Management of Patients with Hepatic Disorders

Learning Objectives

- Identify the metabolic functions of the liver and the alterations in these functions that occur with liver disease.
- Explain liver function tests and the clinical manifestations of liver dysfunction in relation to pathophysiologic alterations of the liver.
- Relate jaundice, portal hypertension, ascites, varices, nutritional deficiencies, and hepatic coma to pathophysiologic alterations of the liver.
- Describe the medical, surgical, and nursing management of patients with esophageal varices.
- Compare the various types of hepatitis and their causes, prevention, clinical manifestations, management, prognosis, and home health care needs.
- Use the nursing process as a framework for care of the patient with cirrhosis of the liver.
- Compare the nonsurgical and surgical management of patients with cancer of the liver.
- Describe the postoperative nursing care of the patient undergoing liver transplantation.

Anatomic and Physiologic Overview

*Figure 39–1* The liver and biliary system.
• The liver, the largest gland of the body (1.4-1.8kg), can be considered a chemical factory that manufactures, stores, alters, and excretes a large number of substances involved in metabolism.

• The location of the liver is essential in this function, because it receives nutrient-rich blood directly from the gastrointestinal (GI) tract and then either stores or transforms these nutrients into chemicals that are used elsewhere in the body for metabolic needs.

• The liver is especially important in the regulation of glucose and protein metabolism.

• The liver manufactures and secretes bile, which has a major role in the digestion and absorption of fats in the GI tract. It removes waste products from the bloodstream and secretes them into the bile.

• The bile produced by the liver is stored temporarily in the gallbladder until it is needed for digestion, at which time the gallbladder empties and bile enters the intestine.

ANATOMY OF THE LIVER

• The liver is located behind the ribs in the upper right portion of the abdominal cavity. It weighs about 1,500 g and is divided into four lobes.

• A thin layer of connective tissue surrounds each lobe, extending into the lobe itself and dividing the liver mass into small units called lobules.

• The circulation of the blood into and out of the liver is of major importance in its function. The blood that perfuses the liver comes from two sources. Approximately 75% of the blood supply comes from the portal vein, which drains the GI tract and is rich in nutrients. The remainder of the blood supply enters by way of the hepatic artery and is rich in oxygen. Terminal branches of these two blood supplies join to form common capillary beds, which constitute the sinusoids of the liver.

• The sinusoids empty into a venule that occupies the center of each liver lobule and is called the central vein. The central veins join to form the hepatic vein, which constitutes the venous drainage from the liver and empties into the inferior vena cava, close to the diaphragm. Thus, there are two sources of blood flowing into the liver and only one exit pathway.

• In addition to hepatocytes, phagocytic cells belonging to reticuloendothelial system are present in the liver. Other organs that contain reticuloendothelial cells are the spleen, bone marrow, lymph nodes, and lungs. In the liver, these cells are called Kupffer cells. Their main function is to engulf particulate matter (such as bacteria) that enters the liver through the portal blood.
The smallest bile ducts, called canaliculi, are located between the lobules of the liver. The canaliculi receive secretions from the hepatocytes and carry them to larger bile ducts, which eventually form the hepatic duct. The hepatic duct from the liver and the cystic duct from the gallbladder join to form the common bile duct, which empties into the small intestine. The sphincter of Oddi, located at the junction where the common bile duct enters the duodenum, controls the flow of bile into the intestine.

FUNCTIONS OF THE LIVER

1. Glucose Metabolism
   • After a meal, glucose is taken up from the portal venous blood by the liver and converted into glycogen, which is stored in the hepatocytes.
   • Subsequently, the glycogen is converted back to glucose and released as needed into the bloodstream to maintain normal levels of blood glucose.
   • Additional glucose can be synthesized by the liver through a process called gluconeogenesis. For this process, the liver uses amino acids from protein breakdown or lactate produced by exercising muscles.

2. Ammonia Conversion
   • Use of amino acids from protein for gluconeogenesis results in the formation of ammonia as a byproduct. The liver converts this metabolically generated ammonia into urea.
   • Ammonia produced by bacteria in the intestines is also removed from portal blood for urea synthesis. In this way, the liver converts ammonia, a potential toxin, into urea, a compound that can be excreted in the urine.

3. Protein Metabolism
   • The liver synthesizes almost all of the plasma proteins (except gamma globulin), including albumin, alpha and beta globulins, blood clotting factors, specific transport proteins, and most of the plasma lipoproteins.
   • Amino acids serve as the building blocks for protein synthesis.
   • Vitamin K is required by the liver for synthesis of prothrombin and some of the other clotting factors.

4. Fat Metabolism
   • Fatty acids can be broken down for the production of energy and the production of ketone bodies.
   • Ketone bodies are small compounds that can enter the bloodstream and provide a source of energy for muscles and other tissues.
   • Breakdown of fatty acids into ketone bodies occurs primarily when the availability of glucose for metabolism is limited, as during starvation or in uncontrolled diabetes.
   • Fatty acids and their metabolic products are also used for the synthesis of cholesterol, lecithin, lipoproteins, and other complex lipids.

5. Vitamin and Iron Storage
   • Vitamins A, B, and D and several of the B-complex vitamins are stored in large amounts in the liver.
   • Iron and copper, are also stored in the liver.
   • Because the liver is rich in these substances, liver extracts have been used for therapy for a wide range of nutritional disorders.

6. Drug Metabolism
   • The liver metabolizes many medications.
   • Metabolism generally results in loss of activity of the medication, although in some cases activation of the medication may occur.
• One of the important pathways for medication metabolism involves conjugation (binding) of the medication with a variety of compounds, such as glucuronic or acetic acid, to form more soluble substances.
• The conjugated products may be excreted in the feces or urine, similar to bilirubin excretion.

8. **Bile Formation**
• Bile is continuously formed by the hepatocytes and collected in the canaliculi and bile ducts.
• It is composed mainly of water and electrolytes such as sodium, potassium, calcium, chloride, and bicarbonate, and significant amounts of lecithin, fatty acids, cholesterol, bilirubin, and bile salts.
• Bile is collected and stored in the gallbladder and is emptied into the intestine when needed for digestion.
• Bile also serves as an aid to digestion through the emulsification of fats by bile salts.
• Bile salts are synthesized by the hepatocytes from cholesterol. After conjugation or binding with amino acids, they are excreted into the bile.
• The bile salts, together with cholesterol and lecithin, are required for emulsification of fats in the intestine, which is necessary for efficient digestion and absorption.
• Bile salts are then reabsorbed, primarily in the distal ileum, into portal blood for return to the liver and are again excreted into the bile.

9. **Bilirubin Excretion**
• Bilirubin is a pigment derived from the breakdown of hemoglobin by cells of the reticuloendothelial system.
• Hepatocytes remove bilirubin from the blood and modify it to be more soluble in aqueous solutions.
• The conjugated bilirubin is secreted by the hepatocytes into the adjacent bile canaliculi and is eventually carried in the bile into the duodenum.
• In the small intestine, bilirubin is converted into urobilinogen, which is in part excreted in the feces and in part absorbed through the intestinal mucosa into the portal blood.
• Some of the urobilinogen enters the systemic circulation and is excreted by the kidneys in the urine.

**Gerontologic Considerations**
• The most common change in the liver in the elderly is a decrease in its size and weight, accompanied by a decrease in total hepatic blood flow. Results of liver function tests do not normally change in the elderly; abnormal results in an elderly patient indicate abnormal liver function and are not the result of the aging process itself.
• The immune system is altered in the aged, and a less responsive immune system may be responsible for the increased incidence and severity of hepatitis B in the elderly and the increased incidence of liver abscesses secondary to decreased phagocytosis by the Kupffer cells.

**Age-Related Changes of the Hepatobiliary System**
• Steady decrease in size and weight of the liver, particularly in women.
• Decrease in blood flow.
• Decrease in replacement/repair of liver cells after injury.
• Reduced drug metabolism.
• Rapid progression of hepatitis C infection and lower response rate to therapy.
• Decline in drug clearance capability.
• Increased prevalence of gallstones.
• Decreased gallbladder contraction after a meal.
• More severe complications of biliary tract disease.
ASSESSMENT HEALTH HISTORY

• If liver function test results are abnormal, the patient may need to be evaluated for liver disease. So look if the client:
  • Was exposed to hepatotoxic substances or infectious agents.
  • Patient’s occupational, recreational, and travel history may assist in identifying exposure to hepatotoxins.
  • Patient’s history of alcohol and drug use.
  • Lifestyle behaviors (Injectable drug use, sexual practices).
  • Current and past medical conditions, previous blood transfusion.

PHYSICAL EXAMINATION

• Assess the patient for pallor, jaundice (skin, mucosa, and sclerae), and the extremities are assessed for muscle atrophy, edema, and skin excoriation secondary to scratching.
• Observe the skin for petechiae or ecchymotic areas (bruises), spider angiomas, and palmar erythema.
• Assess male patient for unilateral or bilateral gynecomastia and testicular atrophy due to endocrine changes.
• Assess patient’s cognitive status (recall, memory, abstract thinking) and neurologic status are assessed.
• Palpate abdomen to assess liver size and to detect any tenderness over the liver. A palpable liver presents as a firm, sharp edge with a smooth surface.
• Tenderness of the liver implies recent acute enlargement with consequent stretching of the liver capsule.
• Enlargement of the liver is an abnormal finding requiring evaluation.

FIGURE 39-3: Technique for palpating the liver. The examiner places one hand under the right lower rib cage and presses downward with light pressure with the other hand.

Diagnostic Evaluation

LIVER FUNCTION TESTS

• More than 70% of the parenchyma of the liver may be damaged before liver function test results become abnormal.
  1. Serum enzyme activity (ie, alkaline phosphatase, lactic dehydrogenase, serum aminotransferases)
  2. Serum concentrations of proteins (albumin and globulins)
  3. Bilirubin, ammonia, clotting factors, and lipids.
4. Serum aminotransferases (also called transaminases) are sensitive indicators of injury to the liver cells and are useful in detecting acute liver disease such as hepatitis.

   A. Alanine aminotransferase (ALT) (formerly called serum glutamic-pyruvic transaminase [SGPT]) (10-40 U/L)
   B. Aspartate aminotransferase (AST) (formerly called serum glutamic-oxaloacetic transaminase [SGOT]) (5-35 U/L)
   C. Gamma glutamyl transferase (GGT) (also called G-glutamyl transpeptidase) (10-48 U/L)
   D. Lactic Dehydrogenase (LDH) (100-200 U/L)

These studies measure the ability of the liver to conjugate and excrete bilirubin. Results are abnormal in liver and biliary tract disease and are associated with jaundice clinically.

<table>
<thead>
<tr>
<th>Pigment Studies</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Serum bilirubin, direct</td>
<td>1. 0–0.3 mg/dL (0–5.1 µmol/L)</td>
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<tr>
<td>2. Serum bilirubin, total</td>
<td>2. 0–0.9 mg/dL (1.7–20.3 µmol/L)</td>
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<tr>
<td>3. Urine bilirubin</td>
<td>3. 0(0)</td>
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<tr>
<td>4. Urine urobilinogen</td>
<td>4. 0.05–2.5 mg/24 h (0.09–4.23 µmol/24 h)</td>
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<tr>
<td>5. Fecal urobilinogen (infrequently used)</td>
<td>5. 40–200 mg/24 h (0.068–0.34 mmol/24 h)</td>
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</table>

CLINICAL FUNCTIONS
Proteins are manufactured by the liver. Their levels may be affected in a variety of liver impairments

- **Albumin:** Cirrhosis
  - Chronic hepatitis
  - Edema, ascites

- **Globulin:** Cirrhosis
  - Liver disease
  - Chronic obstructive jaundice
  - Viral hepatitis

<table>
<thead>
<tr>
<th>Protein Studies</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Total serum protein</td>
<td>7.0–7.5 g/dL (70–75 g/L)</td>
</tr>
<tr>
<td>• Serum albumin</td>
<td>4.0–5.5 g/dL (40–55 g/L)</td>
</tr>
<tr>
<td>• Serum globulin</td>
<td>1.7–3.3 g/dL (17–33 g/L)</td>
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</table>

**Serum protein electrophoresis**

- **Albumin**
  - 4.0–5.5 g/dL (40–55 g/L)
- **α1-Globulin**
  - 0.15–0.25 g/dL (1.5–2.5 g/L)
- **α2-Globulin**
  - 0.43–0.75 g/dL (4.3–7.5 g/L)
- **β-Globulin**
  - 0.5–1.0 g/dL (5–10 g/L)
- **γ-Globulin**
  - 0.6–1.3 g/dL (6–13 g/L)
- **Albumin/globulin (A/G) ratio**
  - A > G or 1.5:1–2.5:

**Serum Aminotransferase or Transaminase Studies**

- **AST (SGOT)**
  - 10–40 units (4.8–19 U/L)
- **ALT (SGPT)**
  - 5–35 units (2.4–17 U/L)
- **GGT, GGTP**
  - 10–48 IU/L
- **LDH**
  - 100–200 units (100–225 U/L)

The studies are based on release of enzymes from damaged liver cells. These enzymes are elevated in liver cell damage. Elevated in alcohol abuse. Marker for biliary cholestasis.
**Cholesterol**

- Ester
- HDL (high-density lipoprotein)
- LDL (low-density lipoprotein)

| 60% of total (fraction of total cholesterol: 0.60) |
| HDL Male: 35–70 mg/dL, Female: 35–85 mg/dL |
| LDL < 130 µg/dL |

Cholesterol levels are elevated in biliary obstruction and decreased in parenchymal liver disease

<table>
<thead>
<tr>
<th><strong>ADDITIONAL STUDIES</strong></th>
<th><strong>CLINICAL FUNCTIONS</strong></th>
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<tbody>
<tr>
<td>Barium study of esophagus</td>
<td></td>
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<td>Abdominal x-ray</td>
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<tr>
<td>Liver scan with radiotagged iodinated rose bengal, gold, technetium, or gallium</td>
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<tr>
<td>Liver biopsy (percutaneous or transjugular)</td>
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<tr>
<td>Ultrasonography</td>
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<tr>
<td>Computed tomography (CT scan)</td>
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<td>For varices, which indicate increased portal blood pressure</td>
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<tr>
<td>To determine gross liver size</td>
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<tr>
<td>To show size and shape of liver; to show replacement of liver tissue with scars, cysts, or tumor.</td>
<td></td>
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<tr>
<td>To determine anatomic changes in liver tissue</td>
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<tr>
<td>To show size of abdominal organs and presence of masses</td>
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<tr>
<td>To detect hepatic neoplasms; diagnose cysts, abscesses, and hematomas; and distinguish between obstructive and nonobstructive jaundice. Detects cerebral atrophy in hepatic encephalopathy.</td>
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**LIVER BIOPSY**

![Liver biopsy image]
Liver biopsy is the removal of a small amount of liver tissue, usually through needle aspiration. It permits examination of liver cells. Liver biopsy is especially useful when clinical findings and laboratory tests are not diagnostic.

- Bleeding and bile peritonitis after liver biopsy are the major complications.

### NURSING ACTIVITIES

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<th>RATIONALE</th>
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<td>- Ascertain that results of coagulation tests (prothrombin time, partial thromboplastin time, and platelet count) are available a) and that compatible donor blood is available.</td>
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<td>- Check for signed consent; confirm that informed consent has been provided.</td>
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<tr>
<td>- Measure and record the patient’s pulse, respirations, and blood pressure immediately before biopsy.</td>
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<tr>
<td>- Describe to the patient in advance: steps of the procedure;</td>
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<tr>
<td>- Support the patient during the procedure.</td>
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<td>- Expose the right side of the patient’s upper abdomen (right hypochondriac).</td>
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<td>- Instruct the patient to inhale and exhale deeply several times, finally to exhale, and to hold breath at the end of expiration. The physician promptly introduces the biopsy needle by way of the transthoracic (intercostal) transabdominal (subcostal) route, penetrates the liver, aspirates, and withdraws.</td>
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<tr>
<td>- Many patients with liver disease have clotting defects and are at risk for bleeding.</td>
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<tr>
<td>- 2. Procedure should be done with agreement of patient</td>
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<tr>
<td>- Pre-biopsy values provide a basis on which to compare the patient’s vital signs and evaluate status after the procedure.</td>
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<tr>
<td>- Explanations allay fears and ensure cooperation.</td>
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<td>- Encouragement and support of the nurse enhance comfort and promote a sense of security.</td>
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<td>- The skin at the site of penetration will be cleansed and a local anesthetic will be infiltrated.</td>
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<tr>
<td>- Holding the breath immobilizes the chest wall and the diaphragm; penetration of the diaphragm thereby is avoided, and the risk of lacerating the liver is minimized.</td>
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</tbody>
</table>

### POSTPROCEDURE

- Immediately after the biopsy, assist the patient to turn onto the right side; place a pillow under the costal margin, and caution the patient to remain in this position and immobile, for several hours. Instruct the patient to avoid coughing or straining.
- Measure and record the patient’s vital signs at a15-minute intervals for the first hour, then every 30 minutes for the next 1 to 2 hours or until the patient’s condition stabilizes.
- **Complications**
  - Pneumothorax
  - Peritonitis
  - Hemorrhage
OTHER DIAGNOSTIC TESTS
- Ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) are used to identify normal structures and abnormalities of the liver and biliary tree.
- A radioisotope liver scan may be performed to assess liver size and hepatic blood flow and obstruction.
- Laparoscopy (insertion of a fiber-optic endoscope through a small abdominal incision) is used to examine the liver and other pelvic structures.

Hepatic Dysfunction
- Results from damage to the liver’s parenchymal cells, either directly from primary liver diseases or indirectly from obstruction of bile flow.
- Liver dysfunction may be acute or chronic; chronic dysfunction is far more common than acute.
- The most common cause of parenchymal damage is malnutrition, especially that related to alcoholism. The parenchymal cells respond to most noxious agents by replacing glycogen with lipids, producing fatty infiltration with or without cell death or necrosis.
- This is commonly associated with inflammatory cell infiltration and growth of fibrous tissue.

Among the most common and significant symptoms of liver disease are the following:
1. Jaundice, resulting from increased bilirubin concentration in the blood
2. Portal hypertension, ascites, and varices, resulting from circulatory changes within the diseased liver and producing severe GI hemorrhages and marked sodium and fluid retention
3. Nutritional deficiencies, which result from the inability of the damaged liver cells to metabolize certain vitamins;
4. Hepatic encephalopathy or coma, reflecting accumulation of ammonia in the serum due to impaired protein metabolism by the diseased liver

I. JAUNDICE
When the bilirubin concentration in the blood is abnormally elevated, all the body tissues, including the sclerae and the skin, become yellow-tinged or greenish-yellow, a condition called jaundice.

Becomes clinically evident when the serum bilirubin level exceeds 2.5 mg/dL.

There are several types of jaundice:
- Hemolytic
- Hepatocellular
- Obstructive

1. Hemolytic Jaundice
- Results from increased destruction of the red blood cells, too much bilirubin reaches the liver, although functioning normally, cannot excrete the bilirubin as quickly as it is formed.
- Occurs with patients with hemolytic transfusion reactions and other hemolytic disorders.
- Prolonged jaundice, however, even if mild, predisposes to the formation of pigment stones in the gallbladder, and extremely severe jaundice (levels of free bilirubin exceeding 20 to 25 mg/dL) poses a risk for brain stem damage.

2. Hepatocellular Jaundice
- Caused by the inability of damaged liver cells to clear normal amounts of bilirubin from the blood. The cellular damage may be from infection, such as in viral hepatitis or other viruses that affect the liver (e.g., yellow fever virus, Epstein-Barr virus), from medication or chemical toxicity (e.g., carbon tetrachloride, chloroform, phosphorus, certain medications), or from alcohol.
- Cirrhosis of the liver is a form of hepatocellular disease that may produce jaundice. It is usually associated with excessive alcohol.

3. Obstructive Jaundice
- Caused by occlusion of the bile duct by a gallstone, an inflammatory process, a tumor, or pressure from an enlarged organ.
- The obstruction may also involve the small bile ducts within the liver (i.e., intrahepatic obstruction), caused, for example, by pressure on these channels from inflammatory swelling of the liver or by an inflammatory exudate within the ducts themselves. Intrahepatic obstruction resulting from stasis and inspissation (thickening) of bile within the canaliculi may occur after the ingestion of certain medications.
- These include phenothiazines, antithyroid medications, sulfonylureas, tricyclic antidepressant agents, nitrofurantoin, androgens, and estrogens.
- Whether the obstruction is intrahepatic or extrahepatic, and whatever its cause may be, bile cannot flow normally into the intestine but is backed up into the liver substance. It is then reabsorbed into the blood and carried throughout the entire body, staining the skin, mucous membranes, and sclerae. It is excreted in the urine, which becomes deep orange and foamy.
- Because of the decreased amount of bile in the intestinal tract, the stools become light or clay-colored. The skin may itch intensely, requiring repeated soothing baths. Dyspepsia and intolerance to fatty foods may develop because of impaired fat digestion in the absence of intestinal bile. AST, ALT, and GGT levels generally rise only moderately, but bilirubin and alkaline phosphatase levels are elevated.
4. Hereditary Hyperbilirubinemia
- Results from several inherited disorders can also produce jaundice. Gilbert’s syndrome is a familial disorder characterized by an increased level of unconjugated bilirubin that causes jaundice.
- Other conditions that are probably caused by inborn errors of biliary metabolism include Dubin–Johnson syndrome (chronic idiopathic jaundice, with pigment in the liver) and Rotor’s syndrome (chronic familial conjugated hyperbilirubinemia without pigment in the liver).

II. PORTAL HYPERTENSION
- Obstructed blood flow through the damaged liver results in increased blood pressure (portal hypertension) throughout the portal venous system.
- It is commonly associated with hepatic cirrhosis, but can also occur with noncirrhotic liver disease.
- Portal hypertension leads to:
  - Splenomegaly (enlarged spleen)
  - Ascites
  - Varices

III. ASCITES - Pathophysiology
- Caused by portal hypertension and the resulting increase in capillary pressure and obstruction of venous blood flow through the damaged liver.
- The failure of the liver to metabolize aldosterone increases sodium and water retention by the kidney. Sodium and water retention, increased intravascular fluid volume, and decreased synthesis of albumin by the damaged liver all contribute to fluid moving from the vascular system into the peritoneal space.
- Loss of fluid into the peritoneal space causes further sodium and water retention by the kidney in an effort to maintain the vascular fluid volume, and the process becomes self-perpetuating.
- As a result of liver damage, large amounts of albumin-rich fluid, 15 L or more, may accumulate in the peritoneal cavity as ascites.
- With the movement of albumin from the serum to the peritoneal cavity, the osmotic pressure of the serum decreases.
- This, combined with increased portal pressure, results in movement of fluid into the peritoneal cavity.

Clinical Manifestations
- Increased abdominal girth and rapid weight gain are common presenting symptoms of ascites.
- The patient may be short of breath and uncomfortable from the enlarged abdomen, and
- Striae and distended veins may be visible over the abdominal wall.
- Fluid and electrolyte imbalances are common.
Assessment and Diagnostic Evaluation
- The presence and extent of ascites are assessed by percussion of the abdomen. When fluid has accumulated in the peritoneal cavity, the flanks bulge when the patient assumes a supine position. The presence of fluid can be confirmed either by percussing for shifting dullness or by detecting a fluid wave. Daily measurement and recording of abdominal girth and body weight are essential to assess the progression of ascites and its response to treatment.

Medical Management A) DIETARY MODIFICATION
- The goal of treatment for the patient with ascites is a negative sodium balance to reduce fluid retention. Table salt, salty foods, salted butter and margarine, and all ordinary canned and frozen foods should be avoided.
- In the meantime, the taste of unsalted foods can be improved by using salt substitutes such as lemon juice, oregano, and thyme.
B) DIURETICS
- Use of diuretics along with sodium restriction is successful in 90% of patients with ascites. Spironolactone (Aldactone), an aldosterone blocking agent, is most often the first-line therapy. When used with other diuretics, it helps prevent potassium loss.
- Daily weight loss should not exceed 1 to 2 kg.
- Possible complications of diuretic therapy include fluid and electrolyte disturbances (including hypovolemia, hypokalemia, hyponatremia).

C) BED REST
- In patients with ascites, an upright posture is associated with activation of the renin-angiotensin-aldosterone system and sympathetic nervous system. This results in reduced renal glomerular filtration and sodium excretion and a decreased response to loop diuretics. Bed rest may be a useful therapy, especially for patients whose condition is refractory to diuretics.

D) PARACENTESIS
- Paracentesis is the removal of fluid (ascites) from the peritoneal cavity through a small surgical incision or puncture made through the abdominal wall under sterile conditions. Ultrasound guidance may be indicated in some patients at high risk for bleeding.
- Use of large-volume (5 to 6 liters) paracentesis has been shown to be a safe method for treating patients with severe ascites. This technique, in combination with the intravenous infusion of saltpoor albumin or other colloid, salt-poor albumin helps reduce edema by causing the ascitic fluid to be drawn back into the bloodstream and ultimately excreted by the kidneys.
- Preprocedure
  1. Prepare the pt by providing the information and instructions about the procedure
  2. Instruct the patient to void.
3. Gather appropriate sterile equipment
4. Place patient in upright position on edge of bed with feet supported on stool, or place in chair. Fowler’s position should be used for the patient confined to bed.
5. Monitoring of blood pressure during the procedure

6. The physician, using aseptic technique, inserts the trocar through a puncture wound below the umbilicus. The fluid drains from the abdomen through a drainage tube into a container.
7. Help the patient maintain position throughout procedure.
8. Measure and record blood pressure frequently.
9. Monitor the patient closely for signs of vascular collapse: pallor, increased pulse rate, or decreased blood pressure.

Postprocedure
1. Return patient to bed or to a comfortable sitting position.
2. Measure, describe, and record the fluid collected.
3. Label samples of fluid and send to laboratory.
4. Continue to monitor vital signs every 15 minutes for 1 hour, every 30 minutes over 2 hours, then every hour over 2 hours and then every 4 hours. Monitor temperature after procedure and every 4 hours.
5. Assess for hypovolemia, electrolyte loss, changes in mental status, and encephalopathy.
6. Check puncture site when taking vital signs for bleeding and leakage.
7. Provide patient education

Nursing Management
- Assessment and documentation of intake and output, abdominal girth, and daily weight to assess fluid status. The nurse monitors serum ammonia and electrolyte levels to assess electrolyte balance, response to therapy, and indicators of encephalopathy.
- PROMOTING HOME AND COMMUNITY-BASED CARE
- Teaching Patients Self-Care.
- Continuing Care.
IV. ESOPHAGEAL VARICES

- Bleeding or hemorrhage from esophageal varices occurs in approximately one third of patients with cirrhosis and varices. The mortality rate resulting from the first bleeding episode is 45% to 50%; it is one of the major causes of death in patients with cirrhosis.

Clinical Manifestations

- The patient with bleeding esophageal varices may present with hematemesis, melena, or general deterioration in mental or physical status and often has a history of alcohol abuse.
- Signs and symptoms of shock (cool clammy skin, hypotension, tachycardia) may be present.
Assessment and Diagnostic Findings
- Endoscopy is used to identify the bleeding site, along with barium swallow, ultrasonography, CT, and angiography.
- Liver function tests

Medical Management
- Bleeding from esophageal varices can quickly lead to hemorrhagic shock and is an emergency. This patient is critically ill, requiring aggressive medical care and expert nursing care, and is usually transferred to the intensive care unit for close monitoring and management.

1. PHARMACOLOGIC THERAPY
- In an actively bleeding patient, medications are administered initially because they can be obtained and administered quickly; other therapies take longer to initiate. Vasopressin (Pitressin) may be the initial mode of therapy because it produces constriction of the splanchnic arterial bed and a resulting decrease in portal pressure.
- Combination of vasopressin and nitroglycerin (administered by the intravenous, sublingual, or transdermal route) has been effective in reducing or preventing the side effects (constriction of coronary vessels and angina) caused by vasopressin alone. Somatostatin and octreotide (Sandostatin) have been reported to be more effective than vasopressin in decreasing bleeding from esophageal varices

2. BALLOON TAMPONADE
- To control hemorrhage in certain patients, balloon tamponade may be used. In this procedure, pressure is exerted on the cardia (upper orifice of the stomach) and against the bleeding varices by a double-balloon tamponade. The tube has four openings, each with a specific purpose: gastric aspiration, esophageal aspiration, inflation of the gastric balloon, and inflation of the esophageal balloon. The balloon in the stomach is inflated with 100 to 200 mL of air. An x-ray confirms
proper positioning of the gastric balloon. Then the tube is pulled gently to exert a force against the gastric cardia.

Dangers and complication of tamponade balloon

- Displacement of the tube and the inflated balloon into the oropharynx can cause life-threatening obstruction of the airway and asphyxiation.
- This may occur if a patient pulls on the tube because of confusion or discomfort.
- It may also result from rupture of the gastric balloon, allowing the esophageal balloon to move into the oropharynx.
- Sudden rupture of the balloon causes airway obstruction and aspiration of gastric contents into the lungs.

3. ENDOSCOPIC SCLEROTHERAPY

- A sclerosing agent is injected through a fiberoptic endoscope into the bleeding esophageal varices to promote thrombosis and eventual sclerosis. The procedure has been used successfully to treat acute GI hemorrhage. After treatment, the patient must be observed for bleeding, perforation of the esophagus, aspiration pneumonia, and esophageal stricture. Antacids may be administered after the procedure to counteract the effects of peptic reflux.
4. **ESOPHAGEAL BANDING THERAPY (VARICEAL BAND LIGATION)**
   - A rubber band–like ligature is slipped over an esophageal varix via an endoscope.
   - (B) Necrosis results and the varix eventually sloughs off.

5. **TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNTING**
   - Transjugular intrahepatic portosystemic shunting (TIPS) is a method of treating esophageal varices in which a cannula is threaded into the portal vein by the transjugular route. An expandable stent is inserted and serves as an intrahepatic shunt between the portal circulation and the hepatic vein, reducing portal hypertension. Complications may include bleeding, sepsis, heart failure, organ perforation, shunt thrombosis, and progressive liver failure.
SURGICAL MANAGEMENT

- Surgical decompression. Surgical Bypass Procedures of the portal circulation can prevent variceal bleeding if the shunt remains patent.

- The distal splenorenal shunt made between the splenic vein and the left renal vein after splenectomy. A mesocaval shunt is created by anastomosing the superior mesenteric vein to the proximal end of the vena cava or to the side of the vena cava using grafting material.

- The goal of distal splenorenal and mesocaval shunts is to drain only a portion of venous blood from the portal bed to decrease portal pressure; thus, they are considered selective shunts.

- The liver continues to receive some portal flow, and the incidence of encephalopathy may be reduced.

- These procedures are extensive and are not always successful because of secondary thrombosis in the veins used for the shunt as well as complications (eg, encephalopathy).

- Partial portacaval shunts with interposition grafts are as effective as other shunts but are associated with a lower rate of encephalopathy.

- If the cause of portal hypertension is the rare Budd-Chiari syndrome or other venous obstructive disease, a portacaval or a mesoatrial shunt may be performed. The mesoatrial shunt is required when the infrahepatic vena cava is thrombosed and must be bypassed.

Devascularization and Transection

- Devascularization and staple gun transection procedures to separate the bleeding site from the high-pressure portal system have been used in the emergency management of variceal bleeding. The lower end of the esophagus is reached through a small gastrostomy incision; a staple gun permits anastomosis of the transected ends of the esophagus. Rebleeding is a risk, and the outcomes of these procedures vary among patient populations.
Nursing Management

- Monitoring the patient’s physical condition and evaluating emotional responses and cognitive status.
- Monitor and record vital signs.
- Assess the patient’s nutritional and neurologic status.
- This assessment will assist in identifying hepatic encephalopathy resulting from the breakdown of blood in the GI tract and a rising serum ammonia level. Manifestations range from drowsiness to encephalopathy and coma.
- Complete rest of the esophagus may be indicated with bleeding, so parenteral nutrition is initiated. Gastric suction usually
- Vitamin K therapy and multiple blood transfusions often are indicated because of blood loss.
- A quiet environment and calm reassurance may help to relieve the patient’s anxiety.
**Hepatic Encephalopathy and Coma**

- Is a life-threatening complication of liver disease, occurs with profound liver failure and may result from the accumulation of ammonia and other toxic metabolites in the blood.
- Represents the most advanced stage of hepatic encephalopathy.

### Table 39-2: Management Modalities and Nursing Care for the Patient With Bleeding Esophageal Varices

<table>
<thead>
<tr>
<th>TREATMENT MODALITY*</th>
<th>ACTION</th>
<th>NURSING PRIORITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nonsurgical Modalities</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pharmacologic agents</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vasopressin (Pitressin)</td>
<td>Reduces portal pressure by constricting splanchnic arteries</td>
<td>Observe response to therapy. Monitor for side effects: vasopressin—angina; nitroglycerin may be prescribed to prevent or treat angina. Propranolol and nadolol—decreased pulse pressure, impaired cardiovascular response to hemorrhage.</td>
</tr>
<tr>
<td>Propranolol (Inderal)/nadolol (Corgard)</td>
<td>Reduces portal pressure by β-adrenergic blocking action</td>
<td></td>
</tr>
<tr>
<td>Somatostatin/octreotide (Sandostatin)</td>
<td>Reduces portal pressure by selective vasodilation of portal system</td>
<td>Support patient during treatment. Explain procedure to patient briefly to obtain cooperation with insertion and maintenance of esophageal tamponade tube and reduce patient’s fear of the procedure. Monitor closely to prevent inadvertent removal or displacement of tube, subsequent airway obstruction, and aspiration.</td>
</tr>
<tr>
<td>Balloon tamponade</td>
<td>Exerts pressure directly to bleeding sites in esophagus and stomach</td>
<td></td>
</tr>
<tr>
<td><strong>Injection sclerotherapy</strong></td>
<td>Promotes thrombosis and sclerosing of bleeding sites by injection of sclerosing agent into the esophageal varices</td>
<td>Observe for aspiration, perforation of the esophagus, and recurrence of bleeding after treatment.</td>
</tr>
<tr>
<td><strong>Variceal banding</strong></td>
<td>Provides thrombosis and mucosal necrosis of bleeding sites by band ligation</td>
<td>Observe for recurrence of bleeding, esophageal perforation.</td>
</tr>
<tr>
<td><strong>Transjugular intrahepatic portosystemic shunting (TIPS)</strong></td>
<td>Reduces portal pressure by creating a shunt within the liver between the portal and systemic venous system.</td>
<td>Observe for rebleeding and signs of infection.</td>
</tr>
<tr>
<td><strong>Surgical Modalities</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Portal-systemic shunting</td>
<td>Reduces portal hypertension by diverting blood flow away from obstructed portal system</td>
<td>Observe for development of portal-systemic encephalopathy (altered mental status, neurologic dysfunction), hepatic failure, and rebleeding.</td>
</tr>
</tbody>
</table>
Pathophysiology

- Ammonia accumulates because damaged liver cells fail to detoxify and convert the ammonia that is constantly entering the bloodstream to urea.
- Ammonia enters the bloodstream as a result of its absorption from the GI tract and its liberation from kidney and muscle cells.
- The increased ammonia concentration in the blood causes brain dysfunction and damage, resulting in hepatic encephalopathy.
- The largest source of ammonia is the enzymatic and bacterial digestion of dietary and blood proteins in the GI tract. Ammonia from these sources is increased as a result of GI bleeding (i.e., bleeding esophageal varices or chronic GI bleeding), a high-protein diet, bacterial infections, and uremia. The ingestion of ammonium salts also increases the blood ammonia level.
- Conversely, serum ammonia is decreased by elimination of protein from the diet and by the administration of antibiotic agents, such as neomycin sulfate, that reduce the number of intestinal bacteria capable of converting urea to ammonia.

Stages of Hepatic Encephalopathy

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical Symptoms</th>
<th>Clinical Signs and EEG Changes</th>
<th>Selected Potential Nursing Diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal level of consciousness with periods of lethargy and euphoria; reversal of day–night sleep patterns</td>
<td>Asterixis; impaired writing and ability to draw line figures. Normal EEG.</td>
<td>Activity intolerance</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Self-care deficit</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Disturbed sleep pattern</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Impaired social interaction</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Ineffective role performance</td>
</tr>
<tr>
<td>2</td>
<td>Increased drowsiness; disorientation; inappropriate behavior; mood swings; agitation</td>
<td>Asterixis; fetor hepaticus. Abnormal EEG with generalized slowing.</td>
<td>Risk for injury</td>
</tr>
<tr>
<td>3</td>
<td>Stuporous; difficult to rouse; sleeps most of the time; marked confusion; incoherent speech</td>
<td>Asterixis; increased deep tendon reflexes; rigidity of extremities. EEG markedly abnormal.</td>
<td>Imbalanced nutrition</td>
</tr>
<tr>
<td>4</td>
<td>Comatose; may not respond to painful stimuli</td>
<td>Absence of asterixis; absence of deep tendon reflexes; flaccidity of extremities. EEG markedly abnormal.</td>
<td>Impaired mobility</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Impaired verbal communication</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Risk for aspiration</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Impaired gas exchange</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Disturbed sensory perception</td>
</tr>
</tbody>
</table>

*Nursing diagnoses are likely to progress, so that most nursing diagnoses present at earlier stages will occur during later stages as well.*
Clinical Manifestations

- The earliest symptoms of hepatic encephalopathy include minor mental changes and motor disturbances. The patient appears slightly confused, has alterations in mood, becomes unkempt, and has altered sleep patterns. The patient tends to sleep during the day and have restlessness and insomnia at night. As hepatic encephalopathy progresses, the patient may be difficult to awaken.
- Asterixis (flapping tremor of the hands) may occur

- Simple tasks, such as handwriting, become difficult. A handwriting or drawing sample (eg, star figure), taken daily, may provide graphic evidence of progression or reversal of hepatic encephalopathy. Inability to reproduce a simple figure is referred to as constructional apraxia.
- In the early stages of hepatic encephalopathy, the deep tendon reflexes are hyperactive; with worsening of hepatic encephalopathy, these reflexes disappear and the extremities may become flaccid.

Assessment and Diagnostic Findings

- (EEG) shows generalized slowing, an increase in the amplitude of brain waves.
- Fetor hepaticus, a sweet, slightly fecal odor to the breath presumed to be of intestinal origin may be noticed. The odor has also been described as similar to that of freshly mowed grass, acetone, or old wine.
- With further progression of the disorder, the patient lapses into frank coma and may have seizures. Approximately 35% of all patients with cirrhosis of the liver die in hepatic coma.

Medical Management

- Lactulose (Cephulac) is administered to reduce serum ammonia levels. It acts by several mechanisms that promote the excretion of ammonia in the stool: (1) ammonia is kept in the ionized state, resulting in a fall in colon pH, reversing the normal passage of ammonia from the colon to the blood;
(2) evacuation of the bowel takes place, which decreases the ammonia to which decreases the ammonia absorbed from the colon.
(3) the fecal flora are changed to organisms that do not produce ammonia from urea.
  
  • **Monitor patient closely for the development of watery diarrheal stools, because they indicate a medication overdose.

• Intravenous administration of glucose to minimize protein breakdown, administration of vitamins to correct deficiencies, and correction of electrolyte imbalances (especially potassium). Additional principles of management of hepatic encephalopathy include the following:
  1. Therapy is directed toward treating or removing the cause.
  2. Neurologic status is assessed frequently. A daily record is kept of handwriting and performance in arithmetic to monitor mental status.
  3. Fluid intake and output and body weight are recorded each day.
  4. Vital signs are measured and recorded every 4 hours.
  5. Potential sites of infection (peritoneum, lungs) are assessed frequently, and abnormal findings are reported promptly.
  6. Serum ammonia level is monitored daily.
  7. Protein intake is restricted in patients who are comatose or who have encephalopathy that is refractory to lactulose and antibiotic therapy.
  8. Reduction in the absorption of ammonia from the GI tract is accomplished by the use of gastric suction, enemas, or oral antibiotics.
  9. Electrolyte status is monitored and corrected if abnormal.
  10. Sedatives, tranquilizers, and analgesic medications are discontinued.
  11. Benzodiazepine antagonists (flumazenil [Romazicon]) may be administered to improve encephalopathy whether or not the patient has previously taken benzodiazepines.

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**Chart 39-4 Nutritional Management of Hepatic Encephalopathy**

- Prevent the formation and absorption of toxins, principally ammonia, from the intestine.
- Keep daily protein intake between 1.0 and 1.5 g/kg, depending on the degree of decompensation.
- Avoid protein restriction if possible, even in those with encephalopathy. If necessary, implement temporary restriction of 0.5 g/kg.
- For patients who are truly protein-intolerant, provide additional nitrogen in the form of an amino acid supplement. Use of branched-chain amino acids is still controversial.
- Provide small, frequent meals and an evening snack of complex carbohydrates to avoid protein loading.
- Substitute vegetable protein for animal protein in as high a percentage as possible.
Nursing Management

- Maintaining a safe environment to prevent injury, bleeding, and infection. The nurse administers the prescribed treatments and monitors the patient for the many potential complications.
- If the patient recovers from hepatic encephalopathy and coma, rehabilitation is likely to be prolonged.

Teaching Patients Self-Care

- If the patient has recovered from hepatic encephalopathy and is to be discharged home, the nurse instructs the family to watch for subtle signs of recurrent encephalopathy.
- In the acute phase of hepatic encephalopathy, dietary protein may be reduced to 0.8 to 1.0 g/kg per day. Instruct the patient in maintenance of a low-protein, high-calorie diet. Vegetable protein intake may result in improved nitrogen balance without precipitating or advancing hepatic encephalopathy.

OTHER MANIFESTATIONS OF LIVER DYSFUNCTION

A. Edema and Bleeding

- Many patients with liver dysfunction develop generalized edema from hypoalbuminemia that results from decreased hepatic production of albumin.
- The production of blood clotting factors by the liver is also reduced, leading to an increased incidence of bruising, epistaxis, bleeding from wounds, and, as described above, GI bleeding.

B. Vitamin Deficiency

- Decreased production of several clotting factors may be due, in part, to deficient absorption of vitamin K from the GI tract. This probably is caused by the inability of liver cells to use vitamin K to make prothrombin.
- Absorption of the other fat-soluble vitamins (vitamins A, D, and E) as well as dietary fats may also be impaired because of decreased secretion of bile salts into the intestine.
- The threat of these avitaminoses provides the rationale for supplementing the diet of every patient with chronic liver disease (especially if alcohol-related) with ample quantities of vitamins A, B complex, C, and K and folic acid.

C. Metabolic Abnormalities

- Abnormalities of glucose metabolism also occur; the blood glucose level may be abnormally high shortly after a meal, but hypoglycemia may occur during fasting because of decreased hepatic glycogen reserves and decreased gluconeogenesis.
- Because the ability to metabolize medications is decreased, medications must be used cautiously and usual medication dosages must be reduced for the patient with liver failure.
- Many endocrine abnormalities also occur with liver dysfunction because the liver cannot metabolize hormones normally, including androgens or sex hormones.
- Gynecomastia, amenorrhea, testicular atrophy, loss of pubic hair in the male, and menstrual irregularities in females may occur.

D. Pruritus and Other Skin Changes

- Patients with liver dysfunction resulting from biliary obstruction commonly develop severe itching (pruritus) due to retention of bile salts. Patients may
develop vascular (or arterial) spider angiomas on the skin, generally above the waistline.

**Management of Patients With Viral Hepatic Disorders**

- Viral hepatitis is a systemic, viral infection in which necrosis and inflammation of liver cells produce a characteristic cluster of clinical, biochemical, and cellular changes. To date, five definitive types of viral hepatitis have been identified: hepatitis A, B, C, D, and E.
- Hepatitis A and E are similar in mode of transmission (fecal–oral route), whereas hepatitis B, C, and D share many characteristics.

<table>
<thead>
<tr>
<th>Previous names</th>
<th>Hepatitis A</th>
<th>Hepatitis B</th>
<th>Hepatitis C</th>
<th>Hepatitis D</th>
<th>Hepatitis E</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epidemiology</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>Hepatitis A virus (HAV)</td>
<td>Hepatitis B virus (HBV)</td>
<td>Hepatitis C virus (HCV)</td>
<td>Hepatitis D virus (HDV)</td>
<td>Hepatitis E virus (HEV)</td>
</tr>
<tr>
<td><strong>Mode of transmission</strong></td>
<td>Fecal–oral route; poor sanitation. Person-to-person contact. Waterborne; foodborne. Transmission possible with oral–anal contact during sex.</td>
<td>Parenterally: by intimate contact with carriers or those with acute disease; sexual and oral–oral contact. Perinatal transmission from mothers to infants. An important occupational hazard for health care personnel.</td>
<td>Transfusion of blood and blood products; exposure to contaminated blood through equipment or drug paraphernalia. Transmission possible with sex with infected partner; risk increased with STD.</td>
<td>Same as HBV. HBV surface antigen necessary for replication; pattern similar to that of hepatitis B.</td>
<td>Fecal–oral route; person to person contact may be possible, although risk appears low.</td>
</tr>
</tbody>
</table>
A. HEPATITIS A VIRUS (HAV)

- HAV accounts for 20% to 25% of cases of clinical hepatitis in the developed world.
- Hepatitis A, formerly called infectious hepatitis, is caused by an RNA virus of the Enterovirus family.
- The mode of transmission of this disease is the fecal–oral route, primarily through the ingestion of food or liquids infected by the virus. The virus has been found in the stool of infected patients before the onset of symptoms and during the first few days of illness.
- The incubation period is estimated to be 15 to 50 days, with an average of 30 days.

Clinical Manifestations

- Many patients are anicteric (without jaundice) and symptomless.
- If symptoms appear, they are of a mild, flu-like upper respiratory tract infection, with low-grade fever.
- Anorexia, an early symptom, is often severe. It is thought to result from release of a toxin by the damaged liver or by failure of the damaged liver cells to detoxify an abnormal product.
- Later, jaundice and dark urine may become apparent.
- Indigestion is present in varying degrees, marked by vague epigastric distress, nausea, heartburn, & flatulence.
- The liver and spleen are often moderately enlarged for a few days after onset; otherwise, apart from jaundice, there are few physical signs.
Prevention

Medical Management

- Bed rest during the acute stage and a diet that is both acceptable to the patient and nutritious are part of the treatment and nursing care.
- During the period of anorexia, the patient should receive frequent small feedings, supplemented, if necessary, by IV fluids with glucose.
- Because this patient often has an aversion to food, gentle persistence and creativity may be required to stimulate the appetite.
- Optimal food and fluid levels are necessary to counteract weight loss and slow recovery.

Nursing Management

- The patient is usually managed at home unless symptoms are severe. Therefore, the nurse assists the patient and family in coping with the temporary disability and fatigue that are common in hepatitis and instructs them to seek additional health care if the symptoms persist or worsen.
B. HEPATITIS B VIRUS (HBV)

- Transmitted primarily through blood (percutaneous and permucosal routes). HBV has been found in blood, saliva, semen, and vaginal secretions and can be transmitted through mucous membranes and breaks in the skin.
- HBV is also transferred from carrier mothers to their babies, especially in areas with a high incidence (ie, Southeast Asia). The infection is usually not via the umbilical vein, but from the mother at the time of birth and during close contact afterward.
- HBV has a long incubation period. It replicates in the liver and remains in the serum for relatively long periods, allowing transmission of the virus.
- Those at risk for developing hepatitis B include surgeons, clinical laboratory workers, dentists, nurses, and respiratory therapists. Staff and patients in hemodialysis and oncology units and sexually active homosexual
- Most people (>90%) who contract hepatitis B infections will develop antibodies and recover spontaneously in 6 months. The mortality rate from hepatitis B has been reported to be as high as 10%. Another 10% of patients who have hepatitis B progress to a carrier state or develop chronic hepatitis with persistent HBV infection and hepatocellular injury and inflammation.
Clinical Manifestations

- Clinically, the disease closely resembles hepatitis A, but the incubation period is much longer (1 to 6 months). Signs and symptoms of hepatitis B may be insidious and variable. Fever and respiratory symptoms are rare; some patients have arthralgias and rashes.
- The patient may have loss of appetite, dyspepsia, abdominal pain, generalized aching, malaise, and weakness. Jaundice may or may not be evident. Light-colored stools and dark urine.
- The liver may be tender and enlarged. The spleen is enlarged and palpable in a few patients; the posterior cervical lymph nodes may also be enlarged.
- Assessment and Diagnostic Findings

HBV is a DNA virus composed of the following antigenic particles:
- HBcAg—hepatitis B core antigen (antigenic material in an inner core)
- HBsAg—hepatitis B surface antigen (antigenic material on surface of HBV)
- HBeAg—an independent protein circulating in the blood
- HBxAg—gene product of X gene of HBV/DNA

- Each antigen elicits its specific antibody and is a marker for different stages of the disease process:
  - Anti-HBc—antibody to core antigen or HBV; persists during the acute phase of illness; may indicate continuing HB in the liver
  - Anti-HBs—antibody to surface determinants on HBV; detected during late convalescence; usually indicates recovery and development of immunity. It appears in the circulation in 80% to 90% of infected patients 1 to 10 weeks after exposure, if continues for > 6 months, pt is considered a HBsAg carrier
  - Anti-HBe—antibody to hepatitis B e-antigen; usually signifies reduced infectivity
• anti-HBxAg—antibody to the hepatitis B x-antigen; may indicate ongoing replication of HBV

**Prevention**

• The goals of prevention are to interrupt the chain of transmission, to protect people at high risk with active immunization through the use of hepatitis B vaccine, and to use passive immunization for unprotected people exposed to HBV.

• **A. PREVENTING TRANSMISSION**
  o Continued screening of blood donors for the presence of hepatitis B antigens
  o The use of disposable syringes, needles, and lancets and the introduction of needleless IV
  o Good personal hygiene is fundamental to infection control. In the clinical laboratory, work areas should be disinfected daily. Gloves are worn when handling all blood and body fluids as well as HBAg positive specimens.
  o Eating and smoking are prohibited in the laboratory and in other areas exposed to secretions, blood products.

• **B. Active Immunization: Hepatitis B Vaccine**
  o Active immunization is recommended for individuals at high risk for hepatitis B (eg, health care personnel and hemodialysis patients). In addition, individuals with hepatitis C and other chronic liver diseases should receive the vaccine.
  o Administered IM (in the deltoid muscle in adults) in three doses, the second and third doses 1 & 6 months after the first dose. The third dose is very important in producing prolonged immunity.
  o Antibody response may be measured by anti-HBs levels 1 to 3 months after completing the basic course of vaccine.
  o Universal vaccination of all infants.

• **C. PASSIVE IMMUNITY: HEPATITIS B IMMUNE GLOBULIN**
  o Hepatitis B immune globulin (HBIG) provides passive immunity to hepatitis B and is indicated for people exposed to HBV who have never had hepatitis B and have never received hepatitis B vaccine. Specific indications for postexposure vaccine with HBIG include:
    1. inadvertent exposure to HBAg-positive blood through percutaneous (needlestick) or transmucosal (splashes in contact with mucous membrane) routes,
    2. sexual contact with people positive for HBAg, and
    3. perinatal exposure (babies born to HBV-infected mothers should receive HBIG within 12 hours of delivery).

**Gerontologic Considerations**

• The elderly patient who contracts hepatitis B has a serious risk of severe liver cell necrosis or fulminant hepatic failure, particularly if other illnesses are present. The patient is seriously ill and the prognosis is poor, so efforts should be undertaken to eliminate other factors (eg, medications, alcohol) that may affect liver function.

**Medical Management**

• The goals of treatment are to **minimize** infectivity, normalize liver inflammation, and decrease symptoms.
• Alpha interferon offers the most promise. It results in remission in approximately one third of patients
• Lamivudine & adefovir are new antiviral agents.
• Adequate nutrition should be maintained; proteins are restricted when the liver’s ability to metabolize protein byproducts is impaired, as demonstrated by symptoms.
• If vomiting persists, the patient may require hospitalization and fluid therapy.

Nursing Management
• Convalescence may be prolonged, with complete symptomatic recovery sometimes requiring 3 to 4 months or longer.
• During this stage, gradual resumption of physical activity is encouraged after the jaundice has resolved.
• The nurse identifies psychosocial issues and concerns, particularly the effects of separation from family and friends if the patient is hospitalized during the acute and infective stages. Even if not hospitalized, the patient will be unable to work and must avoid sexual contact.

C. HEPATITIS C VIRUS (HCV)
• Formerly referred to as non-A, non-B hepatitis.
• Blood transfusions and sexual contact accounted for most cases of hepatitis C in the United States, other parenteral means, such as sharing contaminated needles by IV/injection drug users and unintentional needle-sticks and other injuries in health care workers, now account for a significant number of cases.
• There is no benefit from rest, diet, or vitamin supplements. Recent studies have demonstrated that a combination of interferon (Intron-A) and ribavirin (Rebetol), two antiviral agents, is effective in producing improvement in patients with hepatitis C and in treating relapse.

Chart 39-9
Risk Factors for Hepatitis C
• Recipient of blood products or organ transplant prior to 1992 or clotting factor concentrates before 1987
• Health care and public safety workers after needlestick injuries or mucosal exposure to blood
• Children born to women infected with hepatitis C virus
• Past/current illicit IV/injection drug use
• Past treatment with chronic hemodialysis
• Sex with infected partner, having multiple sex partners, history of STD, unprotected sex
D. HEPATITIS D VIRUS (HDV)

- Hepatitis D (delta agent) occurs in some cases of hepatitis B. Because the virus requires hepatitis B surface antigen for its replication, only individuals with hepatitis B are at risk for hepatitis D. Anti-delta antibodies in the presence of HBAg on testing confirm the diagnosis. It is also common among IV/injection drug users, hemodialysis patients, and recipients of multiple blood transfusions. Sexual contact with those with hepatitis B is considered to be an important mode of transmission of hepatitis B and D.

E. HEPATITIS E VIRUS (HEV)

- Hepatitis E is believed to be transmitted by the fecal–oral route, principally through contaminated water in areas with poor sanitation. The incubation period is variable, estimated to range between 15 and 65 days. In general, hepatitis E resembles hepatitis A. It has a self-limiting course with an abrupt onset. Jaundice is nearly always present. Chronic forms do not develop.

F. HEPATITIS G (HGV) AND GB VIRUS.C

- It has long been believed that there is another non-A, non-B, non-C agent causing hepatitis in humans. The incubation period for post-transfusion hepatitis is 14 to 145 days, too long for hepatitis B or C. In the United States, about 5% of chronic liver disease remains cryptogenic (does not appear to be autoimmune or viral in origin), and half the patients have previously received transfusions. Thus, a new form of hepatitis (hepatitis G or GBV-C) has been described. They are two different isolates of the same virus. Autoantibodies are absent. The clinical significance of this virus remains uncertain. Risk factors are similar to those for hepatitis C.

Management of Patients With Nonviral Hepatic Disorders

- Certain chemicals have toxic effects on the liver and when taken by mouth, inhaled, or injected parenterally produce acute liver cell necrosis, or toxic hepatitis.
- The chemicals most commonly implicated in this disease are carbon tetrachloride, phosphorus, chloroform, and gold compounds.
- Drug-induced hepatitis, is similar to acute viral hepatitis, but parenchymal destruction tends to be more extensive. Some medications that can lead to hepatitis are isoniazide, halothane, acetaminophen, and certain antibiotics, antimetabolites, and anesthetic agents.

TOXIC HEPATITIS

- Resembles viral hepatitis in onset. Obtaining a history of exposure to hepatotoxic chemicals, medications, or other agents assists in early treatment and removal of the offending agent.
- Anorexia, nausea, and vomiting are the usual symptoms; jaundice and hepatomegaly are noted on physical assessment.
- Recovery from acute toxic hepatitis is rapid if the hepatotoxin is identified early and removed or if exposure to the agent has been limited.

DRUG-INDUCED HEPATITIS

- Drug-induced hepatitis is responsible for 20% to 25% of cases of acute hepatic failure in the United States.
- Manifestations of sensitivity to a medication may occur on the first day of its use or not until several months later, depending on the medication.
- Usually the onset is abrupt, with chills, fever, rash, pruritus, arthralgia, anorexia, and nausea. Later, there may be jaundice and dark urine & enlarged and tender liver. When the offending medication is withdrawn, symptoms may gradually subside.

**FULMINANT HEPATIC FAILURE**
- Is the clinical syndrome of sudden and severely impaired liver function in a previously healthy person. According to the original and generally accepted definition, fulminant hepatic failure develops within 8 weeks of the first symptoms of jaundice.
- Three categories are frequently cited: hyperacute, acute, and subacute liver failure.

**FULMINANT HEPATIC FAILURE**
- Is the clinical syndrome of sudden and severely impaired liver function in a previously healthy person.
- In hyperacute liver failure, the duration of jaundice before the onset of encephalopathy is 0 to 7 days; in acute liver failure, it is 8 to 28 days; and in subacute liver failure, it is 28 to 72 days.
- The prognosis for fulminant hepatic failure is much worse than for chronic liver failure. However, in fulminant failure, the hepatic lesion is potentially reversible, with survival rates of approximately 50% to 85% (depending on etiology). Those who do not survive die of massive hepatocellular injury and necrosis.

**FULMINANT HEPATIC FAILURE**
- Fulminant hepatic failure is often accompanied by coagulation defects, renal failure and electrolyte disturbances, infection, hypoglycemia, encephalopathy, and cerebral edema.
- Viral hepatitis is a common cause of fulminant hepatic failure; other causes: toxic medications (eg, acetaminophen) and chemicals (eg, carbon tetrachloride), metabolic disturbances (eg, Wilson's disease, a hereditary syndrome with deposition of copper in the liver), and structural changes (eg, Budd-Chiari syndrome, an obstruction to outflow in major hepatic veins).

**Management**
- The key to optimizing treatment is rapid recognition of acute liver failure and intensive interventions.
- The use of antidotes for certain conditions may be indicated such as N-acetylcysteine for acetaminophen toxicity and penicillin for mushroom poisoning.
- Treatment modalities may include plasma exchanges (plasmapheresis) to correct coagulopathy and to stabilize the patient awaiting liver transplantation.

**HEPATIC CIRRHOSIS**
- Cirrhosis is a chronic disease characterized by replacement of normal liver tissue with diffuse fibrosis that disrupts the structure and function of the liver. There are three types of cirrhosis or scarring of the liver:
  1. Alcoholic cirrhosis, in which the scar tissue characteristically surrounds the portal areas. This is most frequently due to chronic alcoholism and is the most common type of cirrhosis.
2. Postnecrotic cirrhosis, in which there are broad bands of scar tissue as a late result of a previous infection of acute viral hepatitis.
3. Biliary cirrhosis, in which scarring occurs in the liver around the bile ducts. This type usually is the result of chronic biliary obstruction and infection (cholangitis); it is much less common than the other two types.

Pathophysiology
- Several factors have been implicated in the etiology of cirrhosis: alcohol consumption (is the major causative factor), nutritional deficiency with reduced protein intake (excessive alcohol intake is the major causative factor in fatty liver and its consequences).
- Other factors may play a role, including exposure to certain chemicals (carbon tetrachloride, chlorinated naphthalene, arsenic, or phosphorus) or infectious schistosomiasis.
- Twice as many men as women are affected.
- Most patients are between 40 and 60 years of age.
The destroyed liver cells are replaced gradually by scar tissue; eventually the amount of scar tissue exceeds that of the functioning liver tissue.

Islands of residual normal tissue and regenerating liver tissue may project from the constricted areas, giving the cirrhotic liver its characteristic hobnail appearance.

**Clinical Manifestations**

- Signs and symptoms of cirrhosis increase in severity as the disease progresses. The severity of the manifestations helps to categorize the disorder into two main presentations:
  1. Compensated cirrhosis, with its less severe, often vague symptoms, may be discovered secondarily at a routine physical examination.
  2. Decompensated cirrhosis, S & S result from failure of the liver to synthesize proteins, clotting factors, and other substances and manifestations of portal hypertension.

**Compensated**
- Intermittent mild fever
- Vascular spiders
- Palmar erythema (redden palms)
- Unexplained epistaxis
- Ankle edema
- Vague morning indigestion
- Flatulent dyspepsia
- Abdominal pain
- Firm, enlarged liver
- Splenomegaly

** Decompensated**
- Ascites
- Jaundice
- Weakness
- Muscle wasting
- Weight loss
- Continuous mild fever
- Clubbing of fingers
- Purpura (due to decreased platelet count)
- Spontaneous bruising
- Epistaxis
- Hypotension
- Sparse body hair
- White nails
- Gonadal atrophy

**Clinical Manifestations**

- Liver enlargement
- portal obstruction and ascites
- infection and peritonitis
- Gastrointestinal varices
- Edema
- Vitamin deficiency and anemia
- Mental deterioration

**Assessment and Diagnostic Findings**

- The extent of liver disease and the type of treatment are determined after reviewing the laboratory findings. Because the functions of the liver are complex, there are many diagnostic tests that may provide information about liver function.
- In severe parenchymal liver dysfunction, the serum albumin level decreases. Enzyme tests indicate liver cell damage: serum alkaline phosphatase, AST, ALT levels increase, and the serum cholinesterase level may decrease.
• Bilirubin tests are performed to measure bile excretion or bile retention; elevated levels can occur with cirrhosis and other liver disorders.
• Prothrombin time is prolonged.
• Ultrasound scanning is used to measure the difference in density of parenchymal cells and scar tissue.
• CT, MRI, and radioisotope liver scans give information about liver size and hepatic blood flow and obstruction.
• Diagnosis is confirmed by liver biopsy.

Medical Management
• The management of the patient with cirrhosis is usually based on the presenting symptoms. For example, antacids are prescribed to decrease gastric distress and minimize the possibility of GI bleeding.
• Vitamins and nutritional supplements promote healing of damaged liver cells and improve the general nutritional status. Potassium-sparing diuretics (spironolactone [Aldactone], triamterene [Dyrenium]) may be indicated to decrease ascites, if present;
• Preliminary studies indicate that colchicine, an antiinflammatory agent used to treat the symptoms of gout, may increase the length of survival in patients with mild to moderate cirrhosis. Colchicine is believed to reverse the fibrotic processes in cirrhosis, and this has improved survival.

NURSING PROCESS:
THE PATIENT WITH HEPATIC CIRRHOSIS

Nursing diagnoses and goals
1. Activity intolerance related to fatigue, lethargy, and malaise
   Goal: Patient reports decrease in fatigue and reports increased ability to participate in activities
2. Imbalanced nutrition: less than body requirements, related to abdominal distention and discomfort and anorexia
   Goal: Positive nitrogen balance, no further loss of muscle mass; meets nutritional requirements
3. Impaired skin integrity related to pruritus from jaundice and edema.
   Goal: Decrease potential for pressure ulcer development; breaks in skin integrity
4. High risk for injury related to altered clotting mechanisms and altered level of consciousness
   Goal: Reduced risk of injury
5. Disturbed body image related to changes in appearance, sexual dysfunction, and role function
   Goal: Patient verbalizes feelings consistent with improvement of body image and self-esteem
6. Chronic pain and discomfort related to enlarged tender liver and ascites
   Goal: Increased level of comfort
7. Fluid volume excess related to ascites and edema formation
   Goal: Restoration of normal fluid volume
8. Disturbed thought processes related to deterioration of liver function and increased serum ammonia level
   Goal: Improved mental status; safety maintained
9. Risk for imbalanced body temperature: hyperthermia related to inflammatory process of cirrhosis or hepatitis
Goal: Maintenance of normal body temperature, free from infection
10. Ineffective breathing pattern related to ascites and restriction of thoracic excursion secondary to ascites, abdominal distention, and fluid in the thoracic cavity
Goal: Improved respiratory status

Collaborative Problem
1. Gastrointestinal bleeding and hemorrhage
   Goal: Absence of episodes of gastrointestinal bleeding and hemorrhage
2. Hepatic encephalopathy
   Goal: Absence of changes in cognitive status and of injury

Cancer Of The Liver

- Hepatic tumors may be malignant or benign. Benign liver occur with the use of oral contraceptives in women in their reproductive years.
- Malignant tumors could be primary or metastasized from other sites.

Primary Liver Tumors
- Few cancers originate in the liver.
- Usually associated with chronic liver disease, hepatitis B and C infections, and cirrhosis.
- Hepatocellular carcinoma (HCC) is the most common type of primary liver cancer. HCC is usually nonresectable because of rapid growth and metastasis.
- Other types of primary liver cancer include cholangiocellular carcinoma and combined hepatocellular and cholangiocellular carcinoma. If found early, resection may be possible, but early detection is unlikely.

Liver Metastases
- Metastases from other primary sites are found in the liver in about half of all advanced cancer cases.
• Malignant tumors are likely to reach the liver eventually, by way of the portal system or lymphatic channels, or by direct extension from an abdominal tumor.
• Moreover, the liver apparently is an ideal place for these malignant cells to thrive.

Clinical Manifestations
• The early manifestations of malignancy of the liver include pain, a continuous dull ache in the right upper quadrant, epigastrium, or back.
• Weight loss, loss of strength, anorexia, and anemia may also occur.
• The liver may be enlarged and irregular on palpation.
• Jaundice is present only if the larger bile ducts are occluded by the pressure of tumor on bile ducts.
• Ascites develops if tumor obstructs the portal veins or if tumor tissue is seeded in the peritoneal cavity.

Assessment and Diagnostic Findings
• Diagnosis is based on clinical signs and symptoms, the history and physical examination, and the results of laboratory and x-ray studies.
• Increased serum levels of bilirubin, alkaline phosphatase, AST, GGT, and lactic dehydrogenase may occur.
• Leukocytosis, erythrocytosis, hypercalcemia, hypoglycemia, and hypocholesterolemia
• The serum level of alpha-fetoprotein (AFP), which serves as a tumor marker, is elevated in 30% to 40% of patients with primary liver cancer.
• Levels of carcinoembryonic antigen (CEA), a marker of advanced cancer of the digestive tract, may be elevated.
• These two markers together are useful to distinguish between metastatic liver disease and primary liver cancer.
• Assessment and Diagnostic Findings
• X-rays, liver scans, CT scans, ultrasound studies, MRI, arteriography, and laparoscopy may be part of the diagnostic workup and may be performed to determine the extent of the cancer.
• Confirmation of a tumor’s histology can be made by biopsy under imaging guidance (CT scan or ultrasound).

Medical Management
• Surgical resection of the tumor is possible in some patients, but cirrhosis (prevalent in liver cancer), increases the risks associated with surgery.
• Radiation therapy and chemotherapy showed varying degrees of success.
• An implantable pump has been used to deliver a high concentration of chemotherapy to the liver through the hepatic artery. This method provides a reliable, controlled, and continuous infusion of medication that can be carried out in the patient’s home.

Percutaneous Biliary Drainage
• Percutaneous biliary or transhepatic drainage is used to bypass biliary ducts obstructed by liver, pancreatic, or bile duct tumors in patients with inoperable tumors or in those considered poor surgical risks.
• A catheter is inserted through the abdominal wall and past the obstruction into the duodenum. Such procedures are used to reestablish biliary drainage, relieve pressure and pain from the buildup of bile behind the obstruction, and decrease pruritus and jaundice.
Surgical Management

- Surgical resection is the treatment of choice when HCC is confined to one lobe of the liver and the function of the remaining liver is considered adequate for postoperative recovery.
- Capitalizing on the regenerative capacity of the liver cells, some surgeons have successfully removed 90% of the liver.
- However, the presence of cirrhosis limits the ability of the liver to regenerate.
- Lobectomy
- Liver Transplantation

Liver Transplantation

- Liver disease for which no other form of treatment is available.
- The transplantation procedure involves total removal of the diseased liver and its replacement with a healthy liver in the same anatomic location.
- The success of liver transplantation depends on successful immunosuppression (i.e. cyclosporine corticosteroids).

Complications

- The postoperative complication rate is high, primarily because of technical complications or infection.
- Immediate postoperative complication include bleeding, infection, and rejection. Disruption, infection, or obstruction of the biliary anastomosis and impaired biliary drainage may occur. Vascular thrombosis and stenosis are other potential complications.

Nursing Management

1. Preoperative Nursing Interventions

- Provide the patient and family with full explanations about the procedure, the chances of success, and the risks, including the side effects of long-term immunosuppression. The need for close follow-up and lifelong compliance with the therapeutic regimen.
- Malnutrition, massive ascites, and fluid and electrolyte disturbances are treated before surgery to increase the chance of a successful outcome.

2. Post operative;

- The patient is maintained in an environment as free from bacteria, viruses, and fungi as possible to prevent infection.
- Cardiovascular, pulmonary, renal, neurologic, & metabolic functions are monitored continuously.
- Cardiac output, CVP, pulmonary capillary wedge pressure, ABG, O₂ saturation, urine output, V/S are used to evaluate the patient’s hemodynamic status and intravascular fluid volume.
- Liver functions tests and coagulation profile.
- I & O including drainage from T tube.

LIVER ABSCESSES

- Two categories of liver abscess have been identified: amebic and pyogenic.
  1. Amebic liver abscesses are most commonly caused by Entamoeba histolytica. Most amebic liver abscesses occur in the developing countries of the tropics and subtropics because of poor sanitation and hygiene.
  2. Pyogenic liver abscesses are much less common.
Pathophysiology

- Whenever an infection develops anywhere along the biliary or GI tract, infecting organisms may reach the liver through the biliary system, portal venous system, or hepatic arterial or lymphatic system.
- Most bacteria are destroyed promptly, but occasionally some gain a foothold.
- The bacterial toxins destroy the neighboring liver cells, and the resulting necrotic tissue serves as a protective wall for the organisms.
- Meanwhile, leukocytes migrate into the infected area.
- The result is an abscess cavity full of a liquid containing living and dead leukocytes, liquefied liver cells, and bacteria.
- Pyogenic abscesses of this type may be either single or multiple and small.
- Examples of causes of pyogenic liver abscess include cholangitis and abdominal trauma.

Clinical Manifestations

- The clinical picture is one of sepsis with few or no localizing signs. Fever with chills and diaphoresis, malaise, anorexia, nausea, vomiting, and weight loss may occur.
- The patient may complain of dull abdominal pain and tenderness in the right upper quadrant of the abdomen.
- Hepatomegaly, jaundice, anemia, and pleural effusion may develop.
- Sepsis and shock may be severe and life-threatening.

Assessment and Diagnostic Findings

- Blood cultures are obtained but may not identify the organism.
- Aspiration of the liver abscess may be done to assist in diagnosis and to obtain cultures of the organism.
- Percutaneous drainage of pyogenic abscesses is carried out to evacuate the abscess material and promote healing.
- A catheter may be left in place for continuous drainage; the patient must be instructed about its management.

Medical Management

- Treatment includes IV antibiotic therapy; the specific antibiotic used in treatment depends on the organism identified.
- Continuous supportive care is indicated because of the serious condition of the patient.
- Open surgical drainage may be required if antibiotic therapy and percutaneous drainage are ineffective.

Nursing Management

- Depends on the patient’s physical status and the medical management that is indicated.
- For patients who undergo evacuation and drainage of the abscess, monitoring of the drainage and skin care are imperative.
- Strategies must be implemented to contain the drainage and to protect the patient from other sources of infection.
- Vital signs are monitored to detect changes in the patient’s physical status.
- Deterioration in vital signs or the onset of new symptoms such as increasing pain, which may indicate rupture or extension of the abscess, is reported promptly.
- The nurse administers IV antibiotic therapy as prescribed.
- The white blood cell count and other laboratory test results are monitored closely for changes consistent with worsening infection.
## NURSING PROCESS FOR PATIENTS WITH HEPATIC DYSFUNCTIONS

### Nursing Diagnosis: Activity intolerance related to fatigue, lethargy, and malaise

**Goal:** Patient reports decrease in fatigue and reports increased ability to participate in activities

<table>
<thead>
<tr>
<th>Nursing Interventions</th>
<th>Rationale</th>
<th>Expected Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Assess level of activity tolerance and degree of fatigue, lethargy, and malaise when performing routine ADLs.</td>
<td>1. Provides baseline for further assessment and criteria for assessment of effectiveness of interventions</td>
<td>• Exhibits increased interest in activities and events</td>
</tr>
<tr>
<td>2. Assist with activities and hygiene when fatigued.</td>
<td>2. Promotes exercise and hygiene within patient’s level of tolerance</td>
<td>• Participates in activities and gradually increases exercise within physical limits</td>
</tr>
<tr>
<td>3. Encourage rest when fatigued or when abdominal pain or discomfort occurs.</td>
<td>3. Conserves energy and protects the liver</td>
<td>• Reports increased strength and well-being</td>
</tr>
<tr>
<td>4. Assist with selection and pacing of desired activities and exercise.</td>
<td>4. Stimulates patient’s interest in selected activities</td>
<td>• Reports absence of abdominal pain and discomfort</td>
</tr>
<tr>
<td>5. Provide diet high in carbohydrates with protein intake consistent with liver function.</td>
<td>5. Provides calories for energy and protein for healing</td>
<td>• Plans activities to allow ample periods of rest</td>
</tr>
<tr>
<td>6. Administer supplemental vitamins (A, B complex, C, and K).</td>
<td>6. Provides additional nutrients</td>
<td>• Takes vitamins as prescribed</td>
</tr>
</tbody>
</table>

### Expected Outcomes

- Exhibits increased interest in activities and events
- Participates in activities and gradually increases exercise within physical limits
- Reports increased strength and well-being
- Reports absence of abdominal pain and discomfort
- Plans activities to allow ample periods of rest
- Takes vitamins as prescribed

### Nursing Diagnosis: Imbalanced nutrition: less than body requirements, related to abdominal distention and discomfort and anorexia

**Goal:** Positive nitrogen balance, no further loss of muscle mass; meets nutritional requirements

<table>
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<tbody>
<tr>
<td>1. Assess dietary intake and nutritional status through diet history and diary, daily weight measurements and laboratory data.</td>
<td>1. Identifies deficits in nutritional intake and adequacy of nutritional state</td>
<td>• Exhibits improved nutritional status by increased weight (without fluid retention) and improved laboratory data.</td>
</tr>
<tr>
<td>2. Provide diet high in carbohydrates with protein intake consistent with liver function.</td>
<td>2. Provides calories for energy, sparing protein for healing</td>
<td>• States rationale for dietary modifications</td>
</tr>
<tr>
<td>3. Assist patient in identifying low-sodium foods.</td>
<td>3. Reduces edema and ascites formation</td>
<td>• Identifies foods high in carbohydrates and within protein requirements (moderate to high protein in cirrhosis and hepatitis, low protein in hepatic failure)</td>
</tr>
<tr>
<td>4. Elevate the head of the bed during meals.</td>
<td>4. Reduces discomfort from abdominal distention and decreases sense of fullness produced by pressure of abdominal contents and ascites on the stomach</td>
<td>• Reports improved appetite</td>
</tr>
<tr>
<td>5. Provide oral hygiene before meals and pleasant environment for meals at meal time.</td>
<td>5. Promotes positive environment and increased appetite; reduces unpleasant taste</td>
<td>• Participates in oral hygiene measures</td>
</tr>
<tr>
<td>6. Offer smaller, more frequent meals (6 per day).</td>
<td>6. Decreases feeling of fullness, bloating</td>
<td>• Reports increased appetite; identifies rationale for smaller, frequent meals</td>
</tr>
<tr>
<td>7. Encourage patient to eat meals and supplementary feedings.</td>
<td>7. Encouragement is essential for the patient with anorexia and gastrointestinal discomfort.</td>
<td>• Demonstrates intake of high-calorie diet; adheres to protein restriction</td>
</tr>
<tr>
<td>8. Provide attractive meals and an aesthetically pleasing setting at meal time.</td>
<td>8. Promotes appetite and sense of well-being</td>
<td>• Identifies foods and fluids that are nutritious and</td>
</tr>
</tbody>
</table>

- Exhibits improved nutritional status by increased weight (without fluid retention) and improved laboratory data.
- States rationale for dietary modifications
- Identifies foods high in carbohydrates and within protein requirements (moderate to high protein in cirrhosis and hepatitis, low protein in hepatic failure)
- Reports improved appetite
- Participates in oral hygiene measures
- Reports increased appetite; identifies rationale for smaller, frequent meals
- Demonstrates intake of high-calorie diet; adheres to protein restriction
- Identifies foods and fluids that are nutritious and
10. Apply an ice collar for nausea.
11. Administer medications prescribed for nausea, vomiting, diarrhea, or constipation.
12. Encourage increased fluid intake and exercise if the patient reports constipation.

9. Eliminates ‘empty calories’ and further damage from alcohol
10. May reduce incidence of nausea
11. Reduces gastrointestinal symptoms and discomforts that decrease the appetite and interest in food
12. Promotes normal bowel pattern and reduces abdominal discomfort and distention

permitted on diet
- Gains weight without increased edema or ascites formation
- Reports increased appetite and well-being
- Excludes alcohol from diet
- Takes medications for gastrointestinal disorders as prescribed
- Reports normal gastrointestinal function with regular bowel function

7. Nursing Diagnosis: Impaired skin integrity related to pruritus from jaundice and edema
Goal: Decrease potential for pressure ulcer development; breaks in skin integrity.

8. Nursing Interventions

<table>
<thead>
<tr>
<th>Rationale</th>
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</tr>
</thead>
<tbody>
<tr>
<td>1. Assists in determining appropriate interventions</td>
<td>• Exhibits intact skin without redness, excoriations, or breakdown</td>
</tr>
<tr>
<td>2. Provides baseline for detecting changes and evaluating effectiveness of interventions</td>
<td>• Reports relief from pruritus</td>
</tr>
<tr>
<td>3. Prevents skin excoriation and infection from scratching</td>
<td>• Exhibits no skin excoriations from scratching</td>
</tr>
<tr>
<td>4. Removes waste products from skin while preventing dryness of skin</td>
<td>• Uses nondrying soaps and lotions. States rationale for use of nondrying soaps and lotions.</td>
</tr>
<tr>
<td>5. Promotes mobilization of edema</td>
<td>• Turns self periodically. Exhibits reduced edema of dependent parts of the body.</td>
</tr>
<tr>
<td>6. Minimizes prolonged pressure on bony prominences susceptible to breakdown</td>
<td>• Exhibits no areas of skin breakdown</td>
</tr>
<tr>
<td>7. May decrease skin irritation and need for scratching</td>
<td>• Exhibits decreased edema; normal skin turgor</td>
</tr>
<tr>
<td>8. Edematous skin and tissue have compromised nutrient supply and are vulnerable to pressure and trauma</td>
<td>• Exhibits reduced edema; normal skin turgor</td>
</tr>
<tr>
<td>9. Minimizes edema formation</td>
<td>• Exhibits normal gastrointestinal function with regular bowel function</td>
</tr>
<tr>
<td>10. Promotes mobilization of edema</td>
<td></td>
</tr>
</tbody>
</table>

Nursing Diagnosis: High risk for injury related to altered clotting mechanisms and altered level of consciousness
Goal: Reduced risk of injury

1. Assess level of consciousness and cognitive level.

1. Assists in determining patient’s ability to protect self and comply with required self-care.

• Is oriented to time, place, and person
• Exhibits no hallucinations,
2. Provide safe environment (pad side rails, remove obstacles in room, prevent falls).
3. Provide frequent surveillance to orient patient and avoid use of restraints.
4. Replace sharp objects (razors) with safer items.
5. Observe each stool for color, consistency, and amount.
6. Be alert for symptoms of anxiety, epigastric fullness, weakness, and restlessness.
7. Test each stool and emesis for occult blood.
8. Observe for hemorrhagic manifestations: ecchymosis, epistaxis, petechiae, and bleeding gums.
9. Record vital signs at frequent intervals, depending on patient acuity (every 1–4 h).
11. Assist physician in passage of tube for esophageal balloon tamponade, if its insertion is indicated.
12. Observe during blood transfusions.
13. Measure and record nature, time, and amount of vomitus.
14. Maintain patient in fasting state, if indicated.
15. Administer vitamin K as prescribed.
16. Remain with patient during episodes of bleeding.
17. Offer cold liquids by mouth when bleeding stops (if prescribed).
18. Institute measures to prevent trauma:
   a. Maintain safe environment.
   b. Encourage gentle blowing of nose.
   c. Provide soft toothbrush and avoid use of toothpicks.

| Protective actions; may detect deterioration of hepatic function | 2. Minimizes falls and injury if falls occur |
| 3. Protects patient from harm while stimulating and orienting patient; use of restraints may disturb patient further |
| 4. Avoids cuts and bleeding |
| 5. Permits detection of bleeding in gastrointestinal tract |
| 6. May indicate early signs of bleeding and shock |
| 7. Detects early evidence of bleeding |
| 8. Indicates altered clotting mechanisms |
| 9. Provides baseline and evidence of hypovolemia, and hemorrhagic shock |
| 10. Minimizes risk of bleeding and straining |
| 11. Promotes nontraumatic insertion of tube in anxious and combative patient for immediate treatment of bleeding |
| 12. Permits detection of transfusion reactions (risk is increased with multiple blood transfusions needed for active bleeding from esophageal varices) |
| 13. Assists in evaluating extent of bleeding and blood loss |
| 14. Reduces risk of aspiration of gastric contents and minimizes risk of further trauma to esophagus and stomach by preventing vomiting |
| 15. Promotes clotting by providing fat-soluble vitamin necessary for clotting |
| 16. Reassures anxious patient and permits monitoring and detection of further needs of the patient |
| 17. Minimizes risk of further bleeding by promoting vasoconstriction of esophageal varices |

- Exhibits negative results of test for occult gastrointestinal bleeding
- Is free of ecchymotic areas or hematoma formation
- Exhibits normal vital signs
- Maintains rest and remains quiet if active bleeding occurs
- Identifies rationale for blood transfusions and measures to treat bleeding
- Uses measures to prevent trauma (e.g., uses soft toothbrush, blows nose gently, avoids bumps and falls, avoids straining during defecation)
- Experiences no side effects of medications
- Takes all medications as prescribed
- Identifies rationale for precautions with use of all medications
- Cooperates with treatment.
| d. Encourage intake of foods with high content of vitamin C. | and gastric blood vessels and gastric blood vessels and gastric blood vessels and gastric blood vessels |
| e. Apply cold compresses where indicated. | a. Minimizes risk of trauma and bleeding by avoiding falls and cuts, etc. |
| f. Record location of bleeding sites. | b. Reduces risk of nosebleed (epistaxis) secondary to trauma and decreased clotting |
| g. Use small-gauge needles for injections. | c. Prevents trauma to oral mucosa while promoting good oral hygiene |
| 19. Administer medications carefully; monitor for side effects. | d. Promotes healing |
|                               | e. Minimizes bleeding into tissues by promoting local vasoconstriction |
|                               | f. Permits detection of new bleeding sites and monitoring of previous sites of bleeding |
|                               | g. Minimizes oozing and blood loss from repeated injections |
|                               | 19. Reduces risk of side effects secondary to damaged liver's inability to detoxify (metabolize) medications normally |

**Nursing Diagnosis:** Disturbed body image related to changes in appearance, sexual dysfunction, and role function

**Goal:** Patient verbalizes feelings consistent with improvement of body image and self-esteem

| 1. Assess changes in appearance and the meaning these changes have for patient and family. | 1. Provides information for assessing impact of changes in appearance, sexual function, and role on the patient and family |
| 2. Encourage patient to verbalize reactions and feelings about these changes. | 2. Enables patient to identify and express concerns; encourages patient and significant others to share these concerns |
| 3. Assess patient's and family's previous coping strategies. | 3. Permits encouragement of those coping strategies that are familiar to patient and have been effective in the past |
| 4. Assist and encourage patient to maximize appearance and explore alternatives to previous sexual and role functions. | 4. Encourages patient to continue safe roles and functions while encouraging exploration of alternatives |
| 5. Assist patient in identifying short-term goals. | 5. Accomplishing these goals serves as positive reinforcement and increases self-esteem. |
| 6. Encourage and assist patient in decision making about care. | 6. Promotes patient's control of life and improves sense of well-being |
| 7. Identify with patient resources to provide additional support | • Verbalizes concerns related to changes in appearance, life, and lifestyle |
|                               | • Shares concerns with significant others |
|                               | • Identifies past coping strategies that have been effective |
|                               | • Uses past effective coping strategies to deal with changes in appearance, life, and lifestyle |
|                               | • Maintains good grooming and hygiene |
|                               | • Identifies short-term goals and strategies to achieve them |
|                               | • Takes an active role in decision making about self and care |
|                               | • Identifies resources that... |
8. Assist patient in identifying previous practices that may have been harmful to self (alcohol and drug abuse).

7. Assists patient in identifying resources and accepting assistance from others when indicated.

8. Recognition and acknowledgment of the harmful effects of these practices are necessary for identifying a healthier lifestyle.

Nursing Diagnosis: Chronic pain and discomfort related to enlarged tender liver and ascites

Goal: Increased level of comfort

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</tr>
</thead>
<tbody>
<tr>
<td>1. Maintain bed rest when patient experiences abdominal discomfort.</td>
<td>1. Reduces metabolic demands and protects the liver</td>
<td>• Reports pain and discomfort if present</td>
</tr>
<tr>
<td>2. Administer antispasmodic and analgesic agents as prescribed.</td>
<td>2. Reduces irritability of the gastrointestinal tract and decreases abdominal pain and discomfort</td>
<td>• Maintains bed rest and decreases activity in presence of pain</td>
</tr>
<tr>
<td>3. Observe, record, and report presence and character of pain and discomfort.</td>
<td>3. Provides baseline to detect further deterioration of status and to evaluate interventions</td>
<td>• Takes antispasmodic and analgesics as indicated and as prescribed</td>
</tr>
<tr>
<td>4. Reduce sodium and fluid intake if prescribed.</td>
<td>4. Minimizes further formation of ascites</td>
<td>• Reports decreased pain and abdominal discomfort</td>
</tr>
<tr>
<td>5. Prepare patient and assist with paracentesis.</td>
<td>5. Removal of ascites fluid may decrease abdominal discomfort</td>
<td>• Reduces sodium and fluid intake to prescribed levels if indicated to treat ascites</td>
</tr>
<tr>
<td></td>
<td>1. Reduces metabolic demands and protects the liver</td>
<td>• Exhibits decreased abdominal girth and appropriate weight changes</td>
</tr>
<tr>
<td></td>
<td>2. Reduces irritability of the gastrointestinal tract and decreases abdominal pain and discomfort</td>
<td>• Reports decreased discomfort after paracentesis</td>
</tr>
</tbody>
</table>

Nursing Diagnosis: Fluid volume excess related to ascites and edema formation

Goal: Restoration of normal fluid volume

<table>
<thead>
<tr>
<th>Nursing Interventions</th>
<th>Rationale</th>
<th>Expected Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Restrict sodium and fluid intake if prescribed.</td>
<td>1. Minimizes formation of ascites and edema</td>
<td>• Consumes diet low in sodium and within prescribed fluid restriction</td>
</tr>
<tr>
<td>2. Administer diuretics, potassium, and protein supplements as prescribed.</td>
<td>2. Promotes excretion of fluid through the kidneys and maintenance of normal fluid and electrolyte balance</td>
<td>• Takes diuretics, potassium, and protein supplements as indicated without experiencing side effects</td>
</tr>
<tr>
<td>3. Record intake and output every 1 to 8 h depending on response to interventions and on patient acuity.</td>
<td>3. Indicates effectiveness of treatment and adequacy of fluid intake</td>
<td>• Exhibits increased urine output</td>
</tr>
<tr>
<td>4. Measure and record abdominal girth and weight daily.</td>
<td>4. Monitors changes in ascites formation and fluid accumulation</td>
<td>• Exhibits decreasing abdominal girth</td>
</tr>
<tr>
<td>5. Explain rationale for sodium and fluid restriction.</td>
<td>5. Promotes patient’s understanding of restriction and cooperation with it</td>
<td>• Exhibits no rapid increase in weight</td>
</tr>
<tr>
<td>6. Prepare patient and assist</td>
<td></td>
<td>• Identifies rationale for sodium and fluid restriction</td>
</tr>
</tbody>
</table>
with paracentesis.  

| 6. Paracentesis will temporarily decrease amount of ascites present. |  
| • Shows a decrease in ascites with decreased weight |

**Nursing Diagnosis:** Disturbed thought processes related to deterioration of liver function and increased serum ammonia level  

**Goal:** Improved mental status; safety maintained

| 1. Restrict dietary protein as prescribed. | 1. Reduces source of ammonia (protein foods) | • Adheres to protein restriction |
| 2. Give frequent, small feedings of carbohydrates. | 2. Promotes consumption of adequate carbohydrates for energy requirements and spares protein from breakdown for energy | • Demonstrates an interest in events and activities in environment |
| 3. Protect from infection. | 3. Minimizes risk for further increase in metabolic requirements | • Demonstrates normal attention span |
| 4. Keep environment warm and draft-free. | 4. Minimizes shivering, which would increase metabolic requirements | • Follows and participates in conversation appropriately |
| 5. Pad the side rails of the bed. | 5. Provides protection for the patient should hepatic coma and seizure activity occur | • Is oriented to person, place, and time |
| 6. Limit visitors. | 6. Minimizes patient’s activity and metabolic requirements | • Remains in bed when indicated |
| 7. Provide careful nursing surveillance to ensure patient’s safety. | 7. Provides close monitoring of new symptoms and minimizes trauma to the confused patient | • Reports no urinary or fecal incontinence |
| 8. Avoid opioids and barbiturates. | 8. Prevents masking of symptoms of hepatic coma and prevents drug overdose secondary to reduced ability of the damaged liver to metabolize opioids and barbiturates | • Experiences no seizures |
| 9. Awaken at intervals (every 2–4 h) to assess cognitive status. | 9. Provides stimulation to the patient and opportunity for observing the patient's level of consciousness |  

**Nursing Diagnosis:** Risk for imbalanced body temperature: hyperthermia related to inflammatory process of cirrhosis or hepatitis  

**Goal:** Maintenance of normal body temperature, free from infection

| 1. Record temperature regularly (every 4 h). | 1. Provides baseline to detect fever and to evaluate interventions | • Exhibits normal temperature and reports absence of chills or sweating |
| 2. Encourage fluid intake. | 2. Corrects fluid loss from perspiration and fever and increases patient’s level of comfort | • Demonstrates adequate intake of fluids |
| 3. Apply cool sponges or icebag for elevated temperature. |  
| 4. Administer antibiotics as |  
|  

prescribed.  
5. Avoid exposure to infections.  
6. Keep patient at rest while temperature is elevated.  
7. Assess for abdominal pain, tenderness.  

3. Promotes reduction of fever and increases patient's comfort  
4. Ensures appropriate serum concentration of antibiotics to treat infection  
5. Minimizes risk of further infection and further increases in body temperature and metabolic rate  
6. Reduces metabolic rate  
7. May occur with bacterial peritonitis  

local or systemic infection

**Nursing Diagnosis:** Ineffective breathing pattern related to ascites and restriction of thoracic excursion secondary to ascites, abdominal distention, and fluid in the thoracic cavity  
**Goal:** Improved respiratory status

| 1. Elevate head of bed to at least 30 degrees. | 1. Reduces abdominal pressure on the diaphragm and permits fuller thoracic excursion and lung expansion |
| 2. Conserve patient’s strength by providing rest periods and assisting with activities. | 2. Reduces metabolic and oxygen requirements |
| 3. Change position every 2 h. | 3. Promotes expansion and oxygenation of all areas of the lungs |
| 4. Assist with paracentesis or thoracentesis. | 4. Paracentesis and thoracentesis (performed to remove fluid from the abdominal and thoracic cavities, respectively) may be frightening to the patient. |
| a. Explain procedure and its purpose to patient. | a. Helps obtain patient’s cooperation with procedures |
| b. Have patient void before paracentesis. | b. Prevents inadvertent bladder injury |
| c. Support and maintain position during procedure. | c. Prevents inadvertent organ or tissue injury |
| d. Record both the amount and the character of fluid aspirated. | d. Provides record of fluid removed and indication of severity of limitation of lung expansion by fluid |
| e. Observe for evidence of coughing, increasing dyspnea, or pulse rate. | e. Indicates irritation of the pleural space and evidence of pneumothorax or pleuritic pain |

- Experiences improved respiratory status
- Reports decreased shortness of breath
- Reports increased strength and sense of well-being
- Exhibits normal respiratory rate (12–18/min) with no adventitious sounds
- Exhibits full thoracic excursion without shallow respirations
- Exhibits normal arterial blood gases
- Exhibits adequate oxygen saturation by pulse oximetry
- Experiences absence of confusion or cyanosis
Collaborative Problem: Gastrointestinal bleeding and hemorrhage

**Goal:** Absence of episodes of gastrointestinal bleeding and hemorrhage

<table>
<thead>
<tr>
<th>1.</th>
<th>Assess patient for evidence of gastrointestinal bleeding or hemorrhage. If bleeding does occur:</th>
<th>1.</th>
<th>Allows early detection of signs and symptoms of bleeding and hemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td>a.</td>
<td>Monitor vital signs (blood pressure, pulse, respiratory rate) every 4 h or more frequently, depending on acuity.</td>
<td>2.</td>
<td>Minimizes increases in intra-abdominal pressure that could lead to rupture and bleeding of esophageal or gastric varices</td>
</tr>
<tr>
<td>b.</td>
<td>Assess skin temperature, level of consciousness every 4 hours or more frequently, depending on acuity.</td>
<td>3.</td>
<td>Equipment, medications, and supplies will be readily available if patient experiences bleeding from ruptured esophageal or gastric varices.</td>
</tr>
<tr>
<td>c.</td>
<td>Monitor gastrointestinal secretions and output (emesis, stool for occult or obvious bleeding). Test emesis for blood once per shift and with any color change. Hematest each stool.</td>
<td>4.</td>
<td>Gastrointestinal bleeding and hemorrhage require emergency measures (eg, insertion of Blakemore tube, administration of fluids and medications).</td>
</tr>
<tr>
<td>d.</td>
<td>Monitor hematocrit and hemoglobin for trends and changes.</td>
<td>5.</td>
<td>The patient is at high risk for respiratory complications, including asphyxiation if gastric balloon of tamponade tube ruptures or migrates upward.</td>
</tr>
<tr>
<td>2.</td>
<td>Avoid activities that increase intra-abdominal pressure (straining, turning).</td>
<td>6.</td>
<td>The patient who experiences hemorrhage is very anxious and fearful; minimizing anxiety assists in control of hemorrhage.</td>
</tr>
<tr>
<td>a.</td>
<td>Avoid coughing/sneezing.</td>
<td>7.</td>
<td>Risk of rebleeding is high with all treatment modalities used to halt gastrointestinal bleeding.</td>
</tr>
<tr>
<td>b.</td>
<td>Assist patient to turn.</td>
<td>8.</td>
<td>Family members are likely to be anxious about the patient's status; providing information will reduce their anxiety level and promote more effective coping.</td>
</tr>
<tr>
<td>c.</td>
<td>Keep all needed items within easy reach.</td>
<td>9.</td>
<td>Risk of rebleeding is high. Subtle signs may be more quickly identified</td>
</tr>
<tr>
<td>d.</td>
<td>Use measures to prevent constipation such as adequate fluid intake; stool softeners.</td>
<td>10.</td>
<td>Assess patient for evidence of gastrointestinal bleeding or hemorrhage. If bleeding does occur:</td>
</tr>
<tr>
<td>e.</td>
<td>Ensure small meals.</td>
<td>a.</td>
<td>Monitor vital signs (blood pressure, pulse, respiratory rate) every 4 h or more frequently, depending on acuity.</td>
</tr>
<tr>
<td>3.</td>
<td>Have equipment (Blakemore tube, medications, IV fluids)</td>
<td>b.</td>
<td>Assess skin temperature, level of consciousness every 4 hours or more frequently, depending on acuity.</td>
</tr>
<tr>
<td>11.</td>
<td>Avoid activities that increase intra-abdominal pressure (straining, turning).</td>
<td>c.</td>
<td>Monitor gastrointestinal secretions and output (emesis, stool for occult or obvious bleeding). Test emesis for blood once per shift and with any color change. Hematest each stool.</td>
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<tr>
<td>c.</td>
<td>Keep all needed items within easy reach.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
available if indicated.
4. Assist with procedures and therapy needed to treat gastrointestinal bleeding and hemorrhage.
5. Monitor respiratory status every hour and minimize risk of respiratory complications if balloon tamponade is needed.
6. Prepare patient physically and psychologically for other treatment modalities if needed.
7. Monitor patient for recurrence of bleeding and hemorrhage.
9. Once recovered from bleeding episode, provide patient and family with information regarding signs and symptoms of gastrointestinal bleeding.

10. Have equipment (Blakemore tube, medications, IV fluids) available if indicated.
11. Assist with procedures and therapy needed to treat gastrointestinal bleeding and hemorrhage.
12. Monitor respiratory status every hour and minimize risk of respiratory complications if balloon tamponade is needed.
13. Prepare patient physically and psychologically for other treatment modalities if needed.
15. Keep family informed of patient’s status.
16. Once recovered from bleeding episode, provide patient and family with information regarding signs and symptoms of gastrointestinal bleeding.

**Collaborative Problem:** Hepatic encephalopathy

**Goal:** Absence of changes in cognitive status and of injury

<table>
<thead>
<tr>
<th>1. Assess cognitive status every 4–8 h:</th>
<th>1. Data will provide baseline of patient's cognitive status and enable detection of changes.</th>
<th>1. Remains awake, alert, and aware of surroundings</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Assess patient's orientation to person, place, and time.</td>
<td>2. Medications are a common precipitating factor in development of hepatic encephalopathy in patients at risk.</td>
<td>Is oriented to time, place, and person</td>
</tr>
<tr>
<td>b. Monitor patient's level of activity, restlessness, and agitation. Assess for presence of flapping hand tremors (asterixis).</td>
<td>3. Increases in serum ammonia level are associated with hepatic encephalopathy and coma.</td>
<td>Exhibits no restlessness or agitation</td>
</tr>
<tr>
<td>2. Medications are a common precipitating factor in development of hepatic encephalopathy in patients at risk.</td>
<td></td>
<td>Record of handwriting demonstrates no deterioration in cognitive function</td>
</tr>
<tr>
<td>3. Increases in serum ammonia level are associated with hepatic encephalopathy and coma.</td>
<td></td>
<td>States rationale for treatment used to prevent</td>
</tr>
<tr>
<td></td>
<td></td>
<td>constipation such as adequate fluid intake; stool softeners.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ensure small meals.</td>
</tr>
<tr>
<td>c. Obtain and record daily sample of patient's handwriting or ability to construct a simple figure (eg, star).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>d. Assess neurologic signs (deep tendon reflexes, ability to follow instructions).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Monitor medications to prevent administration of those that may precipitate hepatic encephalopathy (sedatives, hypnotics, analgesics).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Monitor laboratory data, especially serum ammonia level.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Notify physician of even subtle changes in patient's neurologic status and cognitive function.</td>
<td></td>
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<tr>
<td>5. Limit sources of protein from diet if indicated.</td>
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<td></td>
</tr>
<tr>
<td>6. Administer medications prescribed to reduce serum ammonia level (eg, lactulose, antibiotics, glucose, benzodiazepine antagonist [Flumazenil] if indicated).</td>
<td></td>
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<tr>
<td>7. Assess respiratory status and initiate measures to prevent complications.</td>
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<td></td>
</tr>
<tr>
<td>8. Protect patient’s skin and tissue from pressure and breakdown.</td>
<td></td>
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</tr>
<tr>
<td>5. Reduces breakdown and conversion of protein to ammonia.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Reduces serum ammonia level.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. The patient who develops hepatic coma is at risk for respiratory complications (ie, pneumonia, atelectasis, infection).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. The patient in coma is at risk for skin breakdown and pressure ulcer formation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or treat hepatic encephalopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Demonstrates stable serum ammonia level within acceptable limits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Consumes adequate caloric intake and adheres to protein restriction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Takes medications as prescribed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Breath sounds are normal without adventitious sounds</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Skin and tissue intact without evidence of pressure or breaks in integrity</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>