- Obesity, hyperlipidemia
- Rapid weight loss
- Female gender; use of oral contraceptives
- Biliary stasis: pregnancy, fasting, prolonged parenteral nutrition
- Diseases or conditions: cirrhosis; ileal disease or resection; sickle cell anemia; glucose intolerance

FOCUS ON CULTURAL DIVERSITY
Gallstones

Native Americans in both the Northern and Southern Hemispheres, and those of the Pima tribe in particular, have a higher incidence of gallstones than do Caucasians of American or European heritage. This is thought to result from genes that promote efficient calorie use and fat storage—a beneficial trait when the availability of adequate food varies over time. Gallstones composed of cholesterol are less common in African Americans, and Asians have a low incidence of the disease (Tierney et al., 2005).

Pathophysiology and Manifestations

Cholelithiasis

Gallstones form when several factors interact: abnormal bile composition, biliary stasis, and inflammation of the gallbladder. Most gallstones (80%) consist primarily of cholesterol; the rest contain a mixture of bile components. Excess cholesterol in bile is associated with obesity, a high-calorie, high-cholesterol diet, and drugs that lower serum cholesterol levels. When bile is supersaturated with cholesterol, it can precipitate out to form stones. Biliary stasis, or slowed emptying of the gallbladder, contributes to cholelithiasis. Stones do not form when the gallbladder empties completely in response to hormonal stimulation. Slowed or incomplete emptying allows cholesterol to concentrate and increases the risk of stone formation. Finally, inflammation of the gallbladder allows excess water and bile salt reabsorption, increasing the risk for lithiasis.

PRACTICE ALERT

Certain very-low-calorie diets are associated with a high risk of cholelithiasis. Increased cholesterol concentration in the bile and decreased gallbladder contractions associated with fasting increase the risk of gallstone formation.

Most gallstones are formed in the gallbladder. They then may migrate into the ducts (Figure 24–1 ■), leading to *cholangitis* (duct inflammation). Although some people with cholelithiasis are asymptomatic, many develop manifestations. Early manifestations of gallstones may be vague: epigastric fullness or mild gastric distress after eating a large or fatty meal. Stones that obstruct the cystic duct or common bile duct lead to distention and increased pressure behind the stone. This causes **biliary colic**, a severe, steady pain in the epigastric region or right upper quadrant of the abdomen. The pain may radiate to the back, right scapula, or shoulder. The pain often begins suddenly following a meal, and may last as long as 5 hours. It often is accompanied by nausea and vomiting.

Obstruction of the common bile duct may cause bile reflux into the liver, leading to jaundice, pain, and possible liver damage. If the common duct is obstructed, pancreatic enzymes will be unable to enter the small intestine, and pancreatitis (discussed later in this chapter) becomes a potential complication.

Cholecystitis

Cholecystitis is inflammation of the gallbladder. *Acute cholecystitis* usually follows obstruction of the cystic duct by a stone. The obstruction increases pressure within the gallbladder, leading to ischemia of the gallbladder wall and mucosa. Chemical and bacterial inflammation often follow. The ischemia can lead to necrosis and perforation of the gallbladder wall.

Acute cholecystitis usually begins with an attack of biliary colic. The pain involves the entire right upper quadrant (RUQ), and may radiate to the back, right scapula, or shoulder. Movement or deep breathing may aggravate the pain. The pain usually lasts longer than biliary colic, continuing for 12 to 18 hours. Anorexia, nausea, and vomiting are common. Fever often is present, and may be accompanied by chills. The RUQ is tender to palpation.

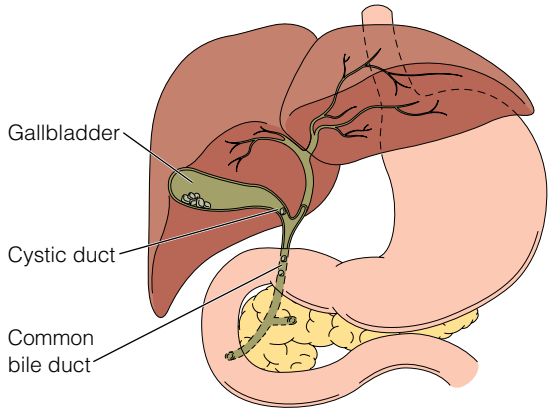


Figure 24–1 ■ Common locations of gallstones.

Chronic cholecystitis may result from repeated bouts of acute cholecystitis or from persistent irritation of the gallbladder wall by stones. Bacteria may be present in the bile as well. Chronic cholecystitis often is asymptomatic.

Complications of cholecystitis include *empyema,* a collection of infected fluid within the gallbladder; gangrene and perforation with resulting peritonitis or abscess formation; formation of a fistula into an adjacent organ (such as the duodenum, colon, or stomach); or obstruction of the small intestine by a large gallstone (*gallstone ileus*). Table 24–1 compares the manifestations and complications of acute cholelithiasis with those of cholecystitis.

INTERDISCIPLINARY CARE

Treatment of the client with cholelithiasis or cholecystitis depends of the acuity of the condition and the client's overall health status. When gallstones are present but asymptomatic and the client has a low risk for complications, conservative treatment is indicated. However, when the client experiences frequent symp-

TABLE 24–1 **Manifestations and Complications of Cholelithiasis and Cholecystitis**

MANIFESTATIONS	CHOLELITHIASIS	CHOLECYSTITIS
Pain	■ Abrupt onset ■ Severe, steady ■ Localized to epigastrium and RUQ of abdomen ■ May radiate to back, right scapula, and shoulder ■ Lasts 30 minutes to 5 hours	■ Abrupt onset ■ Severe, steady ■ Generalized in RUQ of abdomen ■ May radiate to back, right scapula, and shoulder ■ Lasts 12 to 18 hours ■ Aggravated by movement, breathing
Associated symptoms	■ Nausea, vomiting	■ Anorexia, nausea, vomiting ■ RUQ tenderness and guarding ■ Chills and fever
Complications	■ Cholecystitis ■ Common bile duct obstruction with possible jaundice and liver damage ■ Common duct obstruction with pancreatitis	■ Gangrene and perforation with peritonitis ■ Chronic cholecystitis ■ Empyema ■ Fistula formation ■ Gallstone ileus

toms, has acute cholecystitis, or has very large stones, the gallbladder and stones are usually surgically removed.

Diagnosis

Diagnostic tests are ordered to identify the presence and location of stones, identify possible complications, and help differentiate gallbladder disease from other disorders.

- *Serum bilirubin* is measured. Elevated direct (conjugated) bilirubin may indicate obstructed bile flow in the biliary duct system (Box 24–2).
- *Complete blood count (CBC)* may indicate infection and inflammation if the WBC count is elevated.
- *Serum amylase* and *lipase* are measured to identify possible pancreatitis related to common duct obstruction.
- *Abdominal x-ray* (flat plate of the abdomen) may show gallstones that have a high calcium content.
- *Ultrasonography of the gallbladder* is a noninvasive exam that can accurately diagnose cholelithiasis. It also can be used to assess emptying of the gallbladder.
- *Oral cholecystogram* is performed using a dye administered orally to assess the gallbladder's ability to concentrate and excrete bile.
- *Gallbladder scans* use an intravenous radioactive solution that is rapidly extracted from the blood and excreted into the biliary tree to diagnose cystic duct obstruction and acute or chronic cholecystitis.

See Chapter 21 ∞ for more information about and the nursing implications of these diagnostic tests.

Medications

Clients who refuse surgery or for whom surgery is inappropriate may be treated with a drug to dissolve the gallstones. Ursodiol (Actigall) and chenodiol (Chenix) reduce the cholesterol content of gallstones, leading to their gradual dissolution. These drugs act by reducing cholesterol production in the liver, thus reducing the cholesterol content of bile. Consequently, these drugs are most effective in treating stones with high cholesterol content. They are less effective in treating radiopaque stones with high calcium salt content. Ursodiol is generally well tolerated with few side effects, whereas chenodiol has a high incidence of diarrhea at therapeutic doses. It also is hepatotoxic, so periodic liver function studies are required during therapy.

The primary disadvantages of pharmacologic treatment for gallstones include its cost, long duration (2 years or more), and the high incidence of recurrent stone formation when treatment is discontinued. If infection is suspected, antibiotics may be ordered to cure the infection and reduce associated inflammation and edema. Clients with pruritus (itching) due to severe obstructive jaundice and an accumulation of bile salts on the skin may be given cholestyramine (Questran). This drug binds with bile salts to promote their excretion in the feces. A narcotic analgesic such as morphine may be required for pain relief during an acute attack of cholecystitis.

BOX 24–2 Sorting Out Total, Direct, and Indirect Bilirubin Levels

When serum bilirubin levels are drawn, the results usually are reported as the total bilirubin, direct bilirubin, and indirect bilirubin levels. Most bilirubin is formed from hemoglobin, as aging or abnormal RBCs are removed from circulation and destroyed. It is then bound to protein and transported to the liver. This protein-bound bilirubin is called *indirect* or *unconjugated* bilirubin. Once in the liver, bilirubin is separated from the protein and converted to a soluble form, *direct* or *conjugated* bilirubin. Conjugated bilirubin is then excreted in the bile.

- **Total** (serum) **bilirubin,** the total bilirubin in the blood, includes both indirect and direct forms. In adults, the normal total bilirubin is 0.3 to 1.2 mg/dL. Total bilirubin levels increase when more is being produced (e.g., RBC hemolysis), or when its metabolism or excretion are impaired (e.g., liver disease or biliary obstruction).
- **Direct** (conjugated) **bilirubin** levels, normally 0 to 0.2 mg/dL in adults, rise when its excretion is impaired by obstruction within the liver (e.g., in cirrhosis, hepatitis, exposure to hepatotoxins) or in the biliary system.
- **Indirect** (unconjugated) **bilirubin** levels, normally <1.1 mg/dL in adults, rise in RBC hemolysis (e.g., sickle cell disease or transfusion reaction).

Treatments

SURGERY **Laparoscopic cholecystectomy** (removal of the gallbladder) is the treatment of choice for symptomatic cholelithiasis or cholecystitis. This minimally invasive procedure has a low risk of complications and generally requires a hospital stay of less than 24 hours. Not all clients are candidates for laparoscopic cholecystectomy, and there is a risk that a laparoscopic cholecystectomy may be converted to a *laparotomy* (surgical opening into the abdomen) during the procedure. See the following page for nursing care for a client having a laparoscopic cholecystectomy. The Nursing Research box on page 700 discusses evidence-based practice for managing pain in clients undergoing laparoscopic cholecystectomy.

When stones are lodged within the ducts, a cholecystectomy with common bile duct exploration may be done. A T-tube (Figure 24–2 ■) is inserted to maintain patency of the

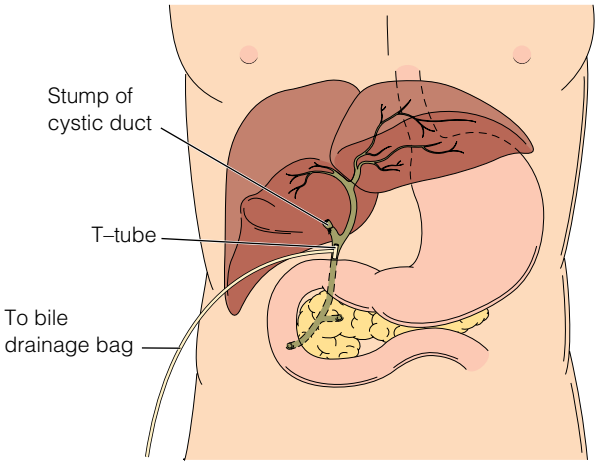


Figure 24–2 ■ T-tube placement in the common bile duct. Bile fluid flows with gravity into a drainage collection device below the level of the common bile duct.

NURSING CARE OF THE CLIENT HAVING Laparoscopic Cholecystectomy

PREOPERATIVE CARE

- Provide routine preoperative care as ordered (see Chapter 4 ∞).
- Reinforce teaching about the procedure and postoperative expectations, including pain management, deep breathing, and mobilization. *Preoperative teaching reduces anxiety and promotes rapid postoperative recovery.*

POSTOPERATIVE CARE

- Provide routine postoperative recovery care as outlined in Chapter 4 ∞.
- Assist to chair at bedside as allowed. *Early mobilization promotes lung ventilation and circulation, reducing the potential for postoperative complications.*
- Advance oral intake from ice chips to regular diet as tolerated. *Oral intake can be rapidly resumed due to minimal disruption of the gastrointestinal tract during surgery.*
- Provide and reinforce teaching: pain management, incision care, activity level, postoperative follow-up appointments. *With early discharge, the client and family assume responsibility for the majority of postoperative care. A clear understanding of this care and expected needs reduces anxiety and the risk of postoperative complications.*
- Initiate follow-up contact 24 to 48 hours after discharge to evaluate adequacy of pain control, incision management, and discharge understanding. *Contact following discharge provides an opportunity to evaluate care and reinforce teaching.*

duct and promote bile passage while the edema decreases. Excess bile is collected in a drainage bag secured below the surgical site. If it is suspected that a stone has been retained following surgery, a postoperative cholangiogram via the T-tube or direct visualization of the duct with an endoscope may be performed. See the box on page 701 for nursing care for a client with a T-tube.

Some clients who are poor surgical risks and for whom laparoscopic cholecystectomy is inappropriate may have either a *cholecystostomy* to drain the gallbladder, or a *choledochostomy* to remove stones and position a T-tube in the common bile duct.

NUTRITION Food intake may be eliminated during an acute attack of cholecystitis, and a nasogastric tube inserted to relieve nausea and vomiting. Dietary fat intake may be limited, especially if the client is obese. If bile flow is obstructed, fat-soluble vitamins (A, D, E, and K) and bile salts may need to be administered.

OTHER THERAPIES In some cases, shock wave lithotripsy may be used with drug therapy to dissolve large gallstones. In *extracorporeal shock wave lithotripsy,* ultrasound is used to align the stones with the source of shock waves and the com-

NURSING RESEARCH Evidence-Based Practice: Client Undergoing Laparoscopic Cholecystectomy

Following ambulatory surgery procedures such as laparoscopic cholecystectomy, clients must self-manage their pain after discharge. In a study of pain severity and management among ambulatory surgery clients, Watt-Watson, Chung, Chan, and McGillion (2004) found that while the most severe pain was experienced within the first 72 hours, some clients reported severe pain episodes up to a week after surgery. For most clients undergoing laparoscopic cholecystectomy, however, by 72 hours postoperatively the worst pain reported was moderate and the interference with usual activities was minimal. Clients tended to significantly reduce their use of analgesics by 72 hours after surgery, perhaps because many experienced adverse effects such as constipation, nausea, and/or drowsiness. Overall, analgesic use was found to be inadequate and inappropriate among study participants (Watt-Watson et al., 2004). Most clients used acetaminophen with codeine, an analgesic with known dose-related adverse effects. Some clients did not fill their prescription for the analgesic or stopped taking it early in the postoperative course because of nausea or constipation experiences. Preoperative teaching about pain management was inadequate for a number of the participants in this study.

IMPLICATIONS FOR NURSING

Effective pain relief is known to promote healing and immune function following surgery. This study indicates a crucial need to carefully prepare clients undergoing ambulatory surgery, including laparoscopic cholecystectomy, for pain management strategies. Effective postoperative pain management requires a combination of good preoperative education, discharge planning related to the client's expectations of pain, and postoperative pain management.

CRITICAL THINKING IN CLIENT CARE

1. Some clients in this study reported purposely not filling their analgesic prescription due to anticipated adverse effects of the drug. How can the nurse intervene to prevent this and promote effective postoperative pain management? What teaching could you provide to help clients manage adverse effects of the prescribed drug?
2. Few clients in this study reported use of adjunctive pain relief measures (nonsteroidal anti-inflammatory drugs, application of heat or cold, etc.). What adjunctive pain relief measures would be appropriate for the nurse to teach clients undergoing laparoscopic cholecystectomy?
3. Some clients in this study expressed concern about becoming addicted to opioid analgesics, citing this as a reason to discontinue their use within 48 to 72 hours after surgery. How would you respond to a client who expresses this as a concern?

Source: Data from Pain management following discharge after ambulatory same-day surgery, by Judy Watt-Watson, Frances Chung, Vincent W. S. Chan, and Michael McGillion (2004). *Journal of Pain Management, 12*(3), 153–161.

NURSING CARE OF THE CLIENT WITH A T-Tube

- Ensure that the T-tube is properly connected to a sterile container; keep the tube below the level of the surgical wound. *This position promotes the flow of bile and prevents backflow or seepage of caustic bile onto the skin. The tube itself decreases biliary tree pressure.*
- Monitor drainage from the T-tube for color and consistency; record as output. Normally, the tube may drain up to 500 mL in the first 24 hours after surgery; drainage decreases to less than 200 mL in 2 to 3 days, and is minimal thereafter. Drainage may be blood tinged initially, changing to green-brown. Report excessive drainage immediately (after 48 hours, drainage greater than 500 mL is considered excessive). *Stones or edema and inflammation can obstruct ducts below the tube, requiring treatment.*
- Place in Fowler's position. *This promotes gravity drainage of bile.*
- Assess skin for bile leakage during dressing changes. *Bile irritates the skin; it may be necessary to apply skin protection with karaya or another barrier product.*
- Teach client how to manage the tube when turning, ambulating, and performing activities of daily living. *Direct pulling or traction on the tube must be avoided.*
- If indicated, teach care of the T-tube, how to clamp it, and signs of infection. *Clients may be discharged home with the tube in place. Reporting early signs of infection facilitates prompt treatment.*

puterized lithotripter. Positioning is of prime importance throughout the procedure, which usually takes an hour. Mild sedation may be given during the procedure. Nursing care after the procedure includes monitoring for biliary colic, which can result from the gallbladder contracting to remove stone fragments; nausea; and transient hematuria. *Percutaneous cholecystostomy,* ultrasound-guided drainage of the gallbladder, may be done in high-risk clients to postpone or even eliminate the need for surgery.

COMPLEMENTARY THERAPIES The herb goldenseal has been used in treating cholecystitis. One of the active ingredients in goldenseal, berberine, stimulates secretion of bile and bilirubin. It also inhibits the growth of many common pathogens, including those known to infect the gallbladder. A study of the effectiveness of berberine in clients with cholecystitis demonstrated relief of all symptoms. Goldenseal can stimulate the uterus, so it is contraindicated for use during pregnancy. It also should not be used by nursing mothers.

NURSING CARE

In addition to the nursing care discussed in this section, a Nursing Care Plan for a client with cholelithiasis is found on page 702.

Health Promotion

Although most risk factors for cholelithiasis cannot be controlled or modified, several can. Modifiable risk factors include obesity, hyperlipidemia, extremely low-calorie diets, and diets high in cholesterol. Encourage clients who are obese to increase their activity level and follow a low-carbohydrate, low-fat, low-cholesterol diet to promote weight loss and reduce their risk for developing gallstones. Discuss the dangers of "yo-yo" dieting, with cycles of weight loss followed by weight gain, and of extremely low-calorie diets. Encourage clients with high serum cholesterol levels to discuss using cholesterol-lowering drugs with their primary care provider.

Assessment

Assessment data related to cholelithiasis and cholecystitis include the following:

- *Health history:* Current manifestations, including RUQ pain, its character and relationship to meals, duration, and radiation, nausea and vomiting, or other symptoms; duration of symptoms; risk factors or previous history of symptoms; chronic diseases such as diabetes, cirrhosis, or inflammatory bowel disease; current diet; use of oral contraceptives or possibility of pregnancy.
- *Physical assessment:* Current weight; color of skin and sclera; abdominal assessment including light palpation for tenderness; color of urine and stool.
- *Diagnostic tests:* Monitor results of WBC, serum bilirubin, liver enzymes, and pancreatic enzymes (amylase and lipase).

Nursing Diagnoses and Interventions

Priority nursing diagnoses for the client with cholelithiasis or cholecystitis often include pain related to biliary colic or surgery, imbalanced nutrition related to the effects of altered bile flow and to nausea and anorexia, and risk for infection related to potential rupture of an acutely inflamed gallbladder. Nursing interventions for the client who has undergone a laparoscopic or open cholecystectomy are similar to those for other clients having abdominal surgery. See Chapter 4 ∞.

Pain

The pain associated with cholelithiasis can be severe. Sometimes a combination of interventions is indicated.

- Discuss the relationship between fat intake and the pain. Teach ways to reduce fat intake (Box 24–3). *Fat entering the duodenum initiates gallbladder contractions, causing pain when gallstones are present in the ducts.*
- Withhold oral food and fluids during episodes of acute pain. Insert nasogastric tube and connect to low suction if ordered. *Emptying the stomach reduces the amount of chyme entering the duodenum and the stimulus for gallbladder contractions, thus reducing pain.*

NURSING CARE PLAN **A Client with Cholelithiasis**

Joyce Red Wing is a 44-year-old married mother of three children. A member of the Chickasaw tribe, she is active in tribal activities and works part time as a cook at a community kitchen. Recently Mrs. Red Wing has noticed a dull pain in her upper abdomen that gets worse after eating fatty foods; nausea and sometimes vomiting accompany the pain. She had a similar pain after the birth of her last child. She is diagnosed with cholelithiasis, and is admitted for a laparoscopic cholecystectomy.

ASSESSMENT

David Corbin, RN, takes Mrs. Red Wing's admission history. It includes intolerance to fatty foods and intermittent "stabbing" abdominal pain that radiates to her back. Her usual diet includes tacos or fried bread and biscuits with gravy for breakfast. She reports "not wanting to eat much of anything lately." She states she has never had surgery before and hopes "everything goes well." Physical assessment includes T 100°F (37.7°C), P 88, R 20, and BP 130/84. She has had a recent 5 lb weight loss, currently weighing 130 lb (59 kg). She is 63 inches (160 cm) tall. Abdominal examination elicits tenderness in the right upper abdominal quadrant. She has no jaundice, chills, or evidence of complications.

DIAGNOSES

- *Imbalanced Nutrition: Less than Body Requirements* related to anorexia and recent weight loss
- *Pain* related to inflamed gallbladder and surgical incisions
- *Risk for Infection* related to potential bacterial contamination of abdominal cavity
- *Anxiety* related to lack of information about perioperative experience

EXPECTED OUTCOMES

- Maintain present weight within 5 lb (2.3 kg) over the next 3 weeks.
- Resume regular diet, decreasing intake of foods high in fat.
- Verbalize adequate pain control after surgery and with activity resumption.
- Remain free of infection.
- Verbalize a decrease in anxiety before surgery.

PLANNING AND IMPLEMENTATION

- Teach about the gallbladder and the function of bile.
- Discuss pre- and postoperative care, including self-care following discharge.
- Promote mobility as soon as allowed after surgery.
- Teach home care of incisions and recognition of signs of infection.
- Review specific high-fat foods to avoid and ways to maintain her weight.
- Provide analgesia as needed postoperatively. Teach appropriate analgesic use after discharge.

EVALUATION

Mrs. Red Wing is discharged the morning after her surgery. She is afebrile, has no signs of infection, and is able to appropriately care for her incisions. She identifies signs of infection and talks about ways to reduce her fat intake while keeping her weight stable. She verbalizes understanding of initial activity restrictions and resumption of normal activities. Mrs. Red Wing states, "It wasn't as bad as I thought it would be at first." She has an appointment to see her surgeon in 1 week.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the rationale for a low-fat diet with cholelithiasis? Discuss nutritional practices as they relate to the medical problem and Mrs. Red Wing's culture.
2. How would your discharge teaching for Mrs. Red Wing differ if she had had an open cholecystectomy instead of a laparoscopic cholecystectomy?
3. Design a nursing care plan for Mrs. Red Wing for the nursing diagnosis *Fatigue.*

See Evaluating Your Response in Appendix C.

BOX 24–3 Examples of High-Fat Foods

- Whole-milk products (e.g., cream, ice cream, cheese)
- Doughnuts, deep-fried
- Avocados
- Sausage, bacon, hot dogs
- Gravies with fat, cream
- Most nuts (e.g., pecans, cashews)
- Corn chips and potato chips
- Butter and cooking oils
- Fried foods (e.g., cheeseburgers, hamburgers, french fries)
- Peanut butter
- Chocolate candies

- For severe pain, administer morphine, meperidine, or other narcotic analgesia as ordered. *Recent research indicates that morphine is no more likely to cause spasms of the sphincter of Oddi than meperidine.*
- Place in Fowler's position. *Fowler's position decreases pressure on the inflamed gallbladder.*
- Monitor vital signs, including temperature, at least every 4 hours. *Bacterial infection often is present in acute cholecystitis, and may cause an elevated temperature and respiratory rate.*

Imbalanced Nutrition: Less than Body Requirements

The client with severe gallbladder disease may develop nutritional imbalances related to anorexia, pain, and nausea following meals, and impaired bile flow that alters absorption of fat and fat-soluble vitamins (A, D, E, and K) from the gut.

- Assess nutritional status, including diet history, height and weight, and skinfold measurements (see Chapters 21 and 22 ∞). *Even though often obese, clients with gallbladder disease may have an imbalanced diet or may have specific vitamin deficiencies, particularly of the fat-soluble vitamins.*
- Evaluate laboratory results, including serum bilirubin, albumin, glucose, and cholesterol levels. Report abnormal results

to the primary care provider. *Elevated serum bilirubin may indicate impaired bilirubin excretion due to obstructed bile flow. A low serum albumin may indicate poor nutritional status. Glucose intolerance and hypercholesterolemia are risk factors for cholelithiasis.*

- Refer to a dietitian or nutritionist for diet counseling to promote healthy weight loss and reduce pain episodes. *A low-carbohydrate, low-fat, higher protein diet reduces symptoms of cholecystitis. While fasting and very-low-calorie diets are contraindicated, a moderate reduction in calorie intake and increased activity levels promote weight loss.*
- Administer vitamin supplements as ordered. *Clients who do not absorb fat well due to obstructed bile flow may require supplements of the fat-soluble vitamins.*

Risk for Infection

An acutely inflamed gallbladder may become necrotic and rupture, releasing its contents into the abdominal cavity. While the resulting infection often remains localized, peritonitis can result from chemical irritation and bacterial contamination of the peritoneal cavity.

PRACTICE ALERT

Rupture of an acutely inflamed gallbladder may be heralded by abrupt but transient pain relief as contents are released from the distended gallbladder into the abdomen. Promptly report this change to the physician.

Following open cholecystectomy (*laparotomy*), the risk for pulmonary infection is significant due to the high abdominal incision.

- Monitor vital signs including temperature every 4 hours. Promptly report vital sign changes or temperature elevation. *Tachycardia, increased respiratory rate, or an elevated temperature may indicate an infectious process.*
- Assess abdomen every 4 hours and as indicated (e.g., when pain level changes abruptly). *Increasing abdominal tenderness or a rigid, boardlike abdomen may indicate rupture of the gallbladder with peritonitis.*
- Assist to cough and deep breathe or use incentive spirometer every 1 to 2 hours while awake. Splint abdominal incision with a blanket or pillow during coughing. *The high abdominal incision of an open cholecystectomy interferes with effective coughing and deep breathing, increasing the risk of atelectasis and respiratory infections such as pneumonia.*
- Place in Fowler's position and encourage ambulation as allowed. *Fowler's position and ambulating promote lung expansion and airway clearance, reducing the risk of respiratory infections.*
- Administer antibiotics as ordered. *Antibiotics may be given preoperatively to reduce the risk of infection from infected gallbladder contents, and may be continued postoperatively to prevent infection.*

Community-Based Care

Teaching varies, depending on the choice of treatment options for cholelithiasis and cholecystitis. If surgery is not an option, teach about medications that dissolve stones, their use and adverse effects (diarrhea is a common side effect), and maintaining a low-fat, low-carbohydrate diet if indicated. Include an explanation about the role of bile and the function of the gallbladder in terms that the client and family can understand.

Provide appropriate preoperative teaching for the planned procedure. Discuss the possibility of open cholecystectomy even when a laparoscopic procedure is planned. Teach postoperative self-care measures to manage pain and prevent complications. If the client will be discharged with a T-tube, provide instructions about its care (see the Nursing Care box on page 701). Discuss manifestations of complications to report to the physician. Stress the importance of follow-up appointments.

Following cholecystectomy, a low-fat diet may be initially recommended. Refer the client and food preparer to a dietitian to review low-fat foods. (See Box 24–3 for examples of high-fat foods to avoid.) Higher fat foods may be gradually added to the diet as tolerated.

THE CLIENT WITH CANCER OF THE GALLBLADDER

Gallbladder cancer is rare, primarily affecting people over age 65. Women are more likely to develop the disorder. Manifestations of gallbladder cancer include intense pain and a palpable mass in the RUQ of the abdomen. Jaundice and weight loss are common. Gallbladder cancers spread by direct extension to the liver, and metastasize via the blood and lymph system.

At the time of diagnosis, the cancer usually is too advanced to treat surgically. Ninety-five percent of clients with primary cancer of the gallbladder die within 1 year. Radical and extensive surgical interventions may be performed, but the prognosis is poor regardless of treatment (Tierney et al., 2005). Nursing care is palliative, focusing on maintaining comfort and independence to the extent possible.

LIVER DISORDERS

The liver is a complex organ with multiple metabolic and regulatory functions. Optimal liver function is essential to health. Because of the significant amount of blood in the liver at all times, it is exposed to the effects of pathogens, drugs, toxins, and possibly malignant cells. As a result, liver cells may become inflamed or damaged, or cancerous tumors may develop.

Physiology Review

The essential functions of the liver include the metabolism of proteins, carbohydrates, and fats. It also is responsible for the metabolism of steroid hormones and most drugs. It synthesizes essential blood proteins, including albumin and clotting

factors in particular. The liver detoxifies alcohol and other toxic substances. Ammonia, a toxic by-product of protein metabolism, is converted to urea in the liver for elimination by the kidneys. The liver produces bile, an essential substance for absorbing fats and eliminating bilirubin from the body. Minerals and fat-soluble vitamins are stored in the liver, as is glycogen (stored carbohydrate for energy reserves). The Kupffer cells that line the sinusoids phagocytize foreign cells and damaged blood cells. See Chapter 21 ∞ for more information about the liver.

Common Manifestations of Liver Disorders

Although many different disorders can disrupt liver function, their manifestations relate to three primary effects: disrupted liver cell function, impaired bilirubin conversion and excretion leading to jaundice, and disrupted blood flow through the liver, with resulting portal hypertension.

Hepatocellular Failure

The liver is vital to digestion and metabolism of nutrients; the production of plasma proteins, including those involved in clotting; and the metabolism and excretion of compounds such as bilirubin, steroid hormones, and ammonia, as well as toxins (such as alcohol) and drugs. Impaired function of liver cells has multiple effects, including:

- Impaired protein metabolism with decreased production of albumin and clotting factors. Low albumin levels contribute to edema in peripheral tissues and **ascites**, accumulation of fluid in the abdomen, as plasma oncotic pressure is reduced. Impaired clotting factor production increases the risk for bleeding.
- Disrupted glucose metabolism and storage with resulting alterations in blood glucose levels (either hyperglycemia or hypoglycemia).
- Reduced bile production that impairs the absorption of lipids and fat-soluble vitamins. Inadequate vitamin K, a fat-soluble vitamin, affects the production of clotting factors, leading to a bleeding tendency.
- Impaired metabolism of steroid hormones (including estrogen and testosterone) leads to feminization in men and irregular menses in women.

Jaundice

Disrupted metabolism and excretion of bilirubin allows it to accumulate in tissues, leading to **jaundice**, yellow staining of tissues. Jaundice (also called *icterus*) often is first noticeable in the sclera of the eyes, then the skin.

When RBCs are destroyed (due to cell aging or disease), hemoglobin is released. The hemoglobin molecule breaks up into globin, a protein, and heme, the iron-containing portion of the molecule. In this process, biliverdin, later converted to fat-soluble bilirubin (*unconjugated bilirubin*), is released. The bilirubin binds with albumin to be transported to the liver. In the liver, it is converted to a water-soluble form (*conjugated bilirubin*) to be excreted in the bile. See Box 24–2 for more information about bilirubin metabolism.

Jaundice can result from disruptions at any point in the production and metabolism of bilirubin:

- *Hemolytic jaundice* develops when excess RBC destruction (hemolysis) releases more bilirubin into circulation than the liver is able to process. High blood levels of unconjugated bilirubin are seen.
- *Hepatic jaundice* occurs when impaired liver cell (*hepatocyte*) function disrupts the conversion and excretion of bilirubin. Blood levels of both conjugated and unconjugated bilirubin may be elevated. Stools may appear normal or clay colored, and urine is dark because the conjugated bilirubin is excreted by the kidneys.
- Obstruction of bile flow within the biliary system (the gallbladder and bile ducts) impairs bilirubin excretion, leading to *obstructive jaundice*. Levels of conjugated bilirubin are elevated. Stools are light or clay colored due to lack of bile pigment; and urine is dark because the kidneys excrete bilirubin.

Portal Hypertension

Impaired blood flow through the liver increases pressure in the portal venous system that drains the gastrointestinal tract, the spleen, and surface veins of the abdomen. **Portal hypertension**, increased pressure in the portal system, has several effects when it is prolonged:

- Dilation of veins in the gastrointestinal tract and the abdominal wall. This congestion tends to suppress the appetite, and lead to formation of collateral vessels in the distal esophagus, stomach, and rectum. The dilated, congested vessels in the esophagus are known as **esophageal varices**; in the rectum, they lead to the development of hemorrhoids. In advanced liver failure, superficial varices may develop around the umbilicus, a feature known as *caput medusae*.
- Splenomegaly, or enlargement of the spleen.
- Ascites, accumulation of fluid in the peritoneal cavity. Increased hydrostatic pressure in abdominal vessels forces fluid out of the vessels and into the peritoneal cavity. Low serum albumin levels (*hypoalbuminemia*) contribute to fluid accumulation by reducing the osmotic draw of fluid back into vessels.
- **Portal systemic encephalopathy** (or hepatic *encephalopathy*), impaired consciousness and mental status due to the accumulation of toxic waste products in the blood (ammonia in particular) as blood bypasses the congested liver. It appears that factors other than elevated ammonia levels contribute, including toxic fatty acids, altered neurotransmitters, and an imbalance of plasma amino acid ratios. Cerebral edema develops late in the course of liver failure, resulting from both the accumulation of toxins and vascular mechanisms. As cerebral edema progresses, intracranial pressure increases, cerebral perfusion decreases, and brain cells become hypoxic.
- **Hepatorenal syndrome** is acute renal failure due to disrupted blood flow to the kidneys. See Chapter 29 ∞ for more information about renal failure.

See the section of this chapter on cirrhosis for more information about the effects and complications associated with portal hypertension.

THE CLIENT WITH HEPATITIS

Hepatitis is inflammation of the liver. It is usually caused by a virus, although it may result from exposure to alcohol, drugs and toxins, or other pathogens. Hepatitis may be acute or chronic in nature. Cirrhosis, discussed in the next section, is a potential consequence of severe hepatocellular damage. Chronic hepatitis also increases the risk for developing liver cancer.

Pathophysiology and Manifestations

The inflammatory process of hepatitis, whether caused by a virus, toxin, or other mechanism, damages hepatic cells and disrupts liver function. Cell-mediated immune responses damage hepatocytes and Kupffer cells, leading to hyperplasia, necrosis, and cellular regeneration. The flow of bile through bile canaliculi and into the biliary system can be impaired by the inflammatory process, leading to jaundice. When the inflammatory process is mild (e.g., hepatitis A), the liver parenchyma is not significantly damaged. The inflammatory processes associated with hepatitis B and hepatitis C, however, can lead to severe liver damage. The metabolism of nutrients, drugs, alcohol, and toxins and the process of bile elimination are disrupted by the inflammation of hepatitis. See Chapter 21 ∞ for more information about the liver, and the preceding section for more information about the effects of disrupted liver function.

Viral Hepatitis

At least five viruses are known to cause hepatitis: hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), the hepatitis B–associated delta virus (HDV), and hepatitis E virus (HEV). With the exception of HBV, all of the hepatitis viruses are RNA viruses; HBV is a DNA virus. The viruses differ from one another in mode of transmission, incubation period, the severity and type of liver damage they cause, and their ability to become chronic or develop a carrier (asymptomatic) state. The illnesses they cause, however, are clinically very similar. Table 24–2 identifies unique features of the primary hepatitis viruses.

FAST FACTS

In the United States, hepatitis A, hepatitis B, and hepatitis C dominate.

- Hepatitis A is slightly more common than hepatitis B, with a reported 7,653 cases in 2003. The rate of hepatitis A infections per 100,000 population has fallen steadily since 1971, when it peaked at 28.9, to a rate of 2.6 in 2003.
- There were 7,526 reported cases of hepatitis B in 2003. In 1966, the rate of reported hepatitis B infections was 0.8 per 100,000 population. It peaked at 11.5 in 1985, since then falling to 2.6.
- In 2003, 891 cases of hepatitis C were reported, for a rate of 0.4 per 100,000 population (Centers for Disease Control and Prevention [CDC], 2006).
- The estimated number of actual cases of viral hepatitis is significantly higher than the number of reported cases for all three types of viral hepatitis.

MEDIALINK Case Study: A Client with Hepatitis

Hepatitis viruses replicate in the liver, damaging liver cells (hepatocytes). The viruses provoke an immune response that causes inflammation and necrosis of hepatocytes as well. Although the extent of damage and the immune response vary among the different hepatitis viruses, the disease itself usually follows a predictable pattern.

No manifestations are present during the incubation period after exposure to the virus. The *prodromal* or *preicteric* (before jaundice) *phase* may begin abruptly or insidiously, with general malaise, anorexia, fatigue, and muscle and body aches. These manifestations often are mistaken for the flu. Nausea, vomiting, diarrhea, or constipation may develop, as well as mild RUQ abdominal pain. Chills and fever may be present.

The *icteric* (jaundiced) *phase* usually begins 5 to 10 days after the onset of symptoms. It is heralded by jaundice of the sclera, skin, and mucous membranes. Inflammation of the liver and bile ducts prevents bilirubin from being excreted into the small intestine. As a result, the serum bilirubin levels are elevated, causing yellowing of the skin and mucous membranes. Pruritus may develop due to deposition of bile salts on the skin.

TABLE 24–2 **Comparison of Types of Viral Hepatitis**

VIRUS	HEPATITIS A (HAV)	HEPATITIS B (HBV)	HEPATITIS C (HCV)	HEPATITIS D (HDV)	HEPATITIS E (HEV)
Mode of transmission	Fecal–oral	Blood and body fluids; perinatal	Blood and body fluids	Blood and body fluids; perinatal	Fecal–oral
Incubation (in weeks)	2–6	6–24	5–12	3–13	3–6
Onset	Abrupt	Slow	Slow	Abrupt	Abrupt
Carrier state	No	Yes	Yes	Yes	Yes
Possible complications	Rare	Chronic hepatitis Cirrhosis Liver cancer	Chronic hepatitis Cirrhosis Liver cancer	Chronic hepatitis Cirrhosis Fulminant hepatitis	May be severe in pregnant women
Laboratory findings	Anti-HAV antibodies present	Positive HBsAg (HBV surface antigen); anti-HBV antibodies present	Anti-HCV antibodies present	Positive HDVAg (delta antigen) early; anti-HDV antibodies later	Anti-HEV antibodies present

The stools are light brown or clay colored because bile pigment is not excreted through the normal fecal pathway. Instead, the pigment is excreted by the kidneys, causing the urine to turn brown. Whereas clients with acute hepatitis A or B are likely to develop jaundice, many people with hepatitis C do not develop jaundice. As a result, the infection may go undiagnosed for an extended period of time.

During the icteric phase, the initial prodromal manifestations usually diminish even though the serum bilirubin increases. The appetite increases, and the temperature returns to normal. When uncomplicated, spontaneous recovery usually begins within 2 weeks of the onset of jaundice.

The *convalescent phase* follows jaundice and lasts several weeks. During this time, manifestations gradually improve: Serum enzymes decrease, liver pain decreases, and gastrointestinal symptoms and weakness subside. See the box on this page for the manifestations of each phase of hepatitis.

HEPATITIS A Hepatitis A, or *infectious hepatitis,* often occurs in either sporadic attacks or mild epidemics. It is transmitted by the fecal–oral route via contaminated food, water, shellfish, and direct contact with an infected person. The virus is in the stool of infected persons up to 2 weeks before symptoms develop. Once jaundice develops, the amount of virus in the stool and the risk of spreading the disease decrease significantly (Kasper et al., 2005). Although hepatitis A usually has an abrupt onset, it is typically a benign and self-limited disease with few long-term consequences. Symptoms last up to 2 months.

HEPATITIS B Hepatitis B can cause acute hepatitis, chronic hepatitis, *fulminant* (rapidly progressive) hepatitis, or a carrier state. In a *carrier state*, the person harbors the active virus and is capable of spreading it to others, even though there are no discernible manifestations of the disease. This virus is spread through contact with infected blood and body fluids. Healthcare workers are at risk through exposure to blood and needlestick injuries. Other high-risk groups for hepatitis B include injection drug users, people with multiple sex partners, men who have sex with other men, and people frequently exposed to blood products (such as people on hemodialysis). Hepatitis B is a major risk factor for primary liver cancer.

In hepatitis B, liver cells are damaged by the immune response to this antigen. Damage may affect only portions or the majority of the liver. The liver shows evidence of injury and scarring, regeneration, and proliferation of inflammatory cells. During the prodromal period, clients with HBV may experience such immune-mediated manifestations as urticaria and other rashes, arthralgias, serum sickness, or glomerulonephritis (Copstead & Banasik, 2005). The disease itself may be asymptomatic.

HEPATITIS C Hepatitis C, formerly known as non-A, non-B hepatitis, is the primary worldwide cause of chronic hepatitis, cirrhosis, and liver cancer (Porth, 2005). It is transmitted through infected blood and body fluids. Injection drug use is the primary risk factor for HCV infection. Acute hepatitis C usually is asymptomatic; if symptoms do develop, they often are mild and nonspecific. The disease often is recognized long after exposure occurred, when secondary effects of the disease (such as chronic hepatitis or cirrhosis) develop. Hepatitis C is unique, in that it does not produce lasting immunity to reinfection (Kasper et al., 2005). Only about 15% of acute infections completely resolve; most progress to chronic active hepatitis (Copstead & Banasik, 2005).

HEPATITIS DELTA Hepatitis delta only causes infection in people who also are infected with hepatitis B. It can cause acute or chronic infection, and can increase the severity of HBV infection (Porth, 2005). It is transmitted in the same manner as HBV. Because of its dependence on HBV, recovery from hepatitis B results in concurrent recovery from hepatitis delta.

HEPATITIS E Hepatitis E is rare in the United States. It is transmitted by fecal contamination of water supplies in developing areas such as southeast Asia, parts of Africa, and Central America. It primarily affects young adults. It can cause fulminant, fatal hepatitis in pregnant women.

MANIFESTATIONS of Acute Hepatitis

PREICTERIC PHASE
- "Flulike" symptoms: malaise, fatigue, fever
- Gastrointestinal: anorexia, nausea, vomiting, diarrhea, constipation
- Muscle aches, polyarthritis
- Mild right upper abdominal pain and tenderness

ICTERIC PHASE
- Jaundice
- Pruritus
- Clay-colored stools
- Brown urine
- Decrease in preicteric phase symptoms (e.g., appetite improves; no fever)

POSTICTERIC/CONVALESCENT PHASE
- Serum bilirubin and enzymes return to normal levels
- Energy level increases
- Pain subsides
- Gastrointestinal: minimal to absent

Chronic Hepatitis

Chronic hepatitis is chronic infection of the liver. Although it may cause few symptoms, it is the primary cause of liver damage leading to cirrhosis, liver cancer, and liver transplantation. Three of the known hepatitis viruses cause chronic hepatitis: HBV, HCV, and HDV. Manifestations of chronic hepatitis include malaise, fatigue, and hepatomegaly. Occasional icteric (jaundiced) periods may occur. Liver enzymes, particularly serum aminotransferase levels, typically are elevated.

In *chronic active hepatitis*, inflammation extends to involve entire hepatic lobules. Chronic active hepatitis usually leads to cirrhosis and end-stage liver failure.

Fulminant Hepatitis

Fulminant hepatitis is a rapidly progressive disease, with liver failure developing within 2 to 3 weeks after the onset of symptoms. Although uncommon, it is usually related to HBV with concurrent HDV infection.

Toxic Hepatitis

Many substances, including alcohol, certain drugs, and other toxins, can directly damage liver cells. Alcoholic hepatitis can result from chronic alcohol abuse or from an acute toxic reaction to alcohol. Alcoholic hepatitis causes necrosis of hepatocytes and inflammation of the liver parenchyma (functional tissue). Unless alcohol intake is avoided, progression to cirrhosis is common.

Other potential hepatotoxins include acetaminophen, benzene, carbon tetrachloride, halothane, chloroform, and poisonous mushrooms. These substances directly damage liver cells, leading to necrosis. The degree of damage often depends on age and the extent of exposure (dose) to the hepatotoxin. Acetaminophen is a common cause of hepatocellular damage.

Hepatobiliary Hepatitis

Hepatobiliary hepatitis is due to cholestasis, the interruption of the normal flow of bile. Cholestasis may result from obstruction of the hepatic duct with stones or inflammation secondary to cholelithiasis. Other agents, such as oral contraceptives and allopurinol (a drug used to lower uric acid levels), also can cause cholestasis. When bile flow is disrupted, the liver parenchyma may become inflamed. Reestablishing bile flow by removing the stone or other causative agent is the treatment for hepatobiliary hepatitis.

INTERDISCIPLINARY CARE

Management of hepatitis focuses on determining its cause, providing appropriate treatment and support, and teaching strategies to prevent further liver damage. Effective management begins with thorough assessment of diagnostic and laboratory data.

Diagnosis

Liver function tests, such as blood levels of bilirubin and enzymes commonly released when liver cells are damaged, are obtained. These include the following:

- *Alanine aminotransferase (ALT)* is an enzyme contained within each liver cell. When liver cells are damaged, ALT is released into the blood. Levels may exceed 1000 U/L or more in acute hepatitis.
- *Aspartate aminotransferase (AST)* is an enzyme found predominantly in heart and liver cells. AST levels rise when liver cells are damaged; with severe damage, blood levels may be 20 to 100 times normal values.
- *Alkaline phosphatase (ALP)* is an enzyme present in liver cells and bone. Serum ALP levels often are elevated in hepatitis.
- *Gamma-glutamyltransferase (GGT)* is an enzyme present in cell membranes. Its blood levels rise in hepatitis and obstructive biliary disease, and remain elevated until function is restored.
- *Lactic dehydrogenase (LDH),* an enzyme present in many body tissues, is a nonspecific indicator of tissue damage. Its isoenzyme, LDH5, is a specific indicator of liver damage.
- *Serum bilirubin* levels, including *conjugated* and *unconjugated,* are elevated in viral hepatitis due to impaired bilirubin metabolism and obstruction of the hepatobiliary ducts by inflammation and edema. The bilirubin level decreases as inflammation and edema subside.
- Laboratory tests for viral antigens and their specific antibodies may be done to identify the infecting virus and its state of activity. These tests are summarized in Table 24–2.
- A *liver biopsy* may be done to detect and evaluate chronic hepatitis. (Nursing implications for this test are outlined in the box on page 617.)

See Chapter 21 ∞ for more information about diagnostic tests and their nursing implications.

Medications

PREVENTION Hepatitis A and hepatitis B are preventable diseases. Vaccines are available, as are preparations to prevent the disease following known or suspected exposure.

Vaccines Hepatitis A vaccine provides long-term protection against HAV infection. It is an inactivated whole-virus vaccine available in pediatric and adult formulations. Although more than 95% of adults achieve immunity after one dose of the vaccine, two doses are recommended for full protection. See Table 24–3.

Three doses of hepatitis B vaccine provide immunity to HBV infection in 90% of healthy adults. Because the hepatitis delta virus requires the presence of the hepatitis B virus, hepatitis B vaccine also protects against HDV. Hepatitis B vaccine is a recombinant vaccine. Vaccines produced by different manufacturers may be used interchangeably, although their dosages differ. Older adults are less likely to achieve immunity than younger adults. Clients on hemodialysis and people who are immunocompromised may need larger or more doses of the vaccine to achieve adequate protection. Serologic testing for immunity is recommended on completion of the series for people in these high-risk groups.

A combined hepatitis A and hepatitis B vaccine is available for use. It is recommended for the same high-risk populations as the single vaccines. Three doses are given: the initial dose, followed by doses no sooner than 4 weeks and 6 months later.

Postexposure Prophylaxis Postexposure prophylaxis may be recommended for household or sexual contacts of people with HAV or HBV and other people who are known to have been exposed to these viruses. It is not necessary if the exposed person has been vaccinated and is known to be immune.

Hepatitis A prophylaxis is provided by a single dose of immune globulin (IG) given within 2 weeks after exposure. IG is recommended for all people with household or sexual contact with a person known to be infected with hepatitis A. See Table 24–3 for further recommendations.

Hepatitis B postexposure prophylaxis is indicated for people exposed to the hepatitis B virus. Hepatitis B immune globulin (HBIG) is given to provide for short-term immunity. HBV vaccine may be given concurrently. Candidates for postexposure prophylaxis include those with known or suspected percutaneous or permucosal contact with infected blood, sexual partners of clients with acute HBV or who are HBV carriers, and household contacts of clients with acute HBV infection (National Immunization Program, 2005).

TABLE 24–3 **CDC Recommendations for Hepatitis Prevention in Adults**

DISEASE/STRATEGY	IMMUNIZATION	ADVERSE REACTIONS	POPULATION RECOMMENDATIONS
Hepatitis A			
Prevention	Hepatitis A vaccine (Havrix; VAQTA), 2 doses (initial dose with booster in 6–12 months) IM into deltoid muscle Combined hepatitis A and hepatitis B vaccine (Twinrix), 3 doses (initial dose followed by doses 4 weeks and 6 months later) given IM into deltoid muscle	Pain at injection site	■ Children > 2 yr ■ International travelers ■ Men who have sex with men ■ Drug users ■ Persons with clotting-factor disorders, chronic liver disease, hepatitis C ■ Persons with occupational risk
Postexposure prophylaxis	Standard immune globulin IM into large muscle mass within 2 weeks of exposure	Rare; risk of anaphylaxis in people with IgA deficiency	■ Close contacts of people with known hepatitis A ■ People potentially exposed to hepatitis A at child care center or restaurant with infected food handler
Hepatitis B			
Prevention	Recombinant hepatitis B vaccine (Recombivax HB; Engerix-B), 3 doses (initial dose followed by doses at 4 weeks and 5 months later) given IM into deltoid muscle Combined hepatitis A and hepatitis B vaccine (Twinrix), 3 doses (initial dose followed by doses 4 weeks and 6 months later) given IM into deltoid muscle	Pain at injection site; fatigue, headache	■ Infants and adolescents ■ Adults with increased risk of HBV ■ Men who have sex with men ■ Prostitutes; heterosexuals with multiple sexual partners ■ People with an STI ■ Injection drug users ■ Long-term male prisoners ■ People on hemodialysis ■ Healthcare workers
Postexposure prophylaxis	Hepatitis B immune globulin (HBIG) given IM into large muscle mass within 24 hours to 7 days of exposure, second dose 28 to 30 days after exposure; concurrent initiation of hepatitis B vaccine series	Infrequent; muscle stiffness, pain	■ Infants born to women with HBV infection ■ Percutaneous or permucosal exposure to HBV when unvaccinated or antibody response is negative or unknown

Source: From *Epidemiology and Prevention of Vaccine-Preventable Diseases* (8th ed.) by Centers for Disease Control and Prevention, January 2005, Atlanta: Department of Health and Human Services.

Treatments

In most cases of acute viral hepatitis, pharmacologic treatment of the infection is not indicated. Acute hepatitis C generally is treated with interferon alpha, an antiviral agent, to reduce the risk of chronic hepatitis C. While treatment with interferon alpha alone is common, it may be combined with the antiviral drug ribavirin (Rebetol, Virazole).

Interferon alpha is used to treat both chronic hepatitis B and chronic hepatitis C. Interferon α interferes with viral replication, reducing the viral load. It is given by intramuscular or subcutaneous injection. Virtually all clients treated with interferon alpha develop a flulike syndrome with fever, fatigue, muscle aches, headache, and chills. Acetaminophen helps alleviate some of these adverse effects. Depression also is a common adverse effect of this drug.

An alternate drug for treating chronic hepatitis B is lamivudine (Epivir HBV), an antiviral drug that can reduce liver inflammation and fibrosis. Although it has minimal side effects, clients may become resistant to the beneficial effects of lamivudine.

The treatment of choice for chronic hepatitis C is combination therapy of interferon alpha with ribavirin, an oral antiviral drug. This combination therapy improves the response rate over either drug used alone. Ribavirin has two major adverse effects: hemolytic anemia and birth defects. Blood counts are obtained before and during treatment to detect early signs of hemolytic anemia. Because of the risk for birth defects, this drug is contraindicated for use during pregnancy, and two reliable methods of birth control must be used by women taking the drug and female sexual partners of men taking the drug.

Treatment of acute hepatitis also includes as-needed bed rest, adequate nutrition as tolerated, and avoidance of strenuous activity, alcohol, and agents that are toxic to the liver. In most cases, clinical recovery takes 3 to 16 weeks.

COMPLEMENTARY THERAPIES Milk thistle, with its active ingredient silymarin, has been used by herbalists to treat liver disease for over 2000 years. Clinical studies have demonstrated that treatment with silymarin promotes recovery and reduces complications in clients with viral hepatitis (Agency for Healthcare Research and Quality, 2000; National Center for Complementary and Alternative Medicine, 2004). It also is beneficial for clients who have liver damage due to toxins, cirrhosis, and alcoholic liver disease. Silymarin's beneficial effects are attributed to its ability to promote liver cell growth, block toxins from entering and damaging liver cells, and reduce liver inflammation. It also is a powerful antioxidant.

Herbalists also may use licorice root to treat hepatitis. It has both antiviral and anti-inflammatory effects. Long-term use of licorice root, however, can lead to hypertension and affect fluid and electrolyte balance.

Herbal preparations also may be used to relieve the adverse effects of interferon alpha. Ginger can help relieve nausea, and St. John's wort is used for the depression associated with interferon alpha.

NURSING CARE

Health Promotion

Nurses play an instrumental role in preventing the spread of hepatitis. Stress the importance of hygiene measures such as hand washing after toileting and before all food handling. Discuss the dangers of injection drug use and, with drug users, of sharing needles or other equipment. Encourage all sexually active clients to use safer sexual practices such as abstinence, mutual monogamy, and barrier protection (such as male or female condoms).

Discuss recommendations for hepatitis A and hepatitis B vaccine with people in high or moderate risk groups for these infections. Ensure that nurses and other healthcare workers at risk for exposure to blood and body fluids are effectively vaccinated against hepatitis A and B. Encourage all people with known or probable exposure to HAV or HBV to obtain postexposure prophylaxis.

Assessment

Collect assessment data related to hepatitis, such as the following:

- *Health history:* Current manifestations, including anorexia, nausea, vomiting, abdominal discomfort, changes in bowel elimination or color of stools; muscle or joint pain, fatigue; changes in color of skin or sclera; duration of symptoms; known exposure to hepatitis; high-risk behaviors such as injection drug use or multiple sexual partners; previous history of liver disorders; current medications, prescription and over the counter.
- *Physical assessment:* Vital signs including temperature; color of sclera and mucous membranes; skin color and condition; abdominal contour and tenderness; color of stool and urine.
- *Diagnostic tests:* Serum bilirubin, liver function tests, serologic antibody–antigen levels.

Nursing Diagnoses and Interventions

Clients with acute or chronic hepatitis usually are treated in community settings; rarely is hospitalization required. Nursing care focuses on preventing spread of the infection to others and promoting the client's comfort and ability to provide self-care.

Risk for Infection (Transmission)

An important goal when caring for clients with acute viral hepatitis is preventing spread of the infection.

- Use standard precautions. Practice meticulous hand washing. *The hepatitis viruses are spread by direct contact with feces or blood and body fluids. Standard precautions and good hand washing protect both healthcare workers and other clients from exposure to the virus.*
- For clients with HAV or HEV, use standard precautions and contact isolation if fecal incontinence is present. *The fecal–oral route is the primary mode of transmission of these viruses. Other hepatitis viruses are transmitted through blood and other body fluids.*
- Encourage prophylactic treatment of all members of household and intimate sexual contacts. *Prophylactic treatment of people in close contact with the client decreases their risk of contacting the disease or, if already infected, the severity of the disease.*

PRACTICE ALERT

If the client diagnosed with hepatitis A is employed as a food handler or child care worker, contact the local health department to report possible exposure of patrons. Maintain confidentiality. Prophylactic treatment of people who have possibly been exposed to the virus can prevent a local epidemic of the disease.

Fatigue

Fatigue and possible weakness are common in acute hepatitis. Although bed rest is rarely indicated, adequate rest periods and limitation of activities may be necessary. Many clients with acute hepatitis may be unable to resume normal activity levels for 4 or more weeks.

- Encourage planned rest periods throughout the day. *Adequate rest is necessary for optimal immune function.*
- Assist to identify essential activities and those that can be deferred or delegated to others. *Identifying essential and nonessential activities promotes the client's sense of control.*
- Suggest using level of fatigue to determine activity level, with gradual resumption of activities as fatigue and sense of well-being improves. *Fatigue associated with activity is an indicator of appropriate and inappropriate activity levels. As recovery progresses, increasing activity levels are tolerated with less fatigue.*

Imbalanced Nutrition: Less than Body Requirements

Adequate nutrition is important for immune function and healing in clients with acute or chronic hepatitis.

- Help plan a diet of appealing foods that provides a high-kilocalorie intake of approximately 16 carbohydrate kilocalories per kilogram of ideal body weight. *Sufficient energy is required for healing; adequate carbohydrate intake can spare protein.*

MEDIALINK Care Plan Activity: A Client with Hepatitis A

- Encourage planning food intake according to symptoms of the disease. Discuss eating smaller meals and using between-meal snacks to maintain nutrient and calorie intake. *Clients with acute hepatitis often are more anorexic and nauseated in the afternoon and evening; planning the majority of calorie intake in the morning helps maintain adequate intake. Limiting fat intake and the size of meals may reduce the incidence of nausea.*
- Instruct to avoid alcohol intake and diet drinks. *Alcohol avoidance is vital to prevent further liver damage and promote healing. Diet drinks (e.g., diet sodas or juice drinks) provide few calories when an increased calorie intake is needed for healing.*
- Encourage use of nutritional supplements such as Ensure or instant breakfast drinks to maintain calorie and nutrient intake. *Nutritional supplement drinks are an additional source of concentrated calories and nutrients.*

Disturbed Body Image

Jaundice and associated rashes and itching can affect the client's body image. Nursing measures to prevent skin breakdown and address body image are discussed in the following section on cirrhosis.

Using NANDA, NIC, and NOC

Chart 24–1 shows linkages between NANDA nursing diagnosis, NIC, and NOC for the client with viral hepatitis.

Community-Based Care

Provide discharge teaching to clients and their families for home care. Include the following topics:

- Recommended prophylactic treatment
- Infection control measures such as frequent hand washing, not sharing eating utensils, avoiding food handling or preparation activities by the client with hepatitis A; abstaining from sexual relations during acute infection and using barrier protection if a carrier or for chronic infection
- Managing fatigue and limited activity
- Managing pruritus and maintaining skin integrity: use warm, not hot water when bathing, use mild or no soap, limit duration of baths and showers; pat dry, do not rub, apply an alcohol-free lotion soon after bathing to retain skin moisture; wear loose cotton garments that allow moisture to evaporate from skin; reduce room temperature, especially at night, to prevent overheating; keep fingernails short, and wear cotton mittens or gloves as needed to prevent scratching during sleep
- Promoting nutrient intake
- Avoiding hepatic toxins such as alcohol, acetaminophen, and selected other drugs; encourage to alert all care providers to presence of infection
- Recommended follow-up.

If chronic hepatitis B or C is being treated with medications, teach how to administer the drug, its dosing schedule, precautions, and management of adverse effects. Stress the importance of keeping follow-up appointments, including recommended laboratory testing.

NANDA, NIC, AND NOC LINKAGES
CHART 24–1 **The Client with Viral Hepatitis**

NANDA

Imbalanced Nutrition: Less than Body Requirements

↓

NIC

Energy Management

Nutrition Management

↓

NOC

Energy Conservation

Nutritional Status: Nutrient Intake

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

THE CLIENT WITH CIRRHOSIS

Cirrhosis is the end stage of chronic liver disease. It is a progressive, irreversible disorder, eventually leading to liver failure.

FAST FACTS

- Cirrhosis is the 12th leading cause of death in the United States overall.
- In adults ages 25 to 64 years, however, cirrhosis/chronic liver disease is the 6th leading cause of death.
- Overall, the death rate due to cirrhosis and chronic liver disease in men is twice that of women (National Center for Health Statistics [NCHS], 2005).

Alcoholic or *Laënnec's* **cirrhosis** is the most common type of cirrhosis in North America and many parts of Europe and South America (Kasper et al., 2005). Cirrhosis also may result from chronic hepatitis B or C; prolonged obstruction of the biliary (bile drainage) system; long-term, severe right heart failure, and other uncommon liver disorders. The incidence and mortality attributable to cirrhosis and chronic liver disease vary significantly among populations. See the accompanying Focus on Cultural Diversity box.

Pathophysiology

In cirrhosis, functional liver tissue is gradually destroyed and replaced by fibrous scar tissue. As hepatocytes and liver lobules are destroyed, the metabolic functions of the liver are lost. Structurally abnormal nodules encircled by connective tissue form. This fibrous connective tissue forms constrictive bands that disrupt blood and bile flow within liver lobules. Blood no longer flows freely through the liver to the inferior vena cava.

FOCUS ON CULTURAL DIVERSITY
Cirrhosis

- Although cirrhosis/chronic liver disease is the 12th leading cause of death overall in the United States, it is the 6th leading cause of death for people of Native American (including Alaska Natives) and Hispanic (or Latino) origin.
- Native American men have the highest incidence and mortality rate from cirrhosis and chronic liver disease, followed by Native American women, Hispanic men, and women of Hispanic or Latino origin (NCHS, 2005).
- At this time, there is no clear explanation for these differences. Contributory factors may include:
  - Socioeconomic factors that lead to greater stress and alcohol consumption among certain populations
  - Patterns of alcohol consumption (e.g., consuming alcohol without food calories)
  - Variations in alcohol metabolism among populations (Mann et al., 2003).

This restricted blood flow leads to portal hypertension, increased pressure in the portal venous system.

Alcoholic Cirrhosis

Alcoholic or Laënnec's cirrhosis is the end result of alcoholic liver disease. Its development is directly related to alcohol consumption: total amount of alcohol consumed, number of years of excessive alcohol consumption, and blood alcohol levels. Women develop cirrhosis at lower overall levels of alcohol use than men. This may relate to less effective metabolism of alcohol in women, resulting in higher blood alcohol levels (Mann et al., 2003).

Alcohol causes metabolic changes in the liver: Triglyceride and fatty acid synthesis increases, and the formation and release of lipoproteins decrease, leading to fatty infiltration of hepatocytes (fatty liver). At this stage, abstinence from alcohol can allow the liver to heal. With continued alcohol abuse, the disease progresses. Inflammatory cells infiltrate the liver (alcoholic hepatitis), causing necrosis, fibrosis, and destruction of functional liver tissue. In the final stage of alcoholic cirrhosis, regenerative nodules form, and the liver shrinks and develops a nodular appearance. Malnutrition commonly accompanies alcoholic cirrhosis. See *Pathophysiology Illustrated: Cirrhosis and Portal Hypertenseion* on the following pages.

Biliary Cirrhosis

When bile flow is obstructed within the liver or in the biliary system, retained bile damages and destroys liver cells close to the interlobular bile ducts. This leads to inflammation, fibrosis, and formation of regenerative nodules.

Posthepatic Cirrhosis

Advanced progressive liver disease resulting from chronic hepatitis B or C or from an unknown cause is known as posthepatic or postnecrotic cirrhosis. Chronic viral hepatitis appears to be the leading cause of posthepatic cirrhosis in the United States (Kasper et al., 2005). The liver is shrunken and nodular, with extensive liver cell loss and fibrosis.

Manifestations and Complications

Early in the course of cirrhosis, few manifestations may be present. The liver usually is enlarged and may be tender. A dull, aching pain in the RUQ may be present. Other early signs include weight loss, weakness, and anorexia. Bowel function is disrupted with diarrhea or constipation (Porth, 2005).

As the disease progresses, manifestations related to liver cell failure and portal hypertension develop. Impaired metabolism causes such manifestations as bleeding, ascites, gynecomastia (breast enlargement) in men and infertility in women, jaundice, and neurologic changes. Portal hypertension accounts from such manifestations as ascites, peripheral edema, anemia, and low WBC and platelet counts. See *Multisystem Effects of Cirrhosis* on page 714.

Portal Hypertension

Portal hypertension causes blood to be rerouted to adjoining lower pressure vessels. This *shunting* of blood involves collateral vessels. Affected veins, which become engorged and congested, are located in the esophagus, rectum, and abdomen. Portal hypertension increases the hydrostatic pressure in vessels of the portal system. Increased hydrostatic pressure in the capillaries pushes fluid out, contributing to ascites formation.

Splenomegaly

The spleen enlarges (splenomegaly) because portal hypertension causes blood to be shunted into the splenic vein. Splenomegaly increases the rate at which red and white blood cells and platelets are removed from circulation and destroyed. This increased blood cell destruction leads to anemia, leukopenia, and thrombocytopenia (Porth, 2005).

Ascites

Ascites is the accumulation of plasma-rich fluid in the abdominal cavity. Although portal hypertension is the primary cause of ascites, decreased serum proteins and increased aldosterone also contribute to the fluid accumulation. *Hypoalbuminemia,* low serum albumin, decreases the colloidal osmotic pressure of plasma. This pressure normally holds fluid in the intravascular compartment; when plasma colloidal osmotic pressure decreases, fluid escapes into extravascular compartments. *Hyperaldosteronism,* an increase in aldosterone, causes sodium and water retention, contributing to ascites and generalized edema. See the box on page 715 for selected manifestations of cirrhosis and their underlying pathophysiology.

Esophageal Varices

Esophageal varices are enlarged, thin-walled veins that form in the submucosa of the esophagus. These collateral vessels form when blood is shunted from the portal system due to portal hypertension. The thin-walled varices may rupture, causing massive hemorrhage; even eating high-roughage foods can precipitate bleeding. Thrombocytopenia, platelet deficiency, and impaired production of clotting factors by the liver contribute to the risk for hemorrhage.

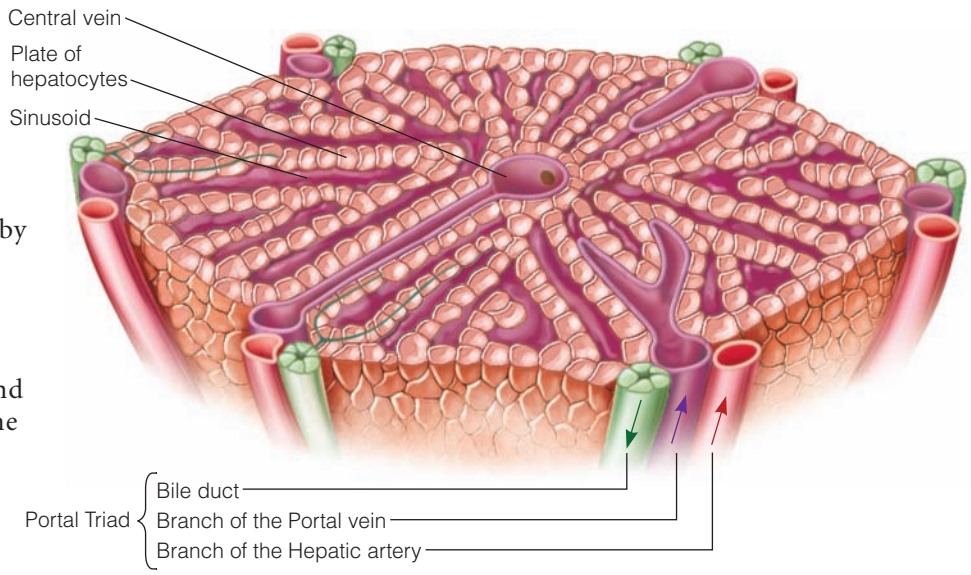
MEDIALINK Cirrhosis Animation

PATHOPHYSIOLOGY ILLUSTRATED

Cirrhosis and Portal Hypertension

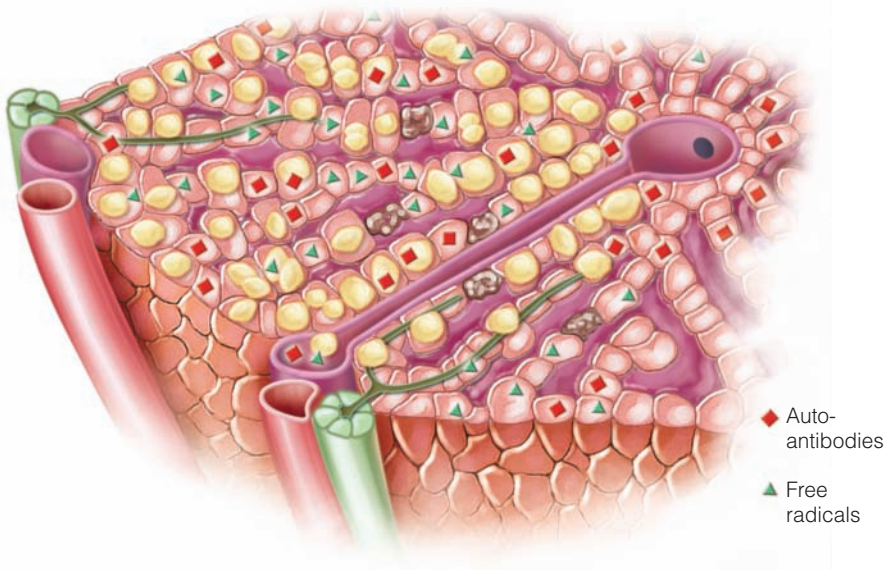
Normal liver

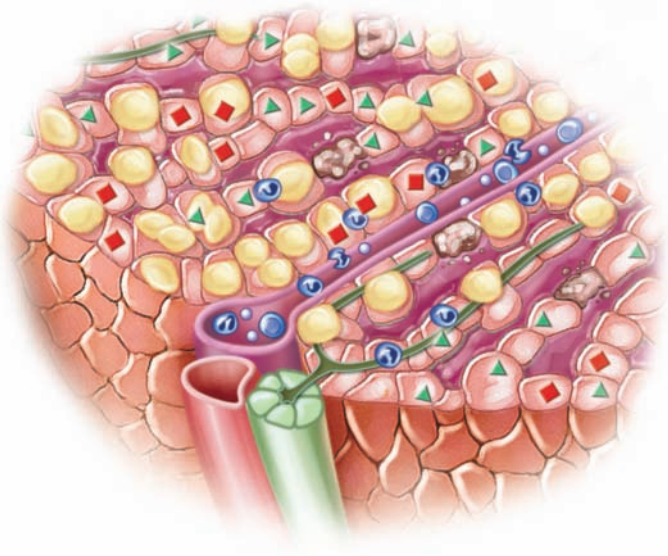
The liver contains multiple lobules made up of plates of hepatocytes, the functional cells of the liver, surrounded by small capillaries called sinusoids. These sinusoids receive a mixture of venous and arterial blood from branches of the portal vein and hepatic artery. Blood from the sinusoids drains into the central vein of the lobule. Hepatocytes produce bile, which drains outward to bile ducts.



Fatty liver

Ingested alcohol is primarily metabolized in the liver. Acetaldehyde, formed when alcohol is metabolized, damages hepatocytes and impairs the oxidation of fatty acids. As a result, fat accumulates within hepatocytes and liver lobules. Other alcohol metabolism byproducts, including oxygen free radicals, promote inflammation and may stimulate autoantibody production.



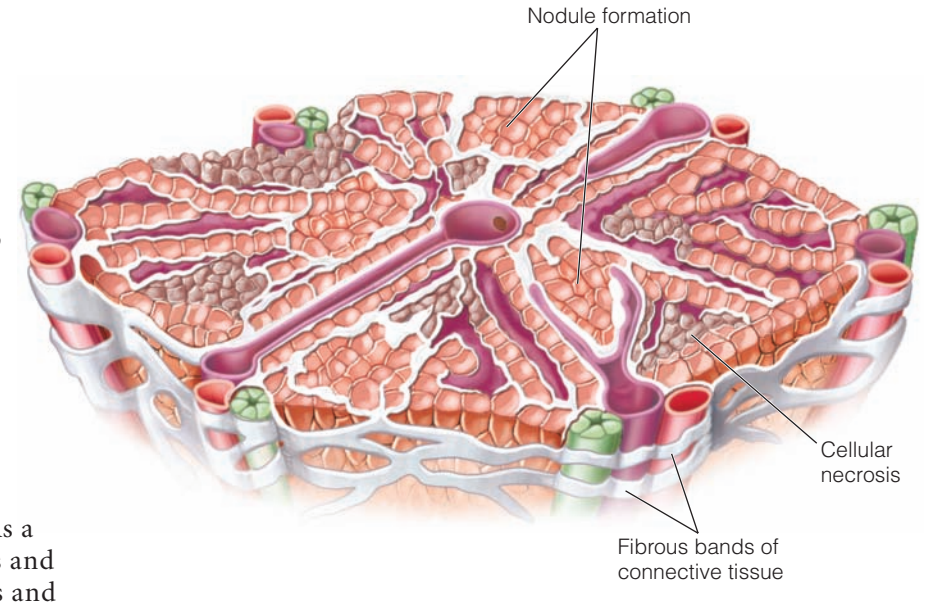


Alcoholic hepatitis

With continued alcohol intake, liver cells degenerate and spotty cellular necrosis occurs. Inflammatory cells such as polymorphonuclear leukocytes and lymphocytes infiltrate the lobule.

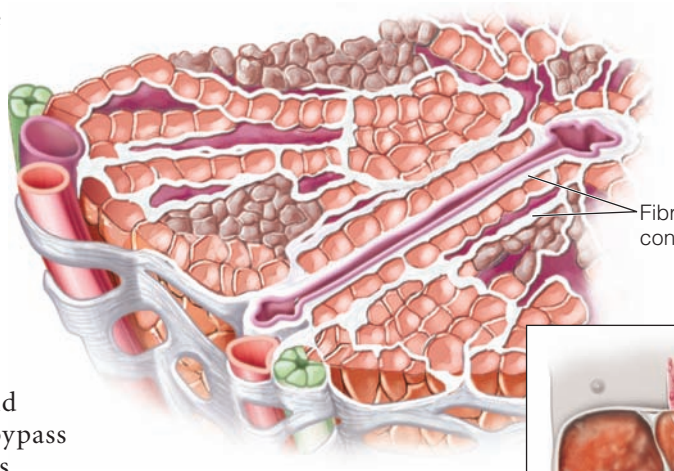
Alcoholic cirrhosis

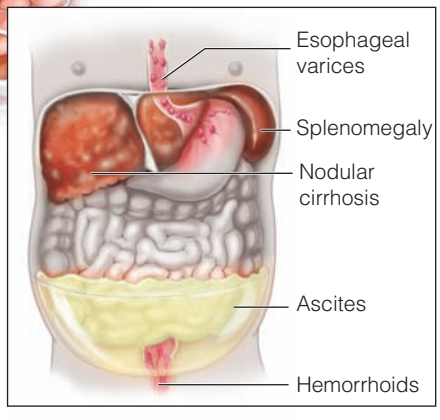
Cellular necrosis and inflammation transform some liver cells into fibroblasts that produce and deposit collagen. Weblike bands of connective tissue develop around the portal triads and central vein, eventually connecting with one another. Small islands of liver cells continue to regenerate, forming nodules. Hepatocyte destruction outpaces regeneration. As a result of cell loss, fibrosis and scarring, the liver shrinks and becomes hard and nodular.



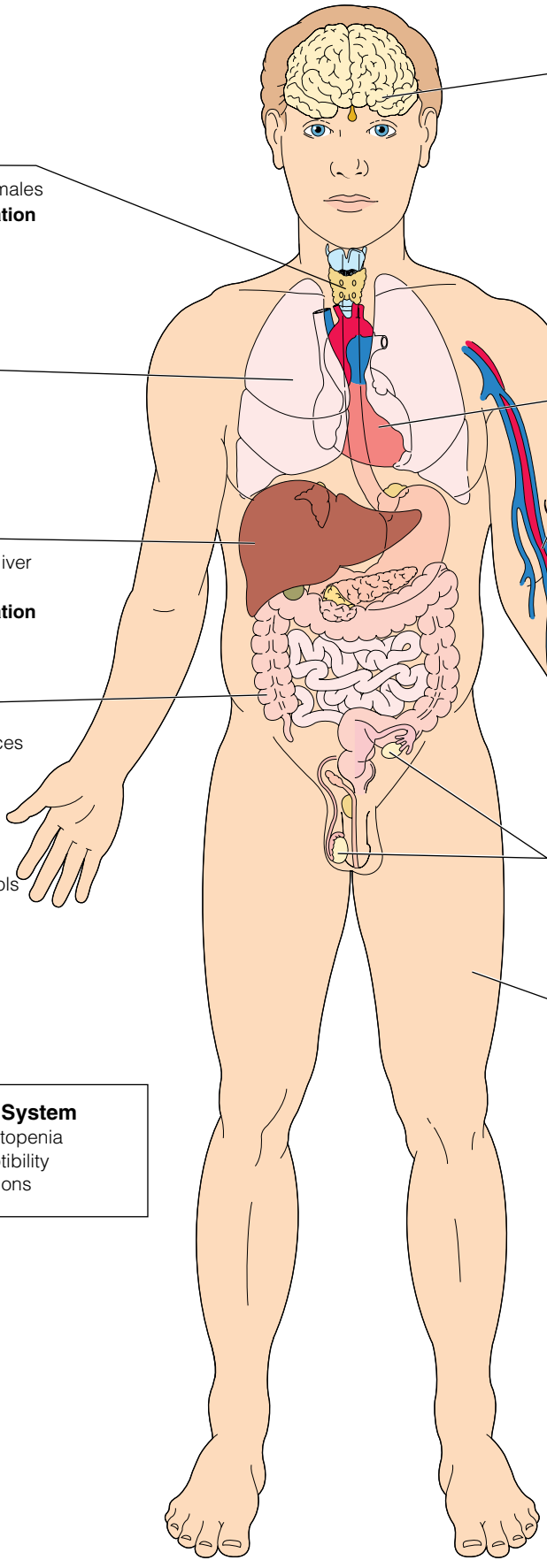
Portal hypertension

Bands of fibrotic scar tissue obstruct the sinusoids and blood flow from the portal vein to the hepatic vein. Pressure in the portal venous system, which drains the gastrointestinal tract, pancreas, and spleen, increases. This increased pressure opens collateral vessels in the esophagus, anterior abdominal wall, and rectum, allowing blood to bypass the obstructed portal vessels. Prolonged portal hypertension leads to the development of (1) varices (fragile, distended veins) in the lower esophagus, stomach, and rectum; (2) splenomegaly (an enlarged spleen); (3) ascites (accumulation of fluid in the abdomen); and (4) hepatic encephalopathy (disrupted CNS function with altered consciousness).





MULTISYSTEM EFFECTS OF **Cirrhosis**



Neurologic
- Portal systemic encephalopathy (agitation → lethargy → stupor → coma)
- Paresthesias
- Sensory disturbances
- Asterixis ("liver flap")

Endocrine
- Gynecomastia in males

Potential Complication
- Diabetes mellitus

Respiratory
- Dyspnea

Cardiovascular
- Bounding pulse
- Pulmonary hypertension
- Portal hypertension
- Dysrhythmias

Hepatic
- Atrophic, nodular liver
- Splenomegaly

Potential Complication
- Liver cancer

Hematologic
- ↓ clotting factors
- Thrombocytopenia
- Anemia

Potential Complication
- Disseminated intravascular coagulation

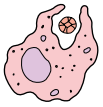
Gastrointestinal

Esophageal:
- Esophageal varices

Stomach/intestines:
- Abdominal pain
- Anorexia
- Ascites
- Nausea
- Clay-colored stools
- Peptic ulcers
- GI bleeding
- Hemorrhoids

Reproductive
- Oligomenorrhea (female)
- Testicular atrophy (male)

Integumentary
- Jaundice (skin, sclera of eyes)
- Erythema of palms
- Spider angioma
- Decreased body hair
- Pruritis
- Ecchymoses
- Caput medusae (dilated veins around the umbilicus)



Immune System
- Leukocytopenia
- ↑ susceptibility to infections

Metabolic Processes
- Fluid and electrolyte imbalances
  - Hypoalbuminemia
  - Hypokalemia
  - Hypocalcemia
- Malnutrition
- Muscle wasting

MANIFESTATIONS of Cirrhosis with Underlying Cause

MANIFESTATION	UNDERLYING PATHOPHYSIOLOGY
Edema, ascites	■ Impaired plasma protein synthesis (hypoalbuminemia) ■ Disrupted hormone balance and fluid retention ■ Increased pressure in portal venous system
Bleeding, bruising	■ Decreased clotting factor synthesis ■ Increased platelet destruction by enlarged spleen ■ Impaired vitamin K absorption and storage
Esophageal varices, hemorrhoids	■ Increased pressure in portal venous system with collateral vessel development
Gastritis, anorexia, diarrhea	■ Engorged veins in gastrointestinal system ■ Alcohol ingestion ■ Impaired bile synthesis and fat absorption
Abdominal wall vein distention (caput medusae)	■ Portal hypertension
Jaundice	■ Impaired bilirubin metabolism and excretion
Malnutrition, muscle wasting	■ Impaired nutrient metabolism ■ Impaired fat absorption ■ Impaired hormone metabolism
Anemia, leukopenia, increased risk for infection	■ Bleeding ■ Increased blood cell destruction by spleen
Asterixis, encephalopathy	■ Accumulated metabolic toxins ■ Impaired ammonia metabolism and excretion
Gynecomastia, infertility, impotence	■ Altered sex hormone metabolism

Portal Systemic Encephalopathy

Portal systemic encephalopathy (*hepatic encephalopathy*) results from accumulation of neurotoxins in the blood and cerebral edema. Ammonia, a by-product of protein metabolism, contributes to hepatic encephalopathy. Ammonium ion is produced as proteins and amino acids are broken down by bacteria in the intestinal tract. Normally, the ammonia produced is then converted by the liver to urea before entering the general circulation. As functional liver tissue is destroyed, ammonia can no longer be converted to urea, and it accumulates in the blood. Other nervous system depressants, such as narcotics and tranquilizers, also can contribute to hepatic encephalopathy. Box 24–4 lists selected precipitating factors for hepatic encephalopathy. Accumulation of other metabolic toxins are thought to contribute as well.

Asterixis (liver flap), a muscle tremor that interferes with the ability to maintain a fixed position of the extremities and causes involuntary jerking movements, is an early sign of portal systemic encephalopathy. Asterixis primarily affects the upper extremities, but also may affect the tongue and feet. Asterixis is elicited by instructing the client to extend the arms and dorsiflex the wrists. If present, asterixis causes a downward flapping of the hands (Figure 24–3 ■). Changes in personality and mentation develop; agitation, restlessness, impaired judgment, and slurred speech also are early manifestations of hepatic encephalopathy. As it progresses, confusion, disorientation, and incoherence develop. Cerebral edema that leads to increased intracranial pressure and cerebral hypoxia is the leading cause of death in people with portal systemic encephalopathy and liver failure.

BOX 24–4 Factors Precipitating Portal Systemic Encephalopathy

- High serum ammonia level
- Constipation
- Blood transfusions
- Gastrointestinal bleeding
- Medications: sedatives, tranquilizers, narcotic analgesics, anesthetics
- Hypoxia
- High-protein diet
- Severe infection
- Surgery

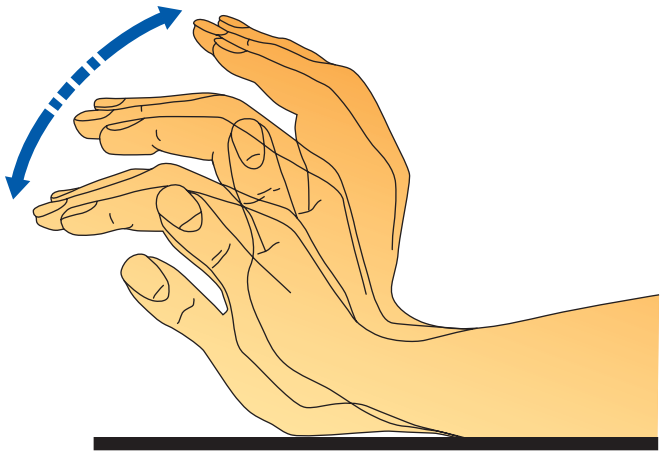


Figure 24–3 ■ Asterixis. Note the downward tremor of the hand on dorsiflexion of the wrist.

Hepatorenal Syndrome

Although the cause is unclear, renal failure with azotemia (excess nitrogenous waste products in the blood), sodium retention, oliguria, and hypotension may develop in clients with advanced cirrhosis and ascites. Hepatorenal syndrome appears to be the result of imbalanced blood flow, leading to constriction of vessels leading to and within the kidneys. The syndrome may be precipitated by gastrointestinal bleeding, aggressive diuretic therapy, or by an unknown cause.

SPONTANEOUS BACTERIAL PERITONITIS Clients with cirrhosis and ascites may develop bacterial peritonitis, even in the absence of known contamination of the peritoneal cavity or other specific risk factors (e.g., paracentesis). The inflammatory response to peritonitis worsens ascites by increasing the permeability of capillaries in the mesentery. The manifestations of spontaneous bacterial peritonitis may be subtle, with increased abdominal discomfort or pain, fever, increasing ascites, worsening encephalopathy, and an overall decline in condition.

INTERDISCIPLINARY CARE

Care for the client with cirrhosis is holistic, addressing physiologic, psychosocial, and spiritual needs. The importance of including the family in the plan of care cannot be overemphasized, particularly if alcohol abuse is identified as the cause. Treatment includes medications to help regulate protein metabolism, maintenance of fluid and electrolyte balance, and supportive therapies, including treatment of underlying problems, such as malnutrition, anemia, bleeding, encephalopathy, renal failure, and infections.

Diagnosis

Studies to confirm the diagnosis of cirrhosis and identify its cause and effects are performed. Diagnostic tests may include the following:

- *Liver function studies* include *ALT, AST, ALP,* and *GGT.* All may be elevated in cirrhosis, but usually not as severely as in acute hepatitis. Elevations in these enzymes may not correlate well with the extent of liver damage in cirrhosis.
- *CBC with platelets* is done. A low RBC count, hemoglobin, and hematocrit demonstrate anemia related to bone marrow suppression, increased RBC destruction, bleeding, and deficiencies of folic acid and vitamin B_{12}. Platelets are low, related to increased destruction by the spleen. Leukopenia (low WBC count) also relates to splenomegaly.
- *Coagulation studies* show a prolonged prothrombin time due to impaired production of coagulation proteins and lack of vitamin K.
- *Serum electrolytes* are measured. Hyponatremia is common, due to hemodilution. Hypokalemia, hypophosphatemia, and hypomagnesemia also are frequently seen, related to malnutrition and altered renal excretion of these electrolytes.
- *Bilirubin* levels are usually elevated in severe cirrhosis, including both direct (conjugated) and indirect (unconjugated) bilirubin.
- *Serum albumin* levels show hypoalbuminemia due to impaired liver production.
- *Serum ammonia* levels are elevated, because the liver fails to effectively convert ammonia to urea for renal excretion.
- *Serum glucose* and *cholesterol* levels frequently are abnormal in clients with cirrhosis.
- *Abdominal ultrasound* is performed to evaluate liver size, detect ascites, and identify liver nodules. Ultrasound may be used in conjunction with *Doppler studies* to evaluate blood flow through the liver and spleen (Tierney et al., 2005).
- *Esophagoscopy* (upper endoscopy) may be done to determine the presence of esophageal varices.
- *Liver biopsy* is not always necessary to diagnose cirrhosis, but may be done to distinguish cirrhosis from other forms of liver disease. See the box on page 617 for nursing implications for a client having a liver biopsy. Figure 21–7 shows the site and position for liver biopsy. Biopsy may be deferred if the bleeding time is prolonged (such as a prothrombin time [PT] greater than 3 seconds over the control).

See Chapter 21 ∞ for more information about and the nursing implications of the above diagnostic tests.

Medications

Medications are used to treat the complications and effects of cirrhosis; they do not reverse or slow the process of cirrhosis itself. Known hepatotoxic drugs and alcohol are avoided, as are drugs metabolized by the liver (e.g., barbiturates, sedatives, hypnotics, and acetaminophen). Several groups of drugs are commonly prescribed. See the Medication Administration box on page 717 for nursing responsibilities and client teaching for commonly used drugs in clients with cirrhosis.

- Diuretics reduce fluid retention and ascites. Spironolactone (Aldactone) is frequently the drug of first choice because it addresses one of the causes of ascites—increased aldosterone levels. If additional diuresis is necessary, a loop diuretic such as furosemide (Lasix) may be added to the regimen.
- Medications to reduce the nitrogenous load and lower serum ammonia levels are added when manifestations of hepatic encephalopathy develop. Two commonly administered medications are lactulose and neomycin. Both exert their effects locally, in the bowel. Lactulose reduces the number of ammonia-forming organisms in the bowel and increases the acidity of colon contents, converting ammonia into ammonium ion. Ammonium ion is not absorbable, and is excreted in the feces. Neomycin sulfate is a locally acting antibiotic that also reduces the number of ammonia-forming bacteria in the bowel.
- The beta-blocker nadolol (Corgard) may be given together with isosorbide mononitrate (Ismo, Imdur, Monoket) to prevent rebleeding of esophageal varices. This drug combination also lowers hepatic venous pressure.
- Ferrous sulfate and folic acid are given as indicated to treat anemia. Vitamin K may be ordered to reduce the risk of bleeding. When bleeding is acute, packed RBCs, fresh frozen plasma, or platelets may be administered to restore blood components and promote hemostasis.
- Antacids are prescribed as indicated. A drug regimen to treat *H. pylori* infection may also be effective (see Chapter 23 ∞).
- Oxazepam (Serax), a benzodiazepine antianxiety/sedative drug, is not metabolized by the liver, and may be used to treat acute agitation.

MEDICATION ADMINISTRATION The Client with Cirrhosis

DIURETICS

Spironolactone (Aldactone) **Furosemide (Lasix)**

Spironolactone is a potassium-sparing diuretic that competes with aldosterone. It reduces ascites by increasing renal excretion of fluid and decreasing aldosterone levels. Furosemide is a loop diuretic that promotes the excretion of potassium. Drugs may be given in combination if serum potassium level permits.

Nursing Responsibilities

- Monitor ECG, serum potassium, BUN, creatinine levels, and hydration status.
- Weigh daily.
- Carefully monitor intake and output.
- Monitor for signs of hyperkalemia if taking spironolactone alone: bradycardia; widening QRS, spiking T waves, or ST segment depression on ECG, diarrhea; and muscle twitching.
- Assess for hyponatremia: confusion, lethargy, apprehension.

Health Education for the Client and Family

- Maintain diet and fluid restrictions as prescribed.
- Report increases in weight or edema.
- Immediately report signs of hyponatremia, hyperkalemia, or hypokalemia (see Chapter 10 ∞).
- Expect increased urinary output; take medications in morning hours to avoid nocturia.

LAXATIVES

Lactulose (Cephulac, Chronulac)

Lactulose is a disaccharide laxative that is not absorbed by the gastrointestinal tract. It reduces the number of ammonia-producing bacteria and lowers the pH in the colon. The lower pH (increased acidity) converts ammonia to ammonium ion, a nonabsorbable form that is excreted in the feces. Lactulose also pulls water into the bowel lumen, increasing the number of daily stools.

Nursing Responsibilities

- Assess bowel sounds and abdominal girth.
- Maintain accurate stool chart.
- Adjust dose to achieve two to four soft stools per day.
- Monitor electrolytes and hydration.

Health Education for the Client and Family

- Drink adequate fluids.
- Report diarrhea; if present, decrease dose. You should have an average of two to four stools per day.
- This drug may cause nausea. Continue taking the drug; taking it with crackers or a soft drink may reduce nausea.

ANTI-INFECTIVE AGENTS

Neomycin sulfate (Neo Tabs)

Neomycin sulfate is a nonsystemic aminoglycoside antibiotic that reduces intestinal bacteria and decreases ammonia production in the bowel lumen. The drug may be administered as an oral or rectal preparation.

Nursing Responsibilities

- Monitor hearing, renal, and neurologic functions. Drug is ototoxic, nephrotoxic, and neurotoxic.
- Prior to administration, check for previous hypersensitivity reaction.
- Monitor intake and output.
- Monitor BUN and creatinine levels.
- If the client is taking digitalis, monitor levels; oral neomycin interferes with its absorption.

Health Education for the Client and Family

- Report dizziness, tinnitus (ringing in ears), hearing loss, headaches, tremors, or vision changes immediately.
- Keep follow-up appointments.
- Maintain fluids, avoid dehydration. (Teach signs of dehydration.)

Treatments

Treatment of cirrhosis is supportive, directed at slowing the progression to liver failure and reducing complications.

NUTRITION Dietary support is an essential part of care for the client with cirrhosis. Dietary needs change as hepatic function fluctuates.

- Sodium intake is restricted to under 2 g/day, and fluids are restricted as necessary to reduce ascites and generalized edema. Fluids are often limited to 1500 mL/day. Fluid needs are calculated based on response to diuretic therapy, urine output, and serum electrolyte values.
- Unless serum ammonia levels are high, a palatable diet with adequate calories and protein is recommended. If hepatic encephalopathy is acute, protein may initially be eliminated from the diet; for chronic hepatic encephalopathy, protein generally is restricted to 60 g/day (Kasper et al., 2005). When encephalopathy resolves and serum ammonia levels stabilize, protein intake is allowed as tolerated. The diet is high in calories and includes moderate fat intake to promote healing. See Table 24–4 for examples of protein-restricted menus. Parenteral nutrition is used as needed to maintain nutritional status when food intake is limited.
- Vitamin and mineral supplements are ordered based on laboratory values. Deficiencies in the B-complex vitamins, particularly thiamin, folate, and B_{12}, and the fat-soluble vitamins A, D, and E are common. These vitamins may need to be administered in a water-soluble form. Clients with alcohol-induced cirrhosis are at high risk for magnesium deficiency, which needs to be replaced.

COMPLICATION MANAGEMENT **Paracentesis**, aspiration of fluid from the peritoneal cavity, may be a diagnostic or a therapeutic procedure. It may be done therapeutically to relieve severe ascites that does not respond to diuretic therapy. The goal of paracentesis is to relieve respiratory distress caused by excess fluid in the abdomen. Ascites fluid may be withdrawn in moderate amounts of 500 mL to 1 L daily to reduce the risk of fluid and electrolyte imbalances. Large-volume paracentesis, withdrawal of 4 to 6 L of fluid at one time, may be used. Albumin is often administered intravenously during large-volume paracentesis to maintain intravascular volume as the pressure of the ascites fluid in the abdomen is relieved. Nursing implications for the client undergoing paracentesis are listed in Box 24–5. Figure 24–4 ■ shows insertion sites and client positioning during paracentesis.

TABLE 24–4 Sample Menus for Protein-Restricted Diets

	SEVERE RESTRICTION	MODERATE RESTRICTION	MILD RESTRICTION
Breakfast	1 orange ½ cup rice or creamed cereal with sugar 1 slice whole-wheat bread with butter or margarine and jam ½ cup milk 1 egg or 1 Tbsp peanut butter Coffee or tea	1 orange ½ to I cup cooked oatmeal with 1 cup milk and sugar 1 slice whole-wheat toast with butter or margarine and jam Coffee or tea	1 orange 1 egg or 1 Tbsp peanut butter ½ to ¾ cup cooked oatmeal with 1 cup milk and sugar 1 slice whole-wheat toast with butter or margarine and jam Coffee or tea
Lunch	1 oz lean fish, poultry, meat, or 1 small piece soft tofu or 3 Tbsp cottage cheese 1 slice whole-wheat bread with butter or margarine and jelly Cooked green vegetable Green salad with oil and vinegar dressing Fresh fruit Coffee or tea	3 oz lean fish, poultry, meat, or 1 large piece soft tofu or ⅓ cup cottage cheese 1 slice whole-wheat bread with butter or margarine and jelly Cooked green vegetable Starch such as potato, squash, or corn Fresh fruit Coffee or tea	4 oz lean fish, poultry, meat, or 1 large piece soft tofu or ½ cup cottage cheese 2 slices whole-wheat bread with butter or margarine and jelly Cooked green vegetable Starch such as potato, squash, or corn Fresh fruit 1 cup milk Coffee or tea
Afternoon snack	Apple juice Soda crackers with margarine or butter and jelly	½ cup milk Graham crackers with margarine or butter and jelly	1 cup milk Graham crackers with margarine or butter and jelly
Dinner	1 oz lean fish, poultry, meat, or 1 small piece soft tofu or 3 Tbsp cottage cheese 1 whole-wheat dinner roll with butter or margarine and jelly Cooked green vegetable Baked potato Fresh or canned fruit ⅓ cup sherbet Coffee or tea	3 oz lean fish, poultry, meat, or 1 large piece soft tofu or ⅓ cup cottage cheese 1 whole-wheat dinner roll with butter or margarine and jelly Cooked green vegetable Baked potato Fresh or canned fruit ⅓ cup sherbet Coffee or tea	4 oz lean fish, poultry, meat, or 1 large piece soft tofu or ½ c cottage cheese 2 whole-wheat dinner rolls with butter or margarine and jelly Cooked green vegetable Baked potato Fresh or canned fruit 1 cup or ⅓ cup ice cream Coffee or tea
Evening snack	1 banana	1 banana	1 banana with ½ cup milk

BOX 24–5 Nursing Implications for Diagnostic Tests

Abdominal Paracentesis

Client Preparation

- Verify presence of an informed consent.
- Weigh prior to paracentesis.
- Assess vital signs for baseline.
- Have client void immediately prior to the test to avoid bladder puncture.
- Position seated, either on the side of the bed or in a chair, with feet supported.

Health Education for the Client and Family

- Describe what to expect during and following paracentesis, blood pressure is monitored during the procedure.
- Following cleansing and local anesthesia, a small incision may be made and a needle or trocar inserted to withdraw fluid. The trocar is connected to tubing and a collection bottle; specimens may be sent to laboratory.
- A small dressing is placed over the puncture site after the needle is withdrawn. There may be some fluid leakage from the site.
- Salt-poor albumin may be given after the procedure to replace lost protein.

Bleeding esophageal varices are life threatening and require intensive care management. Restoration of hemodynamic stability is the first priority. A central line is inserted and central venous and pulmonary artery pressures are monitored (see Chapter 32 ∞). Blood is given to restore blood volume, and fresh frozen plasma may be administered to restore clotting factors. Somatostatin or octreotide, drugs that constrict blood vessels in the gut, are given intravenously to reduce blood flow in the portal venous system. Vasopressin, a drug that produces generalized vasoconstriction, also may be used.

When the blood pressure and cardiac output have stabilized, upper endoscopy is performed to evaluate and treat the varices. A large nasogastric tube is inserted prior to endoscopy, and **gastric lavage** (irrigation of the stomach with large quantities of normal saline) is performed to improve visualization. During endoscopy, the varices may be banded or sclerosed to reduce the risk of recurrent bleeding. In *banding* (*variceal ligation*), small rubber bands are placed on varices to occlude blood flow. *Endoscopic sclerosis* involves injecting a sclerosing agent directly into the varices to induce inflammation and clotting. See Chapter 21 ∞ for the nursing implications of endoscopy.

Balloon tamponade of bleeding varices may be used if bleeding cannot be controlled through vasoconstriction or if

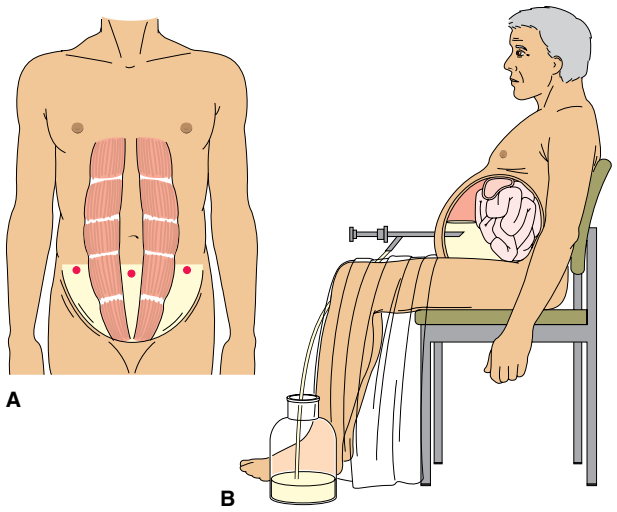


Figure 24–4 ■ Sites and position for paracentesis. *A*, Potential sites of needle or trocar insertion to avoid abdominal organ damage. *B*, The client sits comfortably; in this position, the intestines float back and away from the insertion site.

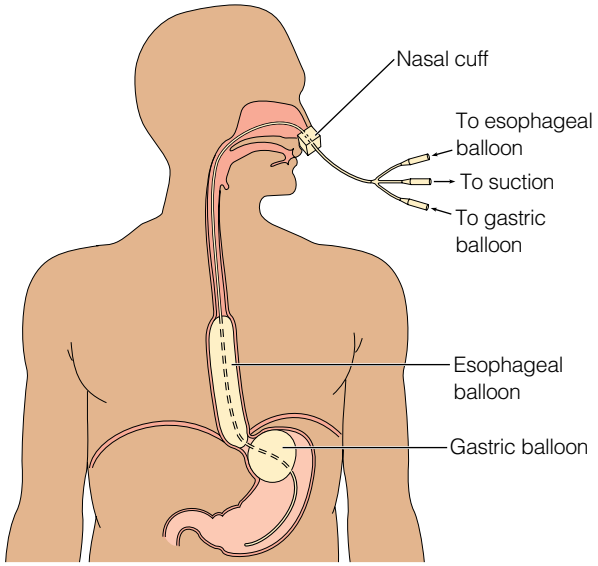


Figure 24–5 ■ Triple-lumen nasogastric tube (Sengstaken-Blakemore) used to control bleeding esophageal varices.

endoscopy is unavailable. A multiple-lumen nasogastric (NG) tube (such as a Sengstaken-Blakemore tube or a Minnesota tube) is inserted, and the gastric and esophageal balloons are inflated to apply direct pressure on the bleeding varices (Figure 24–5 ■). Tension is applied to the tube to further compress the varices. Balloon tamponade carries a number of risks, including aspiration, airway obstruction, and tissue ischemia and necrosis. An endotracheal tube is inserted prior to nasogastric intubation to support the airway and reduce the risk of aspiration. This short-term measure is used only until more definitive treatment can be done.

PRACTICE ALERT

When caring for a client with a multiple-lumen NG tube, always deflate the esophageal balloon before the gastric balloon. This practice prevents the balloon from becoming misplaced and occluding the airway. Always keep an appropriate syringe at the bedside to deflate the esophageal balloon should the client develop respiratory distress.

Transjugular intrahepatic portosystemic shunt (TIPS) is used to relieve portal hypertension and its complications of esophageal varices and ascites. A channel is created through the liver tissue using a needle inserted transcutaneously (Figure 24–6 ■). An expandable metal stent is inserted into this channel to allow blood to flow directly from the portal vein into the hepatic vein, bypassing the cirrhotic liver. The shunt relieves pressure in esophageal varices and allows better control of fluid retention with diuretic therapy. Stenosis and occlusion of the shunt are frequent complications. TIPS also increases the risk of developing hepatic encephalopathy (due to decreased perfusion of the liver and impaired ammonia metabolism) and may reduce long-term survival. It generally is used as a short-term measure until liver transplant is performed.

SURGERY **Liver transplantation** is indicated for some clients with irreversible, progressive cirrhosis. A decline in functional status, increasing bilirubin levels, falling albumin levels, and increasing problems with complications that respond poorly to treatment are indications for liver transplantation. Malignancy,

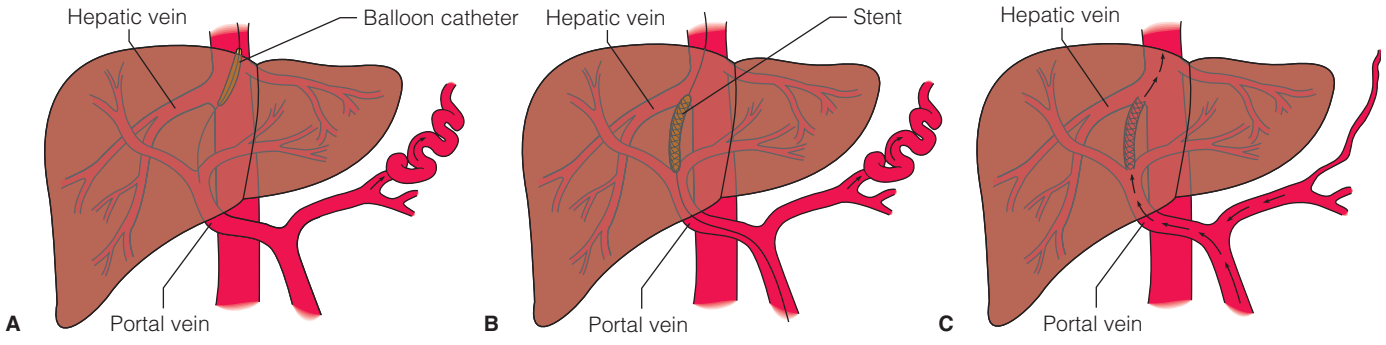


Figure 24–6 ■ Transjugular intrahepatic portosystemic shunt (TIPS). *A*, Guided by angiography, a balloon catheter inserted via the jugular vein is advanced to the hepatic veins and through the substance of the liver to create a portacaval (portal vein-to-vena cava) channel. *B*, A metal stent is positioned into the channel, and expanded by inflating the balloon. *C*, The stent remains in place after the catheter is removed, creating a shunt for blood to flow directly from the portal vein into the hepatic vein.

NURSING CARE OF THE CLIENT UNDERGOING Liver Transplantation

PREOPERATIVE CARE

- Obtain a complete nursing history and physical examination. *A complete preoperative nursing assessment provides baseline data for comparison after surgery.*
- Provide routine preoperative care as ordered (see Chapter 4 ∞). *Preoperative care is similar to that provided for other clients undergoing major surgery.*
- Discuss preoperative and postoperative expectations with the client and family. Introduce to the intensive care unit, and discuss anticipated drainage tubes and supportive measures in the immediate postoperative period. Provide information about visiting policies and family accommodations (if available). *Preoperative teaching helps relieve anxiety in the client and family members. Clients return from surgery to an intensive care or specialized care unit. Restrictions on the number of visitors and the time they may spend with the client are common.*
- Once a donor liver is located, check for evidence of infection; if no infection is present, begin preoperative antibiotics as ordered. *An acute or chronic infection may contraindicate liver transplantation as drugs given postoperatively to suppress rejection of the transplanted organ also impair the ability to fight infection.*

POSTOPERATIVE CARE

- Provide routine postoperative care as outlined in Chapter 4 ∞.
- Maintain airway and ventilatory support until awake and alert. *Until the new liver clears the anesthesia, the client requires measures to support respirations and ventilation.*
- Monitor temperature and implement rewarming measures (such as warming blankets, heating lamps, and head covers) as indicated. *The client often is hypothermic after liver transplant, necessitating careful rewarming while maintaining hemodynamic stability.*
- Frequently monitor hemodynamic pressures, including arterial blood pressure, central venous pressure, and pulmonary artery pressures. *Postoperative fluid volume status may be difficult to determine without careful pressure measurements. The rate and type of fluids administered are determined by hemodynamic status.*
- Monitor urine output hourly; maintain careful intake and output records. Weigh daily. *Urine output and weight provide additional information about fluid volume status. In addition, renal function may be altered after liver transplant; acute renal failure is a significant risk. See Chapter 29 ∞ for more information about acute renal failure and its management.*
- Monitor for signs of active bleeding, including excess drainage, increasing abdominal girth, bloody nasogastric drainage, black tarry stools, tachypnea, tachycardia, diminished peripheral pulses, or pallor. Report immediately. *Altered coagulation in the early postoperative period increases the risk for bleeding. Blood products to replace volume and clotting factors may be necessary.*
- Monitor serum electrolytes and laboratory values related to blood coagulation, liver function, and renal function. Report abnormal results or significant changes immediately. *Electrolyte imbalances are common postoperatively. Altered liver or renal function tests may indicate rejection of the transplanted liver or acute renal failure. Other early signs of transplant rejection include fever, a drop in bile output, or a change in bile color and viscosity (Urden, Stacy, & Lough, 2006).*
- Monitor neurologic status. *With good function of the transplanted organ, mental status should clear within days of the transplant.*
- Provide discharge teaching:
  a. Teach how to reduce risk of infection, and signs of infection to report.
  b. Instruct to recognize and report signs of organ rejection.
  c. Discuss all medications, including their purpose, schedule, adverse effects, and potential long-term effects. Stress the importance of complying with all prescribed medications and postoperative precautions for the remainder of the client's life.
  d. Discuss possible changes in body image and psychologic responses to receiving a transplanted organ. Refer to a counselor or support group as indicated.
  e. Refer for home health services for continued assessment and teaching.
  f. Stress importance of continued follow-up with transplant team and primary care provider.

active alcohol or drug abuse, and poor surgical risk are contraindications for the surgery. See the box above for nursing care of the client having a liver transplant.

NURSING CARE

In addition to the nursing care discussed in this section, a Nursing Care Plan for a client with alcoholic cirrhosis is found on the following page.

Health Promotion

For most clients, high-risk behaviors are the risk factors for cirrhosis. With all clients (including children and young adults), stress the relationship between alcohol and drug abuse and liver disorders. While many clients tolerate alcohol use in moderation with no adverse effects on the liver, excess alcohol use is the leading cause of cirrhosis. Injection drug use also is a significant risk factor, increasing the risk for contracting bloodborne hepatitis (B, C, or D). These types of viral hepatitis can lead to chronic hepatitis and, ultimately, to cirrhosis. Discuss abstinence or safer sex practices as another measure to prevent viral hepatitis and potential liver damage.

Assessment

Assessment data related to cirrhosis includes the following:

- *Health history:* Current manifestations, including abdominal pain or discomfort, recent weight loss, weakness, and anorexia; altered bowel elimination; excess bleeding or bruising; abdominal distention; jaundice, pruritus; altered libido or impotence; duration of symptoms; history of liver or gallbladder disease;

NURSING CARE PLAN A Client with Alcoholic Cirrhosis

Richard Wright is a 48-year-old divorced father of two teenagers. Mr. Wright has been admitted to the community hospital with ascites and malnutrition. He has had three previous hospital stays for cirrhosis, the most recent 6 months ago.

ASSESSMENT

Mr. Wright is lethargic but responds appropriately to verbal stimuli. He complains of "spitting up blood the past week or so" and says, "I'm just not hungry." He has lost 20 lb (9 kg) since his previous admission. He is jaundiced and has petechiae and ecchymoses on his arms and legs. Liz Mowdi, Mr. Wright's nurse, notes pitting pretibial edema. Abdominal assessment reveals a tight, protuberant abdomen with caput medusae. The liver margin is not palpable; the spleen is enlarged. Vital signs are T 100°F (37.7°C), P 110, R 24, and BP 110/70.

Abnormal laboratory results include WBC 3700/mm^3 (normal 4300 to 10,800/mm^3); RBC 4.0 million/mm^3 (normal 4.6 to 5.9 million/mm^3); platelets 75,000/mm^3 (normal 150,000 to 350,000/mm^3); serum ammonia 105 µm/dL (normal 35 to 65 µm/dL); total bilirubin 4.9 mcg/dL (normal 0.1 to 1.0 mcg/dL); and serum sodium 150 mEq/L (normal 135 to 145 mEq/L). Potassium, hemoglobin, hematocrit, total protein, and albumin levels are markedly decreased. Hepatic enzymes are elevated. Blood urea nitrogen and creatinine levels are marginally elevated. Oxygen saturation (O_2 sat) is 88% (normal range: 96% to 100%) per pulse oximetry.

Endoscopy shows bleeding from gastric ulcer, and the diagnosis of alcoholic cirrhosis with gastritis is made. Mr. Wright is started on Aldactone, 25 mg PO q8h; Riopan, 30 mL 2 h p.c. and hs; lactulose, 30 mL qh until onset of diarrhea, then 15 mL tid; and low-protein, 800 mg sodium diet; fluid restriction of 1500 mL/day.

DIAGNOSES

- *Impaired Gas Exchange* related to pressure of ascites fluid on the diaphragm as manifested by tachypnea and decreased oxygen saturation
- *Excess Fluid Volume* related to electrolyte imbalance and hypoalbuminemia as manifested by ascites and peripheral edema
- *Imbalanced Nutrition: Less than Body Requirements* related to anorexia and possible alcohol abuse as manifested by weight loss and low serum protein levels
- *Disturbed Thought Processes* related to effects of high ammonia levels as manifested by lethargy
- *Ineffective Protection* related to impaired platelet formation and malnutrition

EXPECTED OUTCOMES

- Respiratory rate and O_2 sats will be within normal limits.
- Abdominal girth will decrease by 1 to 2 cm per day; peripheral edema will decrease.
- Will gain 1 lb (0.45 kg) per week without evidence of increased fluid retention. Serum albumin levels will return to normal range.
- Will be alert and oriented; serum ammonia levels are within normal range.
- Will demonstrate no further evidence of active bleeding.
- Will verbalize willingness to join a community support group.

PLANNING AND IMPLEMENTATION

- Weigh daily.
- Provide high-calorie, low-salt, low-protein diet with between-meal snacks.
- Maintain stool chart.
- Assign same nurses to care as much as possible to facilitate evaluation of mental status. Promptly report changes in status or laboratory values.
- Measure abdominal girth every 8 h, marking level of measurement.
- Institute bleeding precautions.
- Elevate head of bed; assist to chair with legs elevated tid as tolerated.
- Include significant others in care and teaching; refer to community agencies for discharge follow-up.

EVALUATION

A week after admission, Mr. Wright's ascites has decreased and no further active bleeding is noted. His serum protein levels have increased, and his laboratory values are improving. No further bruising is noted during hospitalization. Although he shows a 5-lb weight loss as excess water is eliminated, he is consuming 100% of his diet. His serum ammonia levels have returned to normal. On discharge, O_2 sat is 96%; respirations are 18. Lactulose will be continued on discharge.

Ms. Mowdi provides both written and verbal information about the medication and cirrhosis, including measures to prevent complications. Mr. Wright and his children express interest in Alcoholics Anonymous and Al-Anon and are referred to those agencies. Prior to discharge, follow-up appointments are made with a psychiatric social worker and a primary caregiver.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe the relationship between portal hypertension, liver dysfunction, and ascites.
2. Outline a 1-day menu for a low-protein, low-sodium high-calorie diet.
3. What is the pathophysiologic basis for hepatic encephalopathy? What are the nursing responsibilities related to lactulose and neomycin?
4. Design a nursing care plan for Mr. Wright for the diagnosis *Ineffective Coping*.

See Evaluating Your Response in Appendix C.

pattern and extent of alcohol or injection drug use; use of other prescription and nonprescription drugs.

- *Physical assessment:* Vital signs; mental status; color and condition of skin and mucous membranes; peripheral pulses and presence of peripheral edema; abdominal assessment including appearance, shape and contour, bowel sounds, abdominal girth, percussion for liver borders, and palpation for tenderness and liver size.

Nursing Diagnoses and Interventions

Nursing care of the client with cirrhosis presents many challenges because liver function affects all body systems. The

nurse is responsible for coordinating care among care providers. Many nursing diagnoses may apply. The diagnoses discussed in this section focus on problems with fluid and electrolyte balance, disturbed thought processes, risk for bleeding, skin integrity, and nutrition.

Excess Fluid Volume

Cirrhosis affects water and salt regulation due to portal hypertension, hypoalbuminemia, and hyperaldosteronism. Signs of fluid volume overload and portal hypertension may develop: ascites, peripheral edema, internal hemorrhoids and varices, and prominent abdominal wall veins. Careful monitoring is necessary, because treatment measures can lead to further fluid and electrolyte imbalances.

- Weigh daily. Assess for jugular vein distention, measure abdominal girth daily, and check for peripheral edema. Monitor intake and output. *Careful assessment is important to detect fluid shifts.*
- Assess urine specific gravity. *Specific gravity measures the concentration of urine, an indicator of hydration.*

PRACTICE ALERT

Monitor the client with cirrhosis for signs of impaired renal function, such as oliguria, a fixed specific gravity of about 1.012, central edema (around the eyes and of the face), and increasing serum creatinine and BUN levels. Such signs may indicate hepatorenal syndrome or acute renal failure from another cause.

- Provide a low-sodium diet (500 to 2000 mg/day) and restrict fluids as ordered. *Excess sodium leads to water retention, and can increase fluid volume, ascites, and portal hypertension.*

Disturbed Thought Processes

Accumulated nitrogenous waste products and other metabolites affect mental status and thought processes. Effects of hepatic encephalopathy can range from mild confusion to agitation to coma.

- Assess neurologic status, including level of consciousness and mental status. Observe for signs of early encephalopathy: changes in handwriting, speech, and asterixis. *Early identification of evidence of encephalopathy allows prompt intervention—subtle changes in neurologic functioning are important!*

PRACTICE ALERT

Closely monitor clients who have experienced gastrointestinal bleeding for signs of hepatic encephalopathy. Blood in the intestinal tract is digested as a protein, increasing serum ammonia levels and the risk for hepatic encephalopathy.

- Avoid factors that may precipitate hepatic encephalopathy. Avoid hepatotoxic medications and CNS depressant drugs. *Cautious use of medications and close monitoring can eliminate iatrogenic causes of encephalopathy.*
- If possible, plan for consistent nursing care assignments. *Consistent care providers facilitate early identification of subtle neurologic changes indicative of hepatic encephalopathy.*
- Provide low-protein diet as prescribed; teach the family the importance of maintaining diet restrictions. *Nitrogenous byproducts from dietary protein increase serum ammonia levels.*
- Administer medications or enemas as ordered to reduce nitrogenous products. Monitor bowel function and provide measures to promote regular elimination and prevent constipation. *Oral or rectally administered (per enema) medications are ordered to reduce intestinal bacteria and the ammonia they produce. Regular bowel elimination promotes protein and ammonia elimination in the feces.*
- Orient to surroundings, person, and place; provide simple explanations and reassurance. *Modification of verbal interactions to level of understanding and mental status may reduce anxiety and agitation.*

Ineffective Protection

Impaired coagulation, esophageal varices, and possible acute gastritis place the client with cirrhosis at significant risk for hemorrhage. Clotting is altered by vitamin K deficiency; impaired manufacture of coagulation Factors II, VII, IX, and X; and increased platelet destruction due to splenomegaly.

- Monitor vital signs; report tachycardia or hypotension. *Increased pulse and decreasing blood pressure may indicate hypovolemia due to hemorrhage.*
- Institute bleeding precautions (Box 24–6). *Preventive measures can decrease the risk for active bleeding.*
- Monitor coagulation studies and platelet count. Report abnormal results. *Coagulation studies help determine the risk for bleeding and the need for treatment.*
- Carefully monitor the client who has had bleeding esophageal varices for evidence of rebleeding: hematemesis, **hematochezia** (bright blood in the stool) or tarry stools, signs of hypovolemia or shock. *Rebleeding is common following variceal hemorrhage, especially within the first week.*

PRACTICE ALERT

Carefully monitor the respiratory status of the client with a Sengstaken-Blakemore or Minnesota tube. Displacement of the tube can obstruct the airway unless an endotracheal tube is in place. The esophageal balloon prevents the client from swallowing oral secretions, increasing the risk for aspiration. Keep the head of the bed elevated to 45 degrees to reduce the risk of aspiration and promote gas exchange.

BOX 24–6 Bleeding Precautions

- Prevent constipation.
- Avoid rectal temperatures or enemas.
- Avoid injections; if needed, use small-gauge needle and apply gentle pressure.
- Monitor platelet count, PT, and aPPT.
- Assess for ecchymotic areas and areas of purpura.
- Apply pressure to bleeding sites. After venipuncture, apply direct pressure for at least 5 minutes.
- Use only a soft toothbrush.
- Avoid blowing nose.
- Assess oral cavity for bleeding gums.

Impaired Skin Integrity

Severe jaundice with bile salt deposits on the skin may cause pruritus. Scratching related to the pruritus damages the skin and impairs its integrity. Malnutrition, particularly protein deficiency, and edema also increase the risk for tissue breakdown and impaired skin integrity.

- Use warm water rather than hot water when bathing. *Hot water increases pruritus.*
- Use measures to prevent dry skin: Apply an emollient or lubricant as needed to keep skin moist, avoid soap or preparations with alcohol, and do not rub the skin. *Dry skin contributes to pruritus.*
- If indicated, apply mittens to the hands to prevent scratching. *Clients with encephalopathy may not understand the need to refrain from scratching.*
- Institute measures to prevent skin and tissue breakdown: Turn at least every 2 hours, use an alternating pressure mattress, and frequently assess skin condition. *Frequent position changes relieve pressure and promote circulation and tissue oxygenation.*
- Administer prescribed antihistamine (to relieve pruritus) cautiously. *Decreased liver function increases the risk for altered drug responses.*

Imbalanced Nutrition: Less than Body Requirements

The client with cirrhosis is at risk for malnutrition for a number of reasons: possible chronic alcohol use, anorexia, impaired vitamin and mineral absorption, and impaired protein metabolism. In addition, salt and protein restrictions may make the diet less palatable and appealing to the client.

- Weigh daily. Instruct to weigh at least weekly at home. *Weight is a good indicator of both nutritional status and fluid balance. Short-term weight fluctuations tend to reflect fluid balance, while longer term changes in weight are more reflective of nutritional status.*
- Provide small meals with between-meal snacks. *A small meal is more appealing for an anorexic client. Between-meal snacks help maintain adequate calorie and nutrient intake.*
- Unless protein is restricted due to impending hepatic encephalopathy, promote protein and nutrient intake by providing nutritional supplements such as Ensure or instant breakfasts. *The sodium and protein content of all meals and snacks must be calculated when maintaining restrictions of these nutrients.*
- Arrange for consultation with a dietitian for diet planning while hospitalized and at home. *The dietitian can provide detailed instructions, sample menus, and suggestions for improving the palatability of the diet and promoting intake.*

Using NANDA, NIC, and NOC

Chart 24–2 shows links between NANDA nursing diagnoses, NIC, and NOC for the client with cirrhosis.

NANDA, NIC, AND NOC LINKAGES
CHART 24–2 **The Client with Cirrhosis**

NANDA

Ineffective Protection

↓

NIC

Environmental Management: Safety

Self-Responsibility Facilitation

Bleeding Precautions

Skin Surveillance

↓

NOC

Neurologic Status: Consciousness

Health-Promoting Behavior

Coagulation Status

Tissue Integrity: Skin and Mucous Membranes

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

Community-Based Care

Cirrhosis is a chronic, progressive disease. As such, the client and family assume major roles in managing the disease and its manifestations and in preventing complications. Teaching topics for home care include:

- The absolute necessity of avoiding alcohol and other hepatotoxic drugs. Suggest inpatient or community-based alcohol treatment programs and Alcoholics Anonymous as indicated.
- Diet and fluid intake restrictions and recommendations. Include suggestions to promote nutritional intake and increase the flavor of food when sodium is restricted.
- Prescribed medications; their timing, intended and adverse effects, and manifestations to report to the primary care provider.
- Bleeding precautions (see Box 24–6).
- Manifestations of potential complications to be reported to the primary care provider. Stress the importance of promptly reporting evidence of gastrointestinal bleeding for prompt intervention for potential hemorrhage.
- Skin care techniques to reduce pruritus and the risk of damage.
- Ways to manage fatigue and conserve energy.

Provide referrals for home health services, dietary consultation, social services, and counseling as needed by the client and family. Suggest local support groups where available. If appropriate, suggest hospice services for the client with end-stage liver disease.

THE CLIENT WITH CANCER OF THE LIVER

Primary liver cancer is uncommon in the United States, accounting for only 0.5% to 2% of all cancers (Porth, 2005). It is,

however, a common malignancy worldwide. Hepatocellular carcinoma is common in parts of Asia and Africa, where the incidence is as high as 500 cases per 100,000 people. This higher incidence is linked to chronic hepatitis B or C infection. The incidence of primary liver cancer is higher in men than it is in women. It typically develops in the fifth or sixth decade of life (Kasper et al., 2005). The prognosis for primary liver cancer is poor, in part because the disease often is advanced at the time of diagnosis. Metastasis to the liver from primary tumors of the lung, breast, and gastrointestinal tract are relatively common.

Pathophysiology

About 80% to 90% of primary hepatic cancers arise from the liver's parenchymal cells (hepatocellular carcinoma); the remainder form in the bile ducts (cholangiocarcinoma). Regardless of the origin, the progress of the disease is similar. Several etiologic factors have been identified (Box 24–7). Most primary liver cancer in the United States is related to alcoholic cirrhosis, HBV, or HCV.

The underlying pathophysiology of primary liver cancer is damage to hepatocellular DNA. This damage may be caused by integration of HBV or HCV into the DNA or by repeated cycles of cell necrosis and regeneration that facilitate DNA mutations. HBV and aflatoxins damage a specific tumor suppressor gene, p53. Tumors may be limited to one specific area, may occur as nodules throughout the liver, or may develop as surface infiltrates. The tumor interferes with normal hepatic function, leading to biliary obstruction and jaundice, portal hypertension, and metabolic disruptions (hypoalbuminemia, hypoglycemia, and bleeding disorders). It also may secrete bile products and produce hormones (paraneoplastic syndrome) that may lead to polycythemia, hypoglycemia, and hypercalcemia. Tumors usually grow rapidly and metastasize early.

Manifestations

Initial manifestations of liver cancer develop insidiously and often are masked by the presence of cirrhosis or chronic hepatitis. Weakness, anorexia, weight loss, fatigue, and malaise are common early manifestations. Abdominal pain and a palpable mass in the right upper quadrant are common presenting symptoms. See the following box for manifestations of primary liver cancer. Ascites and jaundice may be present at diagnosis. Signs of liver failure with portal hypertension, splenomegaly, and altered metabolism develop as the tumor progresses. Most clients die within 6 months of the diagnosis (Porth, 2005).

BOX 24–7 Suspected Causes of Primary Liver Cancer

- Chronic hepatitis C infection
- Chronic hepatitis B infection
- Cirrhosis, regardless of type
- Chronic aflatoxin (a toxin produced by *Aspergillus* molds) exposure
- Arsenic-contaminated water
- Carcinogens in food
- Possible hormonal factors (e.g., long-term use of androgens)

MANIFESTATIONS of Primary Liver Cancer

- Malaise
- Anorexia
- Lethargy
- Weight loss
- Fever of unknown origin
- Jaundice
- Feeling of abdominal fullness
- Painful right upper quadrant mass
- Manifestations of liver failure

INTERDISCIPLINARY CARE

Liver tumors are identified by CT scans and MRI. A liver biopsy may be done to confirm the diagnosis and identify the tumor type or origin. See the box on page 617 for the nursing implications of liver biopsy. Serum alpha-fetoprotein (AFP) levels, normally low in nonpregnant adults, rise in most clients with hepatocellular cancer.

Small, localized tumors may be surgically resected, offering the only viable chance for cure. Most tumors, however, have spread extensively or have distant metastasis at the time of diagnosis, so this is frequently not an option. Liver transplantation may be done; however, the tumor may recur in the transplanted organ.

Radiation therapy is used to shrink the tumor, decreasing pressure on surrounding organs and reducing pain. Chemotherapy may be used as primary treatment or adjunctive therapy. Direct continuous hepatic arterial infusion with an implanted pump has shown promise in prolonging survival rates. See Chapter 14 ∞ for nursing care for clients receiving radiation therapy or chemotherapy.

NURSING CARE

Encourage clients with risk factors for primary liver cancer to avoid alcohol and other substances that may further damage the liver. Urge them to discuss regular screening for liver tumors (such as serum AFP levels) with their primary care physician.

Both the client and the family need extensive nursing support. Controlling pain is a priority. Because of the poor prognosis, early referral for hospice services may be appropriate.

Nursing diagnoses, interventions, and teaching for the client with liver cancer are similar to those for clients with cirrhosis; see pages 721–723.

THE CLIENT WITH LIVER TRAUMA

Blunt or penetrating trauma to the abdomen can damage the liver. Liver trauma is frequently seen in combination with injuries to other abdominal organs. Motor vehicle crashes, stab or gunshot wounds, and iatrogenic sources such as liver biopsy are among the causes of these injuries.

Pathophysiology and Manifestations

Liver trauma generally causes bleeding due to the vascularity of the organ. Liver injury may cause a surface hematoma, hematoma within the liver parenchyma, laceration of liver tissue, or disruption of vessels leading to or from the liver. Severe bleeding can rapidly disrupt hemodynamic stability and lead to shock.

PRACTICE ALERT

Bleeding due to liver trauma may not be immediately apparent. Instruct the client with apparent or potential liver trauma to immediately report light-headedness, rapid heart rate, shortness of breath, thirst, or increasing abdominal pain.

INTERDISCIPLINARY CARE

Diagnostic peritoneal lavage is often used along with CT scan to diagnose liver trauma. The procedure is performed by making a small abdominal incision into the peritoneum (after the bladder has been emptied), and inserting a small catheter into the peritoneal cavity. If blood is immediately detected, the client is taken directly to surgery for abdominal exploration. If frank bleeding is not apparent, a liter of isotonic fluid is instilled into the abdomen, then drained and sent for laboratory analysis.

Intravenous fluids, fresh frozen plasma, platelets, and other clotting factors are administered to restore blood volume and promote hemostasis. Hemodynamic status is closely monitored; continued instability may indicate a need for surgical intervention to control hemorrhage. Postoperative nursing care focuses on preventing pulmonary complications, such as atelectasis, and detecting and preventing infection.

NURSING CARE

Nursing care of the client with liver trauma focuses on fluid management and other supportive care related to shock. Keeping family members informed is an important aspect of care, especially during the period of client instability. Diagnoses include the following:

- *Deficient Fluid Volume* related to hemorrhage
- *Risk for Infection* related to wound or abdominal contamination
- *Ineffective Protection* related to impaired coagulation.

THE CLIENT WITH LIVER ABSCESS

Liver abscesses usually are bacterial or amoebic (protozoal) in origin. Bacterial abscesses may follow trauma or surgical procedures, including biopsy. Multiple or single abscesses occur most commonly in the right lobe. Amoebic abscesses most frequently occur following infestation of the liver by *Entamoeba histolytica*. Amoebic infestation is associated with poor hygiene, unsafe sexual practices, or travel in areas where drinking water is contaminated.

Pathophysiology and Manifestations

Following bacterial or amoebic invasion of the liver, healthy tissue is destroyed, leaving an area of necrosis, inflammatory exudate, and blood. This damaged region becomes walled off from the healthy liver tissue. Pyogenic (bacterial) liver abscess may be caused by cholangitis, or distant or intra-abdominal infections, such as peritonitis or diverticulitis. *Escherichia coli* is the most frequently identified causative organism. The onset of pyogenic abscess is usually sudden, causing acute symptoms such as fever, malaise, vomiting, hyperbilirubinemia, and pain in the right upper abdomen.

The infection pathway for amoebic hepatic abscesses usually is the portal venous circulation from the right colon. Generally, the onset of amoebic abscess is insidious.

INTERDISCIPLINARY CARE

Hepatic abscess is diagnosed through biopsy, hepatic aspirate, blood and fecal cultures, and CT scan and ultrasound studies. Therapy is based on identifying the causative organism through laboratory cultures. Pyogenic abscesses are treated with antibiotics to which the causative organism is sensitive.

Pharmacologic agents used for amebic hepatic abscess are the same as those used for intestinal amebic infestation (see Chapter 26 ∞); combination therapy is commonly used. Two commonly used drugs for treating amebic liver abscesses are metronidazole (Flagyl) and iodoquinol (Diquinol). Both medications can cause gastrointestinal symptoms. Bone marrow suppression is a risk with metronidazole.

If the abscess does not respond to antibiotic therapy, percutaneous aspiration or surgical drainage may be done. In these procedures, a *percutaneous closed-catheter drain* is placed in the abscess to promote drainage of purulent material.

NURSING CARE

A major aspect of nursing care is prevention; teaching clients to avoid contaminated water and foods is especially important. Nursing interventions include teaching hikers to treat water and food handlers to wash hands thoroughly.

Clients who have a liver abscess require supportive care to prevent dehydration from the accompanying fever, nausea, vomiting, and anorexia. Careful monitoring of fluid and electrolyte status is indicated, as are comfort measures for abdominal pain. Possible nursing diagnoses include the following:

- *Risk for Deficient Fluid Volume* related to effects of prolonged fever and vomiting
- *Deficient Knowledge* related to transmission of amebic abscess
- *Activity Intolerance* related to pain and weakness.

EXOCRINE PANCREAS DISORDERS

The pancreas is both an exocrine and an endocrine gland. It is made up of two basic cell types, each having different functions. The exocrine cells produce enzymes that empty through ducts into the small intestine, whereas the endocrine cells produce hormones that enter the bloodstream directly. Disorders of the exocrine pancreas affect the secretion and glandular control of

digestive enzymes, whereas disorders of the endocrine pancreas affect the production of hormones necessary for normal carbohydrate, protein, and fat metabolism. Disorders of the exocrine pancreas are discussed in this section of the chapter; diabetes mellitus, a disorder of the endocrine pancreas, is discussed in Chapter 20 ∞.

THE CLIENT WITH PANCREATITIS

Pancreatitis, or inflammation of the pancreas, is characterized by release of pancreatic enzymes into the tissue of the pancreas itself, leading to hemorrhage and necrosis. Pancreatitis may be either acute or chronic. About 5000 new cases of acute pancreatitis are diagnosed every year in the United States. It is a serious disease, with a mortality rate of approximately 10% (Kasper et al., 2005). Alcoholism and gallstones are the primary risk factors for acute pancreatitis.

The incidence of chronic pancreatitis is less clear, because many people with chronic pancreatitis do not have classic manifestations of the disease. Clients with pancreatitis may have long-term effects of the disease, with chronic changes in enzyme and hormone production.

Physiology Review

Knowledge of the normal structure and functions of the exocrine pancreas is important to understand how inflammation affects it and the client. The exocrine pancreas consists of lobules of acinar cells. The acinar cells secrete digestive enzymes and fluids (pancreatic juices) into ducts that empty into the main pancreatic duct (the duct of Wirsung). The pancreatic duct joins the common bile duct and empties into the duodenum through the ampulla of Vater (in some people the main pancreatic duct empties directly into the duodenum). The epithelial lining of the pancreatic ducts secretes water and bicarbonate to modify the composition of the pancreatic secretions. Pancreatic enzymes are secreted primarily in an inactive form and are activated in the intestine, a modification that prevents digestion of pancreatic tissue by its own enzymes (Porth, 2005). The pancreatic enzymes, with related functions, are as follows:

- Proteolytic enzymes, including trypsin, chymotrypsin, carboxypolypeptidase, ribonuclease, and deoxyribonuclease, which break down dietary proteins
- Pancreatic amylase, which breaks down starch
- Lipase, which breaks down fats into glycerol and fatty acids.

Pathophysiology

Acute Pancreatitis

Acute pancreatitis is an inflammatory disorder that involves self-destruction of the pancreas by its own enzymes through autodigestion. The milder form of acute pancreatitis, *interstitial edematous pancreatitis*, leads to inflammation and edema of pancreatic tissue. It often is self-limiting. The more severe form, *necrotizing pancreatitis*, is characterized by inflammation, hemorrhage, and ultimately necrosis of pancreatic tissue.

Acute pancreatitis is more common in middle adults; its incidence is higher in men than in women. Acute pancreatitis is usually associated with gallstones in women and with alcoholism in men. Some clients recover completely, others experience recurring attacks, and still others develop chronic pancreatitis. The mortality and symptoms depend on the severity and type of pancreatitis: With mild pancreatic edema, mortality is low (6%); with severe necrotic pancreatitis, the mortality rate is high (23%) (Porth, 2005).

Although the exact cause of pancreatitis is not known, the following factors may activate pancreatic enzymes within the pancreas, leading to autodigestion, inflammation, edema, and/or necrosis.

- Gallstones may obstruct the pancreatic duct or cause bile reflux, activating pancreatic enzymes in the pancreatic duct system.
- Alcohol causes duodenal edema, and may increase pressure and spasm in the sphincter of Oddi, obstructing pancreatic outflow. It also stimulates pancreatic enzyme production, thus raising pressure within the pancreas.

Other factors associated with acute pancreatitis include tissue ischemia or anoxia, trauma or surgery, pancreatic tumors, third-trimester pregnancy, infectious agents (viral, bacterial, or parasitic), elevated calcium levels, and hyperlipidemia. Some medications have been linked with this disorder, including thiazide diuretics, estrogen, steroids, salicylates, and NSAIDs.

Regardless of the precipitating factor, the pathophysiologic process begins with the release of activated pancreatic enzymes into pancreatic tissue. Activated proteolytic enzymes, trypsin in particular, digest pancreatic tissue and activate other enzymes such as phospholipase A, which digests cell membrane phospholipids, and elastase, which digests the elastic tissue of blood vessel walls. This leads to proteolysis, edema, vascular damage and hemorrhage, and necrosis of parenchymal cells. Cellular damage and necrosis release activated enzymes and vasoactive substances that produce vasodilation, increase vascular permeability, and cause edema. A large volume of fluid may shift from circulating blood into the retroperitoneal space, the peripancreatic spaces, and the abdominal cavity.

MANIFESTATIONS Acute pancreatitis develops suddenly, with an abrupt onset of continuous severe epigastric and abdominal pain. This pain commonly radiates to the back and is relieved somewhat by sitting up and leaning forward. The pain often is initiated by a fatty meal or excessive alcohol intake.

Other manifestations include nausea and vomiting; abdominal distention and rigidity; decreased bowel sounds; tachycardia; hypotension; elevated temperature; and cold, clammy skin. Within 24 hours, mild jaundice may appear. Retroperitoneal bleeding may occur 3 to 6 days after the onset of acute pancreatitis; signs of bleeding include bruising in the flanks (Turner's sign) or around the umbilicus (Cullen's sign). See the Manifestations box on the following page.

COMPLICATIONS Systemic complications of acute pancreatitis include intravascular volume depletion with acute tubular necrosis and renal failure (see Chapter 29 ∞ for more information about acute renal failure), and acute respiratory distress syndrome (ARDS). Acute renal failure usually develops within 24 hours after the onset of acute pancreatitis. Manifestations of ARDS may be seen 3 to 7 days after its onset, particularly in

MANIFESTATIONS of Acute and Chronic Pancreatitis

ACUTE PANCREATITIS

- Abrupt onset of severe epigastric and left upper quadrant pain, may radiate to back
- Nausea, vomiting; fever
- Decreased bowel sounds; abdominal distention and rigidity
- Tachycardia, hypotension; cold, clammy skin
- Possible jaundice
- Positive Turner's sign (flank ecchymosis) or Cullen's sign (periumbilical ecchymosis)

CHRONIC PANCREATITIS

- Recurrent epigastric and LUQ pain, radiates to back
- Anorexia, nausea and vomiting, weight loss
- Flatulence, constipation
- Steatorrhea

clients who have experienced severe volume depletion. See Chapter 39 ∞ for more information about ARDS.

Localized complications include pancreatic necrosis, abscess, pseudocysts, and pancreatic ascites. Pancreatic necrosis causes an inflammatory mass that may be infected. It may lead to shock and multiple organ failure. A pancreatic abscess may form late in the course of the disease (6 or more weeks after its onset), causing an epigastric mass and tenderness (Tierney et al., 2005). Pancreatic pseudocysts, encapsulated collections of fluid, may develop both within the pancreas itself and in the abdominal cavity. They may impinge on other structures, or may rupture, causing generalized peritonitis. Rupture of a pseudocyst or of the pancreatic duct can lead to pancreatic ascites. Pancreatic ascites is recognized by gradually increasing abdominal girth and persistent elevation of the serum amylase level without abdominal pain.

Chronic Pancreatitis

Chronic pancreatitis is characterized by gradual destruction of functional pancreatic tissue. In contrast to acute pancreatitis, which may completely resolve with no long-term effects, chronic pancreatitis is an irreversible process that eventually leads to pancreatic insufficiency. Alcoholism is the primary risk factor for chronic pancreatitis in the United States. Malnutrition is a major worldwide risk factor. About 10% to 20% of chronic pancreatitis is idiopathic, with no identified cause. A genetic mutation on a gene associated with cystic fibrosis may play a role in these cases. Children or young adults with cystic fibrosis may develop chronic pancreatitis as well.

In chronic pancreatitis related to alcoholism, pancreatic secretions have an increased concentration of insoluble proteins. These proteins calcify, forming plugs that block pancreatic ducts and the flow of pancreatic juices. This blockage leads to inflammation and fibrosis of pancreatic tissue. In other cases, a stricture or stone may block pancreatic outflow, causing chronic obstructive pancreatitis. In chronic pancreatitis, recurrent episodes of inflammation eventually lead to fibrotic changes in the parenchyma of the pancreas, with loss of exocrine function. This leads to malabsorption from pancreatic insufficiency. If endocrine function is disrupted as well, clinical diabetes mellitus may develop.

MANIFESTATIONS Chronic pancreatitis typically causes recurrent episodes of epigastric and left upper abdominal pain that radiates to the back. This pain may last for days to weeks. As the disease progresses, the interval between episodes of pain becomes shorter. Other manifestations include anorexia, nausea and vomiting, weight loss, flatulence, constipation, and **steatorrhea** (fatty, frothy, foul-smelling stools caused by a decrease in pancreatic enzyme secretion).

COMPLICATIONS Complications of chronic pancreatitis include malabsorption, malnutrition, and possible peptic ulcer disease. Pancreatic pseudocyst or abscess may form, or stricture of the common bile duct may develop. Diabetes mellitus may develop, and there is an increased risk for pancreatic cancer. Narcotic addiction related to frequent, severe pain episodes is common.

INTERDISCIPLINARY CARE

Acute pancreatitis often is a mild, self-limiting disease. Treatment focuses on reducing pancreatic secretions and providing supportive care. Treatment to eliminate the causative factor is begun after the acute inflammatory process resolves. Severe necrotizing pancreatitis may require intensive care management. Treatment for chronic pancreatitis often focuses on managing pain and treating malabsorption and malnutrition.

Diagnosis

The laboratory tests that may be ordered when pancreatitis is suspected are summarized in Table 24–5. Diagnostic studies include the following:

- *Ultrasonography* can identify gallstones, a pancreatic mass, or pseudocyst.
- *CT scan* may be ordered to identify pancreatic enlargement, fluid collections in or around the pancreas, and perfusion deficits in areas of necrosis.
- *Endoscopic retrograde cholangiopancreatography (ERCP)* may be performed to diagnose chronic pancreatitis and to differentiate inflammation and fibrosis from carcinoma.
- *Endoscopic ultrasonography* can detect changes indicative of chronic pancreatitis in the pancreatic duct and parenchyma.
- *Percutaneous fine-needle aspiration biopsy* may be performed to differentiate chronic pancreatitis from cancer of the pancreas; the cells that are aspirated are examined for malignancy.

More information about these tests and their nursing implications can be found in Chapter 21 ∞.

Medications

The treatment of acute pancreatitis is largely supportive. Narcotic analgesics such as morphine sulfate are used to control pain. Antibiotics often are prescribed to prevent or treat infection.

Clients with chronic pancreatitis also require analgesics, but are closely monitored to prevent drug dependence. Narcotics are avoided when possible. Pancreatic enzyme supplements are given to reduce steatorrhea (see the Medication Administration

TABLE 24–5 **Laboratory Tests in Exocrine Pancreatic Disorders**

TEST	NORMAL VALUE	SIGNIFICANCE
Serum amylase	0–130 Unit/L	Rises within 2 to 12 hours of onset of acute pancreatitis to two to three times normal. Returns to normal in 3 to 4 days.
Serum lipase	0–160 Unit/L	Levels rise in acute pancreatitis; remain elevated for 7 to 14 days.
Serum trypsinogen	<80 mcg/L	Elevated in acute pancreatitis; may be decreased in chronic pancreatitis.
Urine amylase	4 to 37 Unit/L/2h	Urine amylase levels rise in acute pancreatitis.
Serum glucose	70 to 110 mg/dL	May be transient elevation in acute pancreatitis.
Serum bilirubin	0.1 to 1.0 mg/dL	Compression of the common duct may increase bilirubin levels in acute pancreatitis.
Serum alkaline phosphatase	30 to 95 Unit/L	Compression of the common duct may increase levels in acute pancreatitis.
Serum calcium	8.9 to 10.3 mg/dL or 4.5 to 5.5 mEq/L	Hypocalcemia develops in up to 25% of clients with acute pancreatitis.
White blood cells	4500/mm^3 to 10,000/mm^3	Leukocytosis indicates inflammation and is usually present in acute pancreatitis.

box below). Clients with chronic pancreatitis may need to remain on pancreatic enzyme supplements for life. H_2 blockers such as cimetidine (Tagamet) and ranitidine (Zantac), and proton-pump inhibitors such as omeprazole (Prilosec) may be given to neutralize or decrease gastric secretions. Octreotide (Sandostatin), a synthetic hormone, suppresses pancreatic enzyme secretion and may be used to relieve pain in chronic pancreatitis.

Treatments

NUTRITION Oral food and fluids are withheld during acute episodes of pancreatitis to reduce pancreatic secretions and promote rest of the organ. A nasogastric tube may be inserted and connected to suction. Intravenous fluids are administered to maintain vascular volume, and total parenteral nutrition (TPN) is initiated. Oral food and fluids are begun once the serum amylase levels have returned to normal, bowel sounds are present, and pain disappears. A low-fat diet is ordered, and alcohol intake is strictly prohibited.

SURGERY If the pancreatitis is the result of a gallstone lodged in the sphincter of Oddi, an *endoscopic transduodenal sphincterotomy* may be performed to remove the stone. When cholelithiasis is identified as a causative factor, a cholecystectomy is performed once the acute pancreatitis has resolved. Surgical procedures to promote drainage of pancreatic enzymes into the duodenum or resection of all or part of the pancreas may be done to provide pain relief in clients with chronic pancreatitis. Large pancreatic pseudocysts may be drained endoscopically or surgically.

COMPLEMENTARY THERAPIES Several complementary therapies may be used in conjunction with traditional treatments for clients with acute or chronic pancreatitis. Fasting or use of low-salt, low-fat vegetarian diets may reduce episodes of recurrent pain. Qigong, a system of gentle exercise, meditation, and controlled breathing, is believed to balance the flow of qi (a vital life force) through the body. Qigong lowers the metabolic rate, and may reduce the stimulation of pancreatic enzyme secretion. Magnetic field therapy also may be employed for clients with pancreatitis. All complementary therapies should be prescribed by a trained and competent practitioner.

MEDICATION ADMINISTRATION **The Client with Chronic Pancreatitis**

PANCREATIC ENZYME REPLACEMENT

Pancrelipase (Lipancreatin)

Pancrelipase enhances the digestion of starches and fats in the gastrointestinal tract by supplying an exogenous source of the enzymes protease, amylase, and lipase. The drug promotes nutrition and decreases the number of bowel movements.

Nursing Responsibilities

- Assess for allergy to pork protein.
- Monitor frequency and consistency of stools.
- Weigh every other day. Record weights.
- Give with meals; if not enteric coated, H_2 antagonists or antacids may be given concurrently to prevent destruction of the enzymes by hydrochloric acid.
- Monitor for side effects: rash, hives, respiratory difficulty, hematuria, hyperuricemia, or joint pain.

Health Education for the Client and Family

- Take with meals or snacks.
- If medicine is enteric coated, do not crush, chew, or mix with alkaline foods (e.g., milk, ice cream)
- Be sure to follow prescribed diet.
- Continue taking this drug until or unless advised by physician that it is no longer necessary.

NURSING CARE

In addition to the nursing care discussed in this section, a Nursing Care Plan for a client with acute pancreatitis is found below.

Health Promotion

Teach clients who abuse alcohol about the risk for developing pancreatitis. Advise abstinence to reduce this risk, and refer to an alcohol treatment program or Alcoholics Anonymous.

Assessment

Assessment data related to acute or chronic pancreatitis include the following:

- *Health history:* Current manifestations; abdominal pain (location, nature, onset and duration, identified precipitating factors); anorexia, nausea or vomiting; flatulence, diarrhea, constipation, or stool changes; recent weight loss; history of previous episodes or gallstones; alcohol use (extent and duration); current medications.

NURSING CARE PLAN A Client with Acute Pancreatitis

Rose Schliefer is a 59-year-old wife, mother of three, and grandmother of four. She has been hospitalized for the past 6 weeks for acute hemorrhagic pancreatitis and pseudocyst. The pancreatitis was caused by gallstones. Mrs. Schliefer spent 3 weeks in intensive care, and then underwent surgery to remove the gallstones and to insert drains into the pseudocyst. Prior to discharge, she had progressed to a soft, high-carbohydrate, low-fat diet; had all drains removed; and was able to walk in the hall. Mrs. Schliefer was referred to a community health agency in her home town for continued follow-up.

ASSESSMENT

Lee Quinn, the community health nurse, assesses Mrs. Schliefer at home after discharge. Mrs. Schliefer is thin and appears anxious and tired. She states that she lost 30 lb (13.6 kg) in the hospital and now weighs only 102 lb (46 kg). She is 66 inches (168 cm) tall. Her vital signs are within normal limits. Mrs. Schliefer has a well-healed upper abdominal scar and two small wounds (from drains) on each side of her abdomen. The wounds are closed but still have scabs. Her skin is cool and dry, and turgor is poor. She is alert and oriented and responds appropriately to questions. Blood glucose levels are normal. Mrs. Schliefer states that her main problems are lack of energy and lack of appetite for the low-fat diet that has been ordered. Mrs. Schliefer's husband and daughters express concern about their ability to provide care. Although they have been taught all about the disease and how to provide care, they still are not sure they know exactly what should be done now that Mrs. Schliefer is at home.

DIAGNOSES

- *Fatigue* related to decreased metabolic energy production
- *Imbalanced Nutrition: Less than Body Requirements* related to prolonged hospitalization, dietary restrictions, and impaired digestion
- *Bathing/Hygiene Self-Care Deficit* (Level II: requires help of another person, supervision, and teaching) related to decreased strength and endurance
- *Risk for Caregiver Role Strain* related to inexperience with caregiving tasks

EXPECTED OUTCOMES

- Set priorities for daily and weekly activities, and incorporate a rest period into daily activity.
- Gain 1 to 2 lbs per week.
- Bathe and maintain personal hygiene without assistance.
- Family members will verbalize comfort with providing necessary care.

PLANNING AND IMPLEMENTATION

- Explain causes of fatigue. Review effects of pancreatitis, surgery, and acute illness on energy levels.
- Develop activity goals, incorporating small, incremental steps toward achieving goals. Mrs. Schliefer indicates that she wants to cook a meal for the whole family. To reach this goal, she will:
  a. Schedule the meal when her energy level is highest.
  b. List actions necessary to prepare the meal and delegate difficult tasks to family members.
  c. Ask daughters to reorganize the kitchen to avoid unnecessary steps.
  d. Plan the meal no sooner than the third week after being home.
- Instruct to:
  a. Rest in bed each day from 1:00 P.M. to 3:00 P.M.
  b. Eat six small meals a day with family members or friends.
  c. Sit and rest quietly for 15 minutes before eating.
- Discuss dietary restrictions and how to adapt them to usual diet.
- Advise to use shower chair and develop self-care goals for bathing and hygiene in small steps. Add self-care tasks gradually as tolerated.
- Discuss division of responsibilities for physical care, home maintenance, and medical care with family members.
- Encourage family discussion of concerns about future; acknowledge family strengths.

EVALUATION

One month after discharge, Mrs. Schliefer and her family have established new routines based on her energy levels. Mrs. Schliefer now fixes lunch because she feels best during midday. She and her husband share this time together without interruption. Mrs. Schliefer still rests during the day but can now provide self-care. She has gained only 2 lb, but states that she is getting used to the new diet and that "things are even starting to taste good without butter." She also says that sitting quietly before meals is helpful and that she prefers eating six small meals a day. Mr. and Mrs. Schliefer and their daughters agree that their initial worries about Mrs. Schliefer's care have been resolved; now they all know what they must do, and the future looks much brighter.

CRITICAL THINKING IN THE NURSING PROCESS

1. Your client with acute pancreatitis is also an alcoholic. Describe assessments that indicate the beginnings of withdrawal.
2. Discuss the pathophysiologic basis of hypovolemic shock in acute necrotic pancreatitis.
3. Outline a teaching plan that includes specific foods to omit and to include in a high-carbohydrate, low-protein, low-fat diet.
4. Develop a plan of care for the nursing diagnosis *Impaired Home Maintenance*.

See Evaluating Your Response in Appendix C.

- *Physical assessment:* Vital signs including orthostatic vitals and peripheral pulses; temperature; skin temperature and color, presence of any flank or periumbilical ecchymoses; abdominal assessment including bowel sounds, presence of distention, tenderness, or guarding.

Nursing Diagnoses and Interventions

Nursing care for the client with acute pancreatitis focuses on managing pain, nutrition, and maintaining fluid balance.

Pain

Obstruction of pancreatic ducts and inflammation, edema, and swelling of the pancreas caused by pancreatic autodigestion cause severe epigastric, left upper abdominal, or midscapular back pain. The pain often is accompanied by nausea and vomiting, abdominal tenderness, and muscle guarding.

- Using a standard pain scale (see Chapter 9 ∞), assess pain, including location, radiation, duration, and character. Note nonverbal cues of pain: restlessness or remaining rigidly still; tense facial features; clenched fists; rapid, shallow respirations; tachycardia; and diaphoresis. Administer analgesics on a regular schedule. *Pain assessment before and after analgesic administration measures its effectiveness. Administering analgesics on a regular schedule prevents pain from becoming established, severe, and difficult to control. Unrelieved pain has negative consequences; for example, pain, anxiety, and restlessness may increase pancreatic enzyme secretion.*

PRACTICE ALERT

Regularly assess respiratory status (at least every 4 to 8 hours), including respiratory rate, depth, and pattern; breath sounds; oxygen saturation and arterial blood gas results. Report tachypnea, adventitious or absent breath sounds, oxygen saturation levels below 92%, $Pa_{O_2} < 70$ mmHg or $Pa_{CO_2} > 45$ mmHg. Severe abdominal pain causes shallow respirations and hypoventilation, and suppresses cough effectiveness, which can lead to pooling of secretions, atelectasis, and pneumonia.

- Maintain NPO status and nasogastric tube patency as ordered. *Gastric secretions stimulate hormones that stimulate pancreatic secretion, aggravating pain. Eliminating oral intake and maintaining gastric suction reduce gastric secretions. Nasogastric suction also decreases nausea, vomiting, and intestinal distention.*
- Maintain bed rest in a calm, quiet environment. Encourage use of nonpharmacologic pain management techniques such as meditation and guided imagery. *Decreasing physical movement and mental stimulation decreases metabolic rate, gastrointestinal secretion, pancreatic secretions, and resulting pain. Adjunctive pain relief measures enhance the effectiveness of analgesics (see Chapter 9 ∞).*
- Assist to a comfortable position, such as a side-lying position with knees flexed and head elevated 45 degrees. *Sitting up, leaning forward, or lying in a fetal position tends to decrease pain caused by stretching of the peritoneum by edema and swelling.*
- Remind family and visitors to avoid bringing food into the client's room. *The sight or smell of food may stimulate secretory activity of the pancreas through the cephalic phase of digestion.*

Imbalanced Nutrition: Less than Body Requirements

The effects of pancreatitis and its treatment may result in malnutrition. Inflammation increases metabolic demand and frequently causes nausea, vomiting, and diarrhea. At a time of increased metabolic demand, NPO status and gastric suction further decrease available nutrients. In the client with chronic pancreatitis, loss of digestive enzymes affects the digestion and use of nutrients.

- Monitor laboratory values: serum albumin, serum transferrin, hemoglobin, and hematocrit. *Serum albumin, serum transferrin (which transports iron in the blood), hemoglobin, and hematocrit levels are decreased in malnutrition. Decreased pancreatic enzymes affect protein catabolism and absorption; decreased transferrin affects iron absorption and transport, thereby decreasing hematocrit and hemoglobin levels.*
- Weigh daily or every other day. *Short-term weight changes (over hours to days) accurately reflect fluid balance, whereas weight changes over days to weeks reflect nutritional status.*
- Maintain stool chart; note frequency, color, odor, and consistency of stools. *Protein and fat metabolism are impaired in pancreatitis; undigested fats are excreted in the stool. Steatorrhea indicates impaired digestion and, possibly, an increase in the severity of pancreatitis.*
- Monitor bowel sounds. *The return of bowel sounds indicates return of peristalsis; nasogastric suction usually is discontinued within 24 to 48 hours thereafter.*
- Administer prescribed intravenous fluids and/or TPN. *Intravenous fluids are given to maintain hydration. TPN is used to provide fluids, electrolytes, and kilocalories when fasting is prolonged (more than 2 to 3 days).*
- Provide oral and nasal care every 1 to 2 hours. *Fasting and nasogastric suction increase the risk for mucous membrane irritation and breakdown.*
- When oral intake resumes, offer small, frequent feedings. Provide oral hygiene before and after meals. *Oral hygiene decreases oral microorganisms that can cause foul odor and taste, decreasing appetite. Small, frequent feedings reduce pancreatic enzyme secretion and are more easily digested and absorbed.*

Risk for Deficient Fluid Volume

Acute pancreatitis can lead to a fluid shift from the intravascular space into the abdominal cavity (third spacing). Third spacing of fluid may cause hypovolemic shock, affecting cardiovascular function, respiratory function, renal function, and mental status.

- Assess cardiovascular status every 4 hours or as indicated, including vital signs, cardiac rhythm, hemodynamic parameters (central venous and pulmonary artery pressures); peripheral pulses and capillary refill; skin color, temperature, moisture, and turgor. *These measurements are indicative of fluid volume status and are used to monitor response to treatment. Stable values are as follows: heart rate less than 100; blood pressure within 10 mmHg of baseline; central venous pressure 0 to 8 mmHg; pulmonary wedge pressure 8 to 12 mmHg; cardiac output approximately 5 L/min; and skin warm, dry, with good turgor and color. (See Chapter 11 ∞ for a full discussion of hypovolemic shock.)*

- Monitor renal function. Obtain hourly urine output; report if less than 30 mL per hour. Weigh daily. *Urine output of less than 30 mL per hour indicates decreased renal perfusion or acute renal failure, a major complication of acute pancreatitis. Weight changes are an effective indicator of fluid volume status.*
- Monitor neurologic function, including mental status, level of consciousness, and behavior. *Hypotension and hypoxemia may decrease cerebral perfusion, causing changes in mental status, decreased level of consciousness, and changes in behavior. In addition, alcohol withdrawal is a risk in the client with acute pancreatitis.*

Using NANDA, NIC, and NOC

Chart 24–3 shows linkages between NANDA nursing diagnosis, NIC, and NOC for the client with pancreatitis.

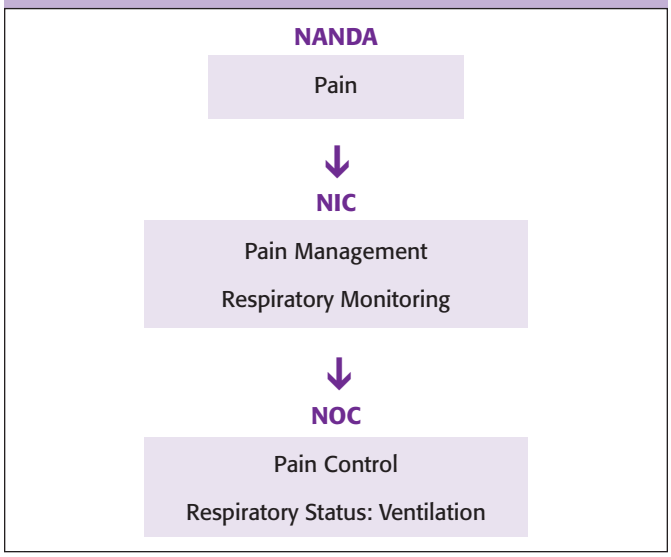
Community-Based Care

The client with pancreatitis is often acutely ill and, along with family members, needs information about both hospital procedures and self-care at home following discharge. During the acute stage, keep explanations brief and simple.

Prior to discharge, teach the client and family about the disease and how to prevent further attacks of inflammation. Include the following topics as appropriate:

- Alcohol can cause stones to form, blocking pancreatic ducts and the outflow of pancreatic juice. Continued alcohol intake is likely to cause further inflammation and destruction of the pancreas. Avoid alcohol entirely.
- Smoking and stress stimulate the pancreas and should be avoided.
- If pancreatic function has been severely impaired, discuss appropriate use of pancreatic enzymes, including timing, dose, potential side effects, and monitoring of effectiveness.

NANDA, NIC, AND NOC LINKAGES
CHART 24–3 **The Client with Pancreatitis**



Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2005), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, & M. Maas (2004), St. Louis, MO: Mosby.

- A low-fat diet is recommended. Provide a list of high-fat foods to avoid. Crash dieting and binge eating also should be avoided as they may sometimes precipitate attacks. Spicy foods, coffee, tea, or colas, and gas-forming foods stimulate gastric and pancreatic secretions and may precipitate pain. Avoid them if this occurs.
- Report symptoms of infection (fever of 102°F [38.8°C]) or more, pain, rapid pulse, malaise) because a pancreatic abscess can develop after initial recovery.

Refer to a dietitian or nutritionist for diet teaching as needed. If appropriate, refer to community agencies, such as Alcoholics Anonymous, or to an alcohol treatment program. Provide referrals to community or home health agencies as needed for continued monitoring and teaching at home.

THE CLIENT WITH PANCREATIC CANCER

Cancer of the pancreas accounts for approximately 2% of all cancers. It is, however, one of the most lethal cancers: More than 98% of people with pancreatic cancer die. An estimated 32,180 new cases occurred in the United States in 2005, with approximately 31,800 deaths from cancer of the pancreas occurring the same year (American Cancer Society, 2005). The incidence of pancreatic cancer increases after age 50. The incidence is slightly higher in women than men, and is higher in blacks than in whites.

FAST FACTS

Identified risk factors for pancreatic cancer include:

- Cigarette smoking—the incidence is twice as high in smokers as in nonsmokers
- Exposure to industrial chemicals or environmental toxins
- Chronic pancreatitis
- Diabetes mellitus
- Obesity, high-fat diet.

In contrast to acute and chronic pancreatitis, alcohol abuse and gallstones are not identified risk factors for pancreatic cancer.

Pathophysiology and Manifestations

Most cancers of the pancreas occur in the exocrine pancreas, are adenocarcinomas, and cause death within 1 to 3 years after diagnosis.

Cancer of the pancreas has a slow onset, with manifestations of anorexia, nausea, weight loss, flatulence, and dull epigastric pain. The pain increases in severity as the tumor grows. Other manifestations depend on the location of the tumor. Cancer of the head of the pancreas, which is the most common site, often obstructs bile flow through the common bile duct and the ampulla of Vater, resulting in jaundice, clay-colored stools, dark urine, and pruritus. Cancer of the body of the pancreas presses on the celiac ganglion, causing pain that increases when the person eats or lies supine. Cancer of the tail of the pancreas often causes no symptoms until it has metastasized. Other late manifestations include a palpable abdominal mass and ascites. Because the manifestations are nonspecific, up to 85% of

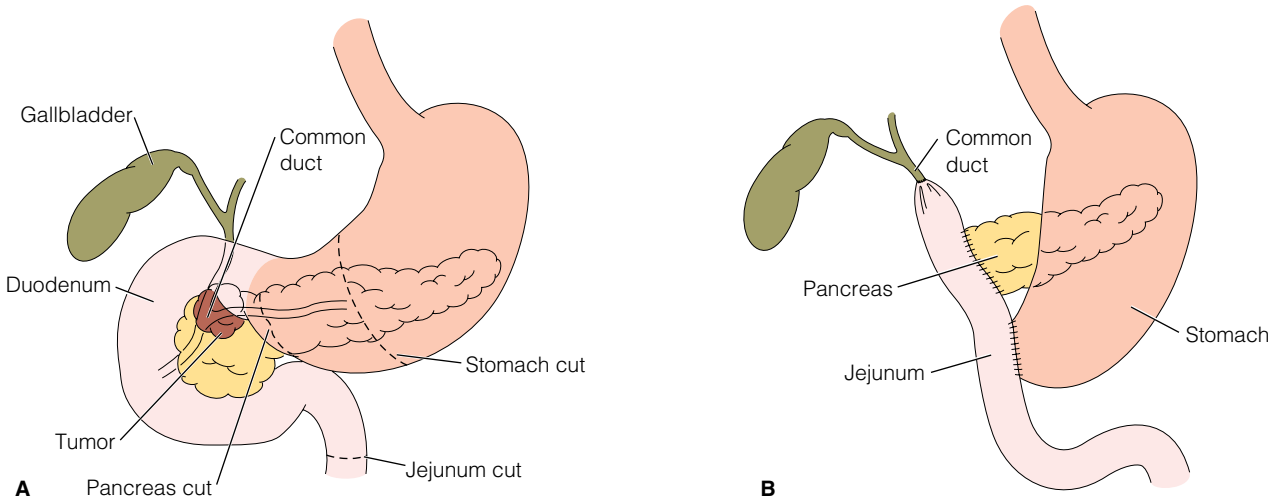


Figure 24–7 ■ Pancreatoduodenectomy (Whipple's procedure). *A*, areas of resection; *B*, appearance following resection.

clients with cancer of the pancreas do not seek health care until the cancer becomes too far advanced for a cure.

INTERDISCIPLINARY CARE

Early cancers of the head of the pancreas may be resectable. A pancreatoduodenectomy (commonly called Whipple's procedure) is performed to remove the head of the pancreas, the entire duodenum, the distal third of the stomach, a portion of the jejunum, and the lower half of the common bile duct. The common bile duct is then sutured to the end of the jejunum, and the remaining pancreas and stomach are sutured to the side of the jejunum (Figure 24–7 ■). Radiation and chemotherapy are often used in addition to surgery.

NURSING CARE OF THE CLIENT UNDERGOING Whipple's Procedure

PREOPERATIVE CARE

- Provide routine preoperative nursing care as outlined in Chapter 4 ∞.
- Clarify teaching and learning as needed. Provide psychologic support for client and family. *The client and family faced with a diagnosis of pancreatic cancer may require reinforcement of teaching as anxiety, fear, and possible denial can interfere with learning.*

POSTOPERATIVE CARE

- Provide postoperative care as outlined in Chapter 4 ∞.
- Maintain in semi-Fowler's position. *Semi-Fowler's position facilitates lung expansion and reduces stress on the anastomosis and suture line.*
- Maintain low gastrointestinal suction. If drainage is not adequate, obtain an order to irrigate, using minimal pressure. Do not reposition nasogastric tube. *Pressure within the operative area from retained secretions increases intraluminal pressure and places stress on the suture line. Forceful irrigations and repositioning of the nasogastric tube may disrupt the suture line.*
- Maintain pain control using analgesics as prescribed (PCA, infusion, or given on a regular basis). Assess effectiveness of pain management. *Doses higher than normal may be required if narcotic analgesics have been used prior to surgery to manage pain.*
  Increased pain may indicate complications such as disruption of suture line, leakage from anastomosis, or peritonitis. Adequate pain management increases resistance to stress, facilitates healing, and increases the ability to cough, deep breathe, and change position.
- Assist with coughing, deep breathing, and changing position every 1 to 2 hours. Splint incision during coughing and deep breathing. *The location of the incision makes coughing and deep breathing more painful. The prolonged surgical procedure, anesthesia, location of incision, and immobility increase the risk of retained secretions, atelectasis, and pneumonia. Changing position facilitates drainage of secretions; effective coughing and deep breathing remove secretions and open distal alveoli.*
- Monitor for complications:
  a. Take vital signs every 2 to 4 hours or as indicated; immediately report changes (such as elevated temperature; hypotension; weak, thready pulse; increased or difficult respirations).
  b. Assess skin color, temperature, moisture, and turgor.
  c. Measure urinary output, gastrointestinal output, and drainage from any other tubes; monitor amount and type of wound drainage.
  d. Assess level of consciousness.
  e. Monitor results of laboratory tests, especially arterial blood gases, hemoglobin, and hematocrit.

  The major complications following Whipple's procedure are hemorrhage, hypovolemic shock, and hepatorenal failure. The assessments listed provide information about the client's status and alert the nurse to abnormal findings that signal the onset of these complications.

Postoperative nursing care of the client undergoing Whipple's procedure is outlined on page 732. Immediate postoperative care is often provided in the intensive care unit.

The client with pancreatic cancer has multiple problems requiring nursing care. Chapter 14 ∞ provides a discussion of care of the client with cancer; the nursing diagnoses and interventions discussed for the client with pancreatitis are also appropriate for the client with pancreatic cancer.

EXPLORE MEDIALINK

DVD-ROM
Audio Glossary
NCLEX-RN® Review

Animation/Video
Cirrhosis

COMPANION WEBSITE www.prenhall.com/lemone

Audio Glossary
NCLEX-RN® Review
Care Plan Activity: A Client with Hepatitis A
Case Studies
Hepatitis B
Traditional Native American Diet and Gallbladder Disease
MediaLink Applications
GI Disorders and Gall Bladder Disease
Treatment of Liver Cancer
Links to Resources

CHAPTER HIGHLIGHTS

- Gallstones (cholelithiasis) are common and often unrecognized until the client develops manifestations of biliary colic or acute cholecystitis. Laparoscopic cholecystectomy is the treatment of choice for symptomatic gallbladder disease.
- Hepatitis, inflammation of functional liver tissue, usually is a viral disease and therefore cannot be cured at this time. Preventing the spread of hepatitis through use of standard and body substance precautions is an important nursing responsibility.
- Hepatitis A, commonly transmitted via the fecal-oral route, generally is a self-limiting disease with few long-term sequelae. Some types of viral hepatitis, most notably hepatitis B and C, can become chronic and ultimately lead to liver failure and an increased risk for liver cancer. Hepatitis B and C can result in a carrier state in which the infected client has no symptoms of the disease, but can spread it to others.
- Alcohol abuse is a significant risk factor for liver and pancreatic disorders. Prevention, early identification, and treatment of alcohol abuse reduces the risk of these disorders. Absolute abstinence from alcohol is an important part of the treatment plan for clients with liver and pancreatic disorders.
- Cirrhosis leads to portal hypertension and liver failure, which, in turn, account for most of the manifestations and complications of the disorder. Complications such as ascites, splenomegaly, esophageal varices, and hepatic encephalopathy affect multiple body systems and significantly contribute to mortality and morbidity associated with cirrhosis.
- Bleeding from esophageal varices may be massive, resulting in a medical emergency and requiring prompt control to maintain cardiac output.
- Acute pancreatitis often develops as a complication of gallstones. Acute pancreatitis often resolves with no long term consequences. Chronic pancreatitis is more frequently related to alcohol abuse and can lead to continuing pain and digestive disruptions.

TEST YOURSELF NCLEX-RN® REVIEW

1 When assessing the client admitted for a laparoscopic cholecystectomy, the nurse would expect to find:
1. a history of intermittent episodes of right upper quadrant pain.
2. significant jaundice of the sclera and skin.
3. complaints of recurrent heartburn and acid reflux.
4. ascites and peripheral edema.

2 Which of the following does the nurse include in her teaching for a client with acute cholecystitis? (Select all that apply.)
1. Avoid consumption of foods high in fat such as gravies and peanut butter.
2. Limit your intake to dry crackers and clear liquids during episodes of acute pain.
3. A low-carbohydrate diet such as the Atkins diet is recommended for weight loss.
4. Call your doctor if you develop severe abdominal pain and a temperature.
5. Surgery for gallstones is optional; they pose little risk when fat intake is minimal.

3 During an outbreak of hepatitis A traced to a food handler at a local restaurant, the nurse teaches staff at the restaurant that the most cost-effective means of protecting customers from further outbreaks is to:
1. insist that all food handlers be immunized against hepatitis A.
2. test all new employees for hepatitis A antigen.
3. wash hands thoroughly before handling food and after using the bathroom.
4. use gloves for handling food if any cuts or scrapes are on hands.

4 The nurse would evaluate teaching as effective when a client with chronic hepatitis C states which of the following?
1. "I will reduce my alcohol intake and use only acetaminophen for pain relief."
2. "I understand that I must return to the doctor every year for a follow-up liver biopsy."
3. "Even though no treatment is available for this disease, I plan to live a long life."
4. "I will avoid donating blood and will use barrier protection during sex."

5 When evaluating for people possibly exposed to hepatitis A by a recently diagnosed client, the nurse inquires about
1. sexual partners within the past 6 months.
2. close household contacts within the past 4 weeks.
3. food preparation activities since the development of jaundice.
4. immunization status of the client.

6 A client hospitalized with cirrhosis, ascites, and mild hepatic encephalopathy suddenly vomits 200 mL of bright red blood. Which of the following should the nurse do first?
1. Insert a nasogastric tube.
2. Place in Fowler's position.
3. Contact the physician.
4. Check stool for occult blood.

7 The nurse caring for a client scheduled for an abdominal paracentesis instructs the client to
1. avoid eating or drinking fluid for 6 hours prior to the procedure.
2. scrub the abdomen with antiseptic soap before the procedure.
3. empty the bladder before the procedure.
4. report excess flatus following the procedure to the physician.

8 A client hospitalized with severe ascites due to cirrhosis develops a fever and confusion. The nurse should
1. auscultate bowel sounds and palpate for abdominal tenderness.
2. inquire about headache and check for nuchal rigidity.
3. observe for neck vein distention and auscultate lung sounds.
4. measure abdominal girth and percuss for shifting dullness.

9 A 54-year-old woman admitted with acute pancreatitis says, "I don't understand how I got this disease. I thought alcoholics got pancreatitis—I never drink." Which of the following is the most appropriate response by the nurse?
1. "Was there a time in your life that you did drink heavily?"
2. "It also is prevalent in smokers; do you smoke cigarettes?"
3. "Gallstones also are a risk factor. We'll evaluate for them."
4. "Intravenous drug use is a risk factor. Do you use drugs by injection?"

10 The nurse caring for a client returning to the unit following Whipple's procedure identifies which of the following as of highest priority in the plan of care?
1. referral to a smoking cessation program
2. frequent turning, coughing, and deep breathing exercises
3. early mobilization including ambulation as tolerated
4. maintaining patency of the nasogastric tube

See Test Yourself answers in Appendix C.

BIBLIOGRAPHY

Agency for Healthcare Research and Quality. (2000). *Milk thistle: Effects on liver disease and cirrhosis and clinical adverse effects. Summary, evidence report/technology assessment* (AHRQ Pub. No. 01-EO24). Retrieved from http://www.ahrq.gov/clinic/epcsums/milktsum.htm

Allen, G. (2004). Evidence for practice. Hepatitis B vaccination schedules. *AORN Journal 79*(6), 1322–1323.

_____. (2005). Evidence for practice. Conversion from laparoscopic to open cholecystectomy. *AORN Journal, 81*(3), 690, 693.

American Cancer Society. (2005). *Cancer facts and figures 2005*. Atlanta: Author.

Atassi, K. A. (2002). Bleeding esophageal varices. *Nursing, 32*(4), 96.

Barthelsson, C., Lutzen, K., Anderberg, B., & Nordstrom, G. (2003). Patients' experiences of laparoscopic cholecystectomy in day surgery. *Journal of Clinical Nursing, 12*(2), 253–259.

Binnekade, J. M. (2005). Review: Enteral nutrition reduces infections, need for surgical intervention, and length of hospital stay more than parenteral nutrition in acute pancreatitis. *Evidence-Based Nursing, 8*(1), 19.

Burruss, N., & Holz, S. (2005). Understanding acute pancreatitis. *Nursing, 35*(3), 32hn2, 32hn4.

Centers for Disease Control and Prevention. (2006). *Reported cases of acute viral hepatitis, by type and year, United States, 1966–2003*. Retrieved from http://www.cdc.gov/ncidod/diseases/hepatitis/resource/PDFs/surv_table.pdf

Cole, L. (2002). Unraveling the mystery of acute pancreatitis. *Dimensions of Critical Care Nursing, 26*(6), 86–89.

Copstead, L. C., & Banasick, J. L. (2005). *Pathophysiology* (3rd ed.). St. Louis, MO: Elsevier/Saunders.

Despins, L. A., Kivlahan, C., & Cox K. R. (2005). Emergency. Acute pancreatitis: Diagnosis and treatment of a potentially fatal condition. *American Journal of Nursing, 105*(11), 54–57.

Durston, S. (2005). What you need to know about viral hepatitis. *Nursing, 35*(8), 36–42.

Fontaine, K. L. (2000). *Healing practices: Alternative therapies for nursing*. Upper Saddle River, NJ: Prentice Hall Health.

Glacken, M., Coates, V., Kernohan, G., & Hegarty, J. (2003). The experience of fatigue for people living with hepatitis C. *Journal of Clinical Nursing, 12*(2), 244–252.

Hainsworth, T. (2005). NT clinical. Improving identification and awareness of hepatitis C. *Nursing Times, 101*(42), 23–24.

Jarrett, M., & Cox, P. (2004). Hepatitis C virus. *Nursing Clinics of North America, 39*(1), 219–229.

Kasper, D. L., Braunwald, E., Fauci, A. S., Hauser, S. L., Longo, D. L., & Jameson, J. L. (Eds.). (2005). *Harrison's principles of internal medicine* (16th ed.). New York: McGraw-Hill.

Knight, J. A. (2005). Liver function tests: Their role in the diagnosis of hepatobiliary diseases. *Journal of Infusion Nursing, 28*(2), 108–117.

Lee, J. M., Botteman, M. F., Xanthakos, N., & Nicklasson, L. (2005). Needlestick injuries in the United States: Epidemiologic, economic, and quality of life issues. *AAOHM Journal, 53*(3), 117–133.

Mann, R. E., Smart, R. G., & Govoni, R. (2003). The epidemiology of alcoholic liver disease. *Alcohol Research & Health, 27*(3), 209–219.

McCance, K. L., & Huether, S. E. (2002). *Pathophysiology: The biologic basis for disease in adults & children* (4th ed.). St. Louis, MO: Mosby.

Medical Encyclopedia: Diet — liver disease. (2005). Retrieved from http://www.nlm.nih.gov/medlineplus/ency/article/oo2441.htm.

Meeker, M. H., & Rothrock, J. C. (1999). *Alexander's care of the patient in surgery* (11th ed.). St. Louis, MO: Mosby.

NANDA International. (2005). *NANDA nursing diagnoses: Definitions & classification 2005–2006*. Philadelphia: Author.

National Center for Complementary and Alternative Medicine, National Institutes of Health. (2004). *Research report. Hepatitis C and complementary and alternative medicine: 2003 update* (NCCAM Pub. No. D004). Retrieved from http://www.nccam.nih.gov/health/hepatitisc/

National Center for Health Statistics. (2005). *Health, United States, 2005, with chartbook on trends in the health of Americans*. Hyattsville, MD: Author.

National Immunization Program. (2005). *Epidemiology and prevention of vaccine-preventable diseases* (8th ed.). Atlanta: Centers for Disease Control and Prevention.

Porth, C. M. (2005). *Pathophysiology: Concepts of altered health states* (7th ed.). Philadelphia: Lippincott.

Smith, D. R., & Leggat, P. A. (2005). Needlestick and sharps injuries among nursing students. *Journal of Advanced Nursing, 51*(5), 449–455.

Stevenson, L., Faucher, Y., Hewlett, S., Klemm, K., & Nelson, D. (2004). Chronic hepatitis C virus and the Hispanic community: Cultural factors impacting care. *Gastroenterology Nursing, 27*(5), 230–238.

Tierney, L. M., McPhee, S. J., & Papadakis, M. A. (2005). *Current medical diagnosis & treatment* (44th ed.). New York: Lange Medical Books/McGraw-Hill.

Urden, L. D., Stacy, K. M., & Lough, M. E. (2006). *Thelan's critical care nursing: Diagnosis and management* (5th ed.). St. Louis, MO: Mosby.

Watt-Watson, J., Chung, F., Chan, V. W. S., & McGillion, M. (2004). Pain management following discharge after ambulatory same-day surgery. *Journal of Nursing Management, 12*(3), 153–161.

Wilkinson, J. M. (2005). *Nursing diagnosis handbook with NIC interventions and NOC outcomes* (8th ed.). Upper Saddle River, NJ: Prentice Hall Health.

Wilson, T. R. (2005). The ABCs of hepatitis. *Nurse Practitioner, 30*(6), 12–15, 18, 20–23.

UNIT 6 BUILDING CLINICAL COMPETENCE
Responses to Altered Nutrition

FUNCTIONAL HEALTH PATTERN: Nutritional—Metabolic

Think about clients with altered nutrition for whom you have cared in your clinical experiences.

- What were the clients' major medical diagnoses (e.g., gastroesophageal reflux disease, cholelithiasis and cholecystitis, hepatitis, pancreatitis)?
- What kinds of manifestations did each of these clients have? Were these manifestations similar or different?
- How did the clients' nutritional problems interfere with their health status? How was their appetite? Were they on a prescribed diet? What was their daily intake of foods and fluids? Did the clients add salt to foods or eat processed foods? What types of fluids were they consuming, including caffeinated beverages? Did they use dietary supplements? Did they have difficulty chewing or swallowing? Were they gaining or losing weight? Did the clients' nutritional disorders affect healing or their immune status?

The Nutritional-Metabolic Health Pattern encompasses those activities and processes involved in taking in, assimilating, and using nutrients to produce energy and to maintain and repair body tissues. Disorders of nutritional intake and disorders of the organ systems involved in consuming, digesting, absorbing, and metabolizing nutrients can disrupt the Nutritional-Metabolic Pattern in several ways:

- Inability to consume and metabolize required nutrients can result in nutritional disorders (e.g., malnutrition) and impaired tissue repair.
- Insufficient nutrient intake can result from functional disorders (e.g., lack of access to appropriate foods), structural disorders (e.g., oral or esophageal cancer, hiatal hernia), inflammatory responses (e.g., cholecystitis, gastritis, hepatitis), or eating disorders (e.g., anorexia nervosa, bulimia nervosa).
- Disorders such as pancreatitis and cirrhosis affect the ability to absorb and metabolize nutrients consumed, impairing nutritional status.
- Excess intake of nutrients, particularly when combined with decreased energy expenditure, can result in nutritional disorders (e.g., obesity, alcoholic cirrhosis).

Nutrients are used by the body to promote growth, maintenance, and repair. Categories of nutrients include proteins, carbohydrates, fats, vitamins, and minerals. Improper intake of these nutrients affects the body's ability to maintain itself, leading to manifestations such as:

- Nausea (unpleasant, wave-like sensations in the stomach ▶ feelings of urge or need to vomit)
- Heartburn (backflow of gastric contents into esophagus ▶ high acidity of stomach contents causes inflammatory response ▶ pain and irritation of mucosa lining the esophagus)
- Pain (viral or bacterial infection, tissue inflammation, obstruction ▶ tissue damage, ischemia, and/or edema ▶ stimulate pain receptors ▶ transmit pain impulses to the brain).

Priority nursing diagnoses within the Nutritional-Metabolic Pattern that may be appropriate for clients with nutritional disorders or disorders of the upper gastrointestinal tract and accessory organs include:

- *Imbalanced Nutrition: Less than Body Requirements* as evidenced by decreased food intake, weight loss 20% or more of ideal body weight, dry and brittle hair, weakness, impaired tissue healing
- *Deficient Fluid Volume* as evidenced by dry mucous membranes, poor skin turgor, thirst, increased body temperature
- *Nausea* as evidenced by complaints of stomach discomfort, increased salivation, tachycardia, and cold and clammy skin
- *Impaired Skin Integrity* as evidenced by disruption of skin surface, pain, itching.

Two nursing diagnoses from other functional health patterns often are of high priority for the client with gastrointestinal disorders because the physiologic responses to these problems interfere with proper nutrition:

- *Acute Pain* (Cognitive-Perceptual)
- *Diarrhea* (Elimination)

Directions: Read the clinical scenario below and answer the questions that follow. To complete this exercise successfully, you will use not only knowledge of the content in this unit, but also principles related to setting priorities and maintaining client safety.

CLINICAL SCENARIO

You have been assigned to work with the following four patients for the 0700 shift. Significant data obtained during report are as follows:

- Thomas Jones, age 56, was transferred to your unit yesterday after treatment in the ICU for esophageal varices. Significant history includes alcohol consumption (6 to 12 beers or a pint of liquor daily for several years) and smoking (2 packs per day for the past 30 years). Current vital signs are T 100°F, P 96, R 28, BP 150/90. He complains of abdominal tenderness and dyspnea. He appears anxious and irritable.
- Ruth Green, age 35, was admitted with right upper quadrant pain radiating to the left shoulder and a feeling of abdominal fullness. She has a history of cholelithiasis and cholecystitis. Her assessment reveals T 99.6°F, P 90, R 24, BP 140/84, with pallor, diaphoresis, and complaints of nausea. She is scheduled for a cholecystectomy at 9:00 A.M.
- Tonya Cooper, age 21, was admitted with dehydration, weakness, fainting. Her weight is 90 lb (40.9 kg) and height is 5'5". Her vital signs are T 97°F, P 70, R 26, BP 90/56 mmHg with orthostatic BP 70/48 mmHg. She has a 3-year history of anorexia nervosa and laxative use. She has an IV of 0.9% NaCl with KCl infusing. She is to be monitored for food intake and watched for 1 hour after meals. She is ringing her call light to get up to the bathroom.
- Joseph Brown, age 82, was admitted 1 hour ago with acute gastritis. He relates a 2-day history of nausea, vomiting, and diarrhea. He has a history of hypertension and angina pectoris. Current vital signs are T 101.6°F, P 102, R 20, BP 130/70. He is pale and listless. His abdomen is distended and tender with decreased bowel sounds. He is requesting medication for nausea.

Questions

1 In what order would you visit these patients after report?

1. _____
2. _____
3. _____
4. _____

2 What top two priority nursing diagnoses would you choose for each of the clients presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Thomas Jones		
Ruth Green		
Tonya Cooper		
Joseph Brown		

3 You need to complete preoperative preparation on Mrs. Green. Which of the following do you need to do?

1. Complete preoperative checklist, witness signed consent, and administer preoperative medication when requested.
2. Explain the procedure, obtain informed consent, and complete the preoperative checklist.
3. Sign the operative consent, explain complications of the procedure, and take vital signs on call.
4. Obtain signed consent, discuss with the family the surgical procedure, and have the client void prior to going to the OR.

4 Mrs. Green understands the postoperative teaching done by the nurse when she states:

1. "I will be on bed rest for two days after surgery."
2. "I will need to cough and deep breathe while splinting my incision."
3. "I will be able to begin eating when I return from surgery."
4. "I will be medicated for pain without having to request it."

5 The nurse explains a diet of low-fat foods to Mrs. Green. She understands this diet when she picks which meal plan?

1. eggs, sausage, and toast
2. turkey, mashed potatoes and gravy, and corn
3. grilled chicken, tossed salad, peaches
4. hamburger with lettuce and tomato, french fries

6 The priority nursing diagnosis for Mr. Brown with acute gastritis is:

1. *Sleep Disturbance*
2. *Acute Pain*
3. *Nausea*
4. *Imbalanced Nutrition: Less than Body Requirements*

7 Which assessment findings on Mr. Brown would be suggestive of dehydration?

1. hypertension, bradycardia, skin taut and dry
2. hypotension, tachycardia, poor skin turgor, dry mucous membranes
3. decreased body temperature, thirst, weakness
4. increased body temperature, slow thready pulse, listless

8 Mr. Brown voices understanding of how to take lansoprazole (Prevacid) after discharge when he states:

1. "I will take it after breakfast to protect my stomach."
2. "I will take it with grapefruit juice at breakfast."
3. "I can stop taking it when my nausea is gone."
4. "I will take it before breakfast to help my stomach feel better."

9 Which of the following laboratory results would be expected findings for Mr. Jones? (Select all that apply.)

1. AST 123 U/L
2. cardiac troponin T 0.8 mcg/L
3. ALP 431 U/L
4. Hct 47%
5. LDH 250 U/L
6. ALT 45 U/L

10 To prepare Mr. Jones for an esophagoscopy, the nurse institutes the following interventions:

1. Explain that it is not a painful procedure but he will be medicated for pain.
2. Keep the client NPO for 12 hours prior to the procedure.
3. Remove dentures and provide mouth care.
4. Place in a supine position with the head slightly hyperextended.

11 With Ms. Cooper's history of anorexia for 3 years, which is a priority nursing intervention in the plan of care for her?

1. Monitor for cardiac dysrhythmias due to electrolyte imbalances.
2. Monitor weight for loss or gain to determine effectiveness of nursing care.
3. Maintain close observation for at least 1 hour after meals.
4. Serve small, frequent meals, increasing serving size gradually.

12 In planning discharge for Ms. Cooper, the family and client participate in teaching and diet counseling sessions. Which is the priority item for the family and Ms. Cooper to follow after discharge?

1. Monitor weight regularly to determine further weight loss.
2. Use rewards for food and caloric intake rather than weight gain.
3. Gradually increase the amount of food taken at meals.
4. Attend support groups for people with eating disorders.

CASE STUDY

Louis Haches, an 82-year-old white male, has a history of hypertension, degenerative arthritis, and angina pectoris for which he takes furosemide, atenolol, extra-strength aspirin, and Nitrostat. He states that his blood pressure is controlled when he takes his medications as prescribed. Lately, he has been having some financial difficulties and does not take his medications as often as he should. He lives with his 80-year-old wife, who also has health problems. He does the cooking and grocery shopping. He has a son who lives nearby who looks in on them every couple of days.

Mr. Haches feels that he cooked some outdated food that made him ill. He states he has been trying to stretch their grocery budget by reducing their serving sizes and shopping less frequently. He says he has had nausea and loose, dark stools for the past 2 days. This morning, he is weak and dizzy, and states he nearly fainted in the shower. He also states he has not been able to take his medications for the past 2 days and feels that his heart is beating too fast. His son brought him to the hospital because he was weak, confused, and very pale when he checked on him prior to going to work in the morning.

Based on Mr. Haches's medical diagnosis and treatment plan, *Imbalanced Nutrition: Less than Body Requirements* is identified as the priority nursing diagnosis at this time.